ANIMAL HEALTH SURVEILLANCE

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PREFACE

Since the first detection in August 2007, much of Australia's veterinary services have been concentrating on the response to equine influenza. The rapid on-the-ground response has contained the affected area to less than a quarter of New South Wales and south-east Queensland. Movement controls and active surveillance programs have played a key role in limiting the spread of the disease. A detailed report on the incursion and the Australian response is provided in this issue of *Animal Health Surveillance Quarterly* (AHSQ). The report is supplemented by further articles providing the perspective of several of the Australian States. Although a large proportion of resources are targeted at equine influenza, work in other areas must be maintained, and AHSQ reports on a variety of activities.

Other topics in this edition include highlights of disease surveillance activities, items of interest from 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

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Equine influenza in Australia

Equine influenza (EI) was first detected in Australia in August 2007. This report provides an overview of the outbreak and the nationally coordinated response to contain and eradicate the disease up to 30 September 2007. The circumstances of the outbreak of EI in Australia are the subject of a Federal Government Commission of Inquiry, headed by retired High Court Judge — The Hon Ian Callinan AC. Mr Callinan is scheduled to report his findings to the Australian Government in April 2008.

Background

EI is an acute respiratory disease caused by the virus subtypes H7N7 and H3N8. The virus has the potential for very rapid spread, particularly in naive populations. Horses in work are more severely affected, and the disease may cause deaths in young foals and debilitated horses. EI is endemic in Europe, North and South America, and parts of Asia and Africa. Occasional outbreaks occur in these areas, but the impacts are minor due to immunity resulting from the high background level of EI infection and vaccination. Periodic epidemics and serious outbreaks can occur when significantly new strains of the virus emerge or there are new incursions. EI is listed by the OIE (World Organisation for Animal Health). As Australia has been free from EI until now, horses are not vaccinated against the disease and therefore the horse population is highly susceptible to infection.

The outbreak

EI was first suspected in imported horses at Eastern Creek Quarantine Station (Sydney, New South Wales) on 17 August and confirmed 3 days later. Further horses showing symptoms of EI were reported on 22 August in central Sydney. H3 equine influenza virus was later confirmed at both locations by viral sequencing tests, undertaken by the CSIRO Australian Animal Health Laboratory (CSIRO AAHL), Geelong, Victoria, and the properties were identified as infected premises (IPs).

In accordance with protocols established under the Emergency Animal Disease Response Agreement, preparations for a nationally coordinated response began immediately, using Australia's veterinary emergency plan (AUSVETPLAN) as a guide.

Following confirmation of EI outside the quarantine station on 25 August, a 72-hour national standstill on movement of horses was activated.

The virus

Sequence analysis of the matrix gene undertaken at the CSIRO AAHL confirmed that the Australian EI isolate was almost identical to the Wisconsin strain of EI (A/equine/Wisconsin/1/03(H3N8)), with 99.6% similarity at the amino acid level. Sequence analysis of the HA1 region of the haemagglutinin gene also shows close identity with the Wisconsin strain (99% at the amino acid level). Work on virus isolation and genome sequencing continues at the CSIRO AAHL. Samples of the isolate have been forwarded to the OIE Reference Laboratory for Equine Influenza in the United Kingdom.

National coordination

In Australia, responses to emergency animal disease (EAD) incursions are the responsibility of the jurisdiction (State or Territory) in which the disease is detected. To ensure a nationally coordinated response, Australia has developed the Emergency Animal Disease Response Agreement (EADRA).[1] The EADRA brings together the Australian Government, State and Territory governments and livestock industry groups to manage the emergency. Parties to the EADRA have agreed to participate in an EAD response, take all reasonable steps to reduce the risk of the occurrence of disease and share the costs of responding to an EAD using an established formula.

Once an EAD incursion is declared and reported to the Australian Chief Veterinary Officer (ACVO), the EADRA is activated and statutory measures are invoked, as necessary, to deal with the situation. The EADRA establishes two high-level decision-making bodies, both of which are integral to facilitating a rapid response. The Consultative Committee on Emergency Animal Disease (CCEAD) agrees a response plan, coordinates the response, and provides technical advice to the National Emergency Animal Disease Management Group (NMG).

Membership of the CCEAD includes Australian Government representatives, State and Territory government representatives, CSIRO and industry experts. The NMG is responsible for financial issues associated with an EAD response and oversees effective management. Membership of the NMG includes senior executives of the Australian Government, State and Territory governments, and horse industries.

The response to date

The national emergency response to the EI outbreak in Australia is in accordance with the AUSVETPLAN *Disease Strategy for Equine Influenza.* As the Australian horse population was naive, immediate control of horse movements was essential. The first step was a nationwide 72-hour equine standstill. Racing clubs and other equine associations were asked to cancel or postpone any meetings or events. Owners were asked to prevent any contact between their horses and other horses and to obey the national equine standstill. Strict biosecurity measures were publicised and implemented for movements of people, equipment and fomites. Fomites are any inanimate objects that are capable of carrying and transferring the EI virus from one horse or individual to another — for example, horse gear such as saddles, blankets and bridles.

In New South Wales, the whole State was declared a control area for EI. This enabled the New South Wales CVO to make enforceable orders within the State. Horses of any type, including pets, show animals, donkeys and horses from riding clubs, were prohibited from moving. Properties where infection was confirmed were declared IPs. Properties with known direct links to an IP were identified as dangerous contact premises, and properties where a disease might be suspected to exist, but were not yet confirmed, were designated as suspect premises. Horses were tested and quarantined.

The declaration of an IP allowed a restricted area with a 10-km radius to be established around the IPs. This allowed strict disease control measures to be taken, including high levels of biosecurity for movement of equipment, fomites and people, and no movement of horses within, into or out of these areas.

Movements of horses in the week before the initial cases were diagnosed spread the disease to horses in New South Wales and Queensland. Direct horse-to-horse contact and passive transfer by people, equipment and fomites are considered to have been the main cause of spread. In some cases, airborne spread has been suspected.

A great deal has been achieved since the initial diagnosis of EI. The disease remains confined to an area around Sydney and a corridor up through the north-eastern part of New South Wales into the south-eastern part of Queensland. A vaccination strategy has been implemented, and action has been taken to allow the future easing of some quarantine measures. Zones have been established in the infected States to help manage the outbreak and the movement of horses.

Infected properties as at 30 September 2007

State	Infected premises[a]	Dangerous contact premises	Suspect premises
NSW	3055	330	369
QLD	594	Not available	Not available
Other jurisdictions	0	0	0

a Premises with horses with fever and respiratory symptoms and positive results to the real-time influenza A polymerase chain reaction; for Queensland infected premises, includes those determined by clinical signs and proximity to infection.

Buffer zones and ring vaccination

In late September, Australia implemented a vaccination strategy to help control and eradicate EI. Buffer and ring zones of vaccinated horses are being used to contain the disease to identified zones in New South Wales and Queensland. The buffer zones were created using natural geographic features (such as escarpments and mountain ranges) and national parks, and included areas of low horse density. Horses vaccinated in these buffer and ring zones will establish a band of immunity around areas where there is a high number of infected horses.

The vaccine selected for use was a live recombinant vaccine produced by Merial Laboratories in France. The vaccine was chosen for several reasons: because the literature and the manufacturer report a rapid onset of immunity with reduced viral shedding; the vaccine strain was considered to be effective against the Australian isolate; the vaccine has demonstrated effectiveness in overseas outbreaks of EI; and there was potential for differentiation of vaccinated horses from infected horses. Emergency permits were obtained from the Australian Quarantine and

Inspection Service, the Australian Pesticides and Veterinary Medicines Authority and the Office of the Gene Technology Regulator to allow importation of the vaccine for use under specific, limited conditions only.

Sufficient vaccine will be imported to ensure that target horses can complete a three-dose primary vaccination schedule.

Movement controls in unaffected States and Territories

Following the national standstill on the movement of horses, the States and Territories that remained free from EI implemented arrangements to allow some horse movements that would not compromise control and eradication. Victoria continues to maintain strict control over the movement of horses and equipment across its border with New South Wales.

Social and economic impact

The Australian Government initially provided $4 million to assist those in the horse industry directly affected by the national standstill on the movement of horses. A further $110 million was provided for a range of assistance measures to ease the widespread social and economic hardships encountered by people in the horse industries.

State and Territory governments have also provided support to people affected by the EI outbreak.

International notification

Notification of the presence of EI in Australia was made to the OIE, ProMED, the South Pacific Commission, the Animal Health Trust and the fellow signatory countries to the International Animal Health Emergency Reserve (Canada, United Kingdom, Ireland, United States and New Zealand). (Note: The Program for Monitoring Emerging Diseases — ProMED is an email alert service that is administered by the International Society for Infectious Diseases.)

Hong Kong, New Zealand, the Philippines, Singapore, the United Arab Emirates, Malaysia, Macau, France and Qatar have notified Australia of the suspension of imports of live horses. Australia is unable to meet requirements for export of equine products to Papua New Guinea, South Africa, Canada, China, Namibia and Singapore.

Summary

Containment and eradication of EI are the focus for the nationally coordinated response to the outbreak in Australia. Zoning, movement restrictions, biosecurity and vaccinations in buffer zones are components of the overall strategy being implemented. Vaccine has been distributed to New South Wales and Queensland, and vaccination in the buffer zones has begun.

As a further measure, vaccination of high-value horses in New South Wales, Queensland and Victoria has started to limit production losses, to protect important community assets, and to help manage consequential loss and animal welfare.

This report covers the Australian EI outbreak up to 30 September 2007; however, the situation has the capacity to change rapidly.

Contributed by: Scott Porteous, Animal Health Australia, and Lyndel Post, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry

Equine influenza postscript — 13 November 2007

EI cases remain confined to New South Wales and Queensland, with the size of the affected areas changing very little. The total number of infected properties is now approximately 7500; however, the rate of new infections has declined significantly. About 60 000 horses have received first-round vaccinations in the two affected States, with second-round vaccinations now progressing. The focus has been on vaccination in buffer zones around infected areas. A further 17 000 horses have received first-round vaccinations to assist in limiting production losses and to protect high-value horses. Eradication of the disease is expected to require a sustained effort for several more months. Movement controls and strict biosecurity requirements remain in place.

The NMG for EI has recently agreed to increased funding for the ongoing eradication and control of EI. The CCEAD continues to meet regularly to coordinate the response and ensure the effectiveness of the response.

[1] http://www.animalhealthaustralia.com.au/programs/eadp/eadra.cfm

Australian Biosecurity Cooperative Research Centre — update

Avian influenza test helps fight equine influenza outbreak

A real-time polymerase chain reaction test developed for avian influenza by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) and the Australian Biosecurity Cooperative Research Centre (AB-CRC) is successfully being used to test horses for equine influenza.

Equine influenza is caused by a strain of influenza virus that is related to, but differs from, the strain causing avian influenza. The nucleic-acid-based test can detect both strains of influenza virus and is complete in a few hours.

The avian influenza test now being used as a first-line diagnostic test for equine influenza was developed within the microbiologically secure environment of CSIRO Livestock Industries' Australian Animal Health Laboratory (CSIRO AAHL) in Geelong, Victoria, and was transferred to all major government veterinary laboratories in Australia and New Zealand. The AB-CRC, CSIRO, the poultry industry and the States supported trials to validate the tests, and ensure accurate and rapid detection of the influenza virus.

