Surveillance

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Contents

Surveillance is published as the Ministry for Primary Industries' authoritative source of information on the ongoing biosecurity surveillance activity and the health status of New Zealand's animal and plant populations in both terrestrial and aquatic environments. It reports information of interest both locally and internationally and complements New Zealand's international reporting.

EDITORIAL

Research in biosecurity surveillance

The recent detection of Queensland fruit fly in Whangarei by MPI's fruit fly surveillance programme demonstrates the value of biosecurity surveillance. Follow-up surveillance was able to determine with certainty that no more Queensland fruit fly were in the area. The credibility of results from the surveillance was underpinned by scientific research. Knowledge of pest biology and trap effectiveness are required to ensure the most effective surveillance programme and to ensure that we are making the most effective use of taxpayers' money by directing resources to where the risk is highest. (More information on MPI's fruit fly surveillance programme can be found in the Annual Report in *Surveillance* 40(3), 72; http://www.sciquest.org.nz/node/89529).

There are strong links between MPI and research providers in the area of biosecurity surveillance. This means surveillance benefits from the research very quickly as research meets surveillance needs and is in step with surveillance work.

A significant group that MPI works closely with is the Better Border Biosecurity (B3) science collaboration (http://b3nz.org/). This is a collaboration between NZ's science agencies (Plant & Food Research, AgResearch, Scion, Bio-Protection Research Centre at Lincoln University – with Landcare Research joining recently) to facilitate biosecurity research in New Zealand. B3's research covers the border spectrum, from risk analysis to eradication. Its surveillance research includes developing:

- new and improved lures for targeted insect trapping;
- self-reporting "smart traps" and automated trap analysis software;
- surveillance methods for plant pathogens and their vectors; and
- methods to measure surveillance efficacy and objectively allocate surveillance effort.

Resources and knowledge from B3's biosecurity surveillance research have been used in incursion responses to great white butterfly, eucalyptus leaf beetle and Australian pasture tunnel moth (see page 40). New technology being developed from research also offers opportunities that will allow the more efficient delivery of programmes in the future. For example, self-reporting traps may increase the speed of reporting and reduce inspection costs.

MPI has invested directly in new research initiatives and enhancements of current surveillance programmes. MPI operational research funding is being used to underpin research on the fruit fly surveillance programme, improve the efficiency of current surveillance programme (e.g. by using multiple lures in surveillance traps – see page 46), and even evaluating the potential of remotely piloted aircraft for biosecurity surveillance.

The one constant with biosecurity is change. New pests are discovered, old pests can become more significant, and trade patterns and volumes are constantly shifting. To ensure that MPI can meet the challenge of these changes requires a good understanding of science and linkages with the most up-to-date research in New Zealand and overseas. One example of these linkages is the Quadrilateral (QUAD) Working Group, made up of biosecurity experts from New Zealand, Australia, Canada and the US. The surveillance-based project within the QUAD working group is the "Lures and Protocols" project. This work shares technical information and research outcomes on surveillance programmes within each respective country to enhance best-practice surveillance for exotic plant pests.

Another way in which research informs biosecurity surveillance is by providing guidelines on optimal levels of surveillance, the trade-off between surveillance and response and when to stop surveillance after a pest is considered eradicated. Recent work in this area includes an international collaboration between New Zealand and US researchers to develop a model for the optimal economic level of surveillance for invasive beetles. This model has also supported the general understanding among surveillance practitioners that greater surveillance is warranted in areas that receive more imported goods that serve as an invasion pathway. This focus on invasion pathways underlies resource allocation in all of MPI's plant pest biosecurity surveillance programmes.

Just as biosecurity surveillance is the precursor to biosecurity responses, so good research underpins good biosecurity surveillance. Without good research backing up biosecurity surveillance we can't be sure that we are doing the best job that we can. And when it comes to protecting New Zealand's primary industries MPI is serious about ensuring that we are doing the best we can.

Paul Stevens

ANIMALS

EXOTIC DISEASES OF POULTRY: A REVIEW

From a surveillance perspective, backyard poultry could provide the vital early warning of an exotic disease incursion. It's important for vets in the field to be able to identify whether any clinical signs they observe are consistent with an endemic disease or an exotic disease requiring notification to the MPI exotic pest and disease hotline.

The possibility of exotic disease should always be considered when these clinical signs are present:

- sudden, unexplained deaths;
- rapid spread of disease throughout the flock;
- depression and loss of appetite;
- a drop in egg production;
- nervous signs;
- swelling and blue combs and wattles; and
- sneezing, coughing and diarrhoea.

Mortality can be extremely high, with entire flocks dying over just a few days. If you suspect exotic disease you should call the exotic pest and disease hotline for further testing.

The following is a summary description of some important exotic diseases of poultry in the New Zealand context. They are diseases that veterinarians and flock owners should keep in mind when presented with diseased poultry especially where multiple birds have been affected. This is not intended to be an exhaustive list. A detailed summary of these diseases is also provided on pages 16–21 (**Table 1**).

Keeping backyard poultry is popular in many New Zealand households. Often the birds have the status of companion animals and many owners will seek veterinary advice if they demonstrate unusual behaviour or disease. This article seeks to highlight some important diseases of poultry that are exotic to New Zealand. It is not intended to be an exhaustive list or in-depth technical review of the diseases. This is the second part of a series on diseases of poultry published in *Surveillance* (see also Watts, 2013) to encourage awareness of diseases of poultry.

Duck virus hepatitis (DHV)

CAUSAL AGENT AND DISTRIBUTION

Duck hepatitis is caused by duck hepatitis virus type 1 (DHV-1), an enterovirus in the *Picornaviridae* family. DHV-1 is the most common type, with two others known to occur regionally; but there are currently four types of the virus known to cause disease in ducks. DHV-1 is a non-enveloped virus that can persist in normal environmental conditions. DHV-1 does not occur in NZ.

CLINICAL SIGNS

The most common sign associated with DHV-1 infection is sudden death following neurological signs such at opisthotonus, paresis and paralysis. Death can occur within 1–2 hours after first appearance of neurological signs. DHV-1 causes significant mortality and morbidity in naïve flocks of young ducklings (< six weeks old but mainly < three weeks old).

At postmortem the key findings are an enlarged, green liver with distinct ecchymotic hemorrhages throughout the parenchyma (**Figure 1**).

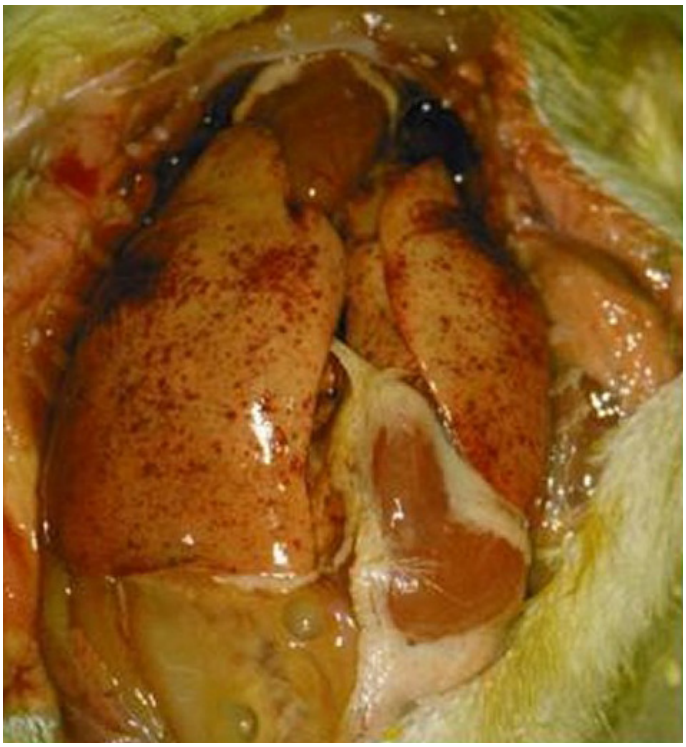

Figure 1: Duck hepatitis A virus type 1 infection in a week-old duckling. Note hemorrhagic lesions on liver.

TREATMENT AND PREVENTION

No treatment is effective once a flock becomes infected, and culling is recommended. There are vaccines available, but a simple effective measure is isolation of ducklings until more than six weeks of age.

Duck viral enteritis (DVE) or duck plague

CAUSAL AGENT AND DISTRIBUTION

Duck viral enteritis is caused by anatid herpesvirus type 1, which is the same as duck viral enteritis virus (DVE). This virus is a member of the *Herpesviridae* family and can occur in all members of the Anatidae, which includes ducks, geese and swans. This virus is endemic in wild waterfowl populations throughout North America, Europe and Asia, and the main risk of transmission is exposure of domestic poultry to infected wild birds. DVE has not been detected in wild or domestic poultry in NZ.

CLINICAL SIGNS

The clinical signs of DVE are quite varied and may include respiratory, reproductive and ophthalmic signs, general ill thrift and bloody, greenish diarrhoea (**Figure 2**). At the flock level there is a pattern of increase of clinical signs as the infection spreads, with older birds often showing more signs of illness (including mortality). In breeding flocks the drop in egg production can be quite significant. The incubation period is 3–7 days, with mortality 1–5 days after the first clinical signs appear.

At postmortem the key finding is yellow-white (diptheritic) plaques on the enteric mucosa (**Figure 3**). There should also be systemic signs of haemorrhage, with petechiae in various tissues including the syrinx, liver (**Figure 4**) and the intestine. There may also be blood in the coelomic cavity and the alimentary tract.

TREATMENT AND PREVENTION

Effective vaccines are used overseas to minimise the effects of outbreaks. Prevention involves screening for infected individuals when establishing flocks, and culling carriers. DVE is best avoided in the first place, by eliminating exposure to wild anatids and water sources they frequent.

Differential diagnoses are:

- duck viral hepatitis;
- pasteurellosis;
- necrotic and haemorrhagic enteritis;
- trauma;
- drake damage; and
- toxicoses.

Newcastle's disease, avian influenza and fowl pox may cause similar lesions but are rarely reported in ducks. (Banda, 2013)

Figure 2: Clinical signs of duck viral enteritis. Note depression and mild dyspnoea.

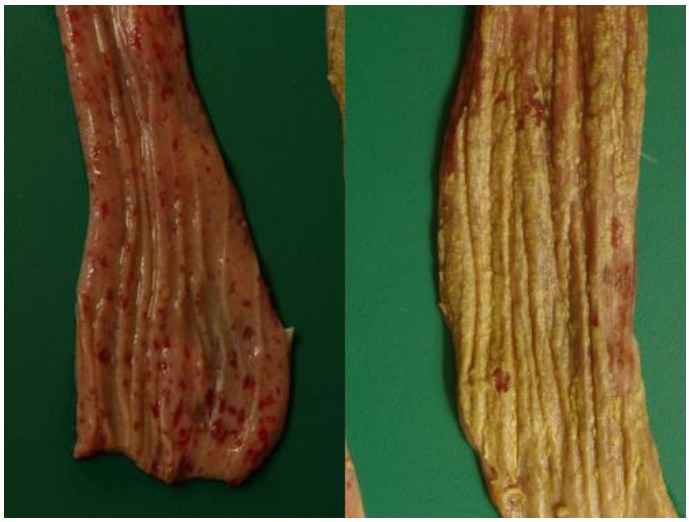

Figure 3: Petechial and ecchymotic haemorrhages (*left*) and diphtheritic membrane (*right*) on oesophagus in duck viral enteritis

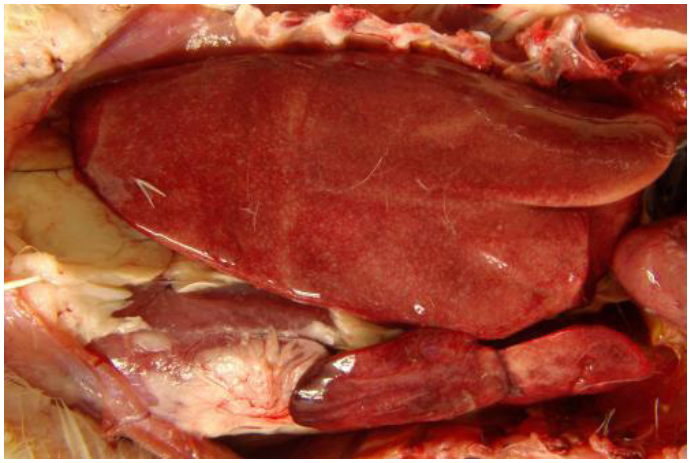

Figure 4: DVE showing diffuse petechiation on liver.

Courtesy of Dr. Alejandro Banda and the Merck Veterinary Manual, published by Merck & Co., Inc., Whitehouse Station, NJ. All rights reserved. Used with permission. Available at: www.MerckManuals.com.

Fowl typhoid and pullorum disease

CAUSAL AGENT AND DISTRIBUTION

Fowl typhoid results from infection by *Salmonella enterica* ssp. *enterica* serovar Gallinarum biovar Gallinarum (*Salmonella* Gallinarum), a Gram-negative bacterial rod in the family Enterobacteriaceae (serogroup D). Pullorum disease is caused by the closely related organism *S. enterica* ssp. *enterica* ser. Gallinarum biovar Pullorum (*Salmonella* Pullorum) (Spickler, 2009). The causal organisms have been eliminated from commercial poultry flocks in many countries. Both these biovars are primarily pathogens of chickens but pullorum infections may also be found, albeit uncommonly, in other species of birds and in mammals. The bacteria can spread readily both horizontally (directly and indirectly) and vertically.

CLINICAL SIGNS

Usually both organisms cause generalised clinical signs of illness such as depression, dehydration, anorexia, ruffled feathers and weakness. Both can also cause diminished egg production, reduced chick survival and decreased hatching. The incubation period is up to a week. While fowl typhoid typically affects older birds, pullorum disease is usually a disease of younger birds. Mortalities can be quite high from both pathogens.

Post-mortem findings can be systemic signs of peritonitis, congestion of liver, spleen (**Figure 5**) and lungs, as well as arthritis and enteritis .The caecum may be enlarged and can contain firm, cheesy material (caecal cores). White necrotic foci or nodules may be found in the liver (**Figure 6**), spleen, lungs, heart (**Figure 7**), pancreas and gizzard, and sometimes in the caecum (Spickler, 2009). Lesions can resemble *Pasteurella* and *Erysipelas* infections. Oedema can occur in the tibiotarsal joints (**Figure 8**).

Figure 5: Avian liver and spleen. Liver is pale with diffuse yellow-brown (bronze) discolouration; splenic congestion and enlargement.

Courtesy of Dr. Andreasen, Iowa State University, College of Veterinary Medicine, Department of Veterinary Pathology and the Center for Food Security and Public Health at Iowa State University, College of Veterinary Medicine.

Figure 6: Acute fowl typhoid. In some cases the size of liver necroses varies from milliary to spots with a diameter of 1–2 cm. Unlike pullorum disease, fowl typhoid lasts for months. Courtesy of Ceva.

Figure 7: Greyish-white nodes on the heart caused by infection with *S. pullorum*. Courtesy of Ceva.

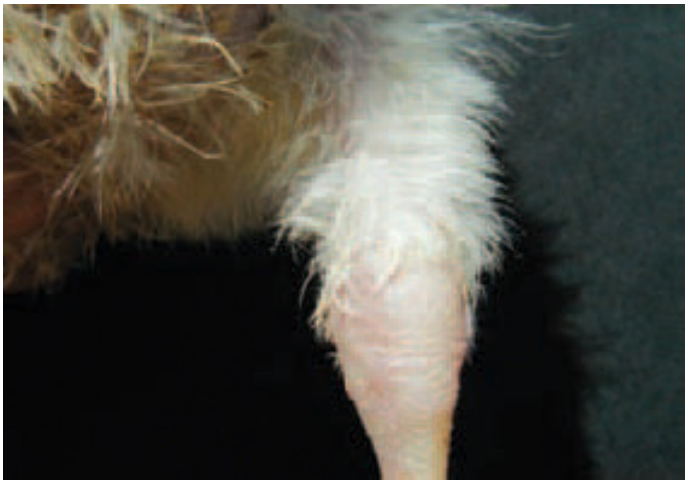

Figure 8: Oedema of the tibiotarsal joints. Courtesy of Ceva.

TREATMENT AND PREVENTION

Antibiotic treatment is possible once a flock becomes infected, but culling is recommended. There are vaccines available in countries where fowl typhoid is endemic, but these do not prevent infection: they just reduce mortality. The best prevention is only to use stock from disease-free flocks or use a test-and-cull process to create flocks free of fowl typhoid and pullorum.

Differential diagnoses are:

- infection with other *Salmonella* species;
- *Mycoplasma synoviae;*
- *Staphylococcus aureus;*
- *Pasteurella multocida;*
- *Erysipelothrix rhusiopathiae;* and
- fungal infections (including *Aspergillus*).

In chicks the white nodules in the internal organs can be confused with Marek's disease or hepatic lesions caused by *Yersinia pseudotuberculosis* (Spickler, 2009).

Turkey rhinotracheitis

CAUSAL AGENT AND DISTRIBUTION

Turkey rhinotracheitis is a clinical syndrome resulting from infection by avian metapneumovirus (aMPV), which is in the *Paramyxoviridae* family. The virus can infect turkeys, chickens (where it is also known as swollen head syndrome), pheasants, Peking ducks and guinea fowl. It is found worldwide except in New Zealand, where it is a declared Unwanted Organism. There are four antigenic subtypes and the severity of clinical signs often depends on the effects of co-infecting bacterial pathogens.

