ANIMAL HEALTH SURVEILLANCE

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PREFACE

The impacts of drought can extend beyond the direct effects on the stock-carrying capacity of farms to impacts on animal health and surveillance programs. The review of the national mastitis and cell count control program in this *Animal Health Surveillance Quarterly* (AHSQ), demonstrates an on-going reduction in the incidence of mastitis in Australia. However, it also highlights the potential for disease spread associated with drought-enforced cost-cutting measures and the movement of cattle for agistment (i.e. an arrangement to pay the owner of a short-term supply of feed to take in livestock and use the feed) to deal with feed shortages. As well, several of the disease reports from this quarter reveal impacts of drought, including botulism in wild birds associated with drying up of water sources, lead poisoning of cattle consuming car batteries while grazing on stock routes, and grain poisoning arising from the supplementary feeding of drought-affected sheep. Even though times are difficult for many of Australia's primary producers, we must not lose our focus on animal health management or on early reporting of significant or unusual animal disease incidents.

Other topics include items of interest from the States and Territories, and summaries of disease monitoring and surveillance programs reported to Australia's National Animal Health Information System (NAHIS). Only summary information is recorded in NAHIS; detailed data are maintained by the source organisations. The information in AHSQ is accurate at the time of publication, but minor discrepancies may occur because of the short reporting and production time. AHSQ is also available on the Animal Health Australia website (http://www.animalhealthaustralia.com.au/status/nahis.cfm).

Bob Biddle, Acting Australian Chief Veterinary Officer

CONTENTS

National Arbovirus Monitoring Program	2
Countdown Downunder	3
World Organisation for Animal Health (OIE) stakeholder workshop	4
Foot-and-mouth disease exercises in Victoria	4
Avian influenza surveillance in wild birds: an update	5
Australian Wildlife Health Network	6
Aquatic animal health	7
State and Territory reports	8
Quarterly disease statistics	20
NAHIS contacts	28

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National Arbovirus Monitoring Program

The Australian National Arbovirus Monitoring Program (NAMP) is an integrated national program that is jointly funded by industry and governments. The program monitors the distribution of economically important insect-borne viruses (e.g. bluetongue, Akabane and bovine ephemeral fever) and their vectors.

This report covers the second half of 2006, a period when arboviral activity in northern Australia is usually low. Previously, there was a very late start to the wet season in the Northern Territory, with no significant rainfall recorded until late in December 2005. Rainfall was well below average in the East Kimberley, and the coastal areas and western half of the Pilbara, but well above average in the West Kimberley and south-eastern areas of the Pilbara. Conditions also remained dry in much of Queensland except for the tropical coast north of Ingham. The south, and the south-west in particular, remained in prolonged drought. The wet season had not started by the end of December. More than 90% of New South Wales continued to be drought affected, with the only significant rainfall recorded on the north coast.

Neither vector activity nor seroconversions to bluetongue or Akabane viruses were detected in the southern states of Victoria, Tasmania and South Australia during the second half of 2006.

Bluetongue virus

In Western Australia, there was increased surveillance in the Kimberley. Seropositive animals were detected in central and northern areas of the Kimberley, but the timing of infection is unknown.

In the Northern Territory, bluetongue activity was very limited. Seroconversions were recorded only in the Victoria River district (in most months during the period) and Katherine (in October 2006).

In Queensland, little bluetongue activity was detected. Seroconversions were mainly confined to the sentinel herds in the far north and north-east. Low numbers of seroconversions were also detected in the south-east at Taroom and Bell.

In New South Wales, no seroconversions to bluetongue were detected.

Akabane virus

In Western Australia, animals seropositive to Akabane virus were detected in central and northern areas of the Kimberley, but the timing of infection is unknown.

In the Northern Territory, Akabane activity was limited. Seroconversions were seen only in the northern herds and Victoria River district.

In Queensland, Akabane activity mirrored the pattern seen with bluetongue; seronversions were detected in the north and at Taroom and Bell. Multiple seroconversions were also detected at McKinlay, but these probably reflected infection earlier in the year.

In New South Wales, a single seroconversion was detected in the Casino sentinel herd in December 2006.

Bovine ephemeral fever virus

In Western Australia, animals seropositive to bovine ephemeral fever (BEF) virus were detected in southern areas of the Kimberley and in the Pilbara.

In the Northern Territory, activity was limited in the first 3 months of the period and increased later in the year. Seroconversions were seen in most northern herds and the Victoria River district. Clinical disease was reported in the Darwin and Katherine regions.

In Queensland, BEF seroconversions were detected in five sentinel herds, mainly in central areas. Clinical disease in these areas was also reported.

In New South Wales, a single seroconversion was detected in the Casino sentinel herd in November 2006.

In South Australia, 10 seropositive animals were detected (from 14 animals) in the recruitment bleed for a new sentinel property in the far north-east of the state. The animals were young, and had probably been exposed during the movement of floodwaters from Queensland's channel country through the north-east of South Australia during the winter. The difference in distribution between BEF and that of Akabane and bluetongue viruses is probably due to differences in vector species for BEF. These most likely are *Culex* or *Anopheles* species (*Culex annulirostris* is particularly suspected), but other

species may be involved, including Culicoides species.

Insect trapping

In Western Australia, traps collected the vector *Culicoides brevitarsis* in the Kimberley (Kalumburu and Wyndham Port) and at Roebuck Plains near Broome, as well as the vectors *C. actoni* and *C. fulvus* at Kalumburu. *C. brevitarsis* was the most common vector species collected in the Northern Territory; large numbers were found in Katherine and the Victoria River District. No vector species were collected at the Darwin port site. *C. brevitarsis* distribution in Queensland remained within previously recorded distributions, and *C. wadai* was detected sporadically in coastal areas. In New South Wales, the first occurrences of *C. brevitarsis* for the quarter were recorded in October 2006 at Grafton and Wauchope.

Additional information can be found at the Animal Health Australia website (http://www.animalhealthaustralia.com.au/programs/adsp/namp/namp_home.cfm).

Contributed by: NAMP group and Jenny Hutchison, National Surveillance Coordinator for Animal Health Australia, AusVet Animal Health Services Pty Ltd

Countdown Downunder

Udder health through the drought and beyond

Countdown Downunder, a project of Dairy Australia, is the national mastitis and cell count control program. It was launched 8 years ago to improve the profitability, sustainability and competitive advantage of the Australian dairy industry.

The goals of the dairy industry are for a cell count in all milk supply below 400 000 cells/mL, and below 250 000 cells/mL in 90% of the milk supply. There has been steady progress toward these goals despite a check in 2003 following the '1-in-100-year' drought in eastern Australia. It is clear that the actions taken to manage herds during the drought can affect udder health and milk quality in subsequent lactations.

Lessons drawn from this experience will help farm businesses protect the quality of their future milk supply as they manage their herds through the current drought.

Working through the immediate issues

Although farm managers' primary concerns are to preserve their core resources and decide how many cows they can afford to feed through the drought, it is also important for them to consider the implications of any planned actions for mastitis control in the herd. In particular, decisions that involve stock movements, cost cutting or changes to staff may pose a considerable risk to udder health.

By the end of 2006, many farmers had already reduced their herd sizes by selling cows or agisting them on other farms. With any stock movement, there is a risk that the bacteria that cause mastitis will transfer between farms. By acknowledging this risk, farmers can take simple steps when moving cows to help safeguard the milk quality of herds; these measures often require only minor modifications to the original strategy. Countdown Downunder has developed a checklist for farmers involved in agistment to assist this process.

It can also be false economy to reduce spending on products and services integral to mastitis management (such as teatcup liners, dry cow treatment or testing of milking machine performance). Reducing costs in the short term may result in significant income reductions in the longer term if bulk milk cell counts and clinical cases increase due to the spread of mastitis.

Changes to the labour force should be managed to ensure that milking routines are consistent, with new milking staff adopting the operating procedures of the farm, including management planning — usually the first task to be dropped from a heavy workload.

Actively seeking support

Farm businesses tend to deal more effectively with emerging issues when they are regularly reviewing their options with the support of relevant professionals. Dairy advisers who have an interest in

mastitis control can help farmers to develop strategies to maintain milk quality.

Nobody can predict how severe or prolonged the current drought will be, and the environment in which decisions are being made is constantly changing. Well-pitched services and advice help managers work through the relentless cycle of decisions that need to be made on multiple fronts, and will help to ensure that the outcomes are appropriate to the current and future farm circumstances.

More information on the reports and resources of Countdown Downunder can be viewed at http://www.countdown.org.au.

Contributed by: Anne Hope, Scientific Officer, Countdown Downunder

World Organisation for Animal Health (OIE) stakeholder workshop

Australia is a longstanding member of the World Organisation for Animal Health (OIE, formerly Office International des Epizooties) and an active and regular contributor to the development of the OIE's animal health and welfare standards and guidelines. Australia's contributions are most effective when Australian stakeholders are involved in their development.

To facilitate the involvement of Australian stakeholders in the standards development process, the Office of the Australian Chief Veterinary Officer hosted a workshop in Canberra on 10 November 2006. The workshop was chaired by Australia's delegate to the OIE, Dr Gardner Murray. Participants came from Australian Government departments, State and Territory governments, industry, universities, animal welfare organisations and other interest groups.

The program covered the mission, structure and processes of the OIE; Australia's role in the OIE; future directions of the OIE; and industry's expectations. A presentation was also given on the World Trade Organization's Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement), which underpins the OIE standards.

The OIE is an intergovernmental organisation that promotes transparency in the global animal disease situation, disseminates veterinary scientific information and develops animal health standards for international trade in animals and animal products. It also works, using a science-based approach, to improve veterinary services in developing countries, the safety of food of animal origin, and animal welfare. Its scope includes both aquatic and terrestrial animal health.

Australian stakeholders who are interested in participating in the development of Australia's comments to the OIE can register on-line (at http://www.daff.gov.au/oieforumregister).

Contributed by: Jill Mortier, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry

Foot-and-mouth disease exercises in Victoria

As part of an ongoing program to build and maintain emergency animal disease (EAD) preparedness in Victoria, the Victorian Department of Primary Industries conducted Exercise Maffra '06 on 10–12 October 2006 and Exercise Bendigo '06 on 20–22 November 2006. Both exercises involved simulated responses to hypothetical outbreaks of foot-and-mouth disease (FMD) in Victoria. Exercise Maffra '06 focused on the dairy industry and Exercise Bendigo '06 on the pig, sheep and beef cattle industries. Both exercises involved setting up local disease control centres and a range of field

activities. They were controlled, scenario-driven exercises that required players to perform a range of tasks related to their emergency roles.