CSIRO AAHL Director, Dr Martyn Jeggo, said the development of the test and the transfer and validation are critical to Australia's national diagnostic capacity.

'The close relationship between CSIRO AAHL and the State veterinary diagnostic laboratories has been critical to the successful validation of the test and its use in this outbreak of equine influenza,' Dr Jeggo said.

This provides a high level of confidence in Australia's capacity to detect equine influenza.

The Chief Executive Officer of the AB-CRC, Dr Stephen Prowse, said quick diagnosis of equine influenza is a key to controlling the disease: 'a rapid diagnostic test for equine influenza ensures that appropriate control and containment measures are implemented quickly'.

Pig industry surveillance improving Australia's biosecurity

An important AB-CRC project is investigating ways of communicating with peri-urban pig farmers to improve their understanding of animal disease and the ability to identify and monitor their pigs. As the world becomes more and more urbanised, the incidence of peri-urban farming will increase. For example, more than 300 peri-urban pig producers with small-scale noncommercial pig herds exist in the Sydney basin alone.

'It is difficult to calculate accurately the number of small "noncommercial" farmers in Australia. There are methods to detect commercial pig farms, such as animal branding registries. However, the small farmer who sells pigs to domestic abattoirs or privately to friends does not even make a blip on the radar,' said Dr Trish Holyoake, the project's principal investigator and senior lecturer in intensive animal health at the University of Sydney.

'Because they are harder to find, it is difficult to provide extension materials to them to assist with prevention and recognition of exotic disease,' she added.

The AB-CRC study focuses on noncommercial farms that sell pigs through live auctions, where commercial pigs, cattle and sheep are often presented for sale, thereby posing a potential risk for disease transmission.

The study has shown a need to provide additional, targeted extension materials to peri-urban farmers to help them recognise signs of exotic disease, improve on-farm quarantine procedures and correctly adhere to swill feeding regulations.

'This project seeks to improve the way that we communicate with noncommercial producers — that way we can get information to them and they are more likely to contact the authorities in the case of a suspected exotic disease,' said Dr Jenny-Ann Toribio, a project leader and senior lecturer in epidemiology, also based at the University of Sydney.

'We also aim to improve the way we track pigs that move off farm. We've designed identification methods for lightweight pigs that currently fall under the radar for individual identification, as they are too small to be tattooed. This year we are measuring the uptake, integration and ultimately the effectiveness of the paper-based tracking system based on the use of the emergency PigPass[2] vendor declaration for noncommercial pigs,' Toribio added.

For more information, contact:
Dr Trish Holyoake, University of Sydney, Camden
Phone: 02 9351 1617
Email: trishh@camden.usyd.edu.au

[2] A scheme for recording movements in the pig industry and providing information to help trace animals in the event of a disease incursion in the Australian pig industry.

Remote area surveillance workshop

In early June, the AB-CRC, Meat & Livestock Australia and Animal Health Australia held a workshop attended by industry, research and government representatives. Participants considered remote area surveillance issues and, in particular, the issues surrounding the adoption of the bovine syndromic surveillance system (BOSSS) developed by the AB-CRC. The primary recommendation of the workshop was for the AB-CRC to work with relevant State and Territory departments and remote area producers to implement BOSSS as a tool for collecting and managing remote surveillance information.

Contributed by: Kimberley Sakzewski, Communication Officer, Australian Biosecurity Cooperative Research Centre

Audit and evaluation of Australia's animal health systems

For many years, Australia's major trading partners have periodically audited and evaluated Australia's animal and public health control and inspection systems. For example, inspection teams from the United States and the European Commission have been regular visitors for more than 20 years. These audits have been common for more regulated commodities, such as meat and dairy products.

These regular evaluations of national sanitary and phytosanitary measures for agri-food exports are an important element of Australia's trading partners' risk-analysis process. Countries may legitimately use risk analysis in their policy formulations that directly apply to animal health and sanitary controls of international trade in animals, animal-derived products, animal genetic material and animal feedstuffs.

The purpose of the audits and evaluations is to provide the importing country with confidence in the Australian Government Department of Agriculture, Fisheries and Forestry (DAFF) as the competent authority with responsibility for ensuring or supervising the implementation of animal health and welfare measures, international veterinary certification and other standards and guidelines.

In particular, the Australian Quarantine and Inspection Service (AQIS), which is a division of DAFF, is the veterinary authority that is repsonsible for providing export veterinary certification to support the importing country's requirements. The evaluation by the importing country will usually aim to demonstrate that AQIS has the capability for effective control of the sanitary and zoosanitary status of animals and animal products. Key elements in this process include resource adequacy, management capability, legislative and administrative infrastructures, and independence in the exercise of official functions and performance history, including disease reporting. AQIS has successfully negotiated for less frequent evaluations with some trading partners by applying system audit approaches, whereby importing countries audit AQIS systems as distinct from each element (or processing establishment) covered by that system.

There is a trend towards a greater number of different trading partners sending inspection teams, and for these inspections to cover a wider range of products, with an increased scope of issues they are inspecting, to verify compliance with their own requirements. Subjects such as animal welfare, residues from

veterinary and agriculture chemicals, animal identification and tracing, animal disease status, and laboratory capacity and product integrity, are just a few major areas that are coming under increasing scrutiny. The OIE (World Organisation for Animal Health) has recently developed guidelines for evaluating member countries' veterinary control systems. The guidelines are called the *Performance, Vision and Strategy Instrument*, and the OIE is encouraging and training member countries to use them.

The introduction of hazard analysis critical control point (HACCP) and quality assurance systems throughout the food chain, from 'farm to fork', has seen a greater emphasis on systems verification. Overseas auditors are just part of the 'check-the-checker system' and add another independent aspect that can only add confidence to Australia's food production system. The major benefit of external auditors is that it ensures Australian standards are on par with the best in the world. Over time, this has helped to improve transparency in the system and ultimately consumer confidence.

In the past year, trading partners have visited to:

- check Australia's bovine tuberculosis status (United States)
- check Australia's tuberculosis and laboratory capacity, and live animal export system (Taiwanese veterinary authorities)
- check Australia's live animal export system (Russian veterinary authorities)
- review Australian production of red meat (including game), dairy, bovine genetic material, fish and shellfish (European Union); this audit looked at animal health systems, traceability (including the National Livestock Identification System) and export certification
- review red meat establishments (United States, Malaysia and China).

In the coming months, Australia is scheduled to have the following visits:

- a check of Australia's live animal export system by the Israeli veterinary authority
- an audit of Australian meat establishments producing pork, ratite and red meat by Taiwan.

A number of other countries, including Korea, Russia, Indonesia, Thailand and the Philippines, have indicated that they are planning to visit to review the Australian meat production system.

More detail on these audits will be provided in subsequent editions of *Animal Health Surveillance Quarterly*.

Contributed by: Andrew Cupit and Laura Timmins, Biosecurity Australia and Australian Quarantine and Inspection Service, 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 causes of death in wildlife in Australia. The information in this report is based on information submitted by network subscribers and network State and Territory coordinators. All contributions are recorded in the AWHN database, the Wildlife Health Information system (WHIS^3). For copies of the network newsletter or digests, please contact Leesa Haynes at lhaynes@zoo.nsw.gov.au. Details about selected incidents and key investigations are provided below.

Avian influenza surveillance in wild birds

Australia maintains a surveillance program for avian influenza (AI) in Australia's wild birds. Investigation of wild bird mortality events is a crucial component of the surveillance plan, and AI virus was excluded as the cause of two of the three reported wild bird mortality events in Australia, between July and September 2007. All the mortality events were atypical of AI and are discussed in detail in 'Wild bird mortalities' below.

The new surveillance season for 2007–08 has begun. During the period July to September 2007, swabs (cloacal, faecal and oropharyngeal) and blood samples were collected from approximately

1284 wild birds in Australia. Sampling occurred at sites in New South Wales, Queensland, South Australia and Western Australia. The majority of samples were collected from waterbirds (e.g. ducks and waders).

Bat mortalities

The previous AWHN report (April–June 2007) discussed flying fox mortalities (more than 500 animals) in June to July 2007 at multiple locations in coastal Queensland. The majority of animals were juvenile (about 100–200 g) and recently weaned black flying foxes (*Pteropus alecto*), with a very small number of spectacled flying foxes (*P. conspicillatus*) also involved. Twelve black flying fox carcases (from Brisbane and Mackay) were examined at the Animal Research Institute, Queensland. The Brisbane animals were mostly adults in moderate to excellent body condition. No consistent underlying condition predisposing the bats to predation was evident. Nevertheless, widespread anecdotal reports of unusual feeding and roosting behaviour during this time support the theory that seasonal food shortages and/or unusually cold weather were implicated in the mortalities. Australian bat lyssavirus was excluded in all cases, and Hendra virus was excluded in the cases from Mackay. The diagnosis remains open.

Eight grey-headed flying foxes (*P. poliocephalus*) were found dead or dying near Sydney. Testing for Hendra and lyssavirus was negative. On necropsy, congestion and haemorrhage in the liver, lungs and eyes was observed. The diagnosis remains open.

Mortalities in mammals

In July and September 2007, six seals in poor condition were reported along the New South Wales coast. Four of the animals died shortly after being taken into care. Emaciation with heavy parasitism was identified in three animals, and the third had a bacterial urethral infection. The remaining two seals were euthanised due to trauma.

A common wombat (*Vombatus ursinus*) from western Sydney was diagnosed with skin and tissue mineralisation associated with chronic kidney disease and nonsuppurative inflammation in the brain and heart.

Wild bird mortalities

Three wild bird mortality events have been reported this quarter. Dead honeyeaters (specific species not recorded) in house gardens on Bathurst Island (Northern Territory) were reported by a tour operator. Avian influenza (AI) was excluded using both an AI antigen detection enzyme-linked immunosorbent assay kit and real-time polymerase chain reaction (PCR). Acute necrotising tracheitis of unknown aetiology was diagnosed on histopathology, and virus isolation testing is pending.

Fifteen crows (specific species not recorded) died at Moura (700 km north-east of Brisbane, Queensland). No significant histology was seen on histopathology, AI and Newcastle disease were excluded by PCR, and fenthion (organophosphate) poisoning was diagnosed.

Around 15 dead rainbow lorikeets (*Trichoglossus haematodus*) were reported in a Sydney suburb. Adequate samples were obtained from one bird, and the cause of death was a syndrome of severe necrotising and haemorrhagic enteritis. This syndrome is seen in lorikeets, usually in the warmer months, and is often associated with an overgrowth of *Escherichia coli* and *Clostridium* sp.

Contributed by: Chris Bunn, Australian Government Department of Agriculture Fisheries and Forestry, and Leesa Haynes, Projects Coordinator, Australian Wildlife Health Network

[3] http://www.wildlifehealth.org.au/

Aquatic animal health

Fish kill protocol

The *National Investigation and Reporting Protocol for Fish Kills* has been developed to provide a consistent national approach in the response to major fish kill incidents in marine, estuarine and freshwater environments in Australia.