CLINICAL SIGNS

The clinical signs in turkeys are mostly respiratory and include sneezing, tracheal rales, gasping, nasal and ocular discharge and swelling around the eyes in the region of the infraorbital sinuses. Clinical signs in chickens can also begin as upper respiratory tract signs but can also include decreased egg production and neurological signs. Invasions of secondary pathogens such as *E. coli,* and concurrent infections with infectious bronchitis virus, may exacerbate the signs of infection, resulting in swollen head syndrome. These signs include swelling of the head, neck and wattles (**Figure 9**) (Anon., 2014). In turkey flocks morbidity is often very high but mortality is less and more variable. Clinical signs vary with the age of the

flock and differing management practices: for example, young turkeys in densely stocked poorly ventilated housing are often the worst affected. The incubation period is very short and even clinical signs can progress through a flock in a matter of hours with a full recovery within 14 days.

Key post-mortem findings are rhinitis and tracheitis with hyperemia with mucopurulent exudate.

Figure 9: Periorbital swelling. © Cornell University.

TREATMENT AND PREVENTION

Vaccines are available in countries where aMPV is endemic, but not in New Zealand. Vaccination is known to be effective in reducing clinical disease and virus excretion.

Differential diagnoses (Rautenschlein, 2013) are:

- paramyxoviruses (particularly Newcastle disease and PM3);
- infectious bronchitis;
- avian influenza; and
- Mycoplasma gallisepticum.

Avian spirochaetosis

CAUSAL AGENT AND DISTRIBUTION

Avian spirochaetosis is caused by a motile spirochaete bacterium, *Borrelia anserine*. The spread of this organism requires some form of mechanical vector such as direct transmission of blood (e.g. by ticks, lice or mosquitoes); contamination with infected blood (e.g. from needles, sharp objects or through cannabalism); or contamination with faeces (e.g. in food and water). These bacteria can infect all poultry species, with free-range birds or birds on litter bedding more likely to be exposed.

CLINICAL SIGNS

Avian spirochaetosis presents as generalised syndromes of ill-thrift (e.g. depressed, anorexic, huddling, dehydrated), respiratory distress (e.g. cyanotic wattles) and gastrointestinal infection (e.g. green or yellow, greasy diarrhoea). On necropsy there is often enteritis and an enlarged spleen with petechial or ecchymotic haemorrhages. The other parenchymal organs may contain areas of necrosis and generally are enlarged and pale.

TREATMENT AND PREVENTION

The organism is treatable with penicillin and tiamulin (Burch, 2006) but prevention through vector control and disinfection of fomites is ideal. There is a vaccine that also is effective in eradication efforts.

Infectious bursal disease (IBD)

CAUSAL AGENT AND DISTRIBUTION

Infectious bursal disease (also known as gumboro disease, infectious bursitis and infectious avian nephrosis) is a highly contagious disease of young chickens caused by infectious bursal disease virus (IBDV). It is economically important to the poultry industry worldwide because it heightens susceptibility to other diseases and causes negative interference with effective vaccination. There are two serotypes, with serotype 2 causing a subclinical infection in chickens and turkeys. Recently very virulent strains (called vvIBDV), causing severe mortality in commercial poultry have emerged in Europe, Latin America, Southeast Asia, Africa and the Middle East. Infection is via the oro-faecal route, with affected birds excreting high levels of the virus for about two weeks after they become infected.

CLINICAL SIGNS

IBD is characterized by immunosuppression and mortality, generally at 3–6 weeks of age after an incubation period of 3–4 days. Affected chickens can seem inco-ordinated or unable to stand and they often have watery diarrhoea. Mortality can be up to 20 percent but most birds recover.

Post-mortem changes are mostly noted in the bursa of Fabricius, which may be swollen and haemorrhagic (**Figure 10**) or reduced in size, depending on the time since infection. Congestion and haemorrhage of skeletal muscle **(Figure 11**) is also often seen.

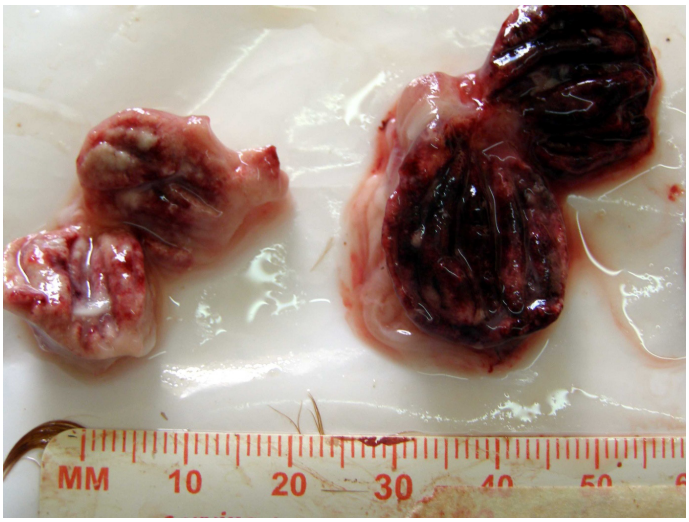

Figure 10: Haemorrhagic bursa of Fabricus. Neil Christensen.

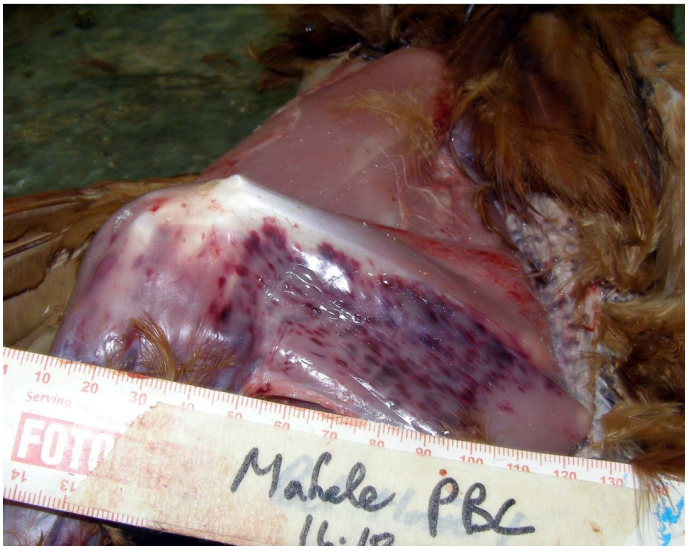

Figure 11: Haemorrhage in skeletal muscle. Neil Christensen.

TREATMENT AND PREVENTION

No treatment is effective once a flock becomes infected, but vaccines are available and widely used.

Highly pathogenic avian influenza (HPAI)

CAUSAL AGENT AND DISTRIBUTION

HPAI is an OIE-listed disease from the virus family *Orthomyxoviridae*. To date, all highly pathogenic isolates in birds have been influenza A viruses of subtypes H5 and H7. Epidemics of HPAI can spread rapidly, devastate the poultry industry and result in severe trade restrictions.

It is possible for wild birds to carry HPAI viruses, but more often wild birds transmit low-pathogenic avian influenza (LPAI) viruses to poultry. These viruses then mutate to become HPAI viruses while circulating in poultry flocks. Although HPAI outbreaks can be devastating, the virus is successfully eradicated in most cases. However, the world is currently experiencing an extensive avian influenza outbreak with no immediate prospects for complete worldwide eradication. (Spickler, 2010)

In birds, avian influenza viruses are shed in the faeces as well as in saliva and nasal secretions. The faeces contain large amounts of virus, and faecal-oral transmission is usually the predominant means of spread in wild bird reservoirs.

CLINICAL SIGNS

Incubation period in individual birds usually is short (2–5 days), and the rate of infection in a flock can be highly variable (days to weeks) depending on the environmental conditions and the ability of the virus to spread between birds (cages vs free-range situations, etc.).

Sudden deaths are common and mortality is high (up to 100 percent). The clinical presentation varies with the species, age, type of bird, viral strain, concurrent infections and environment. Respiratory signs include ocular and nasal discharges, coughing, dyspnoea and swelling of the sinuses and/or head (**Figure 12**). Other signs include severe depression (**Figure 13**), reduced vocalisation, a marked reduction in feed and water intake; cyanosis of unfeathered skin, wattles and comb; inco-ordination, diarrhoea and a drastic decline in egg production.

Post-mortem lesions in turkeys and chickens are variable and resemble those found in other systemic avian diseases. Subcutaneous oedema may be present on the head and neck; oedema and diffuse

subcutaneous haemorrhages on the feet and shanks; fluid (possibly haemorrhagic) in the nares and oral cavity; and congestion, swelling and haemorrhages of the conjunctivae (**Figure 14**). Haemorrhagic tracheitis may be seen in some birds (**Figure 15**); in others the tracheal lesions may be limited to excess mucoid exudates. (Spickler, 2010)

The lungs may be haemorrhagic and congested. Petechiae may be seen throughout the abdominal fat, on serosal surfaces (**Figure 16**), on the peritoneum and occasionally in the muscles (**Figure 17**). Haemorrhages may also be seen on the mucosa and in the glands of the proventriculus (**Figure 18**), beneath the lining of the gizzard and in the intestinal mucosa. The kidneys may be severely congested. The ovaries may be haemorrhagic or degenerated, with areas of necrosis (Spickler, 2010; **Figure 19**).

In domestic ducks, most HPAI viruses produce few clinical signs and virus detection is needed for definitive diagnosis.

ZOONOTIC POTENTIAL

Some avian influenza viruses can infect mammals, including humans, and the severity of zoonotic avian influenza varies with the virus. Although many human infections are limited to conjunctivitis or mild respiratory disease, some viral strains cause severe disease and death. There is strong evidence that people become infected following close contact with infected live poultry, mostly in live bird markets or when slaughtering birds at home.

Generally, avian influenza viruses do not spread efficiently in mammals, and infections are limited to individual animals or small groups. However, some viruses can become adapted to a new species and cause epidemics or pandemics.

TREATMENT AND PREVENTION

There is no treatment. Rapid depopulation, disposal and disinfection is required on infected properties. Vaccination has been used in some control and eradication programmes (Swayne & Suarez, 2000).

The best prevention is by avoiding contact between poultry and wild birds or their fomites, in particular waterfowl. Avoid introducing birds of unknown disease status into a flock. Birds should not be brought back to the farm from live bird markets or from other slaughter channels. Control of human traffic, with strict hygiene and biosecurity measures, is necessary to prevent fomite transmission. One age/species group per farm ("all in/all out") breeding is recommended (Anon., 2014).

Differential diagnoses for HPAI are:

- acute fowl cholera;
- velogenic Newcastle disease;
- respiratory diseases, especially infectious laryngotracheitis; and
- heat exhaustion, water deprivation and some toxins.

Figure 12: HPAI clinical signs: oedema (comb, wattles, eyelids, periorbital), ecchymoses (comb, wattles). © Cornell University.

Figure 13: HPAI clinical signs: oedema (face), ecchymoses (comb, wattles, legs), depression. © Cornell University.

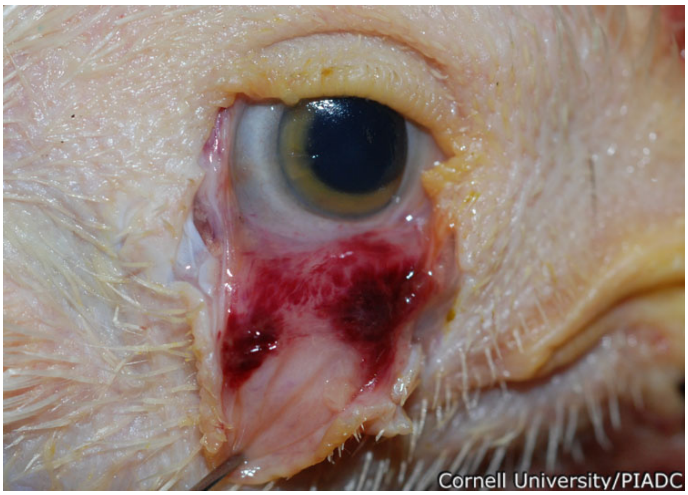

Figure 14: HPAI: conjunctivae severely congested; extensive hemorrhagic lesions on the mucous membranes. © Cornell University.

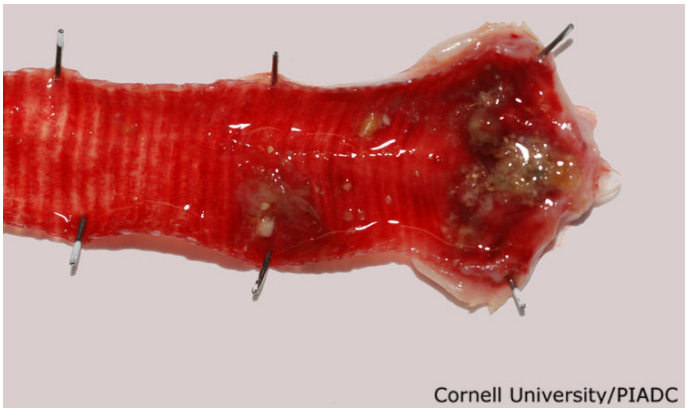

Figure 15: All signs on the trachea: severe acute catarrhal tracheitis with haemorrhage. © Cornell University.

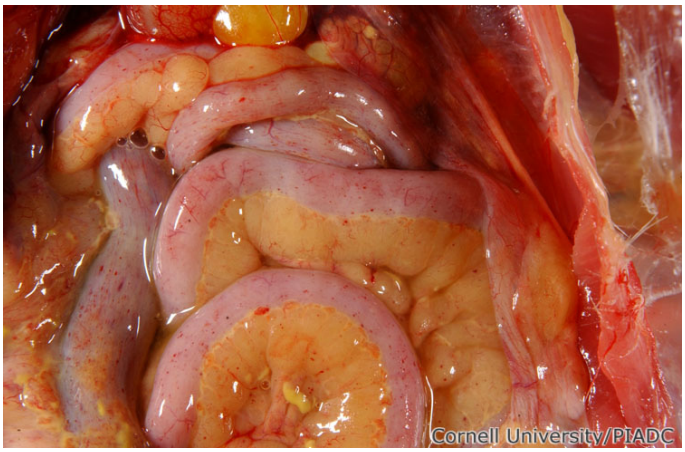

Figure 16: HPAI signs in coelomic cavity. Moderate acute multifocal petechiae; fibrinous coelomitis (presumed secondary to chronic egg-yolk peritonitis). © Cornell University.

Figure 17: HPAI signs in subcutaneous tissue (keel): moderate acute diffuse oedema with multifocal hemorrhage. © Cornell University.

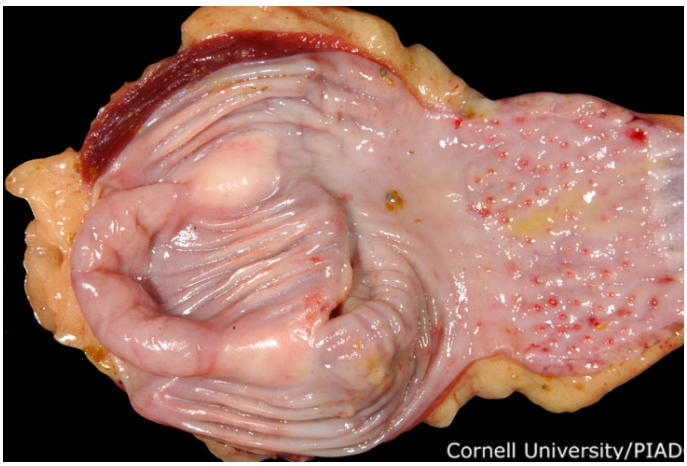

Figure 18: HPAI signs in the proventriculus (mucosa): moderate multifocal acute hemorrhage. © Cornell University.

Figure 19: HPAI signs in the ovary: acute congestion and hemorrhage with follicular atresia. © Cornell University.

Newcastle disease (ND)

CAUSAL AGENT AND DISTRIBUTION

ND is one of the most important poultry diseases worldwide and is an OIE-listed disease. The causal agent is a member of the family *Paramyxoviridae* in the viral genus *Avulavirus*. There are 10 serotypes of avian paramyxoviruses designated APMV-1 to APMV-10, and Newcastle disease virus (NDV) has been designated APMV-1. (Anon., 2009)

NDV has also been categorised into five pathotypes based on clinical signs in infected chickens. These are designated viscerotropic velogenic, neurotropic velogenic, mesogenic, lentogenic or respiratory, and subclinical enteric. However, pathotype groupings are rarely clear-cut. (Anon., 2009)

Velogenic NDV is endemic in areas of Mexico, Central and South America, widespread in Asia, the Middle East and Africa, and has been found in wild double-crested cormorants in the USA and Canada. Lentogenic strains of NDV are distributed worldwide. Mesogenic pathotypes are widespread but appear to be specially adapted to pigeons and do not readily infect other poultry. (Anon., 2009)

ND is spread by direct contact with the droppings or respiratory discharges of infected birds. The virus can live for a long time in the environment and can be spread by fomites such as shoes, clothing and equipment contaminated by infected birds.