Exercise Maffra '06

Exercise Maffra '06 involved 50 government staff and 10 representatives from industry and local government agencies. A significant effort was made to engage local dairy industry stakeholders with EAD preparedness issues, either as players, as observers or through locally broadcast media activities. The main part of the exercise consisted of desktop activities at a local disease control centre (LDCC), which was established at the state government offices at Maffra. There were also field activities at a nearby dairy (Macalister Research Farm), where a team of government officers practised biosecure entry and exit and planned field operations that would be required on FMD-infected premises.

Exercise Bendigo '06

Exercise Bendigo '06 was a smaller exercise, involving 25 government staff and five industry observers: three trained industry liaison officers (beef and sheep) and two members of the Australian Veterinary Reserve. This exercise provided new government animal health staff with training in EAD response field activities. It involved setting up a small LDCC at the Bendigo office of the Department of Primary Industries, from where field teams were dispatched to carry out simulated disease surveillance and control activities in the Bendigo region. An infected premises operations team planned disease control activities at a local piggery, with assistance from the piggery owner and staff. Three field surveillance teams had the opportunity to practise biosecure entry and exit and examination of livestock. A field patrol team was deployed to secure the simulated infected premises.

Exercise evaluation

Evaluation of the exercises confirmed their value in providing opportunities for participants to practise EAD response skills, develop new skills, and to test plans and systems. Trialling of a number of tools and resources as part of the exercises resulted in recommendations for improvements and for additional tools and resources to manage EAD response work more effectively. The exercises received good media coverage in their respective regions, demonstrating the value of having a dedicated regional communication officer with local media experience involved. Effective media coverage and the involvement of local emergency response stakeholders raised awareness of the potential impact of an outbreak of an emergency animal disease. A further two exercises will be held in Victoria in the latter part of 2007, one in the south-west region and one in the north-east region.

Contributed by: Rod Badman, Department of Primary Industries, Victoria

Avian influenza surveillance in wild birds: an update

Avian influenza is not a new disease; however, the widespread infection of production birds around the world has emerged as a significant and increasing problem. The most recent outbreaks of highly pathogenic avian influenza (HPAI) — caused by the subtype H5N1 — in South-East Asia, Europe and Africa since 2003, represent a panzootic that is unprecedented in terms of its duration, the size of the area affected and the high pathogenicity of the virus. Many migratory birds pass through countries with current H5N1 avian influenza infections on their southerly migration from breeding grounds in the Northern Hemisphere to their wintering grounds in Australia. For these reasons, wild bird surveillance for avian influenza has been enhanced through the cooperative efforts of the Australian Government Department of Agriculture, Fisheries and Forestry (including the Australian Quarantine and Inspection Service), the States and universities.

Investigation of wild bird mortality events is a crucial component of the Avian Influenza Wild Bird Surveillance Plan. Avian influenza virus was excluded as a cause of eight wild bird mortality events in Australia between July 2005 and December 2006.

To facilitate collaboration between State and Territory programs, the National Avian Influenza

Wild Bird Steering Committee was established in January 2006. From July 2005 to December 2006, cloacal swabs and blood samples were collected from 5252 wild birds, from 59 different bird species in Australia. Sampling occurred at sites in New South Wales, Victoria, South Australia, Tasmania, Queensland, the Northern Territory and Western Australia. The majority of samples were collected from waterfowl and waders, and a smaller number from other species, such as shearwaters.

Testing using the polymerase chain reaction (PCR) and serology gave positive results for influenza type A in a number of wild bird species throughout Australia. H4N6 was isolated from a juvenile/adult Pacific black duck and H13N6 from a silver gull chick; serological and PCR evidence of other low pathogenic strains was found. No highly pathogenic subtypes were isolated.

Based on these results, the prevalence of avian influenza viruses in Australian wild birds is low, although sampling has detected a wide range of low pathogenic viral types. Major surveillance activities for avian influenza in wild birds are continuing. No evidence of HPAI has been found in Australian poultry.

Contributed by: Chris Bunn, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry

Australian Wildlife Health Network

The Australian Wildlife Health Network (AWHN) receives reports of wildlife incidents and definitive diagnoses of cause of death in wildlife in Australia. The network is interested in receiving such reports. For copies of the network newsletter or digests, contact Rupert Woods at rwoods@zoo.nsw.gov.au. All contributions are recorded in the AWHN database (the Wildlife Health Information System, WHIS, http://www.wildlifehealth.org.au). Details about selected incidents and key investigations (other than avian influenza) are provided below.

Mass mortalities/morbidities in birds

The investigation into a cluster (approximately 45) of deaths of Australian ravens (*Corvus coronoides*), feral pigeons (*Columba livia*) and some other species in central Canberra, Australian Capital Territory (reported in the previous issue of *Animal Health Surveillance Quarterly*), has determined that death was due to intoxication with fenthion, an organophosphate compound.

During October 2006, deaths of approximately 100 red-winged parrots (*Aprosmictus erythropterus*) were reported from the Northern Territory. Birds were noted to be congregating in trees around sprinklers and developed a 'dummy' syndrome of nonresponsiveness, being excessively tame and falling out of trees. Gross autopsy and histological examination of various tissues did not reveal evidence of infectious disease. One bird had erosive ventriculitis, of unknown aetiology. Testing for avian influenza virus was negative, and virus isolation in eggs did not detect haemagglutinating agents. Starvation or other environmental stresses, or exposure to a toxin that did not leave microscopically visible lesions, were suggested as possible causes of morbidity.

A mass mortality event involving mainly insectivorous and nectivorous birds is currently under investigation by the Western Australian departments of Environment and Conservation, and Agriculture at Esperance in Western Australia. The first reports of deaths occurred on 4 December 2006, and few deaths have been reported since 29 December 2006. The species included yellow-throated miners (*Manorina flavigula*), New Holland honeyeaters (*Phylidonyris nigra*), wattlebirds (*Anthochaera* sp.) and some others. No waterfowl are involved. An impact zone of 38 hectares has been identified, centred on the Esperance Port Authority. An accurate estimate of the numbers of deaths is difficult due to scavenging of carcases and the small size of most of the birds, but it varies from approximately 500 to more than 1700 birds. Results are negative for avian influenza and West Nile virus. The most likely cause of mortalities appears at this stage to be a toxin rather than an infectious agent. The investigation is continuing.

Australian bat lyssavirus

In December, a grey-headed flying fox (*Pteropus policephalus*) from Cotton Tree (near Peachester), Queensland, tested positive to Australian bat lyssavirus by fluorescent antibody test (Queensland Health Scientific Services Bat Stats).

Biodiversity/other

One hundred and fifty flying fox pups (27 grey-headed *Pteropus poliocephalus,* and 123 black *P. alecto*) were found dead or comatose at the margins of a large mixed colony near Canungra, Queensland, on the evening of 17 November and morning of 18 November 2006. Deaths followed unseasonal frosts. Affected pups tended to be large (most had forearm lengths of 80–100 mm) and may have been of a size to have been left creched in the colony, rather than carried with the females.

Rescuers of comatose pups reported that they 'were like little icicles'; no rectal temperatures were taken, but some pups had heart rates as low as 60 bpm. Autopsies of eight pups by a private veterinarian identified trauma. No significant gross pathology or histopathology was identified on subsequent necropsies of 11 pups by the Queensland Department of Primary Industries. Testing for Australian bat lyssavirus and Hendra virus, and virus isolation, were all negative. Mortalities are attributed to hypothermia.

The network would like to thank all those who submitted information for this report.

Contributed by: Chris Bunn, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry, and Rupert Woods, Coordinator AWHN

Aquatic animal health

The Department of Agriculture, Fisheries and Forestry's aquatic animal health website

The Aquatic Animal Health Unit (AAHU), within the Division of Product Integrity, Animal and Plant Health of the Australian Government Department of Agriculture, Fisheries and Forestry (DAFF), leads and coordinates the national management of aquatic animal health for finfish, crustacea and molluscs. The AAHU maintains web pages (http://www.daff.gov.au/aquaticanimalhealth) that provide useful information and resources for those with an interest in aquatic animal health. They include information on Australia's National Strategic Plan for Aquatic Animal Health, AQUAPLAN 2005–2010, that aims to build and enhance the capacity for management of aquatic animal health in Australia. The biannual AQUAPLAN newsletter provides updates on the implementation of AQUAPLAN, and recent editions can be downloaded from the website. The aquatic animal health web pages also include information on:

- the consultative and ministerial committees that deal with aquatic animal health management in Australia
- emergency aquatic animal disease response and coordination activities
- the coordination of aquatic animal disease surveillance and reporting within Australia and internationally.

Numerous useful resources can be accessed through the web pages, including:

- the manuals comprising AQUAVETPLAN, the Australian Aquatic Veterinary Emergency Plan
- diagnostic resources such as the field guide *Aquatic Animal Diseases Significant to Australia*
- links to the Disease Watch aquatic animal health awareness multimedia kit.

For further information, contact the AAHU at AAH@daff.gov.au.

Contributed by: Amy Little, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry

State and Territory reports

New South Wales

Contributed by: Rory Arthur, Department of Primary Industries

Hendra virus

A horse showing nervous and respiratory signs consistent with Hendra virus infection was examined near Murwillumbah in November 2006. It had a fever, respiratory distress, ataxia and contorted facial muscles, but no frothy nasal discharge. The horse paddock contained trees that were roosts for flying foxes. The two horses in the paddock were fed from troughs located under the trees. A private practitioner euthanised the affected horse and samples were collected for emergency disease exclusion. The Australian Animal Health Laboratory reported a positive polymerase chain reaction (PCR) test on lung tissue for Hendra virus and subsequently isolated the virus.

Flying foxes most likely transmit Hendra virus to horses through birthing fluids, placental material and aborted pups. Approximately one Hendra virus exclusion test is performed within New South Wales (NSW) each year — this is the first positive case. NSW Health provided advice to the attending veterinarian and the horse's owner because Hendra virus is zoonotic. The other horse in the paddock remained healthy.

Infectious coryza

Infectious coryza was the probable cause of high mortalities and sickness in a flock of 500 free-range poultry in the Mudgee–Merriwa region in October 2006.

