

NEW SOUTH WALES ANIMAL HEALTH SURVEILLANCE

Information contributed by staff of the Rural Lands Protection Boards and the NSW Department of Primary Industries

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SPECIAL REPORT

BHAMC—a lethal heritable congenital 'curly calf' syndrome in Angus cattle

DPI veterinarian Dr Laurence Denholm has collaborated with international experts to identify carriers of a lethal genetic disease in stillborn Angus calves. Its formal name is bovine heritable arthrogryposis multiplex congenita (BHAMC). Most will call these calves 'curly calves', but unfortunately that's also the common name given to calves with a similar appearance that are aborted or born infected with Akabane or other Simbu group viruses. In BHAMC, the brain itself is not damaged as it is in akabane virus infection; rather, the brain and spinal cord fail to develop normally and the calf develops *in utero* without moving normally; as a result it is severely deformed, with fixed and contracted joints of all four limbs (tetramelic arthrogryposis).

Over the last few years several purebred Angus herds in NSW have experienced BHAMC cases that were not caused by Simbu group viruses. At least one Angus cow is reported to have had a BHAMC-affected calf to the same bull in two consecutive years. (This could not happen with Simbu viruses, as the cow would be immune to any second infection.) The owners of two affected Angus herds persisted in their efforts to identify the cause of these Akabane virus-negative cases. RLPB veterinarian David Gardiner drew the cases to the attention of Dr Denholm, who was working on another unrelated heritable syndrome of non-lethal congenital contractures in Angus calves, called 'fawn calf syndrome'. Dr Denholm suspected a syndrome of heritable arthrogryposis, initiated a pedigree analysis in several herds, and identified the US AI sire, GAR Precision 1680,

on both sides of the pedigrees of almost all of these AMC cases, mostly through his popular US-bred son, CA Future Direction 5321.

CA Future Direction 5321 has about 10 000 registered progeny in Australia. His sire GAR Precision 1680 has several hundred progeny in Australia and several US-bred sons in current and recent Australian semen catalogues. If this BHAMC syndrome is indeed a simple recessive heritable disorder in Angus cattle as suspected, there will be several hundred thousand registered and commercial cattle in Australia that are potential carriers of the defect. Both suspect U.S. bulls are also present in some Red Angus and some composite pedigrees.

Dr Denholm has also investigated pedigrees from several reported BHAMC cases in Australia and Missouri, USA, that do not trace through Precision 1680 but trace through different and less common lines of descent to the maternal grandsire of Precision 1680, Rito 9J9 of B156 7T26. The bloodline through Precision 1680 is, however, much more common in Angus pedigrees and is the line apparently responsible for most Australian cases. Rito 9J9 of B156 7T26 appears at least once in the pedigrees of the majority of the top 100 high carcass performance Angus bulls currently in use in Australia.

When Dr Denholm became aware that colleagues in Nebraska and Illinois were working on the same syndrome, he exchanged his findings with them.

On 19 September 2008, the American Angus Association (AAA) announced that it had identified GAR Precision 1680 as the common maternal and paternal ancestor in 47 of 48 cases of this syndrome reported in the USA during a 10-day period after the AAA called for reports of such 'curly calves' on 5 September 2008 (see <http://www.angus.org>). In the near

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future it is expected the AAA will also disclose the names of a number of sons of GAR Precision 1680 that have been used in Australia as well as North America and are now also suspected carriers.

All these U.S. bred bulls appear to carry a defective recessive gene, although the pattern of inheritance may be complex. The suspect Angus bloodlines are very common, a fact that complicates epidemiological investigations within affected herds. Two herds that have reported a 5% incidence of these cases over the last 2 years have a significant level of inbreeding to CA Future Direction 5321 among younger cows in the herd.

Cows carrying affected calves often have unusually swollen abdomens during late pregnancy as a result of hydramnios, and this may be the first sign that something is amiss with the pregnancy.

Affected calves are usually, but not always, stillborn. Dystocia is common. The calves are full term and appear to die shortly before or during birth. Their bodyweight is markedly reduced for gestational age (15 to 25 kg) because of low muscle mass. The forelimbs are invariably in fixed flexion, with fixed flexion of the forelimb pasterns. The hindlimbs are usually in fixed extension, with fixed extension of the hindlimb pasterns, but the hindlimbs may be in fixed flexion in some cases.