Fish kill incidents (unusual mortality events in nonmammalian aquatic animals) are known to occur in natural waterways across Australia. Their occurrence may indicate significant environmental changes, disease incidents or major pollution events.

Often, the causes of fish kill incidents remain unknown, preventing appropriate management decisions to reduce or eliminate the possibility of future fish kills. The *National Investigation and Reporting Protocol* for fish kills sets minimum standards for managing fish kill incidents. Thorough investigation of fish kills can provide early warning of exotic or emerging diseases that could threaten Australia's aquatic ecosystems, fisheries resources or aquaculture industries.

The protocol was developed as part of *AQUAPLAN 2005–2010, Australia's National Strategic Plan for Aquatic Animal Health*. It was a joint government–industry initiative that also engaged academic institutions, and conservation and wildlife agencies. The suggested response to a fish kill incident follows a logical process, as shown in Figure 1, which illustrates the four main stages within a continuous investigation process. The tasks detailed are advisory in nature and are provided as guiding principles. During any incident, the decision to vary or add tasks will be at the discretion of the affected jurisdiction(s).

The protocol has been well received by Australian aquatic animal health specialists and industry professionals, and is now available on the website of the Department of Agriculture, Fisheries and Forestry.[4]

Contributed by: Aquatic Animal Health Unit, Office of the Chief Veterinary Officer, Australian Government Department of Agriculture, Fisheries and Forestry

[4] http://www.daff.gov.au/animal-plant-health/aquatic/resources

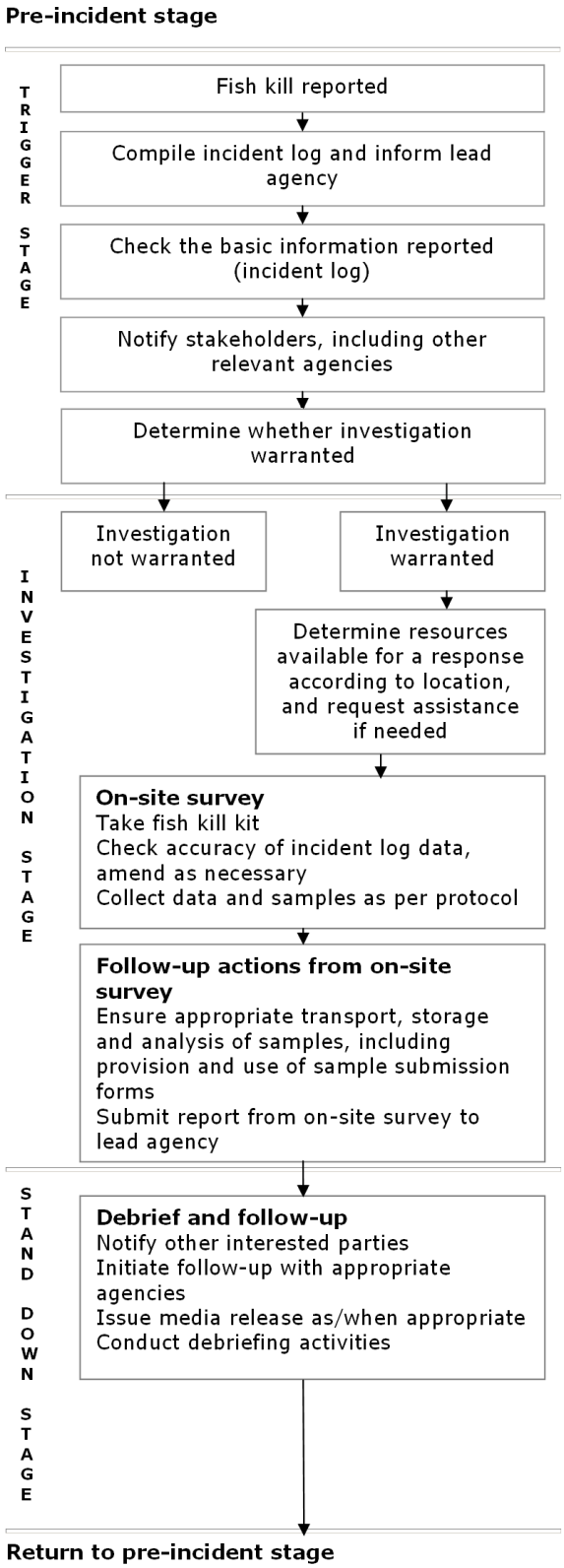

Figure 1 Checklist for responding to a fish kill incident

State and Territory reports

In Australia, the States and Territories are responsible for animal disease control within their borders. National animal health programs are developed through consultation at Animal Health Committee and are managed by Animal Health Australia.

New South Wales

Contributed by: Rory Arthur, Department of Primary Industries

Equine influenza

Summary

Equine influenza (EI) was confirmed in central Sydney, New South Wales (NSW), on 25 August 2007, shortly after cases were detected at Eastern Creek Quarantine Station (ECQS), Sydney. By 29 September, the disease had spread to approximately 2800 premises in NSW, infecting some 25 000 horses.

The original source of infection is suspected to be recently imported horses; however, the route of release is being investigated and is unconfirmed. Possible routes include indirect transmission by personnel handling imported horses and via contaminated equipment. Subsequent spread within NSW is believed to have occurred primarily from the movement of subclinically infected horses associated with equestrian events, before horse movement restrictions were implemented. Direct transmission by horse-to-horse contact and airborne virus, and indirect transmission of infection by movement of people, have all been considered important in the spread of the virus.

NSW continues to work towards eradication of the EI incursion using quarantine measures, movement controls and strategic vaccination.

Epidemiology

Close physical contact between horses at equestrian events is considered to be the main cause of the initial spread of the disease. In contrast, subsequent spread has been accelerated by movement of contaminated people and equipment — for example, between neighbouring properties.

In early September, spread of infection over long distances was observed with no apparent opportunity for transmission by direct or indirect horse contact. Detailed investigations led to the conclusion that airborne spread was occurring over distances of up to 2–3 km. Observations of infection spreading over longer distances (up to 5 km) have also been made; however, these have been uncommon and are difficult to substantiate. Small clusters of infected horses (fewer than five horses) are suspected to be the source of infection in some of these events.

Airborne transmission, which involves suspension of virus in the air for long periods of time, is suspected to occur in the evenings or early mornings when the air is cool and exposure of airborne virus to ultraviolet light is limited. It is expected that airborne transmission will decrease as daytime temperatures rise in the summer months.

Indirect transmission by people is thought to have significantly contributed to the spread of infection, particularly during the early period of the outbreak at premises in central Sydney. Despite community-based biosecurity campaigns, transmission by people continued to be significant in the second month of the outbreak. Suspected circumstances include movement of people for work and social activities between neighbouring communities. Potential spread by mechanical vectors, such as flies and birds, is a concern to the NSW Department of Primary Industries (DPI); however, this is not currently considered to be a significant risk.

Response strategy

The EI disease response plan contained in AUSVETPLAN (Australian Veterinary Emergency Plan) has been a valuable tool in combating the outbreak. NSW DPI is currently implementing AUSVETPLAN quarantine measures, movement restrictions and strategic vaccination to contain further spread and eradicate the disease. Approximately 180 DPI staff manage the response from State and local disease control centres and are supported by many hundred field staff. Most control centre staff are provided on secondment from NSW DPI or other government agencies. Veterinarians in private practice have provided critical expertise to conduct disease surveillance and veterinary investigations for the large number of infected premises.

A vaccination strategy is being implemented to create buffers of immune horses. Additional vaccine use is currently being restricted to horse populations considered to be at a high risk of exposure or likely to suffer severe consequences from infection.

The NSW DPI response to the outbreak has benefited greatly from high levels of community support for movement controls and disease reporting. Thousands of horse owners have been encouraged by the DPI to monitor the health status of their horses using thermometers provided free of charge. The public and stakeholders are well informed on the progress of the response, in large part due to an EI-dedicated website (http://www.dpi.nsw.gov.au/agriculture/livestock/horse/influenza).

Northern Territory

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

Urea poisoning in cattle

Cattle deaths were reported from a station in the gulf country of the Northern Territory. A total of 19 mortalities were recorded, with the deaths occurring in close proximity to supplement troughs. The affected animals were generally better conditioned and possibly the first at the lick troughs after new supplement was added. Most animals showed nervous signs before death, although signs of abdominal discomfort were not observed. Deaths discontinued after 3 days. A suspect diagnosis of urea poisoning was made and supported by necropsy and laboratory findings.

Hard udder syndrome in a goat herd

Several goats from a herd of 28 animals, kept on a rural block in Darwin, developed hard udders soon after kidding. It was first noticed when five of the kids died over a few days. There were no clinical signs in the adult goats other than swollen udders that yielded no milk. The affected animals were bled and tested for caprine arthritis encephalitis virus. Serological results proved negative. The owner was advised to use oxytocin to treat future cases after parturition. A light growth of *Staphylococcus* sp. was cultured from subsequent milk samples. Fortunately, the bacterium is sensitive to a range of antibiotics. The goats' udder condition has improved, but some does continue to experience a chronic form of one-sided hard udders.

Vesicular disease exclusion in pigs

A report of blisters on the snout of a feral pig was made by Indigenous rangers on Elcho Island to members of the Australian Quarantine and Inspection Service (AQIS) Remote Area Team (RAT) present on the island for another purpose. Three pigs were shot, and photos of snout lesions were forwarded by the RAT team to the AQIS office in Darwin. A diagnostic team was organised and flew to the island, where a local hunter shot another two pigs with snout and oral lesions. The investigating pathologist found erosions rather than blisters and no evidence of foot lesions. Samples were collected and forwarded to the Australian Animal Health Laboratory for the exclusion of exotic vesicular diseases. The test results were negative and support the presumptive diagnosis of lesions caused by trauma from foraging on very rough plant material. The pigs also may have had contact with toxins in their swamp feeding grounds.

Queensland

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

Horses

Equine influenza

Equine influenza was diagnosed in Queensland on 25 August 2007 in three New South Wales horses attending an equestrian event in Warwick in southern Queensland. Horse movements from the equestrian event were limited, but resulted in the spread of infection to several other areas of south-east Queensland. Simultaneously, other horse movements from New South Wales resulted in three other outbreaks in south-east Queensland. Control measures put in place were property quarantines, strict movement restrictions on horses and associated horse transport vehicles, and rigorous biosecurity and decontamination measures for horse carers and their motor vehicles. Where infection occurred in dense horse population areas, lateral spread between horses was significant, despite the strict movement restrictions on horses. Where infection occurred on large properties, spread was minimal. New foci of infection seem to be related to movements of people carrying virus, although in some cases airborne spread cannot be discounted. Diagnostic testing is being conducted by polymerase chain reaction on nasal swabs at the Biosecurity Sciences Laboratory in Brisbane. Affected horses show a range of signs from either no fever or a very mild rise in body temperature, serous nasal discharge and occasional cough, through to high fever, significant serous nasal discharge becoming mucopurulant, severe hacking cough and sometimes pneumonia.