The hens were 7 months old. Thirty had died over the previous weeks and 300 were sick, out of a batch of 500. Affected hens were severely depressed and huddled in corners of pens, shaking their heads and making sneezing sounds. There was an associated drop in egg production of 75%. Dead and dying hens had congested and cyanotic wattles. Post mortem findings included whitish exudate in the infraorbital sinuses and moist laryngeal mucosae. There were no consistent lesions in the abdominal tract or in associated organs.

The hens were on dirt due to the drought and had no access to green feed. They were vaccinated against fowl pox and other respiratory diseases. Ducks were concurrently run on the property and were apparently unaffected. *Haemophilus paragallinarum* was not isolated, possibly because of its fragile nature or the chronic nature of the lesions sampled, but coryza was strongly suspected. Both avian influenza and infectious laryngotracheitis were excluded.

Botulism in water birds

Botulism was the suspected cause of death of seven ducks (including Pacific black ducks) and other waterfowl on a residential lake in western Sydney. Avian influenza was excluded as the cause. One moribund bird showed paralysis of the neck that was suggestive of botulism.

Botulism in waterfowl is not uncommon. Changes in water levels in lakes and ponds, especially during dry periods, can result in the death of water microbes or decomposition of vegetation. Both scenarios can create a favourable environment for the growth of *Clostridium botulinum* and toxin production.

Death in feedlot cattle

Aflatoxicosis was suspected as the cause of death of 35 out of 130 feedlot steers and heifers that died in the Scone district following a change in ration. The most consistent symptom in the animals autopsied was liver pathology, although a range of signs was observed, including severe congestion of the intestines, pneumonia, and neurological signs such as ataxia and mania. No infective agent was isolated.

Although the ration formulation appeared suitable for feeding in a feedlot, there were areas of decomposition in the wheaten straw used in the mix.

Lead poisoning

The prolonged drought, the grazing of cattle in unusual places and the improper disposal of old vehicle batteries contributed to unusual reports of lead poisoning in cattle, with 17 cases investigated during the quarter. These included four cattle in a mob of 750 travelling on stock routes in the Wagga Wagga and Gundagai region that died after showing arching of the back and apparent blindness. Laboratory examination showed elevated levels of lead in the blood of 19 companion animals that had not shown clinical signs. These cattle were detained for a determined period in accordance with approved guidelines for management of possible stock lead residues. The lead source was broken battery cases that had been dumped on the stock route.

Lead poisoning was also confirmed in a steer in the Cobar district in November 2006. The 8-month-old steer was found away from the mob, reluctant to move and staggering when pushed. It was wasted, dehydrated and apparently blind. Its blood lead level was 4.7 µmol/L (normal is <1.2 µmol/L). Brain histology revealed a diffuse neuronal necrosis in the cerebral cortex, which is consistent with lead poisoning. The mob was placed under an 8-month detention. Blood lead levels were measured and cattle with acceptable lead levels were released. As a result of these cases, an extension program was instituted to help prevent further occurrences.

Anthrax

Six sporadic and unrelated incidents of anthrax in sheep and cattle were reported from October to December 2006 in areas where anthrax has previously been diagnosed. Four of the incidents occurred in flocks or herds where routine vaccination had been interrupted. In October, five lambs from a flock of 1600 died after being introduced to a property at Hillston. In Finley on 12 October, four dry heifers from a herd of 600 dairy cattle died, 21 days after introduction to a dry heifer feedlot. In November at Nyngan, approximately 20 sheep died out of a mob of 1860 2-year-old crossbred ewes grazing a failed wheat crop. Also in November, 30 sheep from a flock of 1000 were affected at one Condobolin property, and another Condobolin property lost 20 unvaccinated lambs. At Berrigan, four unvaccinated cattle were affected in December. All affected properties were immediately placed under a 42-day quarantine, livestock movement restrictions were applied, carcases were disposed of by burning and at-risk stock were vaccinated. Trace-back confirmed that no stock had left properties within the incubation period.

Theileriasis

A bull introduced from Coolah to the Gloucester area in October 2006 became lethargic, inappetent and very anaemic (packed cell volume of 15%) 6 weeks after introduction. Approximately 5% of its erythrocytes were parasitised with *Theileria buffeli*.

Theileria buffeli is usually considered nonpathogenic and theileriasis is not recognised as a problem in animals bred in the central and mid-north coast areas of NSW. However, problems can sometimes occur with introduced stock with no previous exposure to the organism. Occasionally, such stock can develop severe anaemia which needs to be differentiated from babesiosis. The movement of drought-affected stock from western NSW areas to agistment in the coastal areas may lead to further diagnoses of theileriasis.

Suspected *Salmonella* Typhimurium in calves

The western division of NSW reported cases of salmonellosis in calves during the quarter. Salmonellosis was suspected but not confirmed in a case at Hillston where 10 calves/weaners died in a mob of 80 cows and calves. Diarrhoea and scouring were the predominant signs, along with depression, inappetence and weakness. A post mortem examination of an affected calf showed an inflamed large intestine, and the bile was thickened and granular. There was also a severe tracheitis and pneumonia, suggesting that more than one clinical condition was present. Rotavirus, coccidiosis and cryptosporidiosis were excluded as causes.

Grain poisoning in sheep

Drought feeding and supplementation with grain led to a number of grain poisoning incidents during the quarter. In one case at Forbes, 600 crossbred lambs were affected; 12 lambs died and the remainder of the mob showed depression, lethargy, some scouring and lameness. They were treated twice with a drench of virginiamycin. The next day, two that were recumbent at treatment had died, but the balance of the flock had recovered, with just a few showing signs of lameness.

Fireweed toxicity

Six of 31 Hereford cows with calves at foot died with gastrointestinal disease on the NSW south coast in October–November 2006. The affected cows were in poor condition, and showed scouring and straining during defecation. Rectal prolapses were evident in several before death. The differential diagnoses included Johne's disease, salmonellosis, internal parasites and plant toxins. Post mortem examination showed extensive pulmonary and gastrointestinal tract oedema and a thickened corrugated ileum. The liver was small and fibrotic with an enlarged gall bladder and showed severe histological lesions typical of pyrrolizidine alkaloid toxicity. Dry fireweed was present in the pasture and was diagnosed as the cause of the deaths. Cattle owners in this region are encouraged to report chronic scouring in mature cattle to ensure that fireweed poisoning, an emerging problem in the district, is not mistaken for Johne's disease.

Photosensitisation

Tick fever was excluded and lantana poisoning confirmed in a mob of 47 cattle in the Casino Rural Lands Protection Board. Affected heifers were jaundiced, febrile and sought shade. They were in pain and went down when handled. The skin around the muzzle and ears was peeling and crusting. Blood chemistry showed serious liver disease, and haematology showed a few *Theileria buffeli* but no *Babesia* or *Anaplasma*. Animals recovered when denied access to lantana.

Polioencephalomalacia in lambs

Signs associated with a central nervous system disorder were found in a flock of sheep near Braidwood in November 2006. Of the 80 susceptible lambs, seven died and 17 showed ataxia, inability to stand or maintain sternal recumbency and paddling. Their main diet was processed sheep nuts. They also had access to small amounts of *Phalaris* grass and to a straw/molasses/grass seed mixture that was being used to revegetate a roadside verge. Gross pathology was nonspecific but histopathology revealed polioencephalomalacia associated with thiamine deficiency. The diet was changed, the lambs were injected with thiamine, and losses ceased.

Arsenic poisoning in cattle

Mortalities of six out of 80 9-month-old steers and two out of 29 young calves occurred at different times on the same paddock on a property in the Forbes district. The affected animals were well grown and in good condition at the time of death.

The first deaths occurred in the steers in October 2006. Post mortem investigation on the steers revealed an acute suppurative cholangiohepatitis, suggestive of a bacterial cause; however, the aetiological agent could not be isolated. Losses stopped when the steers were removed from the paddock. Approximately 6 weeks later, a mob of cows and calves was grazed on the same paddock, and mortalities were soon noted in the calves. The first calf was seen with profuse watery diarrhoea and died soon after. The second calf was found dead. None of the cows showed any clinical signs. Post mortem examination of the calves revealed high levels of arsenic in the liver tissues.

Further investigation of the paddock revealed a deposit of white powder which had been uncovered by the construction of a silage pit in the paddock. Testing of the powder confirmed that it contained arsenic. It was assumed that the powder was from unused animal treatment products that had been buried many years ago, and inadvertently exposed by the construction of the silage pit. It was disposed of through approved processes.

Northern Territory

Contributed by: Francois Human, Department of Primary Industry, Fisheries and Mines

Suspected *Swainsona* poisoning in horses

Horses with neurological signs were investigated on a southern Tennant Creek property. About 20 horses had died over a 2-month period. Animals became weak and depressed, lost weight and showed various signs of ataxia; most appeared to be stiff in the hind limbs during movement. Affected animals died within 10–14 days, but a few survived to 4 weeks. The horses overreacted when approached, but would soon settle to their lethargic state. Semi-circular movement was observed when affected animals grazed or moved to the water trough. No gross organ pathology was seen on autopsy of a euthanised horse,

but a histopathological examination of the brain and spinal cord revealed a degenerative axonopathy which is indicative of plant poisoning. This lesion has been described with swainsona and cycad poisoning. Cycads do not occur in the area. There are many *Swainsona* species in Australia, but only five are known to be toxic. In the Northern Territory the implicated species is *Swainsona canescens*, known as grey swainsona. Other toxic species are known as Darling peas.

Ketosis in an extensive beef herd

A group of older cows on a Barkly Tablelands property was moved from the lakes country to open grassland befoe the wet season. Downers and deaths were seen a week later. More than 30 animals died. The majority of cows were in advanced stages of gestation or had just calved. A pale liver was a consistent finding on autopsy, but no other gross lesions were observed. The urine had a very high ketone content as shown on a urine dip-stick. One of the animals examined showed signs of secondary photosensitisation on the muzzle. A diagnosis of ketosis, also known as pregnancy toxaemia, was made. The high protein diet from legumes and shrubs in the lakes area could have been a contributing factor. This metabolic disturbance is mostly found in dairy cattle at the time of peak lactation, but in beef cattle the condition is more likely to develop around the time of calving.

Suspected plant poisonings in cattle

Two Brahman bulls died suddenly on a Katherine property after 40 bulls were transported from Moura and Cloncurry. The bulls had been accustomed to grazing *Leucaena* (a fodder tree) and moved directly towards ironwood suckers and trees (*Erythrophleum chlorostachys*) when unloaded. A post mortem examination revealed evidence of haemorrhage around the valves of the heart and in the rumen. There were abundant ironwood leaves in the rumen and scant contents in the small and large intestines.