BHAMC calf Photo: L. Denholm



BHAMC spinal changes Photo: L. Denholm

There is usually severe curvature, hunching and twisting of the spine (kyphoscoliosis and torticollis), with associated abnormalities of the ribs and sternum. There may also be cleft palate (due to failure of fusion of the bones of the hard palate) and skull bone abnormalities (cranial doming and lateral deviation of the facial bones) in some cases. The ear pinnae are often set lower than normal and closer together on the back of the skull. Unlike in Akabane disease, there is

no destruction of the brain tissue. Accordingly, neither complete absence of the brain nor holes in the brain tissue have been a feature, although hydrocephalus is seen in some BHAMC cases.

Heritable syndromes of arthrogryposis are well recognised in humans and in several cattle breeds. Although this condition has been rare in Angus in the past, it is not new. A significant increase in the incidence of such recessive disorders is also not unexpected with current breeding practices that involve heavy use of a few high-performing AI sires.

NSW DPI is interested in receiving reports of any syndrome of suspected AMC in cattle of any breed. BHAMC cases should be examined and photographed; look for cleft palate and note the pattern of joint contractures and any cranial abnormalities. Collection of the pedigree and some air-dried tail hair from the calf and its mother for DNA analysis is very important. Serum or body cavity fluid from the calf and serum from the cow should be submitted for Simbu virus exclusion, if possible. The pestivirus status of the herd should be recorded if known. The case history should also include the exclusion of access to potentially neuropathic teratogenic plants such as *Conium maculatum* (common hemlock) and *Nicotiana glauca* (wild tree tobacco) or the grazing of lupins or sorghum during the first trimester of gestation.

BHAMC should not be confused with the other recessive heritable syndrome in newborn Angus calves known as 'fawn calf syndrome' (FCS), in which there are less severe contractures of the spine and the proximal joints of the limbs, with the contractures more prominent in the hindlimbs. The hallmark of FCS is the almost complete spontaneous resolution of these congenital contractures within a few months of birth, so long as the affected calf is able to get up and suckle. FCS is a syndrome of non-progressive proximal congenital contractures due to reduced muscle tissue extensibility that reduces the range of joint movements, although not to the extent of complete joint fixation. Most importantly, FCS is characterised by distal joint hyperlaxity (hyperextensibility) at birth despite proximal joint contractures—a very different appearance to the more severe contractures of both the proximal and distal joints that are seen in BHAMC. Finally, the Angus bloodlines identified as suspect in this BHAMC syndrome are quite different from those Angus bloodlines proven to be carriers of FCS in a NSW DPI breeding trial.

Further pathology images of BHAMC are available on Dr Denholm's Photobucket website at <http://s107.photobucket.com/albums/m298/denholml/BHAMC%20in%20Angus%20Calves/> and his images of Fawn Calf Syndrome are at <http://s107.photobucket.com/albums/m298/denholml/Fawn%20Calf%20Syndrome%20in%20Angus%20Calves/>. There is also a NSW DPI video with a text description of FCS at http://www.youtube.com/watch?v=PUt5fQ_3L20

**For further information contact
Dr Laurence Denholm, DPI Orange, on
(02) 6391 3634 or (0418) 641 957 or
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EMERGENCY DISEASE EXCLUSIONS

Hendra virus causes alarm

Many cases of suspect Hendra virus disease of horses were investigated in the quarter following the tragic death of a Queensland veterinarian from Hendra virus infection. All suspect cases were negative.

One example occurred at a racing stable in northern NSW. A private practitioner diagnosed severe pneumonia in one horse. It recovered quickly following antibiotic treatment, but in the meantime a private laboratory requested exclusion of Hendra virus. One of the subsequent tests (PCR on blood) showed a weakly positive result. Until confirmatory testing was conducted, the stable was quarantined and staff were instructed on personal safety issues. When a second sample returned a weak positive, it appeared that either there was a rare case of a recovery in a Hendra virus-infected horse or the PCR test was detecting horse genetic material, not viral genes. The latter was subsequently found to be the case.

At Wauchope, a 3-year-old thoroughbred filly died suddenly. She was undergoing breaking-in and had cantered but not vigorously exercised before death. She fed normally after working but was found dead the following morning with a large amount of frothy and bloody fluid exuding from the nose. The nearest flying fox colony was 20 km away but the horse had arrived from another town only 5 days previously, where possible association with flying foxes could have occurred. At necropsy, the trachea and lungs were full of blood from a massive pulmonary haemorrhage. Samples forwarded to AAHL at Geelong were negative for Hendra virus.