Equine herpesvirus abortion

A thoroughbred mare on a Darling Downs stud had a stillborn full-term foal in mid-September 2007. There was generalised jaundice of the body, and the lungs were deflated, firm and oedematous. There was oedema of the thymus, but no other gross pathology was seen. Sections of the lungs showed a severe necrotising bronchiolitis with extension up the surrounding alveoli. The thymus was oedematous with extensive necrosis. Eosinophilic intranuclear inclusion bodies were seen in large numbers in the bronchiolar epithelial cells. Equine herpesvirus 1 was isolated from lung tissue on tissue culture. Another case occurred on a Cambooya shire thoroughbred stud, which had a foal born dead in July. Equine herpesvirus 1 was isolated from the liver, lung and spleen. In lung tissue, there was a necrotising bronchiolitis and bronchitis, with eosinophilic intranuclear inclusions in bronchiolar epithelial cells. There were also occasional inclusions in hepatocytes in liver sections.

Cattle

Blackleg

In August 2007, a Rosalie shire beef property had five deaths out of a group of 70 cattle aged 8 months. Cattle were not vaccinated for clostridial diseases and showed signs of swelling in leg muscles, ataxia and then recumbency, followed by death. There was serosanguineous oedema extending down the limbs involved, and dark discolouration and dry gaseous crepitation of the affected muscles. Also seen on necropsy were serosanguinous pleural and pericardial effusion and mild fibrinous exudate on the surface of the heart. *Clostridium chauvoei* was isolated from skeletal muscle and subcutaneous tissue.

Botulism

Botulism was diagnosed in a recumbent 3-year-old cow out of 30 at-risk cattle on a property in Beaudesert shire in mid-July 2007. The cow was pyrexic and lethargic. Serum was positive for *Clostridium botulinum* toxin on enzyme-linked immunosorbent assay (ELISA) testing. The cow was on a poultry farm and had gained access to chicken litter contaminated with botulinum toxin.

Botulism was also presumed to have caused the deaths of six dairy cows out of 180 at risk on a property in Caloundra shire in early August. Cattle had access to chicken litter used to fertilise pastures, and the litter was found to be contaminated with botulinum toxin. The cattle were not vaccinated for botulism.

Hepatoencephalopathy

A Warwick shire property had six deaths out of 35 beef weaners aged 3–6 months, as well as two sick animals. Signs included diarrhoea with some blood present, persistent straining to pass motions and nervous signs, such as somnolence. A weaner necropsied at the Toowoomba Animal Disease Surveillance Laboratory (ADSL) showed a shrunken, tan-coloured, firm liver and oedema of the gall bladder, as well as serosal and mesenteric oedema in the upper small intestine. Liver pathology included moderate dissecting fibrosis and bilary proliferation, with remaining hepatocytes showing mild irregular vacuolation. Brain sections showed marked status spongiosis. No significant harmful plant material was detected in the rumen contents, although the suspected cause was previous ingestion of hepatotoxic plants.

Hypomagnesaemia

In early August 2007, a Waggamba shire beef property had 70 deaths, and two sick beef cows and steers, out of 300 at risk. Animals displayed stupor, progressing to stargazing, jaw chomping and convulsions. Necropsy of a recently dead animal found no significant gross pathology. However, blood samples showed marked hypomagnesaemia, hypocalcaemia, hypoproteinaemia and elevated blood urea. Blood was sampled from a further six animals in the group: all except one had marginal to marked hypocalcaemia, and one had marked hypomagnesaemia and hypoproteinaemia.

Nitrate poisoning

Nitrate poisoning caused the death of 16 cattle in three events in July to August 2007. Two cows out of 22 at risk being fed forage sorghum hay on a property in Ipswich shire died suddenly in early July 2007. Two cattle that exhibited clinical signs of trembling and convulsions were saved by treatment. Hay silage tested contained 3.3% potassium nitrate on a dry-matter basis (levels in excess of 1.5% may be toxic to ruminants).

Five cattle out of 30 at risk on a property in Boonah shire died suddenly in mid-July. Cattle were grazing on an oats crop. Feed tested contained 8.1% potassium nitrate on a dry-matter basis.

Nine cows out of 120 at risk on a property in Beaudesert shire died in early August. Pasture oats being fed contained 2.1% potassium nitrate on a dry-matter basis. The levels of nitrate found in the aqueous humour confirmed nitrate poisoning.

Sheep

Encephalomalacia

A Wambo shire property had nine deaths out of 50 lambs aged 3 weeks that were being reared for meat production. Lambs were found dead with no previous signs of illness. The attending veterinarian suspected a bacterial toxin from a clostridial disease, such as tetanus, enterotoxaemia or botulism. As the necropsied lamb had diarrhoea (indicated by faecal staining of wool), colibacillosis was also considered as a possibility by the veterinarian, but this could not be confirmed by laboratory testing. The lamb was found in lateral recumbency with opisthotonus, nystagmus and scouring. On necropsy and submission of samples to the Toowoomba laboratory (ADSL), sections of brain showed multiple areas of severe, focal, bilaterally symmetrical malacia, involving the basal ganglia, thalamus, cerebrum and medulla, which indicates encephalomalacia.

Goats

Mannosidosis

Mannosidosis was diagnosed as the cause of the deaths of two Anglo-Nubian newly born kids on an Ipswich shire property in early September 2007. One kid died at 48 hours of age and the other at 6 days. The kids were unable to stand and developed continual muscle tremors. They had normal suckling reflexes. Mannosidosis is a storage disease of Anglo-Nubian goats due to an inherited deficiency of the enzyme beta-mannosidase. The method of inheritance is autosomal recessive, and affected kids are only born when a carrier buck is mated to a carrier doe.

Nitrate poisoning

A Cambooya shire property had five deaths and two sick goats from a herd of 150 milking goats. Signs observed before death were abortion and miscarriage. A necropsied goat had miscarried 2 days before death. Body fat reserves were depleted, and aqueous humour testing showed a toxic level of nitrate. The source of nitrate was pasture hay being fed to the goats.

Pigs

Colibacillosis

A Warwick shire piggery had 18 deaths and 50 sick pigs out of 500 piglets aged 5 weeks. Signs seen were diarrhoea, weight loss and death. Faecal swabs from four affected piglets were cultured and revealed haemolytic and nonhaemolytic *Escherichia coli*. The organisms cultured were resistant to five different antiobiotics and only sensitive to ceftiofur (cephalosporin antibiotic).

Bronchopneumonia

A Millmerran shire property had 10 deaths and 20 sick piglets out of 100 piglets aged 4 weeks. Signs seen in the piglets were diarrhoea and ill-thrift. On necropsy, one piglet showed a fibrinopurulent pleurisy and bronchopneumonia, and another piglet showed a severe, suppurative bronchopneumonia. *Haemophilus parasuis* was isolated from the lung tissue of the latter pig.

Poultry

Fowl cholera

A Wambo shire property had 27 deaths and two sick layers aged 7–12 months out of 4500 layers. Affected poultry showed respiratory problems followed by death. On necropsy, there were suppurative peritoneal, pericardial, perihepatic and air sac effusions. On histopathology, there were scattered miliary pale foci in livers, consolidation of lung tissue and, in one layer, swelling in the infraorbital sinus. *Pasteurella multocida* was isolated from liver, peritoneum, lung and from the infraorbital sinus. Lung tissue showed a marked, diffuse, necrotising and purulent pneumonia. Livers had mild to moderate multifocal necrotising hepatitis.

A layer flock in the south-east region experienced 100 sudden deaths out of 6000 at-risk layers in mid-July 2007. Nine birds were examined at the ADSL in Toowoomba. Necropsies revealed swollen, mottled livers, enlarged spleens, fibrinous peritonitis, degenerate yolks and cloudy pericardial sacs. Histopathology revealed acute to chronic multifocal hepatitis with occasional granulomas. Other lesions included oophoritis, peritonitis and pericarditis. Fowl cholera was diagnosed with the isolation of *Pasteurella multocida* from the liver.

A free-range flock in the south-east region also lost 200 layers aged 40 weeks out of 1000 at risk in late July from fowl cholera. Birds were found dead, and there was an associated decrease in egg production over 2 months.

South Australia

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

Preventing the spread of equine influenza to South Australia

On 25 August 2007, South Australia (SA) activated a State disease control headquarters (SDCHQ) in response to the equine influenza (EI) outbreak in New South Wales (NSW). The aim of the response was to keep EI out of SA. This initially involved implementing a state-wide livestock standstill for horses (a ban on all horse movements). Representatives from the racing and equestrian industries were recruited to the SDCHQ as industry liaison officers (ILOs) to assist with the response. The various diverse horse industry groups were contacted and informed, and the police were requested to help implement the standstill. All race meetings were also cancelled for that weekend and the following week.

The initial focus of the SDCHQ was on tracing and surveillance. All horses that had arrived from interstate in the preceding 3 weeks were traced and identified, and appropriate surveillance activities were implemented. The ILOs were invaluable in enabling rapid tracing to occur, and the media were instrumental in informing the industry and its associates that the Department of Primary Industries and Resources of South Australia was conducting an intensive scanning surveillance program.

No EI was detected in SA, and horse training and racing were gradually allowed to resume, with the development and implementation of quarantine areas and biosecurity controls. On 3 September, the standstill was lifted and replaced with strict regulation of horse gatherings, and stringent control

measures for the importation of horses, horse products and horse-associated transport from other States. Currently, horses from noninfected States may enter or travel through SA under permit, while horses from infected jurisdictions are prohibited from entering SA. Similar regulations are in place for horse products and horse-related transport. The restriction on horse aggregations has been used as a strategy to both reduce the risk of EI spreading and allow traceability in an efficient and timely manner should EI be introduced into SA.

Surveillance for EI is ongoing. Veterinarians are examining all horses that are reported to have a temperature elevation or respiratory symptoms, and samples are being submitted from these horses for EI exclusion.

The horse industries in SA have been instrumental in keeping EI out of the State, and their ongoing support and cooperation are key to keeping SA free from EI in the future.

Face lesions in pastoral sheep

In September 2007, a sheep producer from the northern SA pastoral region reported three sheep, from a group of 247 mature ewes, with unusual lesions on the nose and mouth. The sheep on the property graze on extensive pasture, and lesions were detected when the sheep were gathered for crutching. The affected sheep were unable to eat, but were otherwise healthy. Other sheep in the group were healthy, with no similar lesions.

Examination of the animals revealed that large areas of skin and flesh on and around the nose and mouth had been eaten away. Foci of necrosis at the end of the nose were considered too localised to be caused by photosensitisation. Neoplasia, chemical burn or toxicity was suspected. The affected animals that were unable to eat or drink were euthanised. Samples from one of the animals were submitted for histopathology, which revealed a moderate multifocal ulcerative dermatitis/rhinitis, suspected to be caused by a chemical burn.

On further investigation with the producer, it was revealed that some cement work was being done in the paddock where the sheep were kept. Sheep tracks were observed in the vicinity of the wet cement, which is a likely cause of the lesions. The area was fenced off so that sheep could not gain access to the cement, and no further cases were observed.