New Zealand has never had an outbreak of ND. Serological surveillance indicates that APMV-1 viruses are widespread in many species of wild birds and are periodically present in commercial poultry. Pathogenicity testing and fusion protein genotyping of the 17 APMV-1 viruses isolated in this country have confirmed that all isolates are avirulent. (Pharo *et al.*, 2000)

CLINICAL SIGNS

The clinical signs vary with the pathogenicity of the isolate and the species of bird. In chickens, lentogenic strains usually cause subclinical infections or mild respiratory disease with coughing, gasping, sneezing and rales. Mesogenic strains can cause acute respiratory disease and neurological signs in some chickens, but the mortality rate is usually low. Lentogenic or mesogenic strains can produce more severe symptoms if the flock is co-infected with other pathogens. (Spickler, 2008)

Velogenic strains cause severe, often fatal disease in chickens. The clinical signs are highly variable. Most birds are lethargic and inappetent, and the feathers may be ruffled (**Figure 20**). Conjunctival reddening and oedema may be an early sign. Some birds develop watery, greenish or white diarrhoea, respiratory signs (including cyanosis) or swelling of the tissues of the head and neck (**Figure 21**). Neurologic signs including tremors, clonic spasms, paresis or paralysis of the wings and/or legs; torticollis (twisted neck) and circling may also be seen. Nervous signs can occur concurrently with other signs but are generally seen later in the course of disease. Egg-laying often declines dramatically and eggs may be misshapen, abnormally coloured and rough or thin-shelled, with watery albumen. Sudden death, with few or clinical signs, is also common. Birds that survive for two weeks usually live but may have permanent neurological damage and/or a permanent decrease in egg production. The clinical signs may be less severe in vaccinated birds. (Spickler, 2008)

Gross lesions are usually only seen with viscerotropic velogenic Newcastle disease. Petechiae may be seen on the serous membranes. Haemorrhages of the proventricular mucosa (**Figure 22**) and intestinal serosa are accompanied by multifocal necrotic haemorrhagic areas on the mucosal surface of the intestine, especially at lymphoid foci such as caecal tonsils. (**Figure 23**)

Figure 20: Newcastle disease. The bird is exhibiting mild depression with a reluctance to stand or move – typical early signs of this infection. There are ruffled feathers on the dorsal neck, consistent with fever. © Cornell University.

Figure 21: Newcastle disease. This image was taken three days after experimental inoculation with viscerotropic velogenic ND virus. The head has a square appearance (best seen over the eyes), which is due to moderate bilateral facial oedema. Oedema is often best appreciated by evaluating the face head-on, as in this view. © Cornell University.

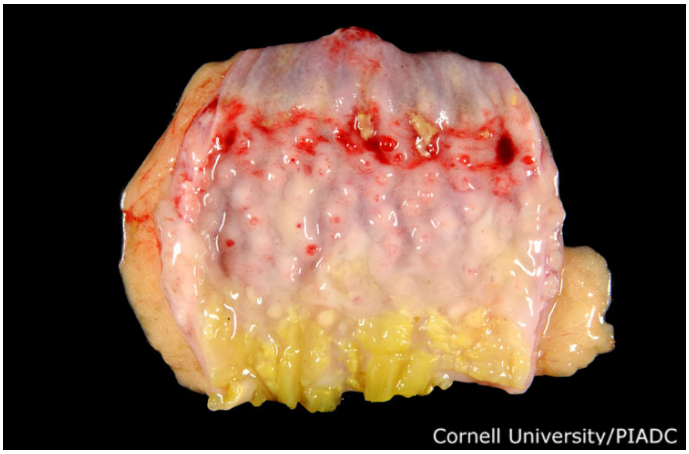

Figure 22: Newcastle disease. This image was taken five days after experimental inoculation with viscerotropic velogenic ND virus. There is hemorrhagic proventriculitis. Often, these hemorrhagic lesions cluster around the oesophageal/proventricular junction, as shown here. © Cornell University.

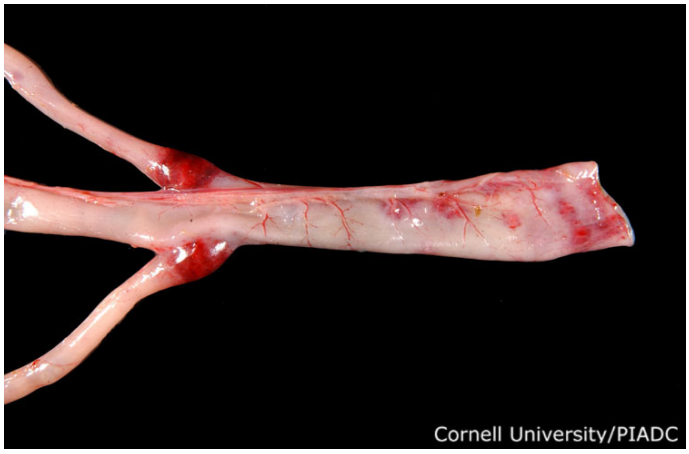

Figure 23: Newcastle disease. This image was taken five days after experimental inoculation with viscerotropic velogenic ND virus. As seen through the serosa, there are severe haemorrhages on the caecal tonsils and the mucosa of the rectum. © Cornell University.

ZOONOTIC POTENTIAL

Newcastle disease infection in people is rare and usually very mild. People in direct contact with infected poultry or other birds can get conjunctivitis (swelling and reddening of the tissues around the eyes). Poultry crews and laboratory workers are at the greatest risk of potential exposure. Handling or eating poultry products does not appear to be a risk. (Spickler, 2008)

When working with birds or poultry, especially when they are diseased, workers should wear protective clothing including gloves and safety glasses. Wash hands after contact with birds or poultry and do not touch your eyes until after handwashing. People working with the virus in laboratories, or in vaccination crews, should take extra precautions.

TREATMENT AND PREVENTION

Biosecurity measures such as cleaning and disinfection of facilities and equipment are very important. New introductions or birds returning to the farm should be isolated for several weeks before being placed into the flock.

Outbreaks are eradicated by quarantine and movement controls, depopulation of all infected and exposed birds, and thorough cleaning and disinfection of the premises.

The differential diagnoses are other causes of septicaemia; enteritis, respiratory disease and/or neurologic signs.

These could include (Spickler, 2008):

- fowl cholera;
- highly pathogenic avian influenza;
- infectious laryngotracheitis;
- the diphtheritic form of fowl pox;
- psittacosis;
- mycoplasmosis;
- infectious bronchitis;
- aspergillosis;
- management problems such as deprivation of water or feed, or poor ventilation;
- salmonellosis;
- adenovirus;
- nutritional deficiencies; and
- other paramyxovirus infections.

TABLE 1: SUMMARY OF EXOTIC DISEASES OF POULTRY

CLINICAL SIGNS	HIGHLY PATHOGENIC AVIAN INFLUENZA*	NEWCASTLE DISEASE#	INFECTIOUS BURSAL DISEASE	MAREKS DISEASE^$
Sudden death	Yes	Yes	–	–
Respiratory signs	Coughing, sneezing	Lentigenic: mild, coughing, gasping sneezing, rales	–	–
Diarrhoea	Green to white	Watery, greenish white	Watery	–
Lymphadenopathy	–	–	–	Neoplasia
Depression	Yes	Yes	–	Yes
Ruffled feathers	Yes	Yes	–	–
Oral and nasal discharges	Blood-tinged	–	–	–
Head/comb/wattle	Cyanosis of head, comb and wattle; oedema of head	Cyanosis	–	–
Sinusitis	Yes	–	–	–
Conjunctivitis	Yes	Yes	–	–
Neurological signs	Can be present	Velogeni: tremors, clonic spasms, paresis/paralysis of wings or legs, torticollis, circling	Prostration, inco-ordination	Asymmetric paralysis
Egg changes	Reduced egg production, loss of egg pigmentation, deformed or shell-less eggs	–	–	–
Musculoskeletal	Ecchymoses on the shanks and feet	–	–	–

* Clinical signs may be minimal in ducks and geese.

Lentogenic strains are least virulent; velogenic most virulent and divided into neurotropic and viscerotropic.

^ Virulent strains of Marek's disease.

	FOWL TYPHOID AND PULLORUM DISEASE	DUCK VIRUS HEPATITIS	DUCK VIRAL ENTERITIS	TURKEY RHINOTRACHEITIS	AVIAN SPIROCHAETOSIS
	Dead and dying chicks after hatching	Yes; rapidly after onset of clinical signs	Yes: 1–5 days after clinical signs	–	–
	Laboured respirations/ gasping	–	–	Sneezing, snicking, trachea gasping	–
	Yes, possibly white and viscous	–	Watery/bloody diarrhoea	–	Green or yellow diarrhoea with increased urates
	–	–	–	–	–
	Yes	Yes	Yes	Yes	Yes
	Yes	–	–	Yes	Possible shivering
	–	–	Nasal discharge	Yes: serous initially, later mucopurulent	–
	Pale, shrunken combs	–	–	–	–
	–	–	–	Yes	–
	–	–	–	Yes	–
	Blindness	Ataxia, opsithotonos	–	Chickens: cerebral disorientation, torticollis, opisthotonus	–
	–	–	–	–	–
	Older birds:lameness/joint swelling	–	–	–	–

TABLE 1: SUMMARY OF EXOTIC DISEASES OF POULTRY (*CONTINUED*)

POST-MORTEM SIGNS	HIGHLY PATHOGENIC AVIAN INFLUENZA*	NEWCASTLE DISEASE#	INFECTIOUS BURSAL DISEASE	MAREKS DISEASE^$
Respiratory system	Lung congestion/haemorrhages	Pulmonary oedema, haemorrhages of caudal pharynx and tracheal mucosa	–	Neoplasia
Nerve pathology	–	–	–	Enlarged peripheral nerves, especially brachial and sciatic plexuses, coeliac plexus, abdominal vagus and intercostal nerves
Lymphoid pathology	–	–	Cloaca bursa swollen, oedmatous, yellowish, occasionally haemorrhagic	Neoplasia
Reproductive organs	Petechiation in ovaries	–	–	Neoplasia
Integumentary	Subcutaneous oedema of head and neck	Subcutaneous oedema of head and neck	–	Firm lesions in feather follicles
Musculo-skeletal	Petechiation in muscles	–	Haemorrhage in pectoral, thigh and leg muscles	–
Alimentary system	Petechiae in abdominal fat, serosal surfaces, peritoneum, gizzard, intestinal mucosa	Haemorrhages proventriculus, cecal tonsils, lymphoid tissues of intestinal wall	–	Hepatic neoplasia
Sensory organs	–	–	–	Iridiocyclitis causes distorted pupil
Circulatory system	–	–	–	Neoplasia of heart
Renal	–	–	–	–
Other	–	–	–	–

* Clinical signs may be minimal in ducks and geese.

Lentogenic strains are least virulent; velogenic most virulent and divided into neurotropic and viscerotropic.

^ Virulent strains of Marek's disease.

	FOWL TYPHOID AND PULLORUM DISEASE	DUCK VIRUS HEPATITIS	DUCK VIRAL ENTERITIS	TURKEY RHINOTRACHEITIS	AVIAN SPIROCHAETOSIS
	Congested lungs, white necrotic foci	–	–	Rhinitis, tracheitis, sinusitis, airsacculitis, pneumonia	–
	–	–	–	–	–
	–	Spleen enlargement	Thymic lobes may be petechiated, spleen may be smaller in size and darkened by congestion; cloacal bursa severely congested or haemorrhagic	Splenomegaly	Spleen enlarged and mottled, petechial or ecchymotic haemorrhages
	–	–	–	Egg peritonitis, ovary/oviduct regression, misshapen eggs	–
	–	–	–	–	–
	Swollen joints containing viscous, creamy fluid	–	–	–	–
	Caecum enlarged, containing cheesy materials. Catarrhal enteritis, slimy intestinal contents, white necrotic foci in liver	Hepatic petechial and ecchymotic haemorrhages	Haemorrhagic annular bands in intestines; petechiae and ecchymotic haemorrhages on liver	Hepatomegaly, perihepatitis	Green catarrhal enteriti;, liver may be swollen
	Exudates in anterior chamber of eye	–	–	–	–
	Heart white necrotic foci, in acute cases fibrinous pericarditis	–	Petechiae and ecchymotic haemorrhages on heart	Pericarditis	–
	–	Swelling of kidneys, congestion of renal blood vessels	–	–	Kidneys enlarged and pale
	Young birds: unabsorbed yolk sacs, peritonitis	–	–	–	–

TABLE 1: SUMMARY OF EXOTIC DISEASES OF POULTRY (*CONTINUED*)

GENERAL CHARACTERISTICS	HIGHLY PATHOGENIC AVIAN INFLUENZA*	NEWCASTLE DISEASE#	INFECTIOUS BURSAL DISEASE	MAREKS DISEASE^$
Incubation period	1–7 days	2–15 days	3–4 days	–
Age groups affected	All	All	Usually 3–6 weeks; possibly up to 16 weeks	3 weeks and older
Mortality rate	High	High (velogenic); low in lentogenic/mesogenic	20–30%	Classical form 10–15%; up to 80% in acute outbreak of virulent MD
Morbidity rate	High	High (velogenic)	High	Usually 10–30% but can be up to 70%
Transmission	Horizontal	–	–	–
Zoonosis	Yes	Yes (mild disease)	–	–
Highly contagious	Yes	Yes	Yes	Yes
Species affected	Chicken: severe; ducks: subclinical	Chickens highly susceptible; turkeys less; varies with ducks and geese	Chickens	Chickens; quail and turkeys can be naturally infected

* Clinical signs may be minimal in ducks and geese.

Lentogenic strains are least virulent; velogenic most virulent and divided into neurotropic and viscerotropic.

^ Virulent strains of Marek's disease.

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	FOWL TYPHOID AND PULLORUM DISEASE	DUCK VIRUS HEPATITIS	DUCK VIRAL ENTERITIS	TURKEY RHINOTRACHEITIS	AVIAN SPIROCHAETOSIS
	4–6 days	18–48 hours	3–7 days	3–7 days	-12 days
	All	6 weeks and younger	7 days (adult)	All (young poults more severely)	All
	Can be as high as 100%	High: up to 95%	-100%)	1–30%	33–77%
	–	No information	–	Up to 100%	Up to 100%
	Horizontal and vertical	–	Horizontal	Horizontal	Vector-borne
	–	–		–	–
	Yes	Yes	Yes	Yes	Yes
	Chickens, turkey, quail, guinea fowl, pheasants, pea fowl	Experimentally in goslings, turkey poults, young pheasants, quail, guinea fowl	Ducks, geese, swans	Chickens, turkeys, pheasants, Muscovy ducks, guinea fowl	All poultry species

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QUARTERLY REPORT OF DIAGNOSTIC CASES: OCTOBER TO DECEMBER 2013

New Zealand Veterinary Pathology

CATTLE

A cow in the Waikato presented in acute recumbence. Blood chemistry demonstrated severe hypocalcaemia, with a calcium level of 0.94 mmol/L (reference range 2.00–2.64). Clinically milk fever was not suspected. Further investigation revealed that the cow had been grazing sorrel (*Rumex* sp.), which contains oxalates. These findings are consistent with **acute oxalate poisoning**.

In Hawke's Bay three yearling bulls lost condition and were coughing. Faecal specimens from each were submitted for parasitology and *Dictyocaulus* sp. larvae were observed in two. The clinical diagnosis of **lungworm infections** was supported by the laboratory findings.

In the Waikato, 12 five-month-old Friesian heifer calves in a mob of 70 became acutely ill and died in less than eight hours despite antibacterial therapy. Post-mortem examination findings included laryneal oedema and hyperaemia, enlarged mediastinal lymph nodes, and fibrinous pericarditis, pleuritis or peritonitis. Histopathology revealed that the affected serosal surfaces were covered by serofibrinous and neutrophilic exudate populated by many colonies of small bacteria. There were bacterial emboli and sometimes fibrin thrombi in high endothelial venules of the lymph nodes, hepatic sinusoids and renal capillaries. Pure growths of *Pasteurella multocida* were readily obtained from pleural fluid, lung tissue and mediastinal lymph nodes. A diagnosis of ***Pasteurella multocida*** **septicaemia** was made. Specimens were forwarded to MPI's Investigation and Diagnostic Centre at Wallaceville for typing. Since 2008 septicaemia in calves caused by *P. multocida* serotype B has been recognised as affecting calves less than eight months of age in New Zealand (McFadden *et al*., 2011).

An adult water buffalo (*Bubalus bubalis*) was found dead on a property in the Auckland supercity. A post-mortem examination was conducted and tissue specimens were submitted for examination. Histopathology found lymphoblastic vasculitis in the brain, retina, lung, kidney, liver, spleen, heart, tongue and diaphragm. Systemic lymphoblastic vasculitis is characteristic of **malignant catarrhal fever** caused by **ovine herpesvirus 2** infection.

A herd of dairy cows in the Waikato experienced cases of mastitis that did not respond to therapy with antibiotics. Milk samples from nine cows were submitted for culture and *Prototheca* sp. was obtained from six, giving a diagnosis of **protothecal mastitis**. *Prototheca* spp. are algae that are widespread in the environment. Occasionally they can be an opportunistic pathogen, and typically causing a small outbreak of mastitis that can become chronic.

Four two-year-old dairy heifers in the Waikato suffered spontaneous fracture of the humerus. Copper deficiency was suspected and serum samples from 11 herdmates were submitted for analysis. The mean serum copper concentration was 7.4 µmol/L (range 8.0–20.0), confirming **copper deficiency**. Copper is a component of the enzyme lysyl oxidase, which is necessary for the cross-linking of collagen precursors that stabilise collagen fibrils. Consequently copper deficiency during skeletal growth results in weak bones that are susceptible to pathological fracture.

A mob of four-month-old dairy calves in the Bay of Plenty presented with crusty scabs on the dorsal surface of the nose and trunk. A specimen of crust was submitted for Gram stain and KOH examination. The Gram stain demonstrated the "railway track" double chains of cocci characteristic of ***Dermatophilus congolensis***. The history and findings are typical of **dermatophilosis**.

In the Waikato, three adult dairy cows presented with acute fever and foul-smelling, bloody diarrhoea that contained sloughed intestinal mucosa. The attending veterinarian suspected **salmonellosis** and submitted a faecal sample. Culture yielded ***Salmonella*** **Typhimurium,** which confirmed the clinical diagnosis.