Fifteen heifer deaths were reported in the Katherine region within 1 week of transport from Halls Creek. Two day-old carcases were examined, but were not suitable for sample collection. There was no evidence of struggle before sudden death. Heavy growth of belly-ache bush (*Jatropha gossypiifolia*) was observed in the paddock, especially around the water point. Deaths ceased when the cattle started grazing and moved away from the water point. Jatrophas contain a toxic lectin in all parts of the plant, particularly the seeds. It affects mainly the gastrointestinal tract with associated signs of gastroenteritis and death in severe cases.

Queensland

Contributed by: John Cronin, Queensland Department of Primary Industries and Fisheries

Cattle

Salmonellosis

Salmonella group B was found to be responsible for the sickness of a 2-year-old dairy cow, one of 100 at risk on a property in Laidley shire in mid-December 2006. Clinical signs of profuse watery diarrhoea and fever were observed. *Salmonella* group B was also associated with the deaths of two Boran cattle in Pine Rivers shire in December.

Botulism

There was increased reporting of botulism in cattle in northern Queensland. The properties involved had not vaccinated in recent years or were not vaccinating at all. Some affected animals were ataxic and gradually became recumbent.

One north Queensland property had 20 deaths in adult cattle. Some animals were autopsied with samples submitted for laboratory examination. Laboratory results in at least one case were positive for *Clostridium botulinum* toxin.

A Brahman-cross herd in the basalt area of Charters Towers shire was suspected of having a problem with botulism. There were 10 sick and four dead animals from a herd of 200 animals. Affected animals were at least 24 months old, and showed signs of lameness, lethargy, recumbency and eventually death. The clinical picture was consistent with botulism, but as can happen with this disease, it could not be confirmed by the laboratory.

Bracken fern poisoning

Twenty out of 40 Holstein Friesian dairy cattle died after showing signs of diarrhoea and jaundice on a property in Eacham shire during October 2006. Autopsy revealed profuse haemorrhaging throughout the body, especially in the skin, eyes and intestine, an enlarged bronzed liver with yellowing around the edges and blood that was not clotting. Arsenic poisoning, anthrax and *Salmonella* were ruled out through negative laboratory results. Given the clinical presentation, histology and pathology, these deaths were highly likely to have been the result of bracken fern (*Pteridium esculentum*) poisoning.

Bracken fern poisoning was presumed to be the cause of death of an 18-month-old heifer from 30 at risk in Cooloola shire in late October. The heifer had access to bracken fern. Clinical signs of recumbency and convulsions were observed before death and serosal haemorrhages of the bladder and oedema of the abomasal and intestinal walls were seen at autopsy. Mucosal haemorrhages were seen in the abomasal and small intestinal walls on histopathological examination. There was also submucosal haemorrhage and oedema and subserosal haemorrhage of the intestinal wall. Multiple septic infarcts were present in the liver. These histological findings are consistent with bracken fern poisoning.

Lantana camara *poisoning*

Lantana camara poisoning was the likely cause of death of 40 cattle, and sickness in another 12 out of 200 at-risk cattle in Ipswich shire in late November 2006. Clinical signs of constipation, purulent nasal discharge and blistering of the nasal mucous membranes were seen, followed by death within 2–3 weeks. Newly introduced animals were affected within 2–3 days of arrival. The clinical chemistry and histopathological results were consistent with poisoning by lantadene, found in toxic *L. camara* plants.

Sheep

Avocado (Persea americana) *poisoning*

A small farm near Townsville was placed under quarantine in November 2006 as a precautionary measure after a dead sheep showed clinical signs suggestive of bluetongue virus infection. These included a bloodstained nasal discharge, ventral oedema, excessive fluid in the thoracic cavity, foam filling the airways and petechial haemorrhages on the omental fat, intestinal mucosa and epicardium. Four sheep out of a flock of 15 had died suddenly over the previous 3 months on the farm. Histopathology findings of cardiomyopathy with congestion, haemorrhage and oedema in many tissues and the history of exposure to avocado leaves and fruit, indicated that the death was most likely due to heart failure caused by avocado (*Persea americana*) poisoning. PCR and virus isolation failed to find evidence of bluetongue virus infection. No cases of disease due to bluetongue virus have ever been recorded in Queensland.

Goats

Salmonellosis

The owner of a property in western Queensland with 7000 goats noticed several of his animals with diarrhoea. The animals were suspected of having internal parasites, and faecal samples were sent to the laboratory. The strongyle egg counts were less than 50 eggs/g. Some samples sent for bacteriology were positive for *Salmonella* Chester.

Horses

Equine herpes virus abortion

A stud on the Darling Downs reported perinatal loss of five of the last six foals born. Tissues from three of the affected foals were sent to the Animal Disease Surveillance Laboratory in Toowoomba. All three foals had pneumonia (two with necrosis of bronchiolar epithelium) and multifocal hepatitis (two associated with hepatic necrosis). Intranuclear inclusion bodies consistent with herpes virus were seen in all three foals. Virus isolation and PCR were positive in all three foals for equine herpes virus 1. This breeding season, there have been nine diagnosed cases of equine herpes virus associated with abortion or perinatal loss on the Darling Downs. These have involved one standardbred and four thoroughbred studs.

Poultry

Nicarbazin poisoning

An accidental overdose of the coccidiostat nicarbazin caused the deaths of 49 000 broilers out of 244 000 at risk on two farms in south-east Queensland in late October 2006. Assays showed that the feed contained 50 times the normal level of nicarbazin. Birds started dying within hours of access to the contaminated

feed batch and the feed was immediately withdrawn. Residue testing was performed on a sample of the remaining broiler birds before approval was granted for their processing for consumption.

Respiratory cryptosporidiosis

Two 6-week-old Sussex chicks were submitted for autopsy with a history of refractory respiratory disease and showing conjunctivitis and gasping respiration. Eight birds from a mixed flock of 80 birds were affected. At autopsy, the birds had bilateral conjunctivitis with eyelids glued together with exudate. Tracheas contained a mild to moderate mucoid exudate. No other gross abnormalities were noted. Histologically, there was a moderate chronic catarrhal tracheobronchitis with large numbers of *Cryptosporidium* sp. on the surface of the hyperplasic mucosal epithelium. Virus isolation was negative as was *Mycoplasma* culture.

South Australia

Contributed by: Celia Dickason, Department of Primary Industries and Resources

Hypophosphataemia in cattle

During spring 2006, a beef cattle producer in the north of the state suffered a spate of 20 cattle deaths over a period of a few weeks. The animals were from a herd of 100 mature cows. The affected cattle were reluctant to move or recumbent, and once they were down they usually died within 24 hours. All cattle were in reasonable condition. The producer suspected calcium deficiency and supplemented the cattle with gypsum, but symptoms and deaths continued. The area was very dry at the time, with little paddock feed, and additional rations of hay and a commercial molasses product containing urea and minerals were being fed. Blood serum samples were collected from a number of cattle, both with and without clinical signs, and all results demonstrated a severe serum phosphorus deficiency. Phosphorus supplementation was given by mixing dicalcic phosphate with molasses and spreading it on the hay being fed to the cattle. Shortly after this, the symptoms began resolving and there have since been very few deaths. The far-north area of the state is known for its phosphorus-deficient soils; this has been demonstrated in previous studies. Phosphorus supplementation is often provided for cattle, especially during dry seasons.

Lead toxicity in cattle

Lead poisoning occurred in cattle on a property in the Murraylands during November and December 2006. Four calves out of a herd of 94 cows and calves died suddenly within 2 weeks of being moved to a stubble paddock. This group of cattle was removed from the paddock and about a month later another group of 83 steers and heifers was placed in the same paddock. A few days after placement, some cattle developed central nervous symptoms and salivation, followed by death. After the death of five animals, this group was also removed from the paddock. Diagnosis was complicated by the lack of any evidence to support the suspicion of an inorganic toxin. After the second incident, the property owner recalled that 2 years previously, some 40-year-old waste fill had been used for a pipe trench that cut through that paddock. This waste fill contained remnants of old batteries. Only on close inspection of the trench line was it apparent that there were small parts of batteries scattered along the length of the trench, with signs of recent scrape marks consistent with cattle chewing. Blood lead levels of affected animals in the second group confirmed lead toxicity. Cattle had grazed this paddock the previous year with no illness occurring. It is assumed that disturbance of the soil in the paddock at sowing had brought the debris to the surface. It is surprising how little battery material was evident, and that this had resulted in such dramatic and acute symptoms. The unaffected cattle in the two groups were considered to be potentially contaminated with lead, and a management program, in accordance with approved guidelines for possible stock lead residues, is being followed, with the producer electing to detain these stock on the property for a determined period.

Enterotoxaemia in the mid-north

Two farmers in the mid-north of the state lost sheep from enterotoxaemia. Twelve of 200 weaner lambs on barley stubble died suddenly in early November 2006. They had received only one clostridial 6-in-1 vaccination, 6 months previously at marking, and had been purchased and brought onto the property

from a pastoral station 3 weeks before the incident. Histopathology revealed nonspecific enteritis, and small intestinal contents tested positive to epsilon toxin. The lambs were also found to have a significant worm burden. A second farmer lost several unvaccinated ewes on a pea stubble over a 1-week period in December. While epsilon toxin was not detected, the ewes had histopathological evidence of severe enteritis, suggestive of enterotoxaemia. These cases reinforce the importance of vaccinating sheep correctly, especially when they will have access to grain.

Nonspecific bacterial wound contamination

Laboratory tests and examinations in sheep from two separate incidents, both with clinical signs involving the musculoskeletal system, revealed cutaneous and muscle lesions attributable to wound contamination. The first incident involved 15 animals out of a flock of 246 sheep, three of which died. The affected animals developed lameness and extensive swelling of one or more limbs. Clostridial myositis or bacterial myositis, secondary to skin and muscle trauma was suspected. Affected areas were swollen, with crusted haemorrhage, bruising, laceration and matting of the wool. Histologically there was pronounced and extensive subacute serofibrinous to fibrinopurulent cellulitis, fasciitis and myositis with multiple microabscesses, thrombotic phlebitis and lymphangitis and bacterial colonisation by gram-positive coccobacilli and gram-negative bacilli. These findings correlated with the isolation of non-spore forming anaerobes from a wound swab. These bacteria form part of an ill-defined group of bacteria that are often associated with superficial infection sites, abscesses, deep wounds or ear infections. They include both gram-negative bacilli such as *Bacteroides* spp. and gram-positive cocci such as *Peptostreptococcus* and related species. The pathology and culture findings excluded clostridial myositis and confirmed severe skin and muscle laceration consistent with trauma, the latter providing conditions suitable for anaerobic bacterial colonisation and growth.