Foot abscesses in multiple sheep flocks

During August 2007, more than a dozen sheep flocks on the Yorke Peninsula were reported with lameness and sudden death. Some of the flocks were wethers, and others were late pregnant ewes. On examination, the animals were found to be severely lame, with interdigital swelling and pain. Many ewes were recumbent and exhibiting respiratory distress and pyrexia. Foot swabs submitted for culture revealed a growth of mixed anaerobic bacteria. A diagnosis was made of foot abscessation, with resultant severe lameness and recumbency, leading to pregnancy toxaemia. Sheep were treated with appropriate antibiotic therapy and glucocorticoids, and were moved into yards to recover.

The Yorke Peninsula had a good, wet spring this year, resulting in moist soils and lush pastures. The ground is also very rocky and this, coupled with soft feet and heavy animals (many pregnant with twins or wethers in good condition), caused foot lesions and resultant abscesses. Lameness also caused some ewes to become recumbent and to develop pregnancy toxaemia.

Selenium deficiency in alpacas

During July 2007, an alpaca property in the Adelaide Hills reported a number of alpacas failing to grow and thrive. This had been occurring over a number of years.

When the property was visited, there were three alpacas showing clinical symptoms including chronic weight loss despite a healthy appetite, stunted growth and an affected gait. Faecal and blood samples were submitted for parasitology and trace element testing. Significant selenium deficiency was found in all three animals, as well as significant worm burdens despite reported drenching. Liver enzymes were elevated, and all three were anaemic. The owner was advised to supplement the herd with selenium annually, as well as to manage internal parasites more carefully and selectively, with worm resistance testing being performed if the problem persists.

Subclinical selenium deficiency is known to cause lowered fertility and production. Under ideal conditions, alpacas generally have few worm problems, but on most small holdings worms can be a health risk.

Vitamin D toxicity in a layer flock

At the beginning of July 2007, an egg layer flock suffered a drop in production from 70% to 50%. Within 2 weeks, birds began to appear ill, with ruffled feathers, depression and wet droppings. Birds with these signs usually died within 24 hours. Mortalities were highest in the oldest birds of 80 weeks of age. Upon further investigation and enquiry, the owner recalled noticing poor eggshell quality 2 weeks before the illness. He had then treated the birds with vitamin D in the water, incorrectly dosing at over 10 times the recommended level. When mortalities began to increase, the correct dose was identified and the treatment adjusted. Necropsy revealed birds with visceral gout and uroliths, leading to renal damage. These findings strongly suggested vitamin D toxicity, and demonstrate the importance of dosing all medications correctly.

Tasmania

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

Suspect primary photosensitisation in beef cattle

Five cases of photosensitivity in a group of 150 1-year-old Hereford heifers occurred during July and August 2007 in the central north of Tasmania. The animals had been rotated between paddocks of almost pure, short-rotation rye grass and then fescue mixed with 15% clover and 10% flat weed (mallow and cape weed). Water was supplied via clean troughs reticulated from the Meander River, which had had a moderate flow over the preceding 4 months. No nutritional or parasitic treatments had been administered to the herd during the previous months.

The animals showed typical signs of photosensitivity including photophobia, agitation, blackening of the face (notably the nose), and thickening of the skin of the ears and, to a lesser extent, other white areas of the body. The condition resolved over the next 4–6 weeks, with affected areas sloughing and healing.

Blood samples were collected from the five affected animals and from five 'normal' cohorts for clinical pathology. Pasture samples were collected on a grid pattern for *Pithomyces chartarum* spore counts.

Liver enzyme levels were marginally outside the normal range in the affected group, but were lower than would be expected in acute bovine liver disease. Thus primary photosensitisation was the most likely cause rather than photosensitivity secondary to hepatopathy. No fungal spores were recovered from the pasture samples.

S-methylcysteine sulfoxide toxicity in cattle

Two instances of *S*-methylcysteine sulfoxide toxicity were reported, one involving two animals, the other a herd of 120, of which four died. Haemolytic anaemia with packed cell volumes of less than 20% was observed. Both cases occured in the Midland area and were associated with grazing rape. Respiratory signs and bloat were also observed.

Rodenticide toxicity in horses

Two of a group of 10 horses ate most of a 1-kg bucket of a common rodenticide containing brodifacoum. One pony died before examination. The surviving animal, a horse of approximately 450 kg, was clinically normal on initial examination apart from continuous bleeding from minor abrasions, but had a prothrombin time of 180 seconds (normal range 12–14 seconds). The horse was treated with oral vitamin K for 6 weeks. Forty-eight hours after the last dose, its prothrombin time was 14 seconds.

Laboratory accessions[a]

Source	Number of accesssions
Aquaculture	89
Companion	111
Livestock	675
Other	7
Wildlife	60

a The number of accessions finalised per animal group during the past quarter

Notifiable diseases

The following table lists finalised investigations in the past quarter in which Tasmanian notifiable diseases were excluded or identified by the diagnostic procedure. Only those diseases not reported elsewhere in this issue are included in the table.

	Investigations	
Disease	Positive	Total
Abalone ganglioneuritis	0	10
American foulbrood	4	5
Avian chlamydophilosis	0	4
Bacterial kidney disease (*Renibacterium salmoninarum*)	0	8
Brucella abortus	0	8
Brucella ovis	0	12
Chalkbrood	0	5
Contagious agalactia	0	4
Devil facial tumour disease	0	1
Enzootic bovine leucosis	0	4
European foulbrood	0	5
Hydatid disease	2	4
Johne's disease (JD culture or histology)	1	4
Leptospira hardjo	2	11
Leptospira pomona	2	11
Listeria monocytogenes	3	13
Marine aeromonad disease (*Aeromonas salmonicida*, marine atypical strain)	1	1
Ovine anthelmintic resistance (macrocyclic lactone resistance)	0	1
Perkinosis of shellfish	0	1
Piscirickettsiosis	0	2
Q fever (*Coxiella burnetii*)	0	1
Rickettsia-like organism of salmonids	3	9
Clinical salmonellosis	50	201
Salmonella Abortusovis	0	2
Salmonella Abortusequi	0	6
Salmonella Enteritidis	0	5
Pullorum disease (*Salmonella* Pullorum)	0	2

Victoria

Contributed by: Cameron Bell, Department of Primary Industries

Oxalate poisoning in Angus cattle

Oxalate poisoning caused the deaths of three Angus cows on a property in south-west Victoria in June 2007. Two hours after 35 cows were moved onto a paddock containing marshmallow weed (*Malva parviflora*), five cows were found recumbent and four had a staggering gait. The recumbent cows had rumen atony, heart rates from 40 to 60 beats per minute and a normal respiratory rate. Despite treatment with methylene blue for suspected nitrate toxicity, and intravenous supplementation with a combined calcium, magnesium, phosphorus and glucose product, three of the cows died. The remaining cattle were removed from the paddock and recovered. Biochemistry showed hypocalcaemia and marked azotaemia, and histopathology revealed crystalline tubular nephropathy, leading to a diagnosis of oxalate nephrosis with renal failure. A return visit to the property identified an expanse of a toxic plant, soursob (*Oxalis pes-caprae*), in a paddock that some of the cattle had broken into 2 days before the deaths.

Perinatal calf losses from in-utero pestivirus infection

In July 2007, six calves from eleven 2-year-old Angus-cross heifers died or were euthanised over a 2-week period, due to pestivirus within 2 weeks of birth, on a property in south-west Victoria. To varying degrees, the affected calves displayed neurological signs consistent with cerebellar hypoplasia. Clinical signs included a poor or absent suckle reflex, inability to stand due to ataxia, a shivering, 'proppy' gait, and intermittent seizures. The longest surviving calf was euthanised at 2 weeks of age, and a necropsy was performed. Grossly, the cerebellum was of a normal size. However, the calf was positive to pestivirus using antigen capture enzyme-linked immunosorbent assay (ACE), with a

histopathological diagnosis of nonsuppurative encephalitis due to in-utero pestivirus infection. The heifer dams tested positive by pestivirus agar gel immunodiffusion, with most samples indicative of a recent pestivirus infection. Four clinically normal calves born earlier in the season were pestivirus ACE (antigen) negative. This disease presentation is typical of a viral incursion into a naive heifer herd during the latter part of the second trimester of pregnancy.

Listeriosis in Hereford calves

Listeriosis was found to be the cause of death of three Hereford calves on a property in southern Victoria at the end of June 2007. Four affected calves had neurological signs, including body tremors, an awkward gait and depression in the 2 days before the deaths occurred. As the illness progressed, blindness became apparent, and sensitivity to touch and temperature increased. These four calves were treated with a single vitamin B1 injection and short-acting antibiotics over 5 days, with one calf surviving. Gross necropsy findings on two of the calves were insignificant, but histopathology revealed acute encephalitis consistent with listeriosis. Although listeriosis is commonly associated with silage feeding, the causative organism, *Listeria monocytogenes*, is ubiquitous in the farm environment, and exposure may have occurred following grazing of rank pasture.

Abortion and death in Angus cattle following ingestion of asphalt

The ingestion of asphalt was implicated in the acute death of one cow and late abortions in three other cows, two of these dying after a chronic illness, in a herd of 60 Angus cows on a property in north-east Victoria in late August 2007. The producer reported that the cows had accessed asphalt before their illness and had been seen passing black, tarry manure. Clinically, the illness was characterised by inappetance and lethargy. Necropsy of one cow revealed ecchymotic haemorrhages on the serosal surface of the rumen and chest, and profuse haemorrhage around the heart and kidney. Serology was negative for *Neospora caninum*, pestivirus and *Brucella abortus*. Kidney histology showed multifocal interstitial nephritis. A presumptive diagnosis of toxicosis due to the ingestion of asphalt was made on the basis of the history given. Access to the the ashphalt was immediately prevented when the clinical cases were detected. It was determined that the cattle were not destined for immediate slaughter, and a management plan was implemented to mitigate the risk of residues. This case highlights the potential hazards posed by materials inadvertently left lying around a property.

Rickets in first-cross lambs

Fifty out of 500 9-month-old Ryland–Merino-cross lambs had a shifting lameness due to rickets, on a property in south-west Victoria in August 2007. The lambs had been grazing a green oat crop supplemented with barley and hay for 6 weeks, following a dry summer on oaten hay and grain. On attempting to move the mob, the producer noticed that many were reluctant to move and would sit down if driven. Twenty lambs had to be moved on a trailer. Biochemistry on three affected lambs revealed hypocalcaemia and hypophosphataemia. Necropsy revealed hyperflexible ribs, enlarged costo-chondral junctions, a soft cranium and severe chronic abomasal lesions. Faecal egg counts were 90 to 1140 eggs per gram, despite drenching of the lambs 3 days before sample collection and the lambs being regularly drenched with levamisole. The severity of the signs seen in this mob resulted from the predisposing factors of green oats being low in calcium and possibly containing a rachitogenic factor, and low levels of natural vitamin D available due to overcast weather and heavy fleece, exacerbated by a high intestinal parasite burden and decreased calcium uptake resulting from gut damage. Feeding of sun-cured hay with the addition of 2% lime to the grain and a revised parasite control program have been implemented.