**For further information contact Rory Arthur,
NSW DPI, on 6391 3823.**

High poultry mortalities: erysipelas

In a flock of 30 000 mature layers, 1000 died over a period of 5 days with no obvious clinical signs. Feed and water consumption remained unchanged and egg production dropped by 10%.

This happened on a free-range farm approximately 40 km from a wetland where low-pathogenicity notifiable avian influenza (LPNAI) was detected in wild waterfowl last year. Avian influenza was urgently excluded by RT-PCR tests.

On necropsy, enlarged spleens in some birds was the only gross pathology observed. Histopathology showed the presence of bacterial emboli in several tissues, along with fibrin and necrosis in the spleen. Culture of various organs revealed the identity of the causative agent: *Erysipelothrix rhusiopathiae*.

This is the fourth case of erysipelas in layers in NSW in the last 2 years. The consistent pattern emerging is the vagueness of clinical features, the lack of gross pathology, and the presence of bacterial emboli in various organs—probably the cause of the acute death.

Erysipelas is believed to be uncommon in chickens; however, the true prevalence of this organism in chickens in Australia is not known. A serological survey in Japan in 2000 found 5.5% of chicken sera to have high titres against *E. rhusiopathiae*.

These findings indicate that the rate of *E. rhusiopathiae* infection among chickens in the field could be higher than what is believed to be the case. Erysipelas is more common in turkeys, which show obvious depression and fever and have signs of septicaemia, such as ecchymotic haemorrhages and liver degeneration and necrosis, in their carcasses.

The source and route of infection of the hens remain unclear. In experimental infection of chickens with erysipelas, septicaemia was produced only by the intrapalpebral and subconjunctival routes and only when injury to these tissues occurred simultaneously.

**For further information contact George
Arzey, NSW DPI, on (02) 4640 6402.**

QUARTERLY HIGHLIGHTS

Arsenic poisoning in cattle

Arsenic poisoning killed thirteen 9-month-old Angus-cross calves in a herd of 130 near Young in July 2008.

The cattle were moved from a pasture paddock to a grazing crop. The following morning, eight were found dead and several were sick with signs of depression, ataxia and collapse with respiratory distress. Losses continued after the mob was moved to another pasture paddock.

On a first visit to the property, nitrate/nitrite poisoning was suspected by a local veterinary practitioner. Blood was taken from a sick calf and aqueous humour from a dead calf for biochemistry, haematology and toxicology. There was evidence of hepatocellular damage, dehydration and azotaemia, but tests for nitrate/nitrite and lead were negative.

Two days later, the District Veterinarian examined a recently deceased calf and found an acute haemorrhagic abomasitis. On inspection of the first paddock, an old tip site was detected, with many rusted-out drums, some of which appeared to contain sump oil residues, as well as a small quantity of a yellowish powder in the middle of the site.

Histopathology on tissues from this calf showed an acute ulcerative abomasitis with fibrinosuppurative and haemorrhagic inflammation of the abomasal peritoneum, moderate multifocal acute hepatic necrosis and mild renal tubulonecrosis.

Abomasal contents, liver and kidney were submitted for toxicology and were tested for lead and arsenic. The Reinsch test for arsenic was negative on liver, but positive on abomasal contents and kidney. A lead test was negative on kidney, and abomasal contents had a lead level of 16 µmol/kg (3.3 mg/kg).

Toxicology testing on a sample from the tip site including yellowish powder revealed that it consisted of 36% arsenic but also contained 71 mg/kg of lead. It couldn't be determined whether the sample contained lead arsenate or a mixture of arsenic powder and sump oil containing lead.

**For further information contact Erika Bunker,
NSW DPI, on (02) 6391 3809 and John Evers,
DV Young RLPB, on (02) 6382 1255.**

Suspected bracken fern poisoning in cattle

A least 50% of a herd of Angus weaners died of bracken fern poisoning on a Mid North Coast property in July 2008, causing substantial economic loss. They had access to bracken fern (*Pteridium aquilinum* var. *esculentum*) but were not observed to be eating it.