The second incident involved six 4-month-old Merino wether lambs (from 300) in the mid-north area. They developed lethargy and perineal oedema and four of them died about 9 days after the application of tail elastrators. Culture of affected tissue yielded mixed enteric and skin flora and histologically there was a septic dermatitis, cellulitis and fasciitis with extensive necrosis and devitalisation of the tail musculature. There was also subcutaneous microabscessation and mixed-type bacterial colonisation. Tetanus may be a complication of elastrator-induced castration or tail docking, but the findings in this case are consistent with wound contamination by bacterial faecal and skin flora.

Tasmania

Contributed by: Mary Lou Conway, Department of Primary Industries, Water and Environment

Urea toxicity in a dairy herd

Acute urea toxicity caused the deaths of 71 cows from a herd of 550 on a north-west dairy farm. The cows died after accidental spillage of fertiliser into a water channel. The contamination occurred in the morning of a relatively hot day with consequential high water intakes by the cows in the affected paddock. The farmer failed to recognise the risk of toxicity at the time of the accident and did not revisit the paddock until 5 hours later. Most mortalities occurred within those 5 hours. Post mortem examination revealed rumen fluid containing between 55 and 106 mmol/L urea and an elevated pH (7.5). Interestingly, there were no significant findings in the vitreous humour samples, probably indicating inadequate time to equilibrate. The urea, pH and ammonia content of water samples taken in the immediate vicinity of the spill and from the adjacent channel were all markedly elevated.

Notifiable diseases

Disease	Investigations	
	Positive	Total
Abalone ganglioneuritis	0	2
American foulbrood	0	1
Avian psittacosis	0	2
Brucella suis	0	1
Brucella abortus	0	3
Classical swine fever	0	1
Contagious agalactia	0	1
Devil facial tumour disease	30	49
Enzootic bovine leucosis	0	1
Equine viral arteritis	0	1
Hydatid disease	1	3
Johne's disease	7	34
Leptospira hardjo	0	12
Leptospira pomona	0	12
Listeria	0	2
Negative finfish bacteriology*	0	52
Ovine macrocyclic lactone resistance	1	2
Clinical salmonellosis	14	71
Pullorum disease (*Salmonella* Pullorum)	0	1
Rickettsia-like organism of salmonids	0	5
Salmonella Abortusovis	0	10
Salmonella Enteritidis	0	1
Viral encephalopathy and retinopathy	0	1

* *Aeromonas salmonicida* ssp. *salmonicida*, goldfish ulcer disease, streptococcosis of salmonids

Laboratory accessions

Source	Number of accessions
Aquaculture	85
Companion	110
Livestock	408
Other	8
Wildlife	103

Suspected ergovaline toxicity in dairy cows

Eighteen of a herd of 300 Friesian cows were lame in the hind legs. Only one paddock on the farm, situated in the Meander Valley in the north of the state, was affected. Hind limbs were swollen and inflamed and some sloughing was evident on both hind and fore legs. Of five affected animals sampled, all were suffering a current inflammatory process and muscle damage. There was no evidence of hepatocellular insufficiency. Post mortem examination of one affected cow that succumbed to ketosis revealed superficial necrosis of the epithelium and marked subcutaneous oedema, as well as kidney pathology (protein-losing nephropathy). The combination of kidney and skin lesions prompted an investigation of possible ergot alkaloid involvement. Pasture samples collected 3 days later were found to have levels of ergovaline close to, but not exceeding, those expected to cause clinical toxicosis. Levels of lolitrem B, a neurotoxin which, like ergovaline, is associated with ryegrass staggers, were considered non-toxic.

Macrocylic lactone-resistant worms from goats

Macrocylic lactone resistance in sheep is a notifiable disease in Tasmania. Since September 2006, a detailed investigation of suspect macrocylic lactone resistance of worms in goats has been undertaken by the Animal Health and Welfare Branch and the Mt Pleasant Animal Health Laboratory. Worm-free sheep were treated with corticosteriods to reduce immune responses to internal parasites. They were subsequently inoculated with worm larvae cultured from the suspect goats' faeces. The sheep were drenched once at 1×, 1.5× or 2× the recommended ivermectin dose. Subsequent total worm counts revealed a 50% reduction in average worm numbers at the 1× dose rate, indicating the larvae were resistant; other dose rates were effective, resulting in a 100% reduction in average worm numbers. The goat flock involved has been treated with an effective regime and moved to a previously uninhabited area where they will stay until their home pastures are shown to be free of larvae.

Victoria

Contributed by: Roger Paskin, Department of Primary Industries

Hardware disease in a heifer

Hardware disease was diagnosed in a supplementary-fed heifer on a property outside Wangaratta in November 2006. The heifer had gradually developed extensive brisket and ventral oedema extending from below the mandible to the udder. The oedema had started on the brisket 2 weeks before calving and progressed with clinical signs of increasing anorexia and abdominal enlargement. The owner noted an intermittent cough with nasal discharge. The heifer had been given a course of long-acting penicillin with no response. She calved with assistance, but because of an absence of milk, the calf was fostered onto a second heifer. Auscultation revealed 'washing machine' heart sounds and reduced lung sounds, and a marked jugular pulse was observed. There was an audible grunt on walking. Autopsy confirmed traumatic reticulo-pericarditis, and a length of no. 8 wire in the reticulum. Other findings included fibrino-purulent pericarditis and a cloudy, blood-stained hydrothorax with associated ascites. This case highlights the need to be vigilant during a drought with stock grazing close to the ground or being fed supplementary feed.

Sudden death in Friesian calves

Cyanide poisoning was the probable cause of death of six out of 18 Friesian calves on the Bellarine Peninsula in December 2006. The calves had been weaned several weeks earlier and transported to the property 2 days before the deaths commenced. The property consisted of approximately 15 hectares of improved pasture, but because of drought conditions, pasture growth was minimal, as was supplementary feeding. Three calves were found dead and submitted for autopsy. The other three displayed varying degrees of weakness, convulsions, muscle tremors and mania before dying. Gross findings included oedema, mucosal haemorrhages and rumen contents containing leaf material. Histological changes were minimal. Differential diagnoses included acute intoxication from sources such as blue-green algae, nitrate and cyanide. Botulism, anthrax and inorganic toxins were also considered.

The day after the calves' arrival, a large branch from a plantation of sugar gums (*Eucalyptus cladocalyx*) bordering the fence line had fallen into the paddock. Given the paucity of other feed sources, the calves had stripped the branch of leaves. Deaths occurred that night. *Eucalyptus cladocalyx* is a recognised source of cyanide, as are many other eucalypts and plant species. Cyanogenic glycosides in the leaves yield free hydrocyanic (HCN or prussic) acid when hydrolysed or when other cell structures are damaged or disrupted physically or chemically. Ruminal microbes can further release free cyanide. Grazing wilted plants during drought is the most common cause of poisoning of livestock by HCN-containing plants. Leaves from the tree in question were submitted for cyanide quantification and were found to contain over 1200 ppm (dry weight). Plants containing greater than 200 ppm (dry weight) cyanide are considered potentially lethal. The cost of this incident to the producer was $5000.

Parasitism in merino wethers

Parasite infestation caused the deaths of 12 of 900 merino wethers and scouring in 5–10% of a flock near Ballarat in south-west Victoria over a week in October 2006. Alarmed by the initial deaths, the producer drenched the mob with the parasiticide abamectin, which failed to stop the deaths. The mob had not previously been drenched for at least 9 months. Four sick wethers examined 4 days post-drenching were weak and inappetent, with pale mucous membranes. Faeces varied from solid to watery diarrhoea, and faecal egg counts ranged from 150 to 12 000 eggs/g. Abomasitis and enteritis consistent with parasitism was diagnosed on histology. The history and clinical and pathological findings supported the diagnosis. Deaths continued for 10 days after the initial examination, costing the producer $1800. Regular faecal counts were recommended, even in symptomless mobs.

Obstructive urolithiasis in merino wethers

Obstructive urolithiasis led to the deaths of 18 3½-year-old merino wethers on agistment at Underbool, north-west Victoria. These deaths

occurred over a 2-week period in October 2006 in wethers grazing pastures that potentially had a high concentration of oxalate-containing plants. On autopsy, urinary calculi were found in the distended bladder. Fibrinosuppurative and necrohaemorrhagic cystitis was diagnosed histologically. Salt blocks and clean water were provided to the sheep on the home property and deaths ceased a week later. The cost to the producer was $1000.

Enterotoxaemia in Boer kids

Enterotoxaemia was the probable cause of the deaths of four 3-week-old Boer kids at Nyah West, in north-west Victoria during November 2006. One kid was presented with circling, head-pressing, slight nystagmus, a temperature of 38°C and minimal suck reflex. The goat was unresponsive to veterinary treatment so was euthanised and the brain submitted for histology, as listeriosis was considered to be a differential diagnosis. Histologically there was mild cortical oedema and a perivascular fluid lake suggestive of vascular damage and cerebral oedema. This finding is characteristic of toxaemia due to epsilon toxin. The cost to the producer was $250.

Superficial dermatitis with vesiculation and ulceration in two piglets

In December 2006, two 8-day-old Duroc-cross piglets were presented to a north-east Victorian veterinary surgery after being inappetent and lethargic for a day. The owner had noticed a nasal discharge for the previous 3 days. Both piglets were reluctant to walk and rested in a dog-sitting position. Clinical examination revealed ulcerations on the nose and suspected burst vesicles on the soles and heel bulbs of all four feet in both piglets. The male piglet had an ulcer on his tongue and the female piglet was hypothermic and had an inflamed, infected umbilicus. The piglets had come from a neighbour who owned other pigs. The piglets were euthanised and sent to Primary Industries Research Victoria, Attwood, for diagnostic tests to exclude foot-and-mouth disease (FMD). Further samples were sent to the Australian Animal Health Laboratory. FMD was excluded and the piglets were diagnosed as having superficial dermatitis consistent with *Staphylococcus hyicus* (greasy pig disease).