Lameness in cross-bred lambs

Twenty 12-week-old cross-bred lambs from a mob of 320 sheep were noticed to be lame in central north-west Victoria in late August 2007. Stiffness in the hindquarters and occasional lameness in the front limbs were noticed a week after yarding (no management procedures had taken place during the yarding). Within a week, this developed into a hunched posture with concurrent weight loss. Joint culture and biochemistry proved unrewarding, and histology diagnosed a neutrophilic synovitis, mild pyelonephritis/hepatitis and myositis. Microabscessation of the ruminal mucosa and abomasal nematodes were also seen. *Mycoplasma* spp., *Chlamydia* spp. and *Erysipelothrix* spp. were all

suspected as possible causes of the lameness; however, none could be confirmed. Treatment with an oxytetracycline antibiotic had some effect in improving affected lambs, and no further cases have occurred.

Copper deficiency in weaned lambs

Copper deficiency was suspected to have caused 15 deaths, mob ill-thrift, and hyperkeratosis on the ears of 20% of weaned lambs on a property in north-west Victoria in July 2007. A single mob of 220 ewes was lambed down in late winter, producing 270 lambs at a lambing percentage of more than 120%. In 15 of the lambs, curled ears were noted at marking in mid-July. All these lambs died over the next 3 weeks, and an additional 30 to 40 lambs developed crusting and curling of the ears, while the entire mob failed to put on weight or condition as expected. A similar syndrome had been observed in the same paddock in 2006. No other lambs on the farm were seen with similar signs. All lambs were marked, mulesed, drenched and vaccinated, and had additional hand feeding that was in excess of requirements. The paddock had a history of no topdressing and was a light sandy loam. There were significant areas in the paddock where the grass was yellowing.

Two lambs were presented to Primary Industries Research Victoria, Attwood, for necropsy. Both lambs had patchy alopecia and crusting on the ears, a distinct indented line about half-way across the hoof and slightly swollen carpal joints with excess synovial fluid. Faecal egg count was zero. Histopathology showed hyperplasia of all levels of the epidermis in the ear, including orthokeratotic hyperkeratosis. No other significant changes were found on histopathology. Blood and fresh liver analysis revealed normal zinc levels but very low copper levels (0.06–0.08 mmol/kg wet weight [normal range 0.23–3.67 mmol/kg]). The producer was advised to have soil analysis performed and to topdress the paddock as indicated by the results.

Pregnancy toxaemia in sheep

Forty-four first-cross ewes died from pregnancy toxaemia on a property in southern Victoria in late July 2007. This flock had a history of high multiple birth rates (205% scanned pregnancy rate) and were in body condition score 3. Necropsies on two ewes showed that both were carrying twins and were in good body condition. Their livers were friable and yellow, and laboratory investigation revealed hepatic lipidosis, renal failure and hypocalcaemia. Recent supplementation with a grain ration came too late to prevent the death of these ewes. A combination of cold weather, multiple births, advanced pregnancy and minimal feed all contributed to this event.

Vesicular lesions on pigs

In July 2007, seven 20-week-old grower pigs on a hobby farm east of Melbourne developed lesions on the dorsum of their snouts, described by the owner as fluid-filled vesicles. The lesions were observed by the owner, and the Department of Primary Industries (DPI) was notified. The property was immediately visited by DPI Animal Health Field Services staff, and physical examination showed that all the piglets, but not their mother, had flaps of skin on the creased area on the dorsum of the snout. There were no other abnormal findings, and no lesions were found on the feet or within the oral cavity. Blood and skin flaps from the lesions were collected and sent to the Australian Animal Health Laboratory, Geelong, to rule out exotic vesicular diseases.

The growers were housed in an outdoor, concrete-floored pen with shelter, and the sow was kept in an adjoining paddock and had nose contact with the growers. All the pigs were isolated from cattle and sheep on the property. The pigs were fed vegetable scraps during the previous week that contained large amounts of parsnip.

Laboratory results were negative for foot-and-mouth disease, vesicular exanthema of swine and swine vesicular disease. It has been shown that furocoumarin derivatives in parsnip and celery can be absorbed into the skin surface of the pig and be activated by ultraviolet light of wavelengths between 334 nm and 440 nm, resulting in the release of energy that may cause skin damage. It is surmised that the cause of the lesions was photosensitivity resulting from the consumption of parsnips.

Multifactorial disease in backyard poultry

A Salmon Faverolle rooster died from a combination of bacterial enteritis, mycotic pneumonia and severe external parasitism in a small poultry-breeding enterprise in north-west Victoria in July 2007. Clinical signs included lethargy, diarrhoea and depression, developing over 7–10 days after introduction to a group of hens. Similar clinical signs

and history had been noted in the deaths of two other roosters in the preceding 3 months. All roosters had been sourced from different flocks. At necropsy, numerous lice (*Menopon gallinae*) were identified, and the liver showed extensive, multifocal, pinpoint white lesions. Faecal culture revealed *Campylobacter* spp. and *Escherichia coli*. Histology described multifocal, granulomatous pneumonia with in situ fungal hyphae and focally extensive necrotic bacterial enteritis. The loss of three roosters from this unusual poultry breed is of significance given that there is only a relatively small gene pool in the breed.

Western Australia

Contributed by: Fiona Sunderman, Department of Agriculture and Food

Laboratory testing was conducted on 283 investigations of animal disease during the quarter. There were 48 investigations of suspected nationally notifiable diseases, although none were confirmed. All 48 exotic disease investigations were a category 1 alert (low index of suspicion). They mostly involved routine exclusion of avian influenza and Newcastle disease in avian species, and equine influenza exclusion in horses with signs of respiratory disease or horses recently introduced from the eastern States. A diagnosis of an endemic disease or management condition (e.g. a toxicity) was made in all cases where the animal was ill.

Sheep

Polioencephalomalacia

Four cases of polioencephalomalacia were reported during the quarter. Four of 300 mature ewes on a farm at Wongan Hills died after exhibiting neurological signs that included blindness, opisthotonos and staggers. Severe and extensive polioencephalomalacia characterised by acute and subacute neuronal necrosis, widespread vascular hyperplasia, malacia and, in some places, infiltration of gitter cells and architectural collapse was diagnosed.

Mortalities were reported in a mob of ewes and two mobs of lambs on an Esperance farm. A clinically affected lamb exhibited neurological signs and was necropsied. Examination of its brain revealed severe and widespread polioencephalomalacia with minor involvement of small to medium-sized hippocampal neurons.

A 2-year-old ewe in a small flock of sheep at Gingin displayed extreme extensor rigidness and paddling. The animal did not respond to medication and was necropsied. Histopathologically, the extent of the lesions was greater than usual for polioencephalomalacia. It was thought that the lesions had progressed because the sheep had not died quickly, allowing the necrotising process to penetrate into the deeper areas of the cortex and extend posteriorly into the midbrain and hindbrain.

Thirty of a mob of 400 4-month-old ram lambs at Esperance had diarrhoea and several displayed neurological signs. Examination of the brain from a lamb that died revealed severe and extensive cerebrocortical neuronal necrosis with widespread vascular prominence and infiltration of gitter cells.

Yersiniosis

Yersiniosis was diagnosed in a flock of 50 hoggets at Cranbrook. The sheep were on a bare paddock and fed lupins and old pea hay, and then moved to a standing wheat crop for grazing. Some of the flock began scouring, and a faecal worm egg count found a maximum of 400 eggs per gram. The flock began to lose condition, and four hoggets died. Two sheep were submitted for necropsy. Culture of the caeca and small intestines of both yielded pure growths of *Yersinia pseudotuberculosis*, a rare disease in Western Australia.

Photosensitivity

Photosensitivity in lambs was reported in a mob of 2200 White Dorper–Suffolk-cross ewes and 2000 lambs running on a green oat and vetch pasture near Esperance. More than 90% of the lambs and some of the ewes developed photosensitisation, especially of their ears. This is very uncommon, but has been seen before when the plants the sheep graze are stressed in some way. It is believed that the plants produce phytoallexins as a defence mechanism when they are stressed or under attack, and that these

induce primary photosensitisation when eaten. In this instance, the crop was heavily infested with aphids.

Phytogenic fluoroacetate poisoning

Fluoroacetate poisoning killed 300 wethers, and a further 200 were affected, when a mob of 600 accessed uncleared land containing a range of poisonous plants at Kendenup. Affected animals were depressed and had 'a short chopping gait, with a few of them goosestepping on both front and hind legs'. Fragments of plants extracted from the submitted rumen contents were identified as prickly poison (*Gastrolobium spinosum*) and Stirling Range poison (*G. velutinum*).

Salmonellosis

Salmonellosis was diagnosed in a flock of 2000 cross-bred hoggets in a feedlot at Kojonup. One hundred died and another 150 had green mucoid diarrhoea. Necropsy of a freshly dead sheep revealed red urine, enlarged mesenteric lymph nodes and a thickened caecum. Microscopic examination revealed widespread inflammatory changes in the large intestine, varying from place to place in severity. Isolation of *Salmonella* sp. from all cultures confirmed the diagnosis, but it is interesting to note that it is the large intestine that was involved rather than the jejunum.

Cattle

Mercury poisoning

Mercury poisoning was diagnosed in a herd of heifers and calves grazing an area with access to an old shed at Mt Barker. In the shed was an old, disintegrating container of a fungicide containing phenyl mercuric chloride. One of the calves in the mob was noted to be lethargic for 5 days and then died. Another animal, an 18-month-old heifer, looked lethargic. Necropsy of the calf revealed extensive hyperaemia and haemorrhage of the oesophagus, abomasum, small intestine, colon and rectum. Examination of a range of tissues revealed widespread and severe full-depth mucosal necrosis of the gastrointestinal tract and patchy but severe renal tubular necrosis, especially noticeable in the descending tubules. Analysis of the kidney and liver detected mercury levels of 46 and 98 mg/kg wet weight, respectively. The herd was placed in quarantine to ensure that no meat or offal containing mercury residue enters the food chain. Producers are reminded to prevent animal access to items that may cause residue issues or ill-health in their stock.

Bovine pestivirus

Mucosal disease was diagnosed in a 5-month-old Murray Grey-cross calf at Margaret River, presenting with crust formation and ulcerations involving the nasal planum, gingiva, oral mucosa, peri-ocular areas, vulva and anus. The animal had been salivating excessively and was reluctant to suckle.

Bovine pestivirus antigen, but no pestivirus antibody, was detected in its blood, an indication that the calf was a persistently infected (carrier) animal. The clinical presentation suggested it had then gone on to develop clinical mucosal disease, and it died a few days after the clinical examination.

Pestivirus also caused wasting in a 16-month-old bull at Denmark that was much smaller than cattle of the same age. The only abnormality seen upon necropsy was consolidation of ventral lung lobes. Microscopically, bronchi were very prominent, with marked epithelial proliferation and folding of the mucosa and submucosal infiltration of plasma cell dominant cellular infiltrate. In the affected lobules, there was fibrin-like material in alveoli and moderate numbers of macrophages in the lumina, together with clumps of fibrin often with a squamous epithelial covering. A positive result was obtained in the pestivirus antigen capture enzyme-linked immunosorbent assay.