Nineteen of the 42 animals died, most quite suddenly, but some were observed sick, showing depression and weight loss. At post-mortem examination by the private practitioner, there were multiple small haemorrhages throughout the body, pale livers and, in some animals, ulcerative lesions on the lips and in the mouth, abomasum and intestines.

Blood samples showed a very low white blood cell count (neutropenia), but no profound anaemia. There was evidence of liver damage (including high fibrinogen levels) and widespread muscle damage, with high blood levels of urea (azotaemia).

On histopathology of a wide range of samples from two animals, there were multi-organ haemorrhages, a severe multifocal acute necrotising hepatitis, abomasitis and pneumonia, enteritis and colitis.

Pasteurella trehalose was cultured from the liver and lung of one animal. There was no growth on routine culture of liver and lung from two other animals, and selective culture for salmonella from the intestines of one was negative. Examination of spleen smears for *Bacillus anthracis* gave negative results.

Bracken fern toxicity was considered the most likely primary cause of disease. The toxic principle of bracken fern, ptaquiloside, can cause acute bone marrow depression, leading to reduction in the numbers of blood platelets (thrombocytopenia) and white blood cells that are normally manufactured in the bone marrow. The lack of platelets prevents clotting and is the cause of the obvious haemorrhages. The secondary infection without any evidence of a boost in white cell counts also signifies bone marrow depression.

The toxicity and palatability of bracken vary with season, growth and locality, making the condition difficult to manage.

For further information contact Erika Bunker, DPI Orange, on (02) 6391 3809.



Bracken fern Photo: DPI image library

Red-gut in sheep on lucerne

Red-gut was diagnosed after post-mortem of lambs in the Benerembah (Riverina) area that died suddenly within days of weaning and being placed on lucerne. Twelve of 400 lambs died. The large intestines were gas filled and the small intestines were brick red with congestion and contained blood. The owner fed hay and administered vitamin A, D and E injections but another three died, so the lambs were provided access to a failed wheat crop to dilute the exposure to straight lucerne.

Red-gut causes sudden death in lambs grazing legume-dominant pastures in late winter and spring. It can be prevented by limiting access to legumes or raising the fibre content of the diet. This ensures that the size and bulk of the rumen-reticulum are sufficient to prevent displacement and torsion of the small intestine.

For further information contact Ian Masters, DV Narrandera RLPB, on (02) 6959 2322.

Metabolic diseases of sheep

Hypocalcaemia in sheep grazing oats and other cereal crops was a significant problem in southern NSW over winter. In one case, 30 of 800 lambs became ataxic and recumbent after being taken off crop and driven along a roadway for several kilometres. The lambs responded well to treatment with intravenous Ca/Mg solution. In another case calcium deficiency was confirmed as causing weakness, trembling and recumbency in 50 of 350 weaner lambs moved off oats for shearing. These lambs also responded well to prompt treatment.

Two outbreaks of rickets were investigated in the Wagga Wagga district. In the first, 20 of 350 crossbred lambs on grazing oats were affected, and in the second eight of 400 were affected. They showed broken bones, stiffness, shifting lameness, hunched back and enlarged joints.

Rickets is a disease of young, growing animals with defective calcification of growing bone. It is caused by deficiencies of calcium, phosphorus

and vitamin D. Lambs grazing green cereal crops during winter may develop rickets from vitamin D deficiency owing to reduced exposure to sunlight and ingestion of anti-rachitic (anti-vitamin D) compounds that may be present in the cereal crop. Vitamin D is necessary for calcium absorption from the intestine and for the regulation of phosphate ion absorption, metabolism and excretion. When these minerals are lacking, or the balance is upset, a failure of calcification of the bones occurs.

Rickets may be prevented by administering vitamin D by injection (usually in combination with vitamins A and E) in situations where vitamin D deficiency is likely and by ensuring lambs have an adequate and balanced dietary calcium and phosphorus intake. A product with Vitamin D at 500 000 IU/mL (10 times the amount in most other products with the same concentrations of vitamins A and E) appears to be the treatment of choice.

For further information contact Tony Morton, DV Wagga Wagga RLPB, on (02) 6923 0900 and Steve Whittaker, DV Hume RLPB, on (02) 6040 4210.

Foot abscesses in the North West

After significant rain, 20% of sheep in a flock on a property in the Brewarrina region developed severe foot abscesses. The sheep were lame and affected in one or more feet; the interdigital space was very inflamed and the feet were swollen and hot to the touch. Abscesses broke out and discharged above the coronet. *Arcanobacterium pyogenes* and *Corynebacterium bovis* were isolated.