SHEEP

At slaughter an adult ewe in the Bay of Plenty was found to have a 200-mm-long tubular thickening of the small intestine. Histopathology demonstrated that the serosa was expanded by tubules and cords of neoplastic epithelial cells embedded in desmoplastic fibrous tissue. **Small intestinal carcinoma** was diagnosed. This is an occasional finding in adult sheep in New Zealand.

In Hawke's Bay, some lambs collapsed and died after a long muster in hot conditions. Post-mortem examinations were carried out on two lambs and fixed tissues were submitted. Both lambs had **intestinal coccidiosis** and there was **embolic hepatitis** as a consequence of the damaged intestinal epithelium.

GOAT

Two adult goats in the Auckland supercity were developing recurrent lymph node abscesses. Caseous lymphadenitis was suspected by the attending veterinarian and swabs of an affected lymph node were taken from each goat. Aerobic culture yielded ***Corynebacterium pseudotuberculosis***, confirming the clinical diagnosis of **caseous lymphadenitis**.

Five out of eight 2-year-old does on a small holding in the Waikato developed ill-thrift and three died. A post-mortem examination was carried out and serum was submitted from the three remaining animals. Histopathology revealed granulomatous lymphadenitis in the mesenteric lymph node, with intracellular acid-fast bacilli characteristic of **Johne's disease**. Serology for caprine arthritis and encephalitis (CAE) and Johne's disease antibodies gave positive results. Johne's disease and CAE are production-limiting chronic infections in dairy goats.

ALPACA

An adult male alpaca developed diarrhoea en route to a farm in the Auckland supercity and was treated for coccidiosis. On arrival it was still lethargic so the attending veterinarian requested CBC and biochemistry, which demonstrated **hypoproteinaemia**. On the recommendation of the pathologist, a further faecal specimen was submitted and numerous coccidian oocysts were found. The oocysts were unusually large (85 x 65 µm) and dark brown, and on the basis of their morphology were identified as ***Eimeria macusaniensis***, an uncommon finding. *E. macusaniensis* oocysts do not normally float on saturated salt solution and flotation on saturated sugar solution should be requested to reliably identify them. In this case only 5 percent of the *E. macusaniensis* oocysts had floated on saturated salt solution.

EQUINE

A six-year-old Thoroughbred horse in the Waikato developed a mass on the neck. A 35 x 20 mm diamond-shaped skin specimen containing multiple small masses was excised and submitted to the laboratory. Microscopic examination revealed a multifocal pyogranulomatous inflammation centred on bacterial club colonies in the subcutis. The club colonies comprised clusters of large cocci resembling *Staphylococcus* sp. that were embedded in Splendore-Hoeppli material. These findings are consistent with a diagnosis of **botryomycosis caused by *Staphylococcus* sp.** Botryomycosis is a descriptive diagnosis for lesions characterised by bacterial club colonies surrounded by pyogranulomatous inflammation. Botryomycosis can also be caused by *Actinobacillus*, *Actinomycosis* and *Nocardia* species.

A nine-year-old Thoroughbred mare in the Waikato presented with bilateral mucopurulent nasal discharge. A swab of the exudate was submitted for culture and a heavy growth of ***Streptococcus equi* ssp. *equi*** was obtained, confirming the clinical suspicion of **strangles**.

A hind-limb tendon sheath of an eight-year-old Warmblood gelding in the Waikato was swollen for two months without causing lameness. Corticosteroid was injected into the swollen tendon sheath but six days later the gelding was markedly lame and the leg was hot to touch. Cytological examination of an exudate sample from the tendon sheath revealed abundant neutrophils consistent with a diagnosis of **suppurative tenosynovitis**. A heavy growth of ***Serratia* sp.** was obtained in aerobic culture. *Serratia* spp. are ubiquitous enterobacteria that sometimes become opportunistic pathogens.

MARSUPIAL

A 14-year-old red-necked wallaby (*Macropus rufogriseus*) from a zoo in the Auckland supercity developed abnormalities of the mandible that were diagnosed as osteomyelitis. Biopsy specimens were submitted for histopathology and demonstrated that **squamous cell carcinoma** had invaded the bone of the mandible and was accompanied by **necrotising bacterial osteomyelitis**.

MONKEY

A 15-year-old cotton-top tamarin (*Sanguinus oedipus*) in an Auckland zoo presented with dry, scurfy skin. Whole blood and serum were submitted for CBC and biochemistry as part of a health check and to screen for diabetes mellitus. A skin biopsy specimen was also submitted for histopathology. The CBC results showed severe lymphocytosis characterised by small to medium-sized lymphocytes. This finding is consistent with lymphoproliferative disease and suggestive of chronic lymphocytic leukaemia. Histopathology revealed sheets of neoplastic small to medium sized lymphocytes effacing

the dermis. Together these findings gave a diagnosis of **non-epitheliotrophic lymphosarcoma with chronic lymphocytic leukaemia**.

REPTILE

Three captive adult tuatara (*Sphenodon punctatus*) in an Auckland zoo developed pesudomembranous skin lesions. Histopathology demonstrated that the skin was covered by a thick serocellular crust populated by branching fungal hyphae, consistent with a diagnosis of **dermatomycosis**. The lesions were cultured and ***Chrysosporium* sp.** was isolated. Recently it has been demonstrated that the fungus affecting tuataras in New Zealand is a unique *Chrysosporium* sp. (Sigler *et al*., 2013).

Gribbles Veterinary Pathology

BOVINE

A group of 30 two-year-old Jersey heifers from the Rodney district presented for veterinary attention because of coughing and ill-thrift. Gross postmortem of one heifer showed multiple abscesses and consolidated areas of lung, combined with large numbers of lungworm nematodes in the airways. Histopathology of lung samples showed severe chronic active eosinophilic bronchiolitis with intraluminal lungworm larvae and eggs, bronchiolitis obliterans, alveolitis and interstitial fibrosis consistent with chronic **dictyocauliasis**.

A nine-year-old dairy cow from the Waikato was anorexic and emaciated. Haematology revealed hyperfibrinogenaemia (9 g/L; reference range 2–7). Serum chemistry showed marked hypochloraemia (60 mmol/L; reference range 97–111), as well as minor changes in total protein (56 g/L; reference range 57–75), calcium (1.96 mmol/L; reference range 2–2.6), beta-hydroxy butyrate (1 mmol/L; reference range 0–0.8) and AST (224 IU/L; reference range 62–206). A Ziehl-Neelsen stain of faeces showed clumps of acid-fast organisms, and a Johne's serum antibody ELISA was positive. An abomasal displacement was suspected, with concurrent **Johne's disease**.

In a mob of 90 seven-month-old weaner Jersey heifers on a Manawatu farm, nine had died after developing respiratory disease. At postmortem the lungs were diffusely reddened and firm. Histopathology revealed diffuse expansion of alveoli and bronchi by large numbers of neutrophils mixed with fibrin and phagocytic alveolar macrophages. In addition, in a few alveoli there were large numbers of syncytial cells. Culture of exudate from the lungs yielded a heavy growth of *Trueperella pyogenes*. This confirmed a dual infection of **bovine respiratory syncytial virus and *Trueperella pyogenes*** infection.

A one-year-old Angus bull from Rangitikei presented in poor condition, with diarrhoea and failing to thrive. A five-day course of antibiotics did not result in any clinical improvement. A serum sample was tested by antigen ELISA for **bovine viral diarrhoea virus**. This revealed a high positive result consistent with a diagnosis of **mucosal disease**.

An 11-year-old Friesian dairy cow from Hawke's Bay had a history of chronic diarrhoea and weight loss. A serum sample tested positive by antigen ELISA for *Mycobacterium paratuberculosis*, confirming a diagnosis of **Johne's disease**.

A nine-month-old Friesian heifer from a central North Island property was examined because of bilateral nasal discharge and oesophageal erosion. A serum sample tested negative by antigen ELISA for bovine viral diarrhoea virus. After the heifer died, samples of lung and oesophagus were collected. Histopathology revealed fibrinoid necrosis and vasculitis consistent with a diagnosis of **malignant catarrhal fever** caused by infection with **ovine herpesvirus 2**.

A mature Friesian milking cow from a Bay of Plenty dairy farm was noticed to be producing less milk. It then developed watery diarrhoea containing pieces of intestinal mucosa. Culture of a faecal sample isolated ***Salmonella* Typhimurium phage type 135**, confirming a diagnosis of **salmonellosis**. In another case, a seven-year-old Freisian cow from the Waikato developed severe diarrhoea. In this case ***Salmonella* Typhimurium phage type 9** was isolated on culture of a faecal sample.

On a Waikato dairy farm, three 5-month-old Jersey x Friesian heifers had died, and eight were showing clinical signs of abdominal pain, from a herd of 120. Examination of the affected heifers revealed they had increased rectal temperatures (39.5°C; normal 38.5). Post-mortem examination of two of the dead heifers revealed excess fibrin in the peritoneal cavity. Culture of peritoneal fluid isolated ***Pasteurella multocida*,** confirming a diagnosis of **pasteurellosis.**

A six-week-old crossbred dairy calf from the Rotorua district was found dead and another one was champing its jaws aimlessly and appeared blind. Whole-blood lead concentration in the live calf was 1.07 mg/L (toxic level > 0.03 mg/L) and the kidney lead concentration of the dead calf was > 30 mg/kg (toxic concentration > 5 mg/kg). Lead fragments were also seen in the rumen of the dead calf. These findings confirmed a diagnosis of **lead toxicity**.

Skin samples were submitted from a Friesian cow from the Wairarapa and a Jersey cow from Taranaki, both with clinically suspected bovine digital dermatitis lesions. Histology revealed marked epidermal hyperplasia, ballooning degeneration and erosion in both samples. Large numbers of elongated, silver-positive bacteria consistent with **spirochaetes** were observed within the affected stratum corneum and stratum spinosum, supporting a diagnosis of **bovine digital dermatitis**.

A lifestyle farmer in Southland sought veterinary attention after seven of 18 bobby calves died suddenly over a two-week period. The calves had been sourced from local dairy farms and were being reared on 3 L of milk daily plus *ad libitum* pasture. The attending clinician noted inflammation and thickening of the rectal mucosa at post-mortem examination of one of the calves. Histological changes included severe necrosuppurative enteritis with crypt abscesses, large numbers of bacteria and large numbers of coccidial life stages. In addition, a heavy growth of ***Salmonella*** **Typhimurium phage type 160** was cultured from the colon. The deaths were attributed to **salmonellosis** complicated by **coccidiosis**.

Twelve animals in a mob of 56 yearling Angus cross heifers grazing on the West Coast developed diarrhoea and became debilitated. Seven died over a period of two weeks. The farmer reported inclement weather and a shortage of quality feed over the period in question. Gastrointestinal tract samples were obtained from two dead heifers and submitted for histopathology. In both cases there was severe multifocal fibrinosuppurative enteritis, particularly in the ileum, with large colonies of fine Gram-negative bacilli and extensive Peyer's patch depletion. In one heifer there was also suppurative inflammation targeting the muscularis mucosa of the abomasum and duodenum and associated with small Gram-positive rods. It was concluded that an outbreak of **yersiniosis** complicated by gastroenteric **listeriosis** was responsible for the clinical signs, with feed shortage and environmental stress considered likely predisposing factors.

Unusual tumours were diagnosed in two cattle from the Manawatu district. A subcutaneous mass the size of a rugby ball was found on the lateral right hind leg of an otherwise clinically well two-year-old Angus heifer. Histopathological examination of a punch biopsy revealed a benign neoplasm composed of sheets of adipose tissue interspersed with interweaving bundles of dense fibrous connective tissue, consistent with a **fibrolipoma.** This is a rare benign connective tissue tumour usually seen in young cattle, suggesting a congenital basis. Another mass was discovered growing from the dorsal vaginal wall of an adult dairy cow. It extended from the level of the distal cervix to the vulva, and was too infiltrative to completely excise. The mass was debulked and submitted for histopathology. Microscopically there were streams and herringbone arrangements of pleomorphic mesenchymal cells separated by collagenous matrix, consistent with a **fibrosarcoma.** Fibrosarcaomas are also not often diagnosed in cattle. They are locally aggressive but infrequently metastasise.

One of a mob of 30 dairy replacement heifers at pasture in the Taranaki region developed severe respiratory distress, while several others developed a cough and increased respiratory rate. The severely affected calf was euthanased and fixed samples from the caudo-dorsal lung lobes were submitted for histopathology. Microscopic examination showed large numbers of adult **nematodes** in the bronchi and many nematode larvae and eggs in the alveoli and bronchioles. There was also significant eosinophilic and granulomatous interstitial inflammation, bronchial mucosal hyperplasia and occasional alveolar type II pneumocyte hyperplasia. These changes were consistent with severe **pulmonary dictyocauliasis.**

Two unusual presentations of **actinobacillosis** were identified in cattle from Waikato and Taranaki. A six-year-old Friesian cow presented with extensive proliferative exudative malodorous dermatitis involving the skin between the udder and the medial thigh. Histopathological examination of a representative biopsy revealed typical dermal and subcutaneous club colonies formed by bacteria surrounded by Splendore-Hoeppli material. Inflammation and fibrosis surrounded and separated the colonies. In another case, at slaughter a three-year-old Friesian cross steer was found to have

extensive fibrosing lesions of the lung, pleura and pericardium. The presumptive gross diagnosis was cancer, but histopathology again revealed club colonies separated by extensive fibrosis. Actinobacillosis frequently presents as woody tongue, but infections involving the skin, draining lymph nodes, oesophageal groove and lower respiratory tract are occasionally reported.

A Wairarapa dairy farmer sought veterinary attention for a mob of 80 three-to-five-month Friesian heifer calves that were not growing as well as expected. Fifteen were judged to be in very poor body condition, 10 had various degrees of diarrhoea, and two had died overnight. The farmer had recently added more than the recommended dose rate of monensin to the water supply. Serum samples were taken from 12 calves and faeces from three. One calf was positive for BVD antigen and the others were negative. Faecal examination and culture were negative for *Salmonella, Yersinia* and coccidia. Post-mortem examination of one of the dead calves revealed about a litre of thin fluid in the pleural space, prominent pulmonary interlobular septae, and foam in the trachea. Histopathology on this calf indicated multifocal myocardial degeneration and necrosis, pulmonary oedema and hepatic centrilobular congestion. The other calf was grossly emaciated, with fluid intestinal contents. Microscopic lesions in this animal included subtle myocardial degeneration, necrotising enteritis with intralesional coccidial life stages, and a serous atrophy and saponification of adipose stores. **Monensin toxicity** was considered the cause of the myocardial lesions and death, while **coccidiosis** and **bovine viral diarrhoea** were likely to have contributed to the ill-thrift and diarrhoea.

A three-year-old Friesian dairy cow from mid-Canterbury had a diffusely and markedly swollen abomasal wall at postmortem. The swelling was due to a proliferation of off-white to cream-coloured tissue. **Lymphoma** was suspected and confirmed histologically. The animal was negative to enzootic bovine leucosis using the ELISA test.

A two-year-old mixed-breed bull from a Canterbury feedlot was euthanased with respiratory distress. At postmortem it had a large fleshy swelling in the larynx. Histologically the swelling had marked oedema, extensive perivascular neutrophil infiltration, thrombosis, and bacterial aggregates in and around vessels. The lesions suggested infection with ***Histophilus somni*** and culture of the lesion yielded a pure growth of this bacterium.

Practitioners are highly alert to **polioencephalomalacia** in dairy calves during the period from December to February each year and this season has been no exception, with cases diagnosed in calves during December throughout Canterbury. Most cases involved calves with obvious neurological disease but some cases were diagnosed in calves found dead.

A Canterbury farm had an outbreak of acute upper respiratory disease in 30 of 150 two-year-old beef cattle. Nasal swabs from five animals were pooled and tested positive by PCR for **infectious bovine rhinotracheitis** (IBR) virus. IBR infection had been diagnosed by paired serum neutralisation test serology on this farm several years previously.

Four yearling beef heifers were found dead on a Central Otago dairy farm. They had been grazing lush pasture of clover and ryegrass and were being supplemented with lucerne hay. Necropsy of one animal that had been dead for at least 12 hours revealed an expanded lung with prominent interlobular septa. Histopathologic examination of this lung showed changes suspicious of a diagnosis of **atypical interstitial pneumonia.**

On a Southland farm there were a number of deaths a few days after 50 yearling dairy heifers were grazed on pasture topdressed with a high-potassium superphosphate fertiliser two days before they were introduced to the paddock. The grass was reasonably long but no rain had fallen. Four affected animals were initially found ataxic and another was recumbent with nervous tremors. After they had been shifted to a new paddock, two more were found dead 24 hours later; and eight days later one of the heifers that had been initially observed as being ataxic was found dead. It was necropsied but no significant gross or histologic changes were seen. **Superphosphate toxicity** was suspected, based on the clinical signs and evidence of severe azotaemia in an affected animal, but this could not be confirmed.

In early December there was an outbreak of diarrhoea in 500 milking cows on a Southland dairy farm. The affected cows were on pasture and no supplements were being fed. Fifteen were noted to have foetid diarrhoea often with traces of blood. Their milk production was affected and two died. *Salmonella* cultures of faeces were all negative. Early treatment of the affected cows with broad-spectrum antibiotics produced a rapid cure in 24 hours and a return to normal milk production. One dead cow was

necropsied without revealing any significant findings, but it had been dead for 24 hours. There were a number of similar but smaller outbreaks of foetid diarrhoea in adult milking cows reported on other dairy farms in Southland but the causative agent (which was likely to be bacterial) was not identified.