Cerebellar atrophy and hydrocephalus

Cerebellar atrophy and hydrocephalus were diagnosed in a 6-year-old Brahman cow near Broome after it displayed ataxia and a high-stepping gait. The brain and other organs were submitted for laboratory testing and exclusion testing for transmissible spongiform encephalopathy. Severe hydrocephalus and absence of a cerebellum was noted at necropsy. Histologically, the cerebral cortex was markedly reduced in thickness, although the normal neuronal architecture was present. A wispy piece of tissue found covering the medulla was found to contain a vestigial cerebellum. The tissue lacked folia and had a few rows of cells comprising a reduced molecular layer and a reduced, often segmentally absent granular layer. Purkinje cells were present, but their normal orderly array was absent. The surprising thing about this case is that the cow managed to survive for so long in a harsh environment without a proper cerebellum.

The aetiology of the condition is very likely viral, and the prime suspect would be Akabane virus infection.

Nephrosis and hypocalcaemia

Eighteen of 300 cross-bred Angus cows in Manjimup died from nephrosis and hypocalcaemia over a 3-month period. Affected animals were weak before becoming recumbent and were not responsive to intravenous supplementation with calcium, magnesium, phosphorus and glucose. Necropsy performed on a recently calved, moribund cow showed dark mucous membranes and enlarged liver and kidneys. Histologically, the renal tubules were dilated and contained proteinaceous casts. A number of both proximal and distal tubules showed either acute epithelial cell necrosis or evidence of repair, with tubules lined by basophilic cuboidal cells. A small number of well-circumscribed foci of necrosis were present in the liver and had been infiltrated by neutrophils. The liver lesions may have been due to bacterial seeding from the omasum, which had multifocal mucosal epithelial lesions characterised by ballooning and necrosis of cells in the cornified layer, which was also infiltrated by neutrophils.

Analysis of vitreous humour showed low calcium levels that would be expected to occur in clinical hypocalcaemia. There is a poorly described syndrome of acute renal tubular necrosis associated with hypocalcaemia. Consideration was also given to toxic causes of nephrosis, such as ingestion of toxic plants like *Isotropis* sp., lesser loosestrife (*Lythrum hyssopifolia*) and melons (*Cucumis* spp.), and other toxic substances, such as superphosphate. Although omasal inflammation may have been due to acidosis secondary to grain poisoning, the combination of omasal and renal lesions may be a feature of *Cucumis* toxicity.

Haemophilus somnii *pneumonia*

Haemophilus somnii pneumonia killed 20 of 100 dairy calves with respiratory signs at Narrikup. Two calves were necropsied with ventral pneumonia described. *H. somnii* was isolated from both calves. Although there is an obvious bacterial pneumonia, serology results also indicate exposure to both infectious bovine rhinotracheitis and parainfluenza III viruses, both of which could be primary pathogens.

Pigs

Pneumonia

Investigation of increased mortality in weaner pigs and finishers, raised in an eco-shelter system in the Porongurup area, revealed pneumonia and porcine circovirus 2 (PCV2) infection. Necropsy of a weaner revealed pericardial effusion, moderate to severe peritonitis with haemorrhage, and enlargement of all lymph nodes. An interesting finding was the detection of PCV2 antigens in a small piece of thymus that was closely attached to the cervical lymph node. No antigen was detected in the lymph node itself. Although detection of PCV2 antigen meant that the weaner was infected, the virus may not have been the cause of the pig's death.

Lesions in three of the 16-week-old pigs consisted of pericardial effusion, severe pleurisy and lung lesions, consistent with enzootic pneumonia. *Pasteurella multocida* and *Mannheimia haemolytica* were cultured from the lungs.

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 30 September 2007

State	Free
ACT	0
NSW	1022
NT	0
QLD	59
SA	514
TAS	0
VIC	514
WA	183
AUS	2292

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 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 or flocks infected with Johne's disease at 30 September 2007[5]

State	Cattle	Deer	Goat	Sheep	Total
NSW	111	1	8	1286	1406
QLD	0	0	1	0	1
SA	72	1	1	77[a]	151
TAS	16	0	3	58	77
VIC	870	2	4	497	1373
WA	0	0	0	17	17
AUS	1069	4	17	1935	3025

a Eight 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, goat and alpacas; the numbers of herds or flocks that have reached a status of monitored negative 1 or higher are shown in Table 3.

Table 3 Herds or flocks with a market assurance program status of at least monitored negative 1 at 30 September 2007

State	Alpaca	Cattle	Goat	Sheep	Total
NSW	32	502	32	325	891
NT[a]	0	0	0	0	0
QLD[a]	0	0	0	0	0
SA	12	266	12	0	290
TAS	6	110	6	31	153
VIC	1	288	1	0	290
WA	0	0	0	0	0
AUS	51	1166	51	356	1624

a Herds or flocks in free or protected zones have a status of monitored negative 1 or better because of the zone status.

[5] This table replaces the incorrect version that was published in the printed version of this issue.

Lists of beef, goat 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 and Western Australia are undertaking a program of bulk milk testing of all dairy herds. Table 4 shows the number of dairy herds tested free from enzootic bovine leucosis at the end of the quarter.

Table 4 Dairy herds tested free from enzootic bovine leucosis at 30 September 2007

State	Infected	Non-assessed	BMT[a] negative	Provisionally clear	Monitored free	Total
NSW	1	27	26	0	874	928
NT	0	0	0	0	0	0
QLD	2	0	0	0	669	671
SA	1	1	0	0	348	350
TAS	0	486	0	0	0	486
VIC	31	37	1829	32	2872	4801
WA	0	0	0	0	203	203
AUS	35	551	1855	32	4966	7439

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 submissions 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
Jul–Sep 2006	2724	392	1302	152	6670	318	723	0	975	1	542	4
Oct–Dec 2006	7493	439	1931	313	10896	365	1946	8	1254	2	386	13
Jan–Mar 2007	2924	304	1512	227	10267	177	2099	1	1172	1	468	2
Apr–Jun 2007	2199	634	1204	254	5572	326	864	0	1360	0	748	6
Jul–Sep 2007												
NSW	2950	1230	225	10	437	12	169	0	451	0	253	0
NT	492	189	440	65	637	168	0	0	1	0	0	0
QLD	221	90	266	98	201	42	72	0	76	0	0	0
SA	15	0	15	0	15	0	1	0	3	0	3	0
TAS	20	0	0	0	20	0	3	0	1	0	0	0
VIC	48	1	36	0	191	0	3801	0	312	0	141	23
WA	742	25	114	0	730	15	30	0	14	0	8	0
AUS	1833	428	1096	173	2231	237	4076	0	858	0	405	23

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 Transmissible spongiform encephalopathy surveillance

	Jul–Sep 2006		Oct–Dec 2006		Jan–Mar 2007		Apr–Jun 2007		**Jul–Sep 2007**	
State	Cattle	Sheep	Cattle	Sheep	Cattle	Sheep	Cattle	Sheep	**Cattle**	**Sheep**
NSW	34	27	24	65	15	33	18	18	**15**	**5**
NT	10	0	3	0	0	0	2	0	**19**	**0**
QLD	76	10	50	11	38	13	35	5	**0**	**0**
SA	13	15	3	41	4	18	9	24	**2**	**9**
TAS	6	0	1	7	2	0	4	1	**2**	**1**
VIC	39	36	15	44	23	21	25	17	**19**	**43**
WA	15	17	5	92	9	18	14	19	**6**	**34**
AUS	193	105	101	260	91	103	107	84	**63**	**92**

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, 96 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
Jul–Sep 2006	120	0	4456	0
Oct–Dec 2006	167	0	2383	0
Jan–Mar 2007	78	0	1632	0
Apr–Jun 2007	62	0	3161	0
Jul–Sep 2007				
NSW	0	0	1452	0
NT	0	0	0	0
QLD	9	0	418	0
SA	13	0	4	0
TAS	10	0	3	0
VIC	1	0	3729	0
WA	63	0	1947	0
AUS	96	0	7553	0

Salmonella surveillance

The National Enteric Pathogen Surveillance Scheme (NEPSS) is operated and maintained on behalf of the Australian Government and State and Territory governments by the Microbiological Diagnostic Unit at the University of Melbourne. Data on isolates of salmonellas 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: Diane Lightfoot, National Enteric Pathogen Surveillance Scheme, Microbiological Diagnostic Unit, University of Melbourne

Table 8 Salmonella notifications, 1 July to 30 September 2007

	Birds	Cats	Cattle	Dogs	Horses	Pigs	Sheep	Other	Total
S. Bovismorbificans	0	1	35	0	5	0	3	1	45
S. Dublin	0	0	25	0	0	0	0	0	25
S. Infantis	0	0	5	3	3	1	0	0	12
S. Typhimurium	8	0	118	4	5	1	4	1	141
Other	2	2	84	14	4	14	4	39	163
Total	10	3	267	21	17	16	11	41	386

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. Table 9 summarises results from the program.

Table 9 Results of the National Granuloma Submission Program

	Jul–Sep 2006	Oct–Dec 2006	Jan–Mar 2007	Apr–Jun 2007	Jul–Sep 2007
Submitted	378	209	178	214	234
TB +ve	0	0	0	0	0

Northern Australia 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. Information is derived from the use of sentinel animals, structured surveys and opportunistic sampling. Table 10 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 10 Summary of recent NAQS activity in Australia

	Jul–Sep 2006		Oct–Dec 2006		Jan–Mar 2007		Apr–Jun 2007		**Jul–Sep 2007**	
Category	Tested	+ve	Tested	+ve	Tested	+ve	Tested	+ve	**Tested**	**+ve**
Aujeszky's disease	147	0	225	0	0	0	0	0	**0**	**0**
Australian bat lyssavirus	0	0	2	0	1	0	0	0	**1**	**0**
Avian influenza — highly pathogenic	413	0	1835	0	0	0	32	0	**364**	**0**
Classical swine fever	147	0	225	0	0	0	107	0	**153**	**0**
Japanese encephalitis	51	0	71	0	45	0	15	0	**0**	**0**
Surra — *Trypanosoma evansi*	124	0	185	0	156	0	96	0	**23**	**0**

Ports Surveillance Program

The Ports Surveillance Program is conducted for *Culicoides* and screw-worm fly by Biosecurity Australia, and for exotic bees and bee mites by Product Integrity, Animal and Plant Health, in the Department of Agriculture, Fisheries and Forestry. 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 11 shows the number of times that insect trap sites were inspected for the Port Surveillance Program; no exotic insects or mites were detected.

Contact: Iain East, Office of the Chief Veterinary Officer, and Howe Heng, Biosecurity Australia, both of the Australian Government Department of Agriculture, Fisheries and Forestry

Table 11 Ports Surveillance Program: number of inspections of insect traps

		Jul–Sep 2006	Oct–Dec 2006	Jan–Mar 2007	Apr–Jun 2007	**Jul–Sep 2007**
Ports	Asian bees	6	17	12	32	**20**
	Varroa mites	17	26	22	26	**10**
	Asian mites	17	26	22	26	**10**
	Tracheal mites	18	43	29	29	**17**
	Culicoides sp.	27	28	27	29	**28**
	Screw-worm fly	23	20	21	21	**23**
NAQS	Screw-worm fly	45	45	45	45	**45**

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 NAHIS each quarter (see Table 12).