Discharging foot abscess Photo: K. Greentree



Foot abscess: interdigital inflammation Photo: K. Greentree

For further information contact Kylie Greentree, NSW DPI Bourke, on (02) 6872 2077.

Grass tetany of cattle

In southern NSW, deaths from grass tetany over autumn and winter have generally been sporadic, but occasionally very serious. In one case investigated by a district veterinarian, 12 of 200 Angus cows died. A number of other properties recorded losses of three or four head overnight. The affected stock were usually found dead, with evidence of paddling.

Cattle losses due to the condition can be \$3 to \$5 million yearly in the Wagga Wagga, Albury and Gundagai districts. The total cost to producers is greater than this, as many different forms of prevention are used, sometimes with ineffective results.

Grass tetany occurs when the level of magnesium in the cerebrospinal fluid around the brain and spinal cord decreases below a critical level, thus causing central nervous system signs. It may arise from a simple deficiency of magnesium or from high levels of potassium in the rumen that may interfere with absorption of magnesium. This can occur on crop or short green pasture during autumn and winter.

For further information contact Steve Whittaker or Brigit Pitman, Hume RLPB, on (02) 6040 4210.

Yersinia pseudotuberculosis in sheep

Yersiniosis caused acute black-green fetid scouring and chronic black scouring, as well as ill thrift and heavy sheep mortalities, in the Hume Rural Lands Protection Board in July and August. Both young and older sheep were affected. In one case, 200 of 3500 weaners died. Other losses included 20 of 500 ewes; 25 of 800 weaners; and 10 of 300 hoggets.

Triggering stress factors included cold wet weather, and most outbreaks were also associated with an accompanying internal parasite problem and poor levels of nutrition. Good responses were reported with both sulfonamide and tetracycline therapies; follow-up treatments were occasionally necessary. Addressing management issues was also important. Secondary pneumonia was a complicating factor in some instances.

For further information contact Steve Whittaker or Brigit Pitman, Hume RLPB, on (02) 6040 4210.

Campylobacter abortions in sheep

Approximately 80 of 1300 crossbred ewes near Guyra began aborting several days after they commenced feeding on silage. Listeriosis associated with poor quality silage was initially suspected, but *Campylobacter fetus* subspecies *fetus* was isolated from a foetal stomach. The diagnosis of campylobacteriosis was consistent with the field observations of vaginal discharge post abortion in association with metritis in otherwise healthy ewes.

Campylobacter abortion in ewes is usually associated with high stocking rates and naïve sheep and occurs in the second half of gestation. This property had no recent history of abortion, and it was likely that the high stocking rate and feeding-out of silage increased exposure to *C. fetus* via the faecal-oral route.

Control was achieved by decreasing stocking density, stopping ground feeding and removing aborted ewes from the flock. Although aborted ewes have lifelong immunity, annual vaccination against *C. fetus* in the future was recommended.

For further information contact Steve Eastwood, DV Armidale RLPB, on (02) 6772 2366.

Suspected marshmallow poisoning

Marshmallow (*Malva parviflora*) poisoning was suspected as the cause of death in three of eight horses with severe muscle tremor, ataxia and pulmonary oedema in the Wagga region. Clinical biochemistry indicated extreme myositis with very high blood levels of creatine kinase, a muscle enzyme released with muscle damage. The investigating practitioner reported that the paddock feed was 90% marshmallow and had been eaten off in an area where it had wilted.

For further information contact Tony Morton, DV Wagga Wagga RLPB, on (02) 6923 0900.

Infectious laryngotracheitis

Infectious laryngotracheitis (ILT) caused severe losses of 400 chickens a day in a flock of 10 000 meat chickens on a poultry farm near Sydney. They showed acute respiratory disease, with watery eyes, sneezing and gasping for air. Histopathologic changes were of an acute tracheitis, typical of ILT. There

was oedema and the ciliated epithelium was sloughed off and replaced with squamous or cuboidal epithelium containing eosinophilic intranuclear inclusions. Some showed mucopurulent exudate on the mucosa.

Virulent Newcastle disease and avian influenza were excluded as causes of the problem. A V4 (vaccine) Newcastle disease virus was identified in tracheal and cloacal swabs.

A number of other cases of ILT were confirmed during the quarter.