On a Southland beef farm, a mob of 50 unweaned calves were noted to be losing weight and many had diarrhoea. They had been with a mob of 60 heifers that had been mixed with the adult cows in mid-gestation. This mob of heifers had also produced 10 stillborn or weak full-term calves that subsequently died earlier in the spring. A full range of tests on these dead calves failed to find a cause. Blood samples from 10 of the older affected calves showed no evidence of exposure to bovine viral diarrhoea virus (BVDV) via a pooled BVDV antibody ELISA. Faecal cultures from three affected calves showed no evidence of *Salmonella* or *Yersinia* so the cause of the stillbirths, weight loss and diarrhoea was not identified.

Twelve yearling beef heifers grazing on a small Southland farm were moved into an area of trees for 24 hours. Next day one was found dead, one was recumbent and another was ataxic and showing tremors. The recumbent animal was found dead next day in spite of supportive treatment. The owner suspected **arsenic toxicity** because among the trees there was an old concrete dip system and rubbish heap. This was confirmed by testing a whole-blood sample from the recumbent calf, which contained very high concentrations of arsenic: 1340 µg/L (normal < 10).

On a Southland dairy farm, a well-grown weaned heifer calf weighing 120 kg was found dull and ataxic. Other calves in the same paddock were unaffected. Clinical examination showed very pale, yellow mucous membranes and a high heart rate. Blood samples showed a severe haemolytic anaemia with large numbers of nucleated red cells and an increased bilirubin concentration of 264 µmol/L (normal range 0–13). *Theileria* PCR and *Leptospira* PCR on an EDTA sample were both negative and serum copper was not elevated so the cause of this severe haemolytic anaemia in this calf was not identified. No further affected calves were found in this mob.

One animal in a mob of 100 well-grown crossbreed heifers on Southland dairy farm was found in poor condition and depressed, with severe crusting lesions on the skin. The entire surface of the cornea of one eye was opaque but there was no evidence of an ocular or nasal discharge. Serum taken for a bovine viral diarrhoea virus antigen ELISA test was negative but a PCR on whole blood was positive for **malignant catarrhal fever** virus**.**

OVINE

A flock of sheep in Northland had a history of low lambing percentages over a number of years, and a particularly low percentage in 2013 combined with an increased percentage of wet-dry ewes (known pregnant ewes with no lambs at foot). The ewes had not been vaccinated for *Campylobacter* spp. or *Toxoplasma gondii*. The rams seemed healthy and tested free of brucellosis. Tissues from an aborted lamb submitted for histopathology showed bronchopneumonia with intralesional bacteria, placentitis with intralesional bacteria, multifocal hepatitis and splenitis consistent with bacterial abortion; but culture of stomach contents produced a mixed growth with no *Campylobacter* spp. Serology was carried out for titres to *Toxoplasma gondii, Leptospira* serovars Hardjo and Pomona, and *Campylobacter fetus* strains 134, 6/1, DL42 and 5915. There were significant titres to *Campylobacter fetus fetus* Strain 6/1 across all age groups, suggesting that despite the negative stomach culture result in one fetus, the abortions were probably due to **campylobacteriosis**.

Liver samples were collected from three 2-month-old Romney cross lambs that had been euthanased after they developed hindlimb ataxia on a Rangitikei farm. Liver copper concentrations were 61, 73 and 55 µmol/kg (adequate range 95–3000 µmol/kg), confirming a diagnosis of **copper-deficiency-induced enzootic ataxia**.

In the Bay of Plenty, proliferative crusting lesions developed on the ears of five-month-old lambs that had been vaccinated at docking with a modified live parapoxvirus vaccine. Dermatophilus was suspected as the cause, so fresh and fixed biopsy samples were collected. Culture of fresh material isolated a heavy growth of ***Staphylococcus aureus*** and ***Trueperella pyogenes***. Histopathology found evidence of ballooning degeneration of keratinocytes, with intracytoplasmic inclusions that confirmed a diagnosis of **parapoxvirus infection** (scabby mouth), along with secondary crusting and bacterial infection. Dermatophilus was excluded as a cause of the problem because the organism did not grow and was not visible in the lesions. The *Staphylococcus aureus* and *Trueperella pyogenes* infections of the

crusts were most likely opportunistic infections of the devitalised tissue.

A Canterbury sheep farm reported a 26 percent death rate in lambs aged under four weeks old. Post-mortem and histology findings from one affected lamb included multifocal embolic suppurative interstitial nephritis and multifocal necrosuppurative myocarditis. Gram-stained sections revealed myriad Gram-positive cocci associated with the lesions, suggestive of ***Staphylococcus aureus* infection**. *S. aureus* is a well-recognised cause of septicaemia in neonates and disseminated abscesses in two-to-four-week-old lambs (Dennis, 1966). It is also associated with ewe mastitis and dermatitis and is an occasional cause of ovine abortion. In lambs, it is thought that infection initially occurs via the umbilicus, with subsequent localisation to many organs (particularly heart, lung, liver and kidneys, but also brain and joints). Adults may carry the organism in the nasal cavity.

About 10 percent of lambs brought in for docking on a central North Island sheep station had proliferative, crusting lesions around the mouth and coronary bands. Samples from two lambs were submitted for histopathology. Both animals had severe, erosive and proliferative dermatitis with ballooning degeneration and intracytoplasmic inclusions, consistent with **contagious ecthyma** caused by **ovine parapoxvirus**. Lesions in affected lambs usually resolve after three to six weeks, by which time they have become non-infective. However, the virus lasts for long periods in the environment (15 years at room temperature) and is potentially zoonotic.

An unacceptably high number of one-month-old lambs on a central North Island sheep station were observed to be weak and daggy. The lambs were euthanased as part of a survey of causes of illness and mortality on the farm. Strongyle egg counts were 450 and 600 eggs per gram, with no *Nematodirus* eggs identified. Histological examination of the distal small intestine of one lamb revealed large numbers of intact and ruptured coccidial megaloschizonts associated with necrosis and inflammation. There were also many megaloschizonts in the subcapsular sinuses of a mesenteric lymph node. **Coccidiosis** was considered to be a contributor to the ill-thrift in the examined lamb.

A North Canterbury sheep farm had about 30 pre-tailing lambs with severe crusty skin lesions and weight loss. The lesions initially developed on the ears and face and then involved the entire body. None of the ewes were affected. **Dermatophilosis** was suspected and the disease was confirmed histologically and via direct examination of crust material.

Four 8-week-old lambs on a Southland sheep farm were found with hind-limb weakness of variable severity. One was unable to walk. Lambs with similar signs had been seen earlier and one had collapsed. Necropsy of the paralysed lamb showed pale skeletal muscles. Selenium concentration in the serum from three affected lambs varied from < 30 to 50 nmol/L (adequate serum concentration > 140 nmol/L), confirming **white muscle disease**.

A Southland sheep farm had a history of poor lamb survival and late abortions, particularly in two-tooth ewes during 2010. An investigation at that time had identified low serum iodine concentration in the two-tooths but not in the older ewes. There were no other significant findings. Since that time the farmer had treated all ewes with iodine and noticed an improvement in lamb survival but was still getting a significant number of small, occasionally "hairy" lambs that did not grow very well. This season there were large numbers of these small lambs among both the two-tooths and late-lambing adult ewe mobs. PCR tests on the serum from some of these affected lambs confirmed **hairy shaker disease** as the cause.

Large numbers of purple lice were found over the scrotum of a two-tooth ram from an Otago sheep farm. The skin over the whole scrotum was very thickened. The other 120 rams in the same mob were unaffected. The lice were identified as the host-specific sucking louse ***Linognathus pedalis***. Although commonly referred to as the "foot louse" of sheep (because it occurs chiefly on the legs and feet), it can also be found on the scrotum and belly. When present in significant numbers, these sucking lice can have a debilitating effect and cause anaemia.

Mature ewes became recumbent after being grazed on a new grass paddock in Otago that had not germinated well and was contaminated by weeds including yarr (***Spergula arvensis***) and fathen (***Chenopodium album***), both of which are known for their high oxalate content. Serum calcium varied from 0.9 to 1.5 mmol/L (normal range 2–3) in five affected ewes tested before intravenous calcium treatment, confirming a diagnosis of **hypocalcaemia** (milk fever). All the treated ewes recovered.

CANINE AND FELINE

A five-year-old American Bulldog from Auckland had a history of three days of anorexia and severe icterus. Serum chemistry abnormalities included severe azotaemia (urea 41.4 mmol/L; reference range 2.5–9), high creatinine (573 umol/L; reference range 48–109) and hyperphosphataemia (3.72 mmol/L; reference range 0.92–1.82). There were marked icterus and enzyme increases, with bilirubin 361.6 umol/L (reference range 1–3), ALP 1365 IU/L (reference range 0–87), ALT 546 IU/L (reference range 0–88), AST 301 IU/L (reference range 0–51), CPK 3562 IU/L (reference range 0–385), amylase 1375 IU/L (reference range 0–1074) and mild hypoalbuminaemia (29 g/L; reference range 33–44). Other tests revealed mild electrolyte decreases (sodium 140 mmol/L; reference range 141–153, and chloride 91 mmol/L; reference range 106–117). All considered together, these changes suggested severe renal or pre-renal disease combined with liver disease. Haematology showed mild thrombocytopenia (109 x 10^9/L; reference range 200–500 x 10^9) and leukocytosis (22.7 x 10^9; reference range 6–15 x 10^9), neutrophilia (21.8 x 10^9/L; reference range 3.6–11.5 x 10^9) and lymphopenia (0.5 x 10^9/L; reference range 0.2–1.5 x 10^9) consistent with inflammation or a stress leukogram. Microscopic agglutination tests for antibodies to the Pomona and Hardjo serovars of *Leptospira* spp. were negative; a MAT for *L copenhageni* was titrated to 1:50, consistent with prior exposure and perhaps early infection. A leptospirosis PCR conducted on the blood sample was positive, confirming a diagnosis of **leptospirosis**.

A 23-week-old Heading dog from Waikato had profuse, watery diarrhoea and inappetance and a 12-week-old Schipperke from Auckland had had intermittent diarrhoea ever since the owner first obtained it. Faecal antigen ELISA tests on both dogs were positive for *Giardia* spp., confirming diagnoses of **giardiasis.** ELISA tests were negative for *Cryptosporidium;* faecal parasitology in both dogs was negative for nematode eggs or coccidial oocysts; and faecal culture from the Heading dog was negative for *Campylobacter and Salmonella* spp.

A 13-week-old Staffordshire Bull Terrier from a rural area near Auckland had diarrhoea. Faecal parasitology was negative for nematode eggs or coccidial oocysts, and faecal culture was negative for *Campylobacter* or *Salmonella* spp. Faecal antigen ELISA tests were negative for *Giardia* but positive for *Cryptosporidium* spp., conferring a diagnosis of **cryptosporidiosis**.

A six-week-old crossbreed puppy from Auckland had diarrhoea. Faecal parasitology was positive for *Trichuris vulpis* eggs and faecal culture was positive for *Campylobacter* spp., indicating a combined infection with **trichuriasis** and **campylobacteriosis**.

A 10-month-old Domestic Shorthaired cat from Auckland had diarrhoea for three days and four-to-six-week-old Burmese kittens from a litter in Auckland were losing weight and feeding poorly. **Giardiasis** was diagnosed in both cases after faecal antigen ELISA tests were negative for *Cryptosporidium* but positive for *Giardia* spp. Faecal parasitology in both was negative for nematode eggs or coccidial oocysts, and faecal culture for *Salmonella* and *Campylobacter* spp. was negative in the kittens.

A five-month-old Hungarian Viszla dog from Taranaki had a history of chronic diarrhoea since the owners had acquired it. Sometimes there was also melena. The dog was clinically well though slightly underweight. A faecal ELISA test for ***Giardia duodenalis*** was positive, confirming **giardiasis**, and culture revealed the presence of ***Campylobacter upsaliensis/helvetica***. ELISA tests for *Cryptosporidium parvum* and faecal egg counts were negative. In another case, from Waikato, a 14-month-old Bearded Collie dog had a history of chronic diarrhoea that had been unresponsive to treatment, and *G. duodenalis* was confirmed in the faeces of this dog as well.

A two-year-old Border Collie dog from Invercargill rapidly developed a progressive hindlimb ataxia, pyrexia and pain over the dorsal upper spine. It had never had contact with farm animals. A full biochemistry screen and radiographs of the spine were unremarkable. Trauma was suspected but four days later the dog showed both hindlimb and forelimb ataxia. It also had developed a marked intention tremor and could not judge distance. In spite of all these problems it was still bright, alert, responsive and playful. A few days later it was euthanased because its condition had deteriorated further. Necropsy failed to find any significant gross findings, so brain and part of the mid spinal cord were fixed in 10 percent buffered formalin and sent to the laboratory. Histopathological examination of brain sections showed severe multifocal gliosis and neuronal necrosis of many of the cerebellar folia. There were similar changes in the spinal cord. In the affected areas there were many large protozoal cysts resembling those of ***Neospora caninum***.

A nine-year-old female Shorthaired cat from the New Plymouth region presented with two small masses in the subcutaneous tissue in the region of the fat pad of the ventral abdomen. The masses were described as about the size of a small fingernail. Fine needle aspirates revealed large numbers of very degenerate neutrophils and smaller numbers of large epithelioid macrophages. Numerous intracellular and extracellular non-staining spicular structures were seen. These were confirmed as ***Mycobacteria*** with a Ziehl-Neelsen stain.

An 11–year-old female Siamese cat in the Tararua district presented with sneezing for the past two weeks. It was bright, alert and responsive. There was a swollen bridge to its nose and a copious nasal discharge that did not respond to doxycycline. Smears from the nasal discharge revealed numerous very degenerate neutrophils, small numbers of large active macrophages and moderate numbers of yeastlike organisms with a dark central region and large clear capsule. Some of these organisms were budding. This gave a diagnosis of ***Cryptococcus***.

PORCINE

Three 6-month-old pigs died suddenly on a Wairarapa farm. One was seen to collapse after developing dyspnoea and soon died with white foam streaming from the nose. At postmortem the lungs were firm, and filled with foam and fluid in the airways. Histopathology confirmed acute pulmonary oedema associated with severe myocardial degeneration consistent with **mulberry heart disease** of pigs. This disease is thought to be associated with insufficient dietary vitamin E or selenium.

A six-year-old Kunekune pig held in a Waikato zoo developed a firm 150 mm diameter mass in the mammary gland. The entire mass was surgically excised and portions were submitted for histopathology. In the centre of the affected tissue were multiple club-colony proliferations of eosinophilic material surrounded by large numbers of neutrophils in an extensive region of necrosis. Gram-positive cocci were seen in the centre of the inflamed tissue, confirming a diagnosis of infectious **mastitis**.

POULTRY

A 10-month-old Silver Spangled chicken from the Rodney district was ill and pyrexic. **Coccidiosis** was identified when faecal parasitology revealed high to extremely high numbers of coccidial oocysts.

NON-POULTRY AVIAN

A male spotted dove from Auckland was lethargic and hanging around a feeder. It was captured for care but died soon after. Gross postmortem revealed yellow plaques on the right cranial thoracic air sac, swelling of the right cranial renal pole, and patchy pale areas on the liver. Histopathology of multiple tissues revealed multifocal necrotising hepatitis with intracellular bacteria, multifocal fibrinonecrotising splenitis with intracellular bacteria and multifocal lymphoplasmacytic serositis consistent with systemic infection. These signs were most likely due to ***Chlamydophila psittaci*** (**chlamydophilosis/psittacosis**).

CAMELID

A two-year-old female alpaca from a small herd in the greater Wellington region lost condition rapidly over a three-week period. During this time it was treated with oral combination and injectable anthelmintics, with no improvement. On veterinary examination, body condition score was judged to be very poor (1/5) and the alpaca was weak and ataxic. Pupillary light reflex was sluggish and there was no menace reflex. Treatment included IV fluids, trimethoprim sulpha, vitamin B1 and dextrose, but the alpaca died later the same day. Histopathological examination of the brain revealed laminar cortical rarefaction and neuronal necrosis, with prominent vascular endothelial cells and infiltration of the perivascular spaces and meninges by Gitter cells. There was also a necrosuppurative bronchopneumonia with plant material in the airways. A diagnosis of **polioencephalomalacia** with secondary aspiration pneumonia was made.

CERVINE

The owner of a deer farm in the central North Island reported the loss of about 30 mixed-age stags from a herd of 200 over a period of 12 months. The stags were apparently found dead without any prior clinical signs, and post-mortem examination failed to determine the cause of death. Multiple fixed tissues were submitted from one stag after veterinary attention was sought. Histopathological examination revealed lymphocytic and necrotising vasculitis in multiple organs, with the most severe lesions found in the brain, abomasum, kidney and lung. These findings were considered consistent with **malignant catarrhal fever** caused by **ovine herpesvirus-2**.

A mature stag on a Southland deer farm had been sedated with a xylazine sedative for de-velveting. It was found dead eight hours later with the thorax filled with straw-coloured fluid. Histopathologic examination of the lung showed congestion and interlobular and subpleural oedema. There were also occasional areas of haemorrhage associated with infiltrates of moderate numbers of eosinophils, and low numbers of plasma cells and lymphocytes around the arterioles surrounding bronchioles. These findings were consistent with an **acute hypersensitivity reaction** and were typical of a delayed reaction to **xylazine** reported in deer. Other deaths were also reported in xylazine-sedated stags on other deer farms in Southland during the velveting season. On one farm four deer were found dead and on another farm six deer died later. Similar gross and microscopic lesions typical of xylazine hypersensitivity were seen on necropsy.

RABBIT

A breeder of rabbits in Christchurch city had several unvaccinated rabbits die suddenly in late November. The tissues of one were examined histologically and the liver had typical lesions of **rabbit haemorrhagic disease**.