Ibis mortalities at a wildlife park

Thirty-five ibis were found dead or displaying degrees of paralysis at a wildlife park 60 km west of Melbourne in December 2006. The birds were part of a large flock of hundreds of ibis that roosted at the park. The birds often flew to nearby coastal areas to feed and particularly favoured a waste-disposal site on the north shore of the Geelong waterfront. The deaths occurred over a 10-day period, with clinical signs ranging from an inability to fly, to ataxia and leg paresis or paralysis. All affected birds died within 2–3 days. Two birds were submitted for autopsy, but there were no gross or histological changes evident.

An examination of the waste-disposal site confirmed large numbers of ibis feeding. There was a significant amount of vegetable matter present, much of which was rotting. Suspected causes of death included botulism, algal or fungal toxins or chemical poisoning. Botulism due to ingestion of preformed toxins produced by *Clostridium botulinum* in the feeding material was considered a likely cause. No other species of birds or animals at the wildlife park were affected, therefore the feeding practice of the ibis away from the park was considered a risk factor. In addition, decaying vegetation is a well-recognised cause of botulism amongst waterbirds worldwide. Routine surveillance of all birds and mammals at the park has been instituted. There have been no further deaths since the initial outbreak.

Infectious laryngotracheitis in broilers

Infectious laryngotracheitis was confirmed as the cause of a sudden increase in mortalities on a north-west Victorian broiler farm in November 2006. The mortalities were confined to one shed of a multi-shed operation and were more severe, for a longer time, in the end of the shed housing female birds. The cooling pads for the shed were also here. Birds presented for autopsy had conjunctivitis, sinusitis and severe mucohaemorrhagic tracheitis. Histology on the trachea and upper airways showed a severe haemorrhagic, necrotising tracheitis with typical herpes-viral inclusions in syncitial cells. A herpes virus producing typical inclusions was isolated and the birds were negative for avian influenza by rapid methods, PCR and viral culture. Infectious laryngotracheitis is an uncommon diagnosis in Victoria and the source of the infection was not determined, but there is evidence that it probably entered the shed via the cooling pads at the female

end of the shed. The outbreak on this farm ended when the birds were sent for emergency slaughter.

Western Australia

Contributed by: Fiona Sundeman, Department of Agriculture and Food

During the quarter, 248 investigations of animal disease led to laboratory testing. There were 11 exotic disease alerts (i.e. investigations of suspected nationally notifiable diseases), but no diseases notifiable nationally or in Western Australia were confirmed. All 11 exotic disease investigations were category 1 alerts (low index of suspicion). Ten involved routine exclusion of avian influenza and Newcastle disease in poultry and other avian species and one case of *Brucella melitensis* in a dog. A diagnosis of an endemic disease was made in all cases.

Cattle

Listeriosis was diagnosed in five cows that died over a single weekend at Busselton. Histopathological examination revealed characteristic lesions in the hindbrain.

Lead poisoning was diagnosed as the cause of death of three of 30 adult cattle on agistment at Walpole. Cohort animals were managed according to the Western Australian lead management policy.

Blindness and neurological signs were reported in a 6-month-old Murray Grey calf from a group of 20 at Denmark. The calves had access to old batteries. The high kidney lead level (520 mg/kg, normal <0.1 mg/kg) confirmed the histological evidence of lead poisoning. This case continues a mini epizootic of lead poisoning, with 10 cases diagnosed in 2005 and 2006 in Western Australia.

Salmonellosis contributed to ongoing morbidity in dairy calves on a Scott River property, with a history of neonatal diarrhoea, dehydration and deaths commencing within the first 48 hours of life. Confirmed diagnoses include infections with *Salmonella* Bovismorbificans and *Salmonella* Kiambu. Heavy *Cooperia* sp. burdens as well as a putative diagnosis of an enterotoxic *Escherichia coli* have previously been made in 1–6-week-old calves from the same property.

A recent case involved a 2-day-old bull calf that commenced scouring at 12 hours of age. Histological lesions consistent with salmonellosis were supported by the recovery of *Salmonella* Bovismorbificans from faeces, liver and spleen. The unusual feature of the case was the very young age of the affected calf. The clinical and histopathological features suggested this animal must have been infected during or soon after birth.

Consumption of *Isotropis atropurpurea* was the likely cause of severe renal disease in 50 of 2000 Brahman heifers on a Pilbara station. The diagnosis of renal failure, as suggested by markedly elevated plasma creatinine and urea levels, was supported by histology. The plant had been identified on the property in 2002 and may have been responsible for losses in previous years.

Pneumonia was diagnosed on several south-west properties. On one farm at Waroona, four Friesian calves died and another 10 of 600 were clinically ill. Autopsy of a calf that died revealed severe pneumonia with consolidation of the dorsal lobes, and pleuritis and adhesions involving the ventral lobes. Mixed growths of *Pasteurella multocida*, *Histophilus somni* and *Arcanobacterium pyogenes* were obtained from culture. On another property at Harvey, *Mannheimia haemolytica* was isolated from severe lung lesions found in one of three 2-year-old dead Friesian cows.

Pneumonia and **pestivirus encephalitis** were diagnosed in a recently purchased cow at Narrikup. Autopsy revealed consolidation of 25% of the cranio-ventral lobes of the lungs. The histopathogical nature of the lesions suggested a bacterial aetiology, but culture was unremarkable. However, results from the pestivirus capture enzyme-linked immunosorbent assay (ELISA) were positive. Widespread distribution of pestivirus antigen associated with an inflammatory response was demonstrated in large neurons in the hippocampus and cerebral cortex by immunohistochemical staining.

Bovine pedal epidermal necrosis was diagnosed in two beef calves at Narrogin. Their owner had

recorded six cases in the past 2 years, mainly from the same paddock on the farm. Typically the condition began at the coronary bands, then extended under the soles and eventually caused sloughing of the hooves. There was no association with ergot-contaminated feed, season or climatic conditions. The histological lesions were suggestive of trauma with secondary bacterial infection.

Sheep

Erysipelas caused sudden death in 15 of 1500 4-month-old weaners at Quairading. *Erysipelothrix rhusopathiae* was cultured from both kidney and liver. Erysipelas in sheep usually presents as polyarthritis, but in rare cases, death may result from septicaemia.

Copper poisoning caused the death of five of 15 rams at Busselton. On autopsy, one carcase was noted to be jaundiced. There was also haematuria, with the kidneys dark and the liver swollen, friable and orange. Histological lesions consistent with copper toxicity were found in the liver and kidneys. Kidney and liver copper levels confirmed the diagnosis.

Enzootic ataxia due to copper deficiency was confirmed in 50 of 300 1–4-month-old Damara/Dorper-cross lambs at Binnu showing clinical weakness. Axonal degeneration was noted along the length of the cord of an affected lamb. Plasma and tissue copper analysis confirmed the diagnosis.

Listeriosis caused the death of 20 of 600 lambs in a Borden feedlot. The sheep were being fed silage and alkalage. Affected animals were seen circling and frothing at their mouths. Microscopic brain lesions typical of listeriosis were present. Listeriosis is often associated with silage feeding.

Pigs

Glässer's disease caused depression and deaths in 5-week-old weaner pigs at Doodlakine. The 100-sow piggery had experienced a rise in post-weaning mortality over the previous month. Pigs became depressed and inappetent and began coughing. Severe pericarditis, pleuritis and peritonitis were seen at autopsy. *Haemophilus* sp. recovered from tissue was identified as *H. parasuis* by PCR.

Pneumonia associated with *Staphylococcus aureus* was seen in a line of 200 22-week-old pigs at slaughter at Kojonup. These animals were from the quarantine facility of a herd in which increased prevalence of lung lesions had been noted at slaughter two weeks previously. *Staphylococcus aureus*, an unusual cause of pneumonia, was recovered from all lung samples. In this instance, *S. aureus* was probably a secondary opportunist. Histological lesions were longstanding and probably initiated by *Mycoplasma* or *Pasteurella* spp.

Poultry

Infectious laryngotracheitis was the cause of respiratory illness and death of nine of 30 backyard poultry near Albany. Autopsy revealed large casts of friable material in the larynx and trachea, completely occluding the airway in two of the birds.

Marek's disease was diagnosed in Japanese bantams at Koorda. A 1-year-old hen examined was in very thin condition with pale comb and wattles. Three-quarters of the liver contained small yellow nodules that had microscopic features typical of Marek's disease.

Quarterly disease statistics

Control activities

Ovine brucellosis

Contagious epididymitis, caused by *Brucella ovis*, is present in commercial flocks at a low level that varies around the country. Voluntary accreditation programs (usually in stud flocks) for ovine brucellosis freedom are operating in all states. Table 1 shows the number of accredited flocks at the end of the quarter.

Table 1 Ovine brucellosis accredited-free flocks at 31 December 2006

State	Free
ACT	2
NSW	904
NT	0
QLD	58
SA	378
TAS	96
VIC	517
WA	173
AUS	2128

Johne's disease

In Australia, Johne's disease occurs primarily in dairy cattle and sheep, and to a lesser extent in beef cattle, goats, deer and camelids. Infection with sheep strains occurs to varying extents across the sheep-producing regions of southern Australia but it has not been detected in Queensland. Cattle strains are endemic in south-eastern Australia but surveillance programs have not identified endemic infection in Queensland, Western Australia or the Northern Territory, and active measures are taken to stamp out any incursions. Table 2 shows the number of herds and flocks known to be infected.

Table 2 Number of herds/flocks infected with Johne's disease at 31 December 2006

	Cattle	Goat	Deer	Sheep	Total
NSW	114	8	1	1286	1409
NT	0	0	0	0	0
QLD	0	1	0	0	1
SA	63	1	1	75[a]	140
TAS	16	3	0	58	77
VIC	946	7	6	476	1435
WA	1	0	0	17	18
AUS	1140	20	8	1912	3080

a Seven of these flocks are infected with 'c' strain.

New approaches based on risk assessment and management have been developed to control Johne's disease. Market Assurance Programs are in operation for cattle, sheep, goats and alpacas; the number of herds or flocks that have reached a status of Monitored Negative 1 or higher is shown in Table 3.

Table 3 Herds/flocks with a Market Assurance Program status of at least Monitored Negative 1 at 31 December 2006

	Alpaca	Cattle	Deer	Sheep	Total
NSW	112	578	33	337	1060
NT[a]	0	0	0	0	0
QLD[a]	2	0	0	0	2
SA	46	266	18	194	524
TAS	1	108	3	31	143
VIC	23	306	1	84	414
WA	0	0	0	0	0
AUS	184	1258	55	646	2143

a Herds/flocks in free or protected zones have a status of 'Monitored Negative 1' or better because of the zone status.