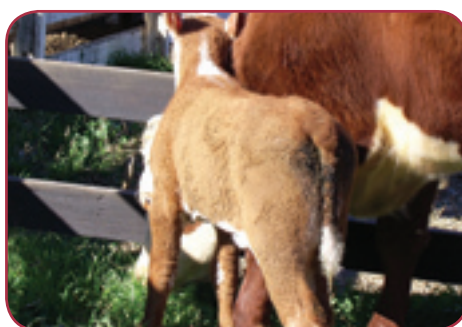
For further information contact George Arzey, NSW DPI Menangle, on (02) 4640 6402.

'Singey' calves

A Hereford beef herd owner near Braidwood reported four calves born almost hairless. The calves grew normally but had lighter, curly coats. Three of the calves were from first-calf heifers and one was from a cow that had had a similar calf the year before. That particular calf had grown normally and had had a normal coat before weaning.

The cows and calves were tested for deficiencies of selenium, copper, iodine and zinc, with all levels being within the normal range. The heifer group had a 50% calving rate. This was very low for this herd. Pestivirus serology gave varying results, not indicative of current infection. One cow had a mid-range titre and the rest of the cows and calves were negative.

'Singey' calves are born looking as though their hair has been singed or burned off at birth. The hair then grows like hair that has been singed: short and curly. Normal coat growth occurs when the next coat change occurs. The cause of the condition is unknown but has been linked to pestivirus infection.



Coat of 'singey' calf Photo: B Templeton

For further information contact Bob Templeton, DV Braidwood RLPB, on (02) 4842 2536.

Footrot surveillance

The footrot control program in NSW continues to be a resounding success. As of 30 June 2008 there were 26 properties in NSW in quarantine for footrot (down from 42 in December 2007). No individual Rural Lands Protection Board had a footrot prevalence above 1% of flocks, although Central Tablelands and Gundagai Boards remain Control Areas until other assurance criteria are met. The total number of sheep in quarantine dropped from 119 736 to 65 079.

A significant initiative is the drafting of surveillance standards for footrot. There are a range of 'active' and 'reactive' surveillance activities that can be undertaken, and these can be allocated points. The proposal is that the minimum standard (target) for most districts will be 50 points per year, made up of:

- 10 points for having competent footrot inspectors
- 10 points for compliance with footrot policy, including finalising all trace-backs reported and attendance at 50% of all sheep sales
- 10 points if each year Board staff investigate 'sheep problems' on 10% of properties with more than 50 sheep
- 2 points for each investigation of lameness or credible complaint undertaken
- 1 point for each random flock survey undertaken in accordance with rules for random surveillance.

For further information, contact Stephen Ottaway, RLPB State Council, Orange, on (02) 6391 3102.

Enzootic bovine leucosis (EBL) surveillance

There are no dairy herds known to be infected with EBL in NSW, following years of surveillance through milk factories and eradication programs managed at the district level. *Table 1* shows the number of dairy herds in various Rural Lands Protection Board Districts and the status of dairy herds in each district.

For further information contact Yuni Yunamu, NSW DPI, on (02) 4828 6628.

Cattle identification and surveillance

A southern NSW cattle producer was convicted recently in Albury Local Court for incorrectly tagging an estimated 177 cattle near Holbrook in NSW. He used NLIS (National Livestock Identification System) tags assigned to his Victorian property. He was fined \$1800 under the Stock Diseases Act and ordered to pay court costs of \$15,500. There is an appeal over costs.

Table 1. EBL status of dairy herds in each RLPB district

Location	Not assessed	BMT negative	Monitored free	Total
Central Tablelands	1	1	4	6
South Coast	5	2	103	110
Casino	0	5	50	55
Murray	7	11	46	64
Hunter	3	0	52	55
Riverina	4	2	47	53
Dubbo	0	0	6	6
Forbes	3	0	10	13
Gloucester	5	11	117	133
Grafton	0	0	38	38
Gundagai	1	0	4	5
Hume	0	2	19	21
Kempsey	0	3	84	87
Maitland	0	2	47	49
Molong	0	0	4	4
Moss Vale	1	5	114	120
Tamworth	0	0	16	16
Tweed-Lismore	0	1	39	40
Wagga Wagga	0	0	12	12
Young	1	0	0	1
TOTAL	31	45	812	888

NLIS regulations stipulate that tags may not be attached to cattle on a property different from the one to which the tags are assigned.