EQUINE

An adult horse from the Auckland region was treated for about a week with corticosteroids and antibiotics for apparent hives, then developed shivering, weakness, lethargy, mild limb oedema and pyrexia. Oral fluids and electrolytes were administered. Haematology showed mild anaemia (HCT 0.3 L/L; reference range 0.32–0.55), neutrophilia (7.9 x 10^9/L; reference range 3–7 x 10^9), lymphopenia (1.0 x 10^9/L; reference range 1.3–6.5 x 10^9), monocytosis (1.3 x 10^9/L; reference range 0–0.5 x 10^9) and hyperfibrinogenaemia (5 g/L; reference range 2–4). These results were consistent with inflammation. A repeat examination three days later revealed that the skin lesions now resembled ringworm, and all analytes were within reference ranges on a follow-up haematology panel. Culture of hair taken at that time produced a growth of *Trichophyton,* which was subsequently confirmed as ***Trichophyton equinum*** by a reference laboratory. A skin biopsy taken before the culture results were known revealed crusts and hair follicles containing acantholytic cells and neutrophils, as well as small fungal hyphae within superficial crusts. A final diagnosis of **pemphigus foliaceus-like dermatophytosis** was made; this has been occasionally reported in horses.

A two-day-old Standardbred filly from Auckland had a 20 x 20 cm mass on the left flank. At surgery this was found to be cavitated, containing blood or serum and it was attached to intercostal vessels. Histopathology revealed endothelium-lined elastic tissue with adjacent haemorrhage, consistent with a **vascular hamartoma**.

Veterinary attention was sought for a two-year-old filly in Southland. Reported clinical findings included very poor body condition, a "saw-horse" stance, pale mucous membranes and laboured breathing, progressing to recumbency. A blood sample taken just before euthanasia revealed significantly increased serum CK, indicating acute muscle damage; and neutrophilia, hyperglobulinaemia and hypoalbuminaemia, consistent with inflammation. Increased serum bile acids suggested hepatic dysfunction. There were 3500 strongyle eggs per gram of faeces and many red worms consistent with **cyathostomes** were observed in the colon. Histological examination revealed marked hepatopathy characterised by megalocytosis, hepatocellular degeneration and loss, portal hepatitis and biliary hyperplasia. These findings were considered consistent with **pyrrolizidine alkaloid toxicity** caused by ragwort ingestion. **Cyathostomiasis** was a likely contributing factor to the clinical deterioration, though sections of colon and intestine were not submitted for histological confirmation.

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QUARTERLY REPORT OF INVESTIGATIONS OF SUSPECTED EXOTIC DISEASES

Exotic disease investigations are managed and reported by MPI Investigation and Diagnostic Centre (IDC) and Response, Wallaceville. The following is a summary of investigations of suspected exotic disease during the period from October to December 2013.

Mycoplasmosis in juvenile yellow-eyed penguin ruled out

A veterinary pathologist reported on a dead juvenile female yellow-eyed penguin with respiratory signs and mucus-like material in the distal trachea. On post-mortem examination the penguin had a ventricular septal defect and possible fungal airsacculitis. Owing to the respiratory signs and recent reports of *Mycoplasma* spp. in penguins, mycoplasma infection was considered as a possible contributing factor. Tracheal samples were submitted to the MPI Animal Health Laboratory (AHL) for fungal, general bacterial and mycoplasma culture. Culture was negative for mycoplasma and fungal species. General bacterial culture isolated a *Corynebacterium* sp. that was most consistent with *C. falsenii*. This bacterium has been isolated from the upper respiratory tract of some bird species, and is thought to be a normal commensal species. In this case, *C. falsenii* is not thought to have been a primary pathogen.

Anthrax ruled out

A farmer called the MPI exotic pest and disease hotline to report sudden death in a rising-three-year-old Hereford bull that had been imported from Australia two months previously. The bull had been at pasture with cows, and was found recently dead with dark, bloodstained faeces. Differential diagnoses included endemic diseases such as haemorrhagic jejunitis (jejunal haemorrhage syndrome, definitive aetiology unknown, likely *Clostridium perfringens*), gastrointestinal haemorrhage and torsion. Exotic differential diagnoses were limited to anthrax, which has not been reported in New Zealand since 1954 (Gill, 1993). Given the period of time since importation, if anthrax were found in this bull it would have to have originated from New Zealand soil. Peripheral blood smears were taken and the carcass was deeply buried without being opened, in order to minimise environmental contamination. Blood smears were negative for *Bacillus anthracis* spores by Gram, Giemsa and polychrome methylene blue stains. The cause of death was considered to be endemic and the investigation was stood down.

Enterohaemorrhagic *E. coli* ruled out

A veterinarian reported a possible enterohaemorrhagic *Escherichia coli* infection in two age groups of calves. One group was aged from a few days to two weeks, which is within the usual age range for disease caused by *E. coli*, while the older group was aged about 3–4 weeks. There was high morbidity (over 90 percent) and low mortality, with some chronic scouring and depressed calves being euthanased. Rotavirus was circulating concurrently but testing on 10 sick calves was negative for *Coccidia*, *Salmonella* and coronavirus. Histopathology from two calves demonstrated acute severe fibrinonecrotic colitis. Of concern was an emerging enterohaemorrhagic presentation of *E. coli* infection, or the presence of O157:H7 *E.coli*, a serious human pathogen of which cattle can be reservoirs, though usually subclinically.

Faecal samples from five affected animals were taken for testing at the Animal Health Laboratory's Investigation and Diagnostic Centre (AHL IDC), Wallaceville. The outbreak had been running for 3–4 weeks by the time of notification and none of the older group of calves were affected at the time of sampling. *E. coli* was cultured from all five animals but latex agglutination testing was negative for serotype O157. *E. coli* verocytotoxin testing was subcontracted to establish whether a non-O157 serogroup verotoxigenic *E. coli* strain (VTEC) was present. Enterohaemorrhagic *E. coli* are VTEC strains with additional virulence factors that enable them to cause haemorrhagic colitis and haemolytic uraemic syndrome in humans. However, PCR tests for these toxins were negative for all samples. Most of the affected animals recovered uneventfully with antibiotics, and no human illness was reported from contact with the animals.

Enzootic bovine leucosis excluded

An export veterinarian called the MPI exotic pest and disease hotline to report a heifer with a suspicious pre-export result for enzootic bovine leucosis (EBL) virus. The heifer was part of a shipment of 4000 heifers bound for China. The initial test included ELISA testing of serum. The sample was sent to a confirmatory laboratory, which determined by ELISA and PCR tests that the heifer was negative for EBL. The rest of the heifers were shipped uneventfully. The investigation was stood down and the

dairy herd of New Zealand is still considered to be free of EBL.

Pulmonary amoebiasis confirmed

A veterinary pathologist rang the MPI exotic pest and disease hotline to report a potentially new disease of dairy cows in New Zealand, after a Taranaki veterinarian found an unusual pneumonia at post-mortem of a cow. Histopathology on samples of affected lung revealed that amoeba were associated with the lesions. Amoeba were present both throughout the parenchyma of the lung tissue and in the airspaces, thus excluding the possibility that this was due to the inhalation of rumen fluid. Pulmonary amoebiasis caused by *Acanthamoeba* sp. has been described in the USA and other countries but never been reported in New Zealand. Furthermore, *Acanthamoeba* sp. are linked to the spread of *Legionella* bacteria. At the IDC AHL (Wallaceville) *Acanthamoeba* sp. was successfully detected from the lung tissue by PCR. This is a ubiquitous organism and was a case of an opportunistic infection, so no further work was required and the investigation was stood down.

Unusual encephalitis investigated

A veterinary pathologist working at Massey rang the MPI exotic pest and disease hotline after suspecting that *Chlamydophila pecorum* was the cause of sporadic encephalomyelitis in a group of 3–5-month-old calves in the Manawatu. Over a period of four weeks, 40 out of a group of 150 calves presented with hindlimb ataxia, with knuckling over on the fetlocks, depression, recumbency, opisthotonous and blindness. A total of 13 had died or been euthanased. Three of these calves were referred to Massey University for neurological and post-mortem examination. Gross postmortem and histology revealed a polyserositis, leading to a suspicion of septicaemic pasteurellosis caused by *Pasteurella multocida* or *Histophilus somni*, but bacteriological culture failed to isolate these organisms. Subsequently a fourth calf was necropsied in the field and *Histophilus somni* was suspected based on the brain histology. The brains from the original calves were then examined and found to have histological lesions that were more suggestive of sporadic bovine encephalomyelitis caused by *Chlamydophila pecorum*. As a result, PCR tests were carried out at the MPI AHL, firstly on stored fresh lung and later on fixed brain tissue from the original three calves. All these tests were negative. However, fresh brain from a seventh similarly affected calf was positive on PCR for *Chlamydophila pecorum*. Histology was not performed on this brain. Over the course of the investigation, repeated bacteriology failed to isolate *Pasteurella* or *Histophilus* from these cases.

C. pecorum strains in cattle cause numerous diseases including sporadic bovine encephalomyelitis, polyarthritis, pneumonia, enteritis, vaginitis and endometritis. Sporadic bovine encephalomyelitis is most common in calves six to 12 months of age although younger calves and older cattle can be affected. Morbidity is highest in calves and mortality highest in adult cattle. Clinically affected cattle can exhibit neurological, respiratory and/or gastrointestinal signs. Neurological signs are characterised by depression and hindlimb ataxia and often affected cattle knuckle over on the fetlocks. Gross postmortem frequently reveals a polyserositis and meningeal congestion. Histologically there is meningoencephalitis and vasculitis with foci of neutrophilic encephalitis. Differential diagnoses include *Histophilus somni*, septicaemic pasteurellosis, polioencephalomalacia, lead toxicity, listeriosis and malignant catarrhal fever.

C. pecorum is not new to New Zealand and was first reported in January 2002 (Mackereth & Stanislawek, 2002) but does not appear to have been linked to disease in cattle. Having only one positive PCR test is a concern. The failure of PCR to detect this organism in the fixed brain tissue could be explained in that formalin fixation for more than two weeks has been shown to reduce the sensitivity of *Chlamydophila* PCR; this could result in false negatives (Ortega *et al.*, 2007). The affected calves were placed on Tetravet 100 soluble antibiotic powder in the milk for 10 days, after which there were no further cases.

Practitioners concerned about *C. pecorum* as a possible diagnosis should in the future submit fresh brain tissue along with other samples.

Brucellosis excluded

A member of the public rang the MPI exotic pest and disease hotline to report an apparent outbreak of fistulous withers in a pony herd at a polytechnic institute. Further investigation determined that about 10 horses were affected with skin lesions at the withers, although there was no discernible discharging tract associated with the lesions. Furthermore, these cases were not all in one mob of horses and earlier cases treated by the institute's

staff had responded to an iodine wash, suggesting a fungal aetiology. Blood samples tested negative by ELISA for *Brucella abortus* and *B. suis* antibodies at the AHL (Wallaceville). Histopathology on skin biopsies of lesions collected from affected horses at the time of the field visit showed a lymphocytic eosinophilic vesicular dermatitis with dermal oedema. No diagnosis was reached, but a contact irritant was suspected. As a result of exotic differentials being excluded the investigation was stood down.

EIA ruled out

A veterinary pathologist phoned MPI to report anaemia in a three-year-old Thoroughbred horse with a large hematoma caused by injury. Blood work indicated thrombocytopenia and anaemia. The horse was subsequently euthanased. An equine infectious anaemia (EIA) agar-gel immunodiffusion test was negative and EIA was ruled out.

EIA/EVA ruled out

A Gribbles veterinary pathologist contacted the MPI exotic pest and disease hotline to report suspected equine viral arteritis (EVA) and equine infectious anaemia (EIA) in a New Zealand-born nine-year-old Thoroughbred horse with borderline anaemia (HCT = 0.32; reference range 0.31–0.41) and oedema of distal limbs. All other horses on the property were in good health. This horse had competed on the equestrian circuit in New Zealand and internationally. Paired serum samples were submitted to the AHL and tested by serology for EVA (using VNT) and EIA (using AGID). Both tests yielded negative results.

A veterinary pathologist notified MPI of a one-year-old male sport horse with a five-week history of pyrexia and limb oedema, and an inflammatory leucogram. The horse had no history of travel outside of New Zealand. Differential diagnoses for equine subcutaneous oedema include exotic aetiologies, e.g. equine viral arteritis (EVA) virus and equine infectious anaemia (EIA) virus, and endemic conditions such as septicaemia caused by several bacterial pathogens (e.g. *Streptococcus equi*). AGID for EIA was negative, as was a VNT for EVA. Having ruled out the two major exotic differential aetiologies, the investigation was stood down.

Equine herpes virus 5 excluded

A pathologist called the MPI exotic pest and disease hotline to report an unusual interstitial fibrosis on lung necropsy and histopathology of a two-month-old Thoroughbred filly. Equine herpes virus 5 (EHV-5) was identified as a possible cause, and if confirmed would have been a new detection in New Zealand in such a young animal. Samples of fresh lung tissue were sent to Massey University for testing by PCR for EHV-5, 2, 1 and 4, and for virus isolation. Further bacterial culture by the referring lab yielded the common equine neonatal respiratory pathogen *Rhodococcus equi*. PCR testing was positive for EHV-2 (which in New Zealand is known to cause immunosuppression in equine neonates) and PCR was negative for EHV-1, 4 and 5. Virus isolation was also negative and the investigation was closed.

Histoplasmosis excluded

A veterinary pathologist called the MPI exotic pest and disease hotline to report cases of nodular pyogranulomatous dermatitis in two young dogs. Grossly the lesions resembled canine cutaneous histiocytomas. The dogs were unrelated and from separate households. The first case was a one-year-old male castrated Greyhound that had three well demarcated alopecic nodules on its head and face. The lesions were variably ulcerated and measured 1 cm in diameter. The second case occurred in a four-month-old male Australian Silky terrier. The lesion was a single well-demarcated ulcerated nodule near the right eye. Clinically, the nodules from both dogs resembled histiocytomas but in both cases cytology showed a pyogranulomatous inflammatory infiltrate with small numbers of fungal yeasts. The primary exotic differential diagnosis for the fungal yeasts was Histoplasma capsulatum (a notifiable organism). H. capsulatum is a soil-borne dimorphic fungus considered to be exotic to New Zealand. Fungal culture excluded H. capsulatum. Microsporum canis, an endemic dermatophyte and the primary aetiological agent of canine ringworm, was cultured from both dogs. Well-circumscribed nodular lesions such as these are an uncommon manifestation of ringworm infection in dogs.

Babesia gibsonii ruled out

A member of the MPI Animal Imports team phoned the MPI exotic pest and disease hotline to report a call from a person with a dog imported from Singapore and infested with ticks. The owners found two live ticks, which they removed but unfortunately destroyed. An investigation was opened and urgent measures undertaken to prevent

establishment, because there were no ticks available for identification, the dog had only been in the country for one month and the owner said that the ticks looked like those they used to find on the dog in Singapore.

The dog was placed back in quarantine, treated and inspected, with no further ticks found. The owner's vehicle and house were fumigated. The dog was negative for *Babesia gibsonii* on both PCR and ELISA tests as per the import health standard and the SOP for border tick detections. The investigation was closed after a final negative post-detection check for ticks six weeks later.

Infectious bursal disease excluded

A scientist with the IDC AHL advised the Animals Incursion Investigation team of an infectious bursal disease (IBD) surveillance farm with seropositive VNT results . An investigation was initiated using epidemiological evidence from the farm and repeat testing to verify that there was no clinical IBD circulating on the farm. As the farm was raising broiler chickens, the cohort tested with the non-specific reactor results had already been processed and the animals were no longer available for testing. However, the current cohort were housed in the same sheds and on the same litter as previous, meaning that if IBD was present the current cohort would most likely have been exposed. However, daily inspection of all sheds on the farm for 14 days found no evidence of clinical IBD. Samples of birds were also culled from each shed and fully post-mortemed, with no evidence of IBD. Repeat serological testing also found no evidence of IBD infection. The investigation was closed and the results confirmed as a non-specific reaction.

Avian bornavirus excluded

MPI was notified of a black cockatoo with a history of a dilated proventriculus, thought initially to be consistent with avian bornaviral infection (also known as proventricular dilatation disease or PDD). Feathers and a blood sample were submitted for testing by PCR. Shortly after notification, the proventricular dilation appeared to resolve. PCR testing was negative for avian bornavirus. Based on resolution of clinical signs and negative PCR results, the investigation was stood down.

Exotic salmonellosis excluded

A veterinary pathologist notified the MPI exotic pest and disease hotline of a die-off of finches that had gross lesions resembling salmonellosis. Up to 20 finches were found dead near Palmerston North Hospital, and six were submitted for post-mortem examination. Gross lesions were similar in all birds, including multifocal caseous nodules adhering to the mucosa of the oesophagus and crop, and also scattered within the hepatic and splenic parenchyma. Histologically the nodules were consistent with areas of necrosis surrounded by mainly heterophils, with intralesional coccobacilli. The lesions were cultured and all yielded *Salmonella*, including from a maggot found on one of the birds. Subtyping identified *Salmonella* Typhimurium phage 56 (formerly phage type RDNC). This serotype is frequently isolated from small numbers of New Zealand poultry (Mulqueen, 2013; Watts, 2012). Salmonella outbreaks are not uncommon among wild passerine birds worldwide, and in this case the occurrence near a hospital was a public health concern. However, contact with the hospital was initiated as soon as salmonellosis was suspected, and no clinical cases of salmonellosis occurred in the area.