Contact: National Notifiable Diseases Surveillance System, Australian Government Department of Health and Ageing (http://www9.health.gov.au/cda/Source/CDA-index.cfm)

Table 12 Notification of zoonotic disease in humans

	Q3 2006	Q4 2006	Q1 2007	Q2 2007	**Q3 2007**	Current quarter (July–September 2007)							
			AUS			ACT	NSW	NT	QLD	SA	TAS	VIC	WA	AUS
Brucellosis	14	10	15	6	**7**	0	0	0	4	1	1	1	0	7
Chlamydophilosis	38	46	30	26	**17**	0	3	0	1	0	0	12	1	17
Leptospirosis	20	15	48	33	**11**	0	1	0	7	1	0	1	1	11
Listeriosis	14	13	17	8	**9**	0	5	0	2	1	0	1	0	9
Q fever	100	104	121	126	**110**	2	45	0	41	12	0	9	1	110

National Residue Survey

During the third quarter of 2007, 3091 samples were collected and analysed in the National Residue Survey Random Monitoring Program (see Table 13). Two samples were found with residues above the relevant standard in the *Australian Food Standards Code*.

One sample of liver from sheep was found with a lead residue over the Australian maximum level (ML) of 0.5 mg/kg. This sample contained 0.6 mg/kg. No specific cause for this residue could be identified.

One sample of sheep liver had a cadmium level above the ML of 1.25 mg/kg. Cadmium residues above the ML are a common finding in older sheep across southern Australia. While this cadmium detection was above the ML for sheep liver, it was below the action level of 2.5 mg/kg required to initiate a trace-back investigation.

A third sample, of pig muscle, contained a residue of dimetridazole at 0.001 mg/kg, which is above the temporary maximum residue level of 0.0001 mg/kg recently gazetted by the Australian Pesticides and Veterinary Medicines Authority. A trace-back investigation is continuing. It should be noted that use of dimetridazole is being phased out.

Contributed by: Jim Derrick, National Residue Survey, Australian Government Department of Agriculture, Fisheries and Forestry

Table 13 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	67	0	2	0	119	0	14	0	5	0	23	0	10	0	240
	pigs	0	29	0	0	0	13	0	1	0	1	0	10	0	15	0	69
	poultry	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	sheep	0	74	0	0	0	8	0	25	0	2	0	38	0	25	0	172
	other	0	11	0	0	0	12	0	2	0	0	0	11	0	0	0	36
	Total	0	181	0	2	0	152	0	42	0	8	0	82	0	50	0	517
Antimicrobials	cattle	0	89	0	3	0	168	0	19	0	13	0	92	0	16	0	400
	pigs	0	66	0	0	1	46	0	13	0	0	0	26	0	32	1	183
	poultry	0	16	0	0	0	0	0	0	0	0	0	0	0	0	0	16
	sheep	0	71	0	0	0	6	0	23	0	0	0	22	0	24	0	146
	other	0	3	0	0	0	6	0	4	0	0	0	10	0	0	0	23
	Total	0	245	0	3	1	226	0	59	0	13	0	150	0	72	1	768

		NSW		NT		QLD		SA		TAS		VIC		WA		AUS	
Growth promotants	cattle	0	103	0	1	0	165	0	13	0	6	0	19	0	14	0	321
	pigs	0	46	0	0	0	30	0	9	0	2	0	18	0	22	0	127
	poultry	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1
	sheep	0	81	0	0	0	15	0	20	0	2	0	19	0	20	0	157
	other	0	0	0	2	0	2	0	1	0	0	0	7	0	0	0	12
	Total	0	231	0	3	0	212	0	43	0	10	0	63	0	56	0	618
Insecticides	cattle	0	97	0	1	0	176	0	20	0	11	0	60	0	18	0	383
	pigs	0	23	0	0	0	18	0	3	0	1	0	9	0	9	0	63
	poultry	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	sheep	0	95	0	0	0	17	0	26	0	1	0	28	0	46	0	213
	other	0	13	0	0	0	28	0	2	0	0	0	11	0	1	0	55
	Total	0	228	0	1	0	239	0	51	0	13	0	108	0	74	0	714
Metals	cattle	0	23	0	0	0	55	0	1	0	2	0	16	0	2	0	99
	other	0	17	0	1	0	13	0	0	0	0	0	4	0	2	0	37
	pigs	0	27	0	0	0	17	0	3	0	0	0	10	0	13	0	70
	poultry	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	sheep	1	21	0	0	0	1	1	7	0	3	0	6	0	12	2	50
	Total	1	88	0	1	0	86	1	11	0	5	0	36	0	29	2	256
Miscellaneous	cattle	0	20	0	1	0	49	0	4	0	3	0	14	0	6	0	97
	pigs	0	14	0	0	0	18	0	4	0	0	0	11	0	8	0	55
	poultry	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	sheep	0	29	0	0	0	4	0	9	0	0	0	9	0	6	0	57
	other	0	1	0	1	0	3	0	0	0	0	0	4	0	0	0	9
	Total	0	64	0	2	0	74	0	17	0	3	0	38	0	20	0	218
Total		1	1037	0	12	1	989	1	223	0	52	0	477	0	301	3	3091

Suspect exotic or emergency disease investigations

There were 69 investigations (other than for equine influenza) of diseases, suspected to be either exotic or a possible emergency, reported during the quarter, as shown in Table 14. More details about some of these investigations can be found in the State and Territory reports. One hundred and seventy-two equine influenza investigations are reported; these represent exclusion testing of clinically suspect cases in States that are, to date, free from equine influenza. In Queensland, the tests reported are those conducted up to and including the identification of the first case of disease; testing conducted subsequently is not reported here. Details of testing for equine influenza in New South Wales are described in the State report.

Table 14 Exotic or emergency disease investigations reported, 1 July to 30 September 2007

Disease	Species	State	Month	Response code	Finding
African horse sickness	Equine	WA	Aug	3	negative
Avian influenza — highly pathogenic	Avian	NSW	Jul	2	negative (6 unrelated investigations)
	Avian	NSW	Aug	2	negative (2 unrelated investigations)
	Avian	QLD	Jul	3	negative
	Avian	TAS	Aug	2	negative (2 unrelated investigations)
	Avian	TAS	Jul	3	negative
	Avian	VIC	Jul	3	negative
	Avian	VIC	Aug	3	negative (6 unrelated investigations)
	Avian	VIC	Sep	3	negative (11 unrelated investigations)
	Avian	WA	Aug	2	negative (7 unrelated investigations)
	Avian	WA	Jul	2	negative (5 unrelated investigations)
	Avian	WA	Sep	2	negative (5 unrelated investigations)
Brucellosis — bovine (*B. abortus*)	Ovine	WA	Aug	2	negative
	Porcine	WA	Aug	2	negative
Equine herpes-virus 1 — abortigenic and neurological strains	Equine	QLD	Jul	2	positive
	Equine	QLD	Sep	2	positive
	Equine	TAS	Aug	2	negative (2 unrelated investigations)
	Equine	VIC	Sep	2	negative
Equine influenza	Equine	NT	Aug	2	negative (2 unrelated investigations)
	Equine	NT	Sep	2	negative
	Equine	QLD	Aug	3	positive
	Equine	SA	Aug	3	negative (11 unrelated investigations)
	Equine	SA	Sep	3	negative (24 unrelated investigations)
	Equine	TAS	Aug	1	negative (2 unrelated investigations)
	Equine	TAS	Aug	2	negative
	Equine	TAS	Aug	3	negative
	Equine	TAS	Aug	5	negative
	Equine	TAS	Sep	1	negative (3 unrelated investigations)
	Equine	TAS	Sep	3	negative (2 unrelated investigations)
	Equine	VIC	Aug	1	negative (2 unrelated investigations)
	Equine	VIC	Aug	3	negative (35 unrelated investigations)
	Equine	VIC	Sep	1	negative (6 unrelated investigations)
	Equine	VIC	Sep	3	negative (72 unrelated investigations)
	Equine	WA	Aug	3	negative (6 unrelated investigations)
	Equine	WA	Sep	3	negative (2 unrelated investigations)

Disease	Species	State	Month	Response code	Finding
Foot-and-mouth disease	Bovine	NT	Sep	3	negative
	Bovine	VIC	Aug	3	negative (2 unrelated investigations)
	Bovine	VIC	Sep	2	negative
	Porcine	VIC	Jul	3	negative
Hendra virus	Equine	NSW	Aug	2	negative
	Equine	QLD	Jul	3	positive
	Equine	QLD	Aug	3	negative
	Equine	WA	Aug	3	negative
Newcastle disease — virulent	Avian	VIC	Sep	3	negative
Rabies	Bats	WA	Sep	3	negative
	Feline	NSW	Aug	2	negative
Tuberculosis in any mammal	Bottlenosed dolphin	TAS	Jul	4	negative
Vesicular diseases of swine	Porcine	NT	Jul	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

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
Ian Langstaff	Animal Health Australia Program Manager	02 6203 3903	02 6232 5511	ilangstaff@animalhealthaustralia.com.au
Ingo Ernst	Aquatic Animal Health	02 6272 5615	02 6272 3372	ingo.ernst@daff.gov.au
Iain East	Australian Government NAHIS Coordinator	02 6272 3106	02 6272 3150	iain.east@daff.gov.au
Kristy Venten	Australian Milk Residue Analysis Survey	03 9810 5919	03 9819 4299	kventen@dairysafe.vic.gov.au
Rupert Woods	Australian Wildlife Health Network	02 9978 4749	02 9978 4516	rwoods@zoo.nsw.gov.au
Chris Bunn	Emergency Disease Preparedness, DAFF	02 6272 5540	02 6272 3372	chris.bunn@daff.gov.au
David Kennedy	Johne's Disease Coordinator	02 6365 6016	02 6365 6088	david@ausvet.com.au
Diane Lightfoot	National Enteric Pathogen Surveillance Scheme	03 8344 5701	03 8344 7833	dligh@unimelb.edu.au
Neville Spencer	National Granuloma Submission Program	02 6271 6650	02 6272 5442	neville.spencer@aqis.gov.au
Krissa O'Neil	National Notifiable Diseases Surveillance System	02 6289 1555	02 6289 7791	www.health.gov.au
Jim Derrick	National Residue Survey	02 6272 4019	02 6272 4023	jim.derrick@daff.gov.au
Jenny Hutchison	National Surveillance Coordinator	02 6287 4483	02 6287 4468	jenny@ausvet.com.au
Jane Parlett	Northern Australia Quarantine Strategy	02 6272 3494	02 6272 3468	jane.parlett@aqis.gov.au
State Coordinators				
Barbara Moloney	NSW State Coordinator	02 6391 3687	02 6361 9976	barbara.moloney@dpi.nsw.gov.au
Francois Human	NT State Coordinator	08 8999 2246	08 8999 2024	francois.human@nt.gov.au
John Cronin	QLD State Coordinator	07 4688 1220	07 4688 1199	john.cronin@dpi.qld.gov.au
Celia Dickason	SA State Coordinator	08 8391 7125	08 8388 8455	dickason.celia@saugov.sa.gov.au
Mary Lou Conway	TAS State Coordinator	03 6233 6330	03 6278 1875	marylou.conway@dpiw.tas.gov.au
Cameron Bell	VIC State Coordinator	03 5430 4545	03 5430 4520	cameron.bell@dpi.vic.gov.au
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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