For further information contact Steve Whittaker or Brigit Pitman, Hume RLPB, on (02) 6040 4210.

Sheep lice surveillance

Lice (*Bovicola ovis*) infestations with a history consistent with resistance to pesticide treatments were seen on three properties in the Wagga district. Two of these flocks also had a history of strays into or from the flock.

There are anecdotal reports of more lice problems than usual in the southern part of the State. There is a huge surge in lice-related inquiries to District Veterinarians and Rangers and consistent reports from shearing contractors, shearers, contract plunge-dippers and stock and station agents that sheep lice are very common and infestations are present, despite treatment at the last shearing. Resistance to insect growth regulator (IGR) lousicidal products has been confirmed in several lice populations and may be one of several causes of the apparent increase in lice prevalence.

Extinosad® backline (spinosad) has recently entered the market and may offer a solution, provided that reinfestation can be prevented. Unlike the IGR backlines it provides very little protection against reinfestation. Extinosad® is also available as a dip. Other off-shears backline products available to

wool producers contain pyrethroids or low-volume spray formulations containing diazinon. Other chemicals unrelated to IGRs or spinosad have reached the field trial stage and should find a ready market when registered. One of these alternatives is a dip containing the organophosphate temephos (Assassin™). Assassin™ is due for commercial release very soon. Dip products containing magnesium fluorosilicate are registered for lice control but require very thorough agitation during use to prevent settling out. The use of diazinon dip products has been suspended or regulated by Pesticide Permit. Products purchased before 9 May 2007 may be used according to label instructions until May 2009, provided that they are still within their use-by dates. Products manufactured after that date must not be used for dipping or jetting sheep. If wet dipping is preferred, thorough treatment, such as can be achieved by contract cage- or plunge-dippers, is recommended. For optimum wetting, sheep should carry no more than 2 to 4 weeks' wool growth and should have their heads dunked twice. It is advisable to dip all sheep on the property at the same time and ensure that fences provide an effective barrier to reinfestation. Any introduced sheep should be shorn and treated and/or held separately from lice-free mobs until their lice-free status is assured.

For further information contact Tony Morton, DV Wagga Wagga RLPB, on (02) 6923 0900 or Garry Levot, NSW DPI Menangle, on (02) 4640 6376.

12th anniversary of *Salmonella enteritidis* surveillance

In June 1996, the then NSW Department of Agriculture and the poultry industry commenced a program for the accreditation of *Salmonella enteritidis* (SE)-free flocks. In the 12 years since the inception of the program SE has never been detected in participating flocks.

The voluntary program attracted support from all sectors of the commercial egg industry as well as the broiler industry. The program provides opportunities to market eggs from accredited flocks as Accredited SE-Free and also provides valuable intelligence on the potential spread of SE from the northern parts of Australia, where a relatively benign phage type of SE, phage type 26, has been reported.

Currently, 95% of breeders (holding 75% of all egg-laying stock) in NSW and Victoria participate in the scheme.

One of the most significant findings is that despite the intensive level of movements of poultry and poultry products between the northern parts of Australia and NSW/Victoria, *Salmonella enteritidis* has not crossed the borders from Queensland.

The net present value of maintaining the SE-free status of the Australian egg industry has been estimated to be \$965 million over 20 years.

A significant element of the accreditation program has been the accumulation of valuable data on the ecology and epidemiology of other *Salmonella* serovars on layers farms.

These data show that the *Salmonella* serovar involved mostly in human outbreaks in Australia, *Salmonella typhimurium*, does not persist on layer farms. It has also shown that there is a poor correlation between farm cleanliness, quality assurance programs and the presence of salmonella of any type.

Salmonella serovars other than *Salmonella typhimurium* are found more consistently on layer farms and appear to have a better capacity to persist in the environment of the layer shed, especially in controlled-environment sheds. The role of these other serovars in compromising the ability of *Salmonella typhimurium* to persist in the layer environment requires further assessment.

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

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 Rural Lands Protection Board District Veterinarian, Departmental Senior Regional Animal Health Manager, Regional Health Leader, or Regional Veterinary Laboratory.

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'.

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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 (October 2008). 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.



Indonesian veterinarian, Dr Heru Rachmadi, peruses the NSW SQ at an ACIAR sponsored workshop in Lombok. Photo: B Christie.



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