Lists of beef, dairy and alpaca herds and sheep flocks assessed in the Market Assurance Programs are available at http://www.animalhealthaustralia.com.au/programs/jd/maps.cfm

Information about components of the National Johne's Disease Control Program can be obtained from State coordinators and Animal Health Australia's Johne's disease coordinator, David Kennedy (02 6365 6016).

Enzootic bovine leucosis

Enzootic bovine leucosis accreditation programs have been operating in the dairy industries in Queensland and New South Wales for several years. Victoria, South Australia, Western Australia and Tasmania are undertaking a program of bulk milk testing of all dairy herds. Table 4 shows the number of dairy herds tested free of enzootic bovine leucosis at the end of the quarter.

Table 4 Dairy herds tested free of enzootic bovine leucosis at 31 December 2006

State	Infected	Non-assessed	BMT[a] negative	Provisionally clear	Monitored free	Total
NSW	0	21	20	0	860	901
NT	0	0	0	0	0	0
QLD	2	0	0	0	882	884
SA	0	3	0	0	366	369
TAS	0	486	0	0	0	486
VIC	39	47	1914	32	3082	5114
WA	0	0	0	0	241	241
AUS	41	557	1934	32	5431	7995

a bulk milk test

Laboratory testing

Table 5 shows the results of serological testing for a range of viral diseases from routine laboratory submissions for the quarter.

Table 5 Serological testing from routine submission to State laboratories

	Akabane[a]		Bovine ephemeral fever[a]		Bluetongue[a]		Enzootic bovine leucosis		Equine infectious anaemia		Equine viral arteritis	
	Tests	+ve	Tests	+ve	Tests	+ve	Tests	+ve	Tests	+ve	Tests	+ve
Oct–Dec 2005	4926	383	1586	252	8429	272	1526	3	719	14	343	6
Jan–Mar 2006	1667	394	1321	291	5669	254	1889	0	462	0	273	9
Apr–Jun 2006	1970	460	1398	290	2492	297	1341	3	740	1	281	6
Jul–Sep 2006	2724	392	1302	152	6670	318	723	0	975	1	542	4
Oct–Dec 2006												
NSW	5561	83	441	49	8483	36	267	8	660	0	191	0
NT	535	139	597	107	647	239	0	0	19	2	0	0
QLD	555	201	595	96	591	80	384	0	168	0	14	1
SA	67	0	67	10	70	0	1217	0	2	0	1	0
TAS	3	0	0	0	1	0	76	0	0	0	0	0
VIC	6	0	5	0	36	0	0	0	323	0	118	12
WA	766	16	226	51	1068	10	2	0	82	0	62	0
Total	7493	439	1931	313	10896	365	1946	8	1254	2	386	13

a http://www.animalhealthaustralia.com.au/programs/adsp/namp/namp_home.cfm

Surveillance activities

National Transmissible Spongiform Encephalopathies Surveillance Program

The National Transmissible Spongiform Encephalopathies Surveillance Program (NTSESP) is an integrated national program jointly funded by industry and governments to demonstrate Australia's ongoing freedom from bovine spongiform encephalopathy and scrapie, and to provide early detection of these diseases should they occur. Table 6 summarises the activity of the program over the past five quarters. All specimens tested were negative for transmissible spongiform encephalopathies. Information about the NTSESP is available at http://www.animalhealthaustralia.com.au/aahc/programs/adsp/tsefap/ntsesp.cfm.

Contact: Duncan Rowland, Animal Health Australia's NTSESP National Coordinator

Table 6 Transmissable spongiform encephalopathy surveillance

	Oct–Dec 2005		Jan–Mar 2006		Apr–Jun 2006		Jul–Sep 2006		Oct–Dec 2006	
State	Ovine	Bovine	Ovine	Bovine	Ovine	Bovine	Ovine	Bovine	Ovine	Bovine
NSW	27	25	19	18	16	17	27	34	61	20
NT	0	5	0	0	0	10	0	10	0	3
QLD	1	37	1	34	7	49	11	77	11	49
SA	7	4	4	3	4	5	15	13	37	3
TAS	0	0	1	2	1	2	0	6	5	1
VIC	52	78	13	23	47	32	36	39	44	15
WA	67	11	14	11	23	7	17	14	92	5
Total	154	160	52	91	98	122	106	193	250	96

Bovine brucellosis

Although bovine brucellosis is now exotic to Australia, surveillance is maintained through abortion investigations and miscellaneous testing of cattle for export or other reasons. As shown in Table 7, 167 abortion investigations were performed during the quarter, all with negative results for bovine brucellosis.

Table 7 Surveillance for bovine brucellosis

	Abortion		Other reasons	
	Tests	+ve	Tests	+ve
Oct–Dec 2005	201	0	1038	0
Jan–Mar 2006	274	0	2215	0
Apr–Jun 2006	204	0	1702	0
Jul–Sep 2006	120	0	4456	0
Oct–Dec 2006				
NSW	1	0	679	0
NT	0	0	1125	0
QLD	15	0	289	0
SA	2	0	0	0
TAS	13	0	23	0
VIC	0	0	51	0
WA	136	0	216	0
Total	167	0	2383	0

Salmonella surveillance

The National Enteric Pathogen Surveillance Scheme (NEPSS) is operated and maintained on behalf of the Australian, State and Territory governments by the Microbiological Diagnostic Unit at the University of Melbourne. Data on isolates of salmonellae and other pathogens are submitted to NEPSS from participating laboratories around Australia. Quarterly newsletters and annual reports of both human and nonhuman isolates are published, and detailed data searches are provided on request to NEPSS. Table 8 summarises *Salmonella* isolations from animals notified to NEPSS for the quarter.

Contact: National Enteric Pathogen Surveillance Scheme, Microbiological Diagnostic Unit, University of Melbourne

Table 8 *Salmonella* notifications, 1 October to 31 December 2006

	Avian	Bovine	Canine	Equine	Feline	Ovine	Porcine	Other	Total
S. Bovismorbificans	0	13	0	0	0	0	0	0	13
S. Dublin	0	20	0	0	0	0	0	0	20
S. Infantis	0	1	1	0	0	0	0	0	2
S. Typhimurium	49	26	2	4	2	2	7	0	92
Other	12	12	10	7	1	0	5	16	63
Total	61	72	13	11	3	2	12	16	190

Tuberculosis

Australia was declared free from bovine tuberculosis (TB) on 31 December 1997, exceeding the World Organisation for Animal Health (OIE) requirements for declaration of country freedom. The last outbreaks of TB were detected in buffalo in January 2002 and in cattle in December 2000, and trace-forward and trace-back slaughter were carried out according to the Tuberculosis Freedom Assurance Program (TFAP).

All Australian laboratories supporting TFAP are accredited for veterinary testing by the National Association of Testing Authorities under ISO/IEC 17025. Laboratories approved for culture of *Mycobacterium bovis* must pass an external quality assurance program run by the Australian reference laboratory for TB on an annual basis.

The National Granuloma Submission Program has been the major surveillance tool for TB since 1992. Tables 9 and 10 summarise results from the program.

Table 9 National case register for bovine tuberculosis

	2002	2003	2004	2005	2006
NSW	0	0	0	0	0
NT	2[a]	0	0	0	0
QLD	1[b]	0	0	0	0
SA	0	0	0	0	0
TAS	0	0	0	0	0
VIC	0	0	0	0	0
WA	0	0	0	0	0
AUS	3	0	0	0	0

a Buffalo herd

b These cases are secondary to the case detected in Queensland in December 2000

Definitions: Primary cases are those detected in case herds. Case herd is a herd in which a case of TB has been found, that was previously tested negative, monitored negative or confirmed free 1, 2 or 3 (excluding secondary case herds) as defined in the TB standard definitions and rules. Secondary cases are those detected in secondary case herds. A secondary case herd means a herd found to be infected with tuberculosis following tracing from a case herd.

Table 10 Results of the National Granuloma Submission Program

	Oct–Dec 2005	Jan–Mar 2006	Apr–Jun 2006	Jul–Sep 2006	Oct–Dec 2006
Submitted	488	362	371	378	169
TB +ve	0	0	0	0	0

Northern Australian Quarantine Strategy

In recognition of the special quarantine risks associated with Australia's sparsely populated northern coastline, the Australian Quarantine and Inspection Service conducts an animal disease surveillance program as an integral component of the Northern Australia Quarantine Strategy (NAQS). The NAQS surveillance program provides early warning of disease threats to livestock industries and, in some cases, to human health. NAQS surveillance activities include both offshore and onshore components. Table 11 summarises NAQS activity in Australia over the past five quarters.

Contact: Jane Parlett, Australian Quarantine and Inspection Service, Australian Government Department of Agriculture, Fisheries and Forestry

Table 11 Summary of recent NAQS activity in Australia

	Oct–Dec 2005		Jan–Mar 2006		Apr–Jun 2006		Jul–Sep 2006		Oct–Dec 2006	
Category	Tested	+ve	Tested	+ve	Tested	+ve	Tested	+ve	Tested	+ve
Aujeszkys disease	13	0	19	0	16	0	147	0	225	0
Avian influenza — highly pathogenic	710	0	15	0	0	0	413	0	1833	0
Classical swine fever	13	0	19	0	16	0	147	0	225	0
Japanese encephalitis[a]	89	0	79	1	193	0	51	0	71	0
Surra — *Trypanosoma evansi*	84	0	10	0	334	0	124	0	185	0

a The positive result noted in the table for Japanese encephalitis (JE) occurred in a pig bled on one of the northern islands in the Torres Strait. These islands experience seasonal incursions of JE. JE remains exotic to the Australian mainland.

Ports Surveillance Program

Biosecurity Australia conducts the Ports Surveillance Program for *Culicoides*, screw-worm fly, exotic bees and bee mites. Seaports, particularly those servicing returning livestock vessels and those dealing with high-risk deck cargo such as timber, mining equipment and containers, are considered to be high-risk locations for incursions of such pests. The program increases the capacity to detect any incursions at an early stage, and this in turn increases the probability of a successful eradication program. The *Culicoides* surveillance also supports the livestock export trade by confirming the continuous or seasonal absence of *Culicoides* vectors at ports from which livestock are loaded. Table 12 shows the number of times that insect trap sites were inspected for the Port Surveillance Program; no exotic insects or mites were detected.