Chelonian herpesvirus excluded

A veterinarian at a zoo called the the MPI exotic pest and disease hotline to notify an inconclusive result from generic PCR for herpesvirus in a Galapagos tortoise (*Chelonoidis nigra*). Chelonian herpesvirus had been suspected because it had been exhibiting signs of depression and rhinitis. As a result a nasal flush had been undertaken and the material tested by PCR. Chelonian herpesvirus is highly host-specific to chelonians, which are often subclinically infected. There is no past record of testing for chelonian herpesviruses in tortoises in New Zealand. There are no native tortoises in New Zealand but animals have been imported in the past for zoological collections and it is possible that the virus could have been brought in with zoo stock. There is no mention of testing for chelonian herpesvirus in the import health standard for tortoises imported from specific zoos in Australia. The MPI AHL was consulted and advised sequencing of the generic PCR product at Massey University to see if it matched a sequence from chelonian herpesvirus. However, it was not possible to sequence the PCR product from the faintly positive test. The animal was resampled and the generic PCR returned a negative result. Three other Galapagos tortoises were tested twice and returned negative results for all tests. The investigation was stood down as the repeat testing indicated that chelonian herpesvirus was not associated with this disease incident.

European foulbrood ruled out

An apiary specialist called the MPI exotic pest and disease hotline to report disease resembling European foulbrood (EFB, caused by *Melissococcus plutonius*). The disease was present in one hive of 25 on the apiary site located in Canterbury. Disease signs included a foul smell, dead larvae, larvae laid inappropriately at the entrance to cells, and larvae with developmental abnormalities including twisting and deformation. Some larvae were roped as is seen in both EFB and American foul brood (AFB). Samples from the hive were sent to the AHL for culture and PCR. PCR returned suspicious results for EFB so urgent measures were initiated, including collection of further samples, movement restrictions on the apiary site, deployment of an Incursion Investigator on site, and initial tracing of hive movements to and from the property. The second set of samples was express-couriered to the AHL for testing as above. DNA amplified in the laboratory was sequenced, but analysis of the suspicious DNA indicated that it was not consistent with *M. plutonius*. The best match was with a *Bacillus* species suspected of being common in that locality; this species shared some binding sites with primers used for the OIE-recommended PCR test. Consequently the response was stood down. Follow-up investigation was performed to establish the distribution and likely surveillance impact of the *Bacillus* species found. The diagnosis in this case was *Bacillus* overgrowth secondary to halfmoon syndrome, a common endemic disease resulting from mislaying of eggs by a defective queen.

Southern saltmarsh mosquito ruled out

A regional council biosecurity officer phoned MPI to request confirmation of a mosquito identification. The mosquitoes had banding on the legs and were active in the daytime. They were found in possible southern saltmarsh mosquito (SSM) habitat. Initial identification from pictures ruled out SSM, but specimens were sent to IDC PHEL Tamaki and confirmed to be *Aedes antipodes*, a native species. The investigation was closed after all SSM surveillance partners were notified.

Risk goods incidents

Live insect larvae were discovered in packages of loose feathers sold for craft purposes from a shop in Dunedin. The purchaser notified MPI's exotic disease and pest hotline. The presence of live larvae in these packages of sealed feathers, which were from China, meant that the import health standard had not been met as the necessary treatment requirements for import, e.g. fumigation, would have killed any insects present. The import was traced to the supplier and all the stores in a chain throughout New Zealand that had received the products. The stores in Auckland and the supplier's warehouse were visited by an MPI technical coordinator and the feathers there were found to be similarly infested and were seized. Larvae were sent to IDC PHEL Tamaki for identification by the entomology team, and feathers and larvae were tested at the AHL (Wallaceville) for infectious bursal disease virus and *Chlamydophila psittaci* (a zoonotic pathogen that can be spread to people from feathers). Both tests were negative and the larvae were identified as *Tineola bisselliella* (webbing clothes moth), which is present in New Zealand. Investigation determined that the feathers had been misdeclared as plastic craft when imported, hence the failure to meet the required procedures for natural feather imports. The feathers constituted risk goods as undeclared and unauthorised goods. A complete recall of all packages from the consignment was undertaken and the feathers were destroyed. The case was referred to ITOC for assessment of non-compliance with MPI's import protocols. Recommendations were made regarding future inspections of the company's imports and further compliance investigations.

A member of the MPI Food Operational Coordination group phoned the MPI exotic pest and disease hotline to report an apparently unauthorised honey product in the post-border space. This notification was followed up with the company distributing the product, which was subsequently found to contain less than 2 percent honey. This meant that the product was an authorised product in compliance with the Import Health Standard and the investigation was closed.

An MPI Food Officer phoned the MPI exotic pest and disease hotline to report that an Environmental Health Officer (EHO) had received a complaint regarding a small bone in a 25 kg bag of bulk imported peanuts. The peanuts were traced to an importer and it was determined that they had been imported in late 2012 from China in vacuum-sealed 25 kg bags. All of the bags of the suspect consignment had been sold with no other reports of contaminants known to the importer. Inspection of the

bone indicated that it was of rodent origin, and since it had been roasted, it was a negligible biosecurity risk. An investigator visited the premises of the importer, thoroughly inspected the available peanut consignments and found nothing further therefore it is unknown when it became a contaminant but it is suspected it did so at the NZ repacking facility. The MPI food safety team has been notified of the inspection report and the recommendations for increased vigilance for foreign matter at the NZ repacking facility as part of the MPI-approved Food Safety Programme.

A dairy farmer in the Bay of Plenty discovered an animal part remaining in the feeder after a batch of palm kernel expeller (PKE) had been fed out to his cows. He alerted the supplier of the product, who then notified MPI Border Clearance Services, who in turn notified the Incursion Investigation team. An investigation was undertaken to determine whether the animal part was of foreign or domestic origin, and the risks involved if it was from overseas. A response was also launched in case a foreign origin was confirmed so that risk mitigation could proceed quickly.

The shipment of PKE had originated in Indonesia and Malaysia and had been held for five days in bulk storage before being delivered to the farmer. It had been hammer-milled during production and screened with a 4 mm mesh before loading. Both the exporting facilities had been recently audited and approved for export to New Zealand. MPI had a high degree of confidence in the biosecurity procedures of the exporting facilities. The PKE had been trucked directly to the farmer and stored overnight in an uncovered bin on his property before the animal part was discovered the next day. The farmer had dogs that had in the past scavenged dead animals, and had previously been known to bury items in PKE.

The animal part was desiccated and mummified and could not be identified by gross examination alone. X-rays were taken and it was found to comprise the shoulder region and proximal front leg of a small mammal. DNA sequencing at the IDC (Wallaceville) to determined the species of the animal confirmed it was *Bos taurus* (domestic cow). Owing to the size of the animal, it was probably an aborted calf foetus.

There were 150 mixed-age dairy cows on the property, all of which were fed PKE daily. Only one feeding had taken place before the animal part was discovered and removed. All cows were in good health and milk production had been increasing on the farm since the discovery. The only animals to have left the farm recently were calves that had been milk-fed, and the farm owner voluntarily stopped any further animal movements until the investigation was concluded.

Meanwhile, a risk assessment was undertaken by the Incursion Investigation team to determine the likely biological risk of disease transmission if the animal part was from overseas. This depended on the likelihood of the animal part harbouring viable exotic pathogens. For risk to be present, micro-organisms would have needed to survive the animal's post-mortem changes, desiccation and the length of time the animal had been dead, at ambient temperatures. Diseases of concern would have to be present in the exporting regions and the animal would have needed to be infected with a disease at the time of death. There would also have to have been a suitable exposure event to susceptible animals in New Zealand. None of these factors were considered likely given the situation and the risk was determined to be negligible. The overall conclusion was that the animal part was probably of New Zealand origin, not accidentally imported from Indonesia or Malaysia. However, in the unlikely event that it was of overseas origin, the risk of its transmitting disease was negligible.

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MARINE AND FRESHWATER

QUARTERLY REPORT OF INVESTIGATIONS OF SUSPECTED EXOTIC MARINE AND FRESHWATER PESTS AND DISEASES

Exotic marine pest and aquatic disease investigations are managed and reported by MPI's Investigation and Diagnostic Centre and Response, Wallaceville. The following is a summary of investigations of suspected exotic marine diseases and pests during the period from October to December 2013.

Exotic sea squirt excluded

After reading an MPI marine pest identification brochure, a member of the public phoned the MPI pest and disease hotline reporting something that resembled a sea squirt in the brochure. The caller said that he had never seen this organism before. It was located on a jetty in the Marlborough Sounds, but the caller lived in Upper Hutt, so he brought a sample directly to the MPI Animal Health Laboratory. It was identified as *Pyura pachydermatina*, an ascidian native to New Zealand. There was no biosecurity risk so the investigation was closed.

Phoronid worm confirmed

NIWA notified MPI that a non-indigenous species of phoronid worm had been identified from samples taken during the Marine High Risk Site Surveillance winter survey at Marsden Cove Marina, Whangarei Harbour. It appeared to be either *Phoronis hippocrepia* or *P. ijimai*, and neither species had been reported in New Zealand previously. Histology was undertaken and the species confirmed as *P. ijimai*. The Phoronida are a small, exclusively marine phylum comprising only 10 species worldwide. Superficially they may resemble polychaete worms but can be readily distinguished by their lack of segmentation and bristles. They also have a characteristic U-shaped gut, from which their common name of horseshoe worms is derived. *P. ijimai* prefers hard substrata, was first described from Japan in the 1890s, and is thought to be native to the northern Pacific (Gordon, 2013). It has also been recorded from Botany Bay, Australia, and the eastern seaboard of the US (Emig, 2005). In New Zealand it was reported colonising wharf pilings at a depth of about 5 m. The risk of its establishing in New Zealand (if it has not already done so) is unknown, but a literature search did not reveal a particularly invasive history. It is, however, possible that it may compete with other fouling species of hard substrates for space and resources. Three other phoronids are already known from New Zealand waters (Emig & Roldán, 1992; Gordon, 2009) – a small shell-boring species, *Phoronis ovalis* Wright, and the soft sediment dwellers *Phoronis psammophila* Cori and *Phoronopsis albomaculata* Gilchrist. Owing to the difference in habitat preference, *P. ijimai* is unlikely to compete directly with New Zealand phoronids. Its spread beyond the known range is notable as horseshoe worms have not previously been reported from vessel biofouling, nor have their larvae been detected in ballast water (Gordon, 2013), so the manner in which it was introduced to New Zealand remains unknown. The Northland Regional Council was informed of the detection and will monitor any observed spread. As *P. ijimai* appears to pose a low biosecurity risk to New Zealand, no further action was recommended and the investigation stood down.

Tadpole shrimp intercepted

A member of the public notified MPI of an online auction for tadpole shrimp, *Triops longicaudatus*, a North American freshwater crustacean in the order Notostraca. The eggs are highly resistant to desiccation and have to be completely dried out then re-immersed in water to be hatched. This means *Triops* eggs are easily transported and they are sold in kits with the name Aquasaurs, Trigons or Triops, to be raised as aquarium pets. This ornamental aquatic species is not permitted for import into New Zealand and instances of aquarium kits of this species entering the country have been investigated previously by MPI. A preliminary risk assessment of *T. longicaudatus* during the 2006 investigation and response found that this species had the potential to cause unwanted harm to New Zealand should it escape from captivity and become established. The person who won the online auction was contacted by MPI, voluntarily surrendered the *Triops* kit and provided the contact details of the seller. The original packaging indicated the kit had been imported from the USA by airmail. Attempts to locate the original seller were unsuccessful. It is likely that there will be future interceptions of *Triops* from the pet trade in the future. This case highlights the importance of the general public in post-border surveillance.

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PLANTS AND ENVIRONMENT

A PRAGMATIC APPROACH TO DELIMITATION

Biosecurity surveillance involves special challenges, in particular that the target organism is frequently absent from the surveyed area. This means surveys must be designed to detect small target populations and to estimate the likelihood that failing to detect the population means it really is absent. Biosecurity incursion investigators often need to assess the spatial extent of populations to evaluate whether eradication is a viable management option, but strict delimitation of the occupied area requires substantial sampling effort. Furthermore, with little prior information about the biology of many exotic organisms it is often difficult to know where to begin.

A Herculean task

An exotic caterpillar was reported to MPI in March 2010 and identified as one of the many undescribed Australian pasture tunnel moths (*Philobota* spp. – **Figure 1**) that cause occasional damage to Australian pastures (Popay & Gunawardana 2011; Gerard 2013). The larvae (**Figure 2**) hide by day in silken tunnels burrowed into the ground under thick vegetation, emerging by night to feed nearby. Their subterranean habits make them very difficult to find and control. Although initial experiments following its detection suggested suction samplers might be effective in dry conditions, it was eventually concluded that the most reliable detection method was laborious digging and manual searching of turves.

Figure 1: Adult Australian pasture tunnel moth, *Philobota* sp., reared from a larva collected in the field. Photo: T. Oliver, MPI

Figure 2: A *Philobota* larva beside its tunnel, which consists of a silken tube embedded with soil particles. Photo: A. Pathan, MPI

MPI investigators appreciated the magnitude of the task when they stood at the site of discovery and surveyed the thousands of hectares of suitable habitat surrounding them: the rolling pastures of the Waikato on one side and the hundreds of private lawns in the city of Hamilton close by on the other (**Figure 3, page 34**). How could we effectively delimit the extent of an almost invisible population in such a vast and uniformly suitable habitat?

Getting the question right

We sought assistance from researchers in New Zealand's Better Border Biosecurity (B3) science collaboration. Together, investigators and scientists realised that they didn't actually need to know the extent of the population – at least, not initially. Immediate decision making needed only the knowledge of whether or not the population was likely to be eradicable. With that in mind, the question to be addressed was not "Where in this vast landscape is the organism present?" but the much more manageable "Is the organism present further than 1 km from the site of discovery, or on the urban boundary; in which case eradication would not be feasible?" This pragmatic approach focused on getting the delimitation question right, narrowing the scope of the investigation to something feasible.

Answering with confidence

With the right question, the task became to survey the perimeter of a 1 km buffer around the discovery site,

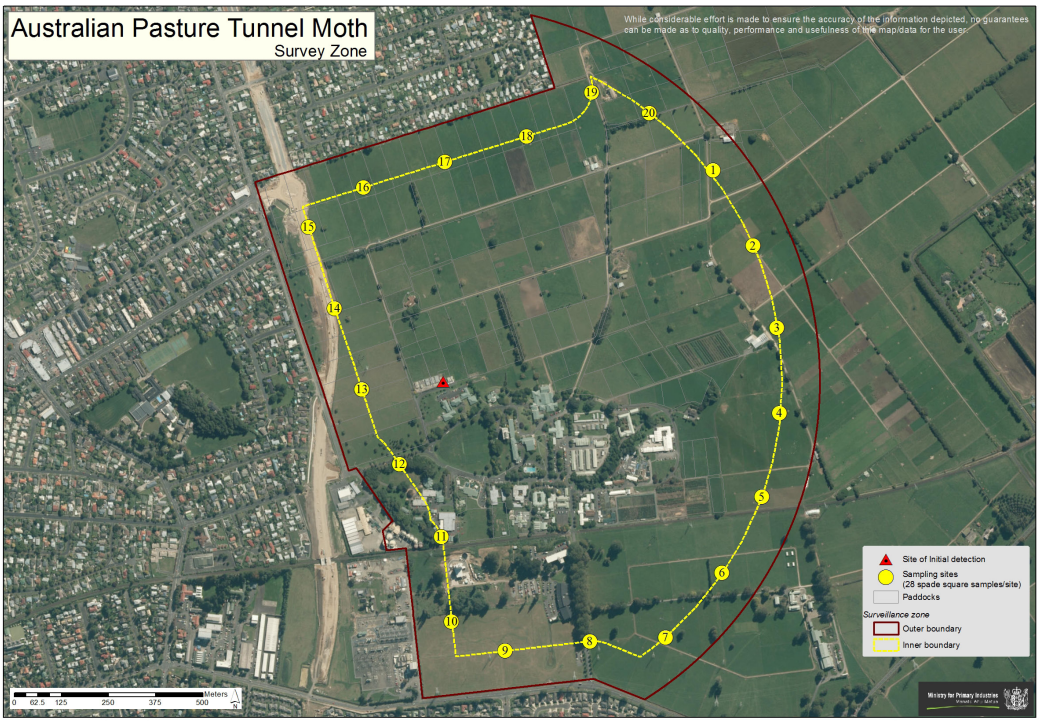

Figure 3: Map showing the location of the initial *Philobota* detections and the zone within which surveillance was undertaken. H. Acosta, MPI

including the edge of urban Hamilton. But armed only with spades for detecting larvae, how many holes would need to be dug to be confident that any population present would be found?

B3 researchers designed a multi-level sampling plan that would give 95 percent confidence of detecting the target population. All surveys rely on careful specification of the target population size to be detected, and this should reflect the aims of the survey. Here, the aim was to detect a healthy, spreading population (if present), so the target was specified as one larva per square metre present in 20 percent of the 70 perimeter paddocks. Some straightforward mathematics determined that 28 spade squares taken in each of 20 paddocks would achieve the desired 95 percent survey sensitivity (Kean *et al.*, 2014). Now investigators were faced with the task of digging a maximum (since once the organism was detected the survey could stop) of 560 small holes. This was a huge improvement from the apparently insurmountable delimitation survey that initially challenged them.

Successful outcomes

In early July 2010 a team of trained searchers started digging, under the supervision of professional pasture entomologists (**Figure 4**). Pasture tunnel moths were found at two sites on opposite sides of the 1 km perimeter and at a third site that was not part of the formal survey. Based on this information, MPI concluded that eradication was not feasible, and subsequent efforts have been directed towards long-term pest management (Gerard, 2013).