Contact: Leigh Nind and Howe Heng, Biosecurity Australia, Australian Government Department of Agriculture, Fisheries and Forestry

Table 12 Ports Surveillance Program: number of inspections of insect traps

		Oct–Dec 2005	Jan–Mar 2006	Apr–Jun 2006	Jul–Sep 2006	Oct–Dec 2006
Ports	Asian bees	12	7	7	6	17
	Varroa mites	22	28	34	17	26
	Asian mites	22	28	34	17	26
	Tracheal mites	22	22	33	18	43
	Culicoides sp.	28	27	30	27	28
	Screw-worm fly	24	22	24	23	20
NAQS	Screw-worm fly	45	45	45	45	45

Suspect exotic or emergency disease investigations

There were 43 investigations of diseases suspected to be either exotic or a possible emergency reported during the quarter, as shown in Table 13. More details about some of these investigations can be found in the State and Territory reports.

Table 13 Exotic or emergency disease investigations reported, 1 October to 31 December 2006

Disease	Species	State	Month	Response	Finding
Australian bat lyssavirus	Feline	NSW	Oct	3	negative
Avian influenza — highly pathogenic	Avian	NSW	Oct	2	negative
	Avian	NSW	Nov	2	negative (2 unrelated investigations)
	Avian	NSW	Dec	2	negative
	Avian	NT	Nov	3	negative
	Avian	QLD	Oct	2	negative (2 unrelated investigations)
	Avian	QLD	Dec	1	poison
	Avian	SA	Nov	3	negative
	Avian	VIC	Oct	2	negative
	Avian	VIC	Nov	2	infectious laryngotracheitis (2 unrelated investigations)
	Avian	VIC	Dec	1	poison
	Avian	VIC	Dec	2	negative
	Avian	WA	Oct	2	negative (2 unrelated investigations)
	Avian	WA	Nov	2	negative (5 unrelated investigations)
	Avian	WA	Dec	2	negative
	Avian	WA	Dec	3	negative (2 unrelated investigations)
Bluetongue — clinical disease	Ovine	QLD	Nov	3	avocado poisoning
Classical swine fever	Porcine	TAS	Oct	3	negative
Foot-and-mouth disease	Bovine	NSW	Dec	3	negative
	Ovine	VIC	Dec	2	negative
	Porcine	VIC	Dec	3	*Staphylococcus hyicus*
	Porcine	VIC	Dec	3	exudative dermatitis
Hendra virus	Equine	NSW	Nov	3	positive
	Equine	NT	Dec	3	negative
	Equine	QLD	Nov	3	negative (2 unrelated investigations)
	Equine	QLD	Dec	3	negative
	Equine	VIC	Nov	2	acute pulmonary oedema
	Equine	VIC	Nov	3	negative
	Equine	VIC	Dec	3	negative
Newcastle disease — virulent	Avian	NSW	Oct	3	negative
Rabies	Feline	NSW	Oct	3	negative
	Feline	NSW	Dec	3	negative
Transmissible gastroenteritis	Porcine	VIC	Oct	3	negative

Key to response codes

1: Field investigation by government officer; 2: Investigation by State or Territory government veterinary laboratory; 3: Specimens sent to the Australian Animal Health Laboratory (or CSIRO Division of Entomology); 4: Specimens sent to reference laboratories overseas; 5: Regulatory action taken (quarantine or police); 6: Alert or standby; 7: Eradication

Zoonoses

The National Notifiable Diseases Surveillance System (NNDSS) of the Communicable Diseases Network Australia collects statistics about many human diseases. A summary of information about five important zoonoses is submitted to the National Animal Health Information System each quarter (see Table 14).

Contact: National Notifiable Diseases Surveillance System, Australian Government Department of Health and Ageing (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/Nationally+notifiable+diseases+(NNDSS)-2)

Table 14 Notification of zoonotic disease in humans

	Q4 2005	Q1 2006	Q2 2006	Q3 2006	Q4 2006	Current quarter (Oct – Dec 2006)								
			AUS			ACT	NSW	NT	QLD	SA	TAS	VIC	WA	AUS
Brucellosis	18	14	5	14	10	0	4	0	6	0	0	0	0	10
Chlamydophilosis	39	41	39	38	46	0	22	0	0	0	0	23	1	46
Leptospirosis	28	51	59	20	15	0	1	0	11	0	1	2	0	15
Listeriosis	18	25	7	14	13	1	3	0	3	1	0	2	3	13
Q fever	85	101	89	100	104	0	51	1	44	4	0	3	1	104

National Residue Survey

There were 4115 samples collected and analysed in the National Residue Survey Random Monitoring Program for the quarter (see Table 15). Five samples were found with residues above the relevant standard in the Australian Food Standards Code.

One sample of kidney from a pig was found to have residues of sulfadiazine (0.33 mg/kg; Australian maximum residue level (MRL) 0.1 mg/kg) and lincomycin (0.45 mg/kg; MRL 0.2 mg/kg). A trace-back investigation is ongoing but it has been determined that the residues occurred as a result of failure to observe the withholding period.

One sample of liver from a sheep had lead residues of 1.65 mg/kg and a sample of liver from a game pig had residues of 0.91 mg/kg. These levels exceed the Australian maximum level (ML) of 0.5 mg/kg. Lead residues in animals are often the result of exposure to old lead batteries or other lead sources dumped inappropriately. A trace-back investigation on the sheep identified the cause of the residue as animals having access to a site containing old lead batteries. The owner has been required to clean up the area and fence off any areas containing mechanical debris. A trace-back investigation was unable to establish the cause of the residue in the pig.

Two samples of sheep liver had cadmium levels above the ML of 1.25 mg/kg, but below the residue action level of 2.5 mg/kg. A trace-back investigation was not initiated. Cadmium residues above the ML are a common finding in older sheep across southern Australia.

Table 15 National Residue Survey (each pair of figures gives the number of residues above the maximum residue limit (or the maximum level), and the number of samples tested)

		NSW		NT		QLD		SA		TAS		VIC		WA		AUS	
Anthelmintics	cattle	0	56	0	1	0	81	0	7	0	4	0	48	0	13	0	210
	other	0	19	0	1	0	1	0	2	0	0	0	26	0	3	0	52
	pigs	0	26	0	1	0	19	0	11	0	3	0	26	0	6	0	92
	sheep	0	120	0	0	0	4	0	36	0	0	0	86	0	79	0	325
	Total	0	221	0	3	0	105	0	56	0	7	0	186	0	101	0	679
Antimicrobials	cattle	0	87	0	1	0	106	0	15	0	8	0	89	0	15	0	321
	other	0	13	0	1	0	4	0	3	0	0	0	35	0	1	0	57
	pigs	0	74	0	2	2	48	0	49	0	3	0	71	0	31	2	278
	poultry	0	64	0	0	0	24	0	17	0	8	0	23	0	13	0	149
	sheep	0	89	0	0	0	1	0	28	0	5	0	46	0	48	0	217
	Total	0	327	0	4	2	183	0	112	0	24	0	264	0	108	2	1022
Growth promotants	cattle	0	88	0	3	0	123	0	9	0	7	0	43	0	18	0	291
	other	0	5	0	0	0	3	0	2	0	0	0	15	0	0	0	25
	pigs	0	46	0	0	0	35	0	20	0	3	0	50	0	24	0	178
	poultry	0	8	0	0	0	3	0	2	0	1	0	2	0	2	0	18
	sheep	0	94	0	0	0	4	0	27	0	5	0	46	0	66	0	242
	Total	0	241	0	3	0	168	0	60	0	16	0	156	0	110	0	754
Insecticides	cattle	0	99	0	3	0	132	0	25	0	11	0	87	0	28	0	385
	other	0	44	0	0	0	35	0	8	0	0	0	25	0	5	0	117
	pigs	0	19	0	0	0	22	0	19	0	1	0	19	0	12	0	92
	sheep	0	167	0	0	0	4	0	53	0	4	0	115	0	115	0	458
	Total	0	329	0	3	0	193	0	105	0	16	0	246	0	160	0	1052
Metals	cattle	0	20	0	2	0	29	0	2	0	3	0	23	0	7	0	86
	other	1	12	0	1	0	18	0	1	0	0	0	10	0	5	1	47
	pigs	0	25	0	0	0	19	0	19	0	1	0	23	0	7	0	94
	sheep	0	38	0	0	0	1	0	8	0	0	1	26	2	16	3	89
	Total	1	95	0	3	0	67	0	30	0	4	1	82	2	35	4	316
Miscellaneous	cattle	0	18	0	0	0	35	0	6	0	1	0	23	0	5	0	88
	other	0	10	0	0	0	12	0	0	0	0	0	7	0	0	0	29
	pigs	0	23	0	1	0	18	0	13	0	0	0	26	0	12	0	93
	sheep	0	29	0	0	0	1	0	13	0	1	0	19	0	19	0	82
	Total	0	80	0	1	0	66	0	32	0	2	0	75	0	36	0	292
Total		1	1293	0	17	2	782	0	395	0	69	1	1009	2	550	6	4115

NAHIS CONTACTS

The National Animal Health Information System (NAHIS) collects summaries of animal health information from many sources. NAHIS is on the internet (http://www.animalhealthaustralia.com.au/status/nahis.cfm). Because NAHIS does not duplicate the data in the other systems, the relevant person below should be contacted if further details are required.

Name	Role	Phone	Fax	email
Chris Bunn	Emergency Disease Preparedness, DAFF	02 6272 5540	02 6272 3372	chris.bunn@daff.gov.au
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Kristy Venten	Australian Milk Residue Analysis Survey	03 9810 5919	03 9819 4299	kventen@dairysafe.vic.gov.au
Jenny Hutchison	National Surveillance Coordinator	02 6287 4483	02 6287 4468	jenny@ausvet.com.au
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Fiona Sunderman	WA State Coordinator	08 9368 3805	08 9474 2479	fsunderman@agric.wa.gov.au

EMERGENCY ANIMAL DISEASE WATCH HOTLINE — 1800 675 888

The Emergency Animal Disease Watch Hotline is a toll-free telephone number that connects callers to the relevant State or Territory officer to report concerns about any potential disease situation. Anyone suspecting an exotic disease outbreak should use this number to get immediate advice and assistance.

For information about the Emergency Animal Disease Watch Hotline, contact Scott Porteous, Animal Health Australia.

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