Figure 4: Training the sampling team in spade square sampling to detect the soil inhabiting larvae. Photo: G. Burnip, MPI

The success of this investigation has led to the same approach being used in other incursion events. A contrasting example is the eucalyptus leaf beetle (*Paropsisterna beata*), which was discovered near Wellington in August 2012. In this case, the delimitation survey formulated by investigators and B3 researchers suggested that the population was confined to a very small area, and eradication treatments were applied (Williams, 2013). Post-treatment monitoring continues, including further detection surveys, and there is currently cause for optimism that in this case a pragmatic approach to delimitation may have contributed to the successful eradication of an unwanted exotic pest.

Publication of a book chapter describing the detail of this sampling approach and how it was used when *Philobota* was detected in Hamilton is pending (Kean, Burnip & Pathan, 2014)

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A BIOSECURITY POST-BORDER SUCCESS STORY: EARLY DETECTION AND REMOVAL OF *RUGONECTRIA* CANKER FUNGUS IN AN OAK TREE

In 2011, an oak tree (*Quercus robur x canariensis*) with elongate, sunken cankers on the trunk (**Figures 1 & 2**) was discovered in urban Auckland through the High Risk Site Surveillance Programme conducted by the Ministry for Primary Industries (MPI). The affected tree was located near a hub of registered Transitional Facilities for overseas shipping containers. This article describes how the fungus was identified, contained and treated. It appears likely that the result is a successful eradication of this new to New Zealand organism before it had an opportunity to become established.

Figure 1: The oak tree (*Quercus robur x canariensis* when infection was detected.

Figure 2: Cankers on lower trunk.

Identification of the fungus

The trunk cankers were filled with orange-red nectriaceous fruit bodies (**Figures 3 & 4**) that did not match the description of species known to cause or be associated with oak cankers in New Zealand. Morphological characteristics of the specimen fitted the broad description of a fungus, *Rugonectria castaneicola*, which is associated with stem cankers or cracked bark of a range of tree hosts in Japan (Chaverri *et al.*, 2011). When the DNA sequences of this fungus from the oak tree were examined and compared with sequences retrieved from international databases, it was revealed to be a species new to science and present in China, subsequently named *R. sinica* (Zeng *et al.*, 2012).

Figure 3: Clusters of orange-red, warted fruit bodies (perithecia) of R. sinica formed on bark.

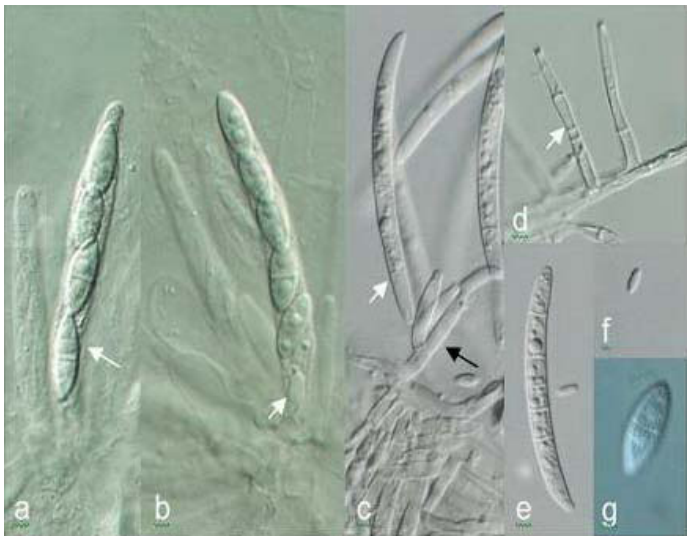

Figure 4: *Rugonectria sinica*. a. Ascus containing 4 sexually reproduced spores (ascospores; arrowed); b. Ascus with 4 mature and a few aborted ascospores (arrowed); c. Large asexual spore (macroconidium; white arrow) and branched macroconidium-producing structure (conidiophore; black arrow); d. Unbranched conidiophores (arrowed) that produce small asexual spores (microconidia); e. A macroconidium with 6 cross walls (septa); f. A microconidium; g. An ascospore with stripes on the spore wall.

This is the first record of a species of *Rugonectria* in New Zealand and the first association with a stem canker of *Q. robur* x *canariensis*.

Pathogenicity of *Rugonectria sinica*

Species of *Rugonectria* are likely tree pathogens as they are associated with stem and trunk cankers, or found growing on recently killed trees (Chaverri *et al.*, 2011). *R. castaneicola* is associated with stem cankers or cracked bark on a range of tree hosts in Japan. Its pathogenicity has been demonstrated in *Abies veitchii* and *Acer crataegifolium* (Kobayashi *et al.*, 2005). *R. rugulosa* causes bark death and is associated with trunk cankers of *Macadamia integrifolia* in Hawaii (Ko & Kunimoto, 1991). The third previously recorded species, *R. neobalansae,* occurs on the bark of living and recently killed trees and is known only in Indonesia (Chaverri *et al.*, 2011).

The crown of the infected tree was healthy but a cross-section of the sapwood beneath the cankers was stained brown (**Figure 5**). In China, *R. sinica* has been found on dead twigs of plants, including *Quercus* species. The potential for *R. sinica* to be a serious pathogen of *Quercus* spp. is unknown.

Figure 5: Cross-section of tree trunk showing brown staining of sapwood.

Removal of infected tree and treatment of material

Permission was obtained from the lessees of the Auckland site to fell and dispose of the infected tree. Branches were removed and the tree felled (**Figure 6**) in December 2011. All plant material including stump, branches and leaves was wrapped in tarpaulins and transported in a covered truck for deep burial at an approved site (**Figure 7**). Sawdust and wood chips were removed from chainsaws and added to the material sent to the landfill. Chainsaws were stripped and, along with other equipment used at the site, were sprayed with a quaternary ammonium compound solution. The parts were left for 30 minutes, rinsed with water and left to dry. The stump was cut below ground level and a heavy coating of picloram herbicide applied to the surface. In subsequent, regular follow-up surveys, *R. sinica* has not been found on surrounding trees.

MPI considers *R. sinica* to be eliminated from this site and the fungus will be declared eradicated if no further finds are detected during follow-up surveys.

Figure 6: Removal of the infected oak.

Figure 7: Plant material wrapped in tarpaulins and transported in a covered truck.

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USING MULTIPLE LURES IN SURVEILLANCE TRAPS TO IMPROVE EFFICIENCY AND EFFECTIVENESS

MPI is investigating opportunities to combine lures in MPI surveillance programmes to increase surveillance efficiency without compromising effectiveness. Progress to date includes a literature review summarising data in this area and the set up of an international network of trials.

The Ministry for Primary Industries (MPI) undertakes fruit fly and gypsy moth surveillance for early detection of any incursion, to facilitate timely eradication and to demonstrate continued freedom from this range of pests. New Zealand's fruit fly and gypsy moth-free status is valuable to producers marketing fresh produce and timber products overseas. The presence of these insects could cause prohibition or require treatments for many export markets. The current MPI fruit fly surveillance programme uses three different lures to target various species according to lure response, with a separate trap for each lure. The total number of traps used is about 7500.

Since the cost of fruit fly surveillance is borne by the taxpayer, MPI has an obligation to ensure that the programme is carried out in the most cost-effective manner. Currently MPI contracts out the work of servicing all gypsy moth and fruit fly traps fortnightly.

What MPI wants to achieve

Surveillance trapping for multiple species by baiting traps with numerous lures will likely increase the number of species that can be targeted and reduce the costs without affecting surveillance coverage (Brockerhoff *et al.*, 2013; Suckling *et al.*, 2012). For example, if a single lure incorporating all three fruit-fly attractants could be used, the effectiveness of the programme could be significantly increased because of the increased density of traps targeting all species, with only marginal increases in operating costs. Alternatively, the cost of the programme could be reduced by maintaining the present probability of detection.

There are a number of other organisms that pose a risk to New Zealand that are potential candidates for combining lures with current surveillance programmes (e.g. Brockerhoff *et al.*, 2013). As we move into the era of GIA with partnerships with multiple primary-sector industries, the cost of specific targeted trapping programmes for each pest for which there is a lure could be prohibitive. Multiple-species trapping offers the opportunity to trap a wider range of pests at moderate cost increases.

Many trials are going on in the US to combine fruit-fly lures. Research by Vargas *et al.* (2012) on the effectiveness of combining into a single trap the fruit-fly lures trimedlure, methyl eugenol and raspberry ketone (which attracts the same species as the cuelure that New Zealand uses) has had promising results.

Gypsy moth surveillance is conducted using (+) disparlure for attraction, targeting *Lymantria dispar* and other *Lymantria* spp. Recent work by Brockerhoff *et al.* (2013) has indicated that lures for some non-lymantrid species are not likely to significantly reduce the catch of *Lymantria* spp., and vice-versa when lures from different species are presented together.

Progress on an MPI operational research programme

MPI has initiated an operational research programme building on previous B3 (b3nz.org) and other research in this area. Plans have been developed in conjunction with Plant & Food Research (PFR), Scion and various overseas collaborators (e.g. USDA). Work was initiated in mid-2013.

The approach initially has been to review the combinations of lures that have been previously tested and produce a literature review. This part of the project referenced more than 60 documents and produced recommendations for the next stages of the project.

Next, experimental methods for combining lures for the top-priority organisms such as fruit flies and cross-sectoral and forestry pests were developed. Then an experimental trapping network was established with an international team based in the US, Europe and Japan, targeting current and likely priority pests for New Zealand.

In the first year of research, various lures were combined for target groups that go to similar traps, e.g. tephritid flies to Lynfield traps, moths to delta traps, and beetles and brown marmorated stink bugs (BMSB) to panel traps (see **Figure 1** for examples of the trap types). A summary of the field trials is shown in **Table 1**, including countries we are collaborating with. After the first year comparisons will be made between the different groups to see whether there are any interactions among the different group/trap types, to determine whether traps from these different groups may be placed in close proximity to each other.

From this research, recommendations will be made on the lures that can be combined in traps to target multiple pest species with estimates of potential reduction in efficacy due to the presence of another species' lure, as well as whether traps targeting different groups can be placed close to each other.

Based on the research carried out to date there appear to be good prospects for improving surveillance through the combination of lures for multiple species.

Figure 1: Examples of trap designs for different target groups insect groups. 1a – Lynfield traps (tephritid flies); 1b – delta traps (moths); 1c – pyramid (background) (BMSB) and panel (foreground) (beetles/ BMSB) traps from one of the field trials (Source: 1a - MPI website http://www.biosecurity.govt.nz/pests/surv-mgmt/surv/fruit-fly; 1b - Plant & Food Research /1c – Shearer PW Oregon State University)

1a – Lynfield

1b – Delta

TABLE 1: GENERAL DETAILS OF TRIALS PLANNED OR UNDERWAY BY COUNTRY OR STATE FOR THE FRUIT FLY, MOTH AND BMSB SUBPROJECTS

	SPECIES	TRAPS	COMBINATIONS	LOCATION
Subproject 1	Fruit flies	Lynfield	Trimedlure (e.g. Mediterranean fruit fly, *Ceratitis capitata*) Methyl eugenol (e.g. oriental fruit fly, *Bactrocera dorsalis*) Cuelure (e.g. Queensland fruit fly, *Bactrocera tryoni* and other cuelure-responsive species) Vargas *et al.* (2012) combined lure with raspberry ketone	Hawai'i and Australia
Subproject 2	Moths	Delta	Fall web worm, *Hyphantria cunea* European grapevine moth, *Lobesia botrana* Pine processionary moth, *Thaumetopoea pityocampa* Gypsy moths *Lymantria* spp.	Italy, France, Japan, Portugal
Subproject 3	BMSB/ Beetles	Panel; pyramid	Brown marmorated stink bug, *Halyomorpha halys*; other beetle species	Oregon

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PLANTS AND ENVIRONMENT INVESTIGATION REPORT: OCTOBER TO DECEMBER 2013

Suspect brown marmorated stink bug found in Auckland warehouse

An unusual bug was found in an Auckland warehouse belonging to an importer of bathroom goods. The warehouse was a Transitional Facility (TF), where the Approved Person noticed a solitary bug crawling on a pallet. The AP called the Ministry for Primary Industries (MPI) about the bug because a fact sheet on brown marmorated stink bug (BMSB, *Halyomorpha halys*) had recently been distributed by MPI to all TFs nationwide. However, although similar in appearance to BMSB, the bug was identified as *Erthesina fullo*, the yellow-spotted stink bug. Like BMSB, *E. fullo* is not present in New Zealand and both are Unwanted Organisms under the Biosecurity Act 1993. However, *E. fullo* is considered of lesser risk to New Zealand than *H. halys,* based on its biology and interception data. Both species are known to overwinter as diapausing adults, with aggregations of multiple adults sometimes seen on inanimate objects. Species exhibiting such behaviour are considered to have a high establishment risk. The imported goods concerned originated mostly from China and Thailand and were mainly packaged in purpose-built wood-composite crates, a type of packaging considered to have low biosecurity risk. The pallet on which the bug was found came from a consignment imported in September 2013, but as the bug was mobile and adjacent goods and packaging were from multiple consignments, it could have originated from any of them. The warehouse typically receives four or five 40-foot sea containers weekly; in this case, nine arrived during the week prior to the detection.

Goods typically arrive shrinkwrapped in plastic on pallets. These are broken down in the warehouse, stored, and distributed as individual items to plumbing merchants nationwide. This intensity of handling increases the likelihood that any further bugs would have been seen if present. MPI staff inspected the warehouse but found no more bugs. The day after the first find, three light traps and some passive sticky traps were placed in the warehouse but they caught no insects after three nights. Available information suggests this case was a solitary individual, which is consistent with all previous interceptions of live *E. fullo* bugs.

Live borer found in imported spades

MPI was notified of beetles emerging from hardwood spade handles at a Waikato hardware store. Specimens were couriered to the Plant Health Environment Laboratory and identified as *Lyctus sinensis* (Coleoptera: Bostrichidae), a species not present in New Zealand. The goods originated from China. The store immediately contacted all affected branches to remove affected goods from display and storage, and to secure all spades and forks in insect-proof wrapping. This mitigated the risk of establishment from goods held in retail outlets. The store also arranged for the secured items to be returned to Auckland by dedicated transport for fumigation and assessment. Tracing of the infested goods revealed that they originated from two containers, but systems did not allow differentiation of exactly which goods in the supply chain originated from those containers. As a result, all of those product lines were withdrawn for treatment, as well as wooden products that had been in proximity to them in the stores. All potentially affected material in the supply chain was fumigated with methyl bromide. Inspection of returned goods showed that the infestation rate was probably less than 1 percent. Given this low level of exposure, a public recall was not considered to be warranted. Fumigation was completed and the investigation was closed.

Snail from Portugal found in packing material

The notifier found an unusual live snail among packing materials borrowed from a neighbour who had used the materials when recently emigrating from Portugal. The snail was submitted to MPI and identified as *Otala lactea* (milk snail), an exotic fruit- and plant-feeding species native to southern Europe, including Portugal. It is considered a nuisance in many places and has been invasive in locations with a hot Mediterranean climate, e.g. California, where it may displace local native snails. An MPI quarantine inspector was sent to inspect the property but found no more snails. He provided images of the snail and asked the residents to notify MPI if they saw any more like it, but none have subsequently been found. The notifier searched the packaging material for further snails and then destroyed the material by burning it.

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PEST WATCH: 9 NOVEMBER 2013 – 14 FEBRUARY 2014

Biosecurity is about managing risks: protecting New Zealand from exotic pests and diseases that could harm our natural resources and primary industries. MPI's Investigation & Diagnostic Centres and Response (IDC & R) directorate devotes much of its time to ensuring that new organism records come to its attention, and to following up as appropriate.

This information was collected from 9 November 2013 to 14 February 2014. The plant information is held in the MPI Plant Pest Information Network (PPIN) database. Wherever possible, common names have been included. Records in this format were previously published in the now discontinued magazine *Biosecurity*.

To report suspect new pests and diseases to MPI phone 0800 80 99 66.

Validated new to New Zealand reports

Type	Organism	Host	Location	Submitted by	Comments
Insect	*Micromimetus* sp. mirid bug	*Alnus cordata* alder	Auckland	S Thorpe (General Surveillance)	Similar specimens known from Australia and Cook Islands. The species is awaiting description.
Insect	*Tuberolachnus salignus* giant willow aphid	*Salix fragilis* crack willow	Auckland	S Thorpe (General Surveillance)	First reported on willow trees at Western Springs Park, Auckland, on 24 December 2013. Subsequently recorded from Northland, Waikato, Wellington to mMid Canterbury. Further distribution reports are awaiting validation.
Mite	*Eriophyes* sp. eriophyid mite	*Pennantia corymbosa* kaikōmako	Wellington	IDC & R (High Risk Site Survey)	Undescribed species. Likely native.
Mite	*Phytoseius* sp. phytoseiid mite	*Chrysanthemoides monilifera* boneseed	Auckland	IDC & R (High Risk Site Survey)	Undescribed species.

If you have any enquiries regarding this information please contact surveillance@mpi.govt.nz.

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NEW ZEALAND VETERINARY PATHOLOGY

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- HAMILTON
  Courier: Cnr Anglesea and Knox Streets, Hamilton
  Postal: PO Box 944, Hamilton
  Tel: 07 839 1470 Fax: 07 839 1471

- PALMERSTON NORTH
  Courier: IVABS Building, 1st Floor, Massey University, Tennant Drive, Palmerston North
  Postal: PO Box 325, Palmerston North

To report suspected exotic land, freshwater and marine pests, or exotic diseases in plants or animals, call:

0800 80 99 66

Investigation and Diagnostic Centre – Wallaceville
66 Ward Street
Upper Hutt
Tel: 04 526 5600

Investigation and Diagnostic Centre – Tamaki
231 Morrin Road
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