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INSIDE:

Diseases of backyard pigs in New Zealand
Clarifying the pest status of some exotic termite species for New Zealand
3D printing and biosecurity



Biosecurity New Zealand

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Manatū Ahu Matua



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Editorial

Innovation in biosecurity

In June 2017, applications opened for the MPI graduate development programme. As a Lincoln University student studying ecology, plant pathology and biosecurity, this programme was an obvious choice for me and something I'd had my eye on for a number of years. After a thorough and robust process of selection, 700 applicants were whittled down to 11 graduates, starting our new roles at MPI in January this year. The high number of applicants reflects the range of opportunities on offer at MPI and is further reinforced by the MPI Graduate Development Programme winning the NZAGE best graduate programme in the 2017 industry awards.

The graduate programme is 18 months long and consists of three 6-month rotations within MPI. The rotations are selected for each graduate, based on their interests and backgrounds as well as their tertiary study, resulting in huge variation across our cohort. My primary focus has been working in the Surveillance and Investigation group to develop surveillance traps using 3D printing technology for the National Invasive Ant Surveillance programme (NIAS). I have also had the opportunity to be involved in other work, including investigations dealing with biosecurity-risk animals and analysing marine surveillance and diagnostic data to build on epidemiological information about specific marine diseases. Using 3D printing technology to develop surveillance traps is an innovative approach to a real-world problem and provides an opportunity to reflect on the relevance of innovation to biosecurity – hence I am writing this editorial.

Innovation can be defined as the introduction of something new. It can be simple, like pre-slicing bread, or complex, like Rocket Lab 3D-printing rocket-engine parts. For biosecurity, innovation can be in the form of new science, new technology, alterations or transfers of existing technology, or changes in the way we think, interact or operate. Innovations are becoming increasingly prevalent across society, and there is huge potential for applying innovation to the improvement of biosecurity.

Biosecurity is facing some complex and difficult issues. Global warming is making our climate more amenable to many invasive species, supply chains are becoming more complex and our trade and tourism pathways have switched from a largely European origin, to increasing volumes from Asia and the Pacific. These areas pose a higher biosecurity risk because, as trade and tourism increase, so too does the pressure of invasive species arriving in New Zealand.

To remain effective, MPI has to adapt to address these complex and difficult issues. Traditional adaptation has involved ramping-up resource inputs into the biosecurity system to result in incremental increases in efficiency. Until now this has

worked, but this approach is becoming ineffective owing to limited funding, tools and resources becoming unavailable or outdated, and biosecurity facilities reaching staffing capacity. Innovation and changing systems and processes through behaviour will empower MPI to work “smarter, not harder” across the biosecurity system. Key areas for improvement include tools for early detection in surveillance and advanced techniques to more quickly and accurately deal with throughput in diagnostics, but there is potential for improvements across the biosecurity system. Innovation, rather than increased resource inputs, will drive these improvements.

For innovation to occur, there must be an environment that nurtures innovation and provides the opportunity to challenge the status quo where there is room for improvement. An environment that promotes thinking at all levels results in new ideas that reflect the needs of the user. This has to be combined with a regulatory approach to pursue options that are most successful, cost-effective and socially acceptable.

Collaboration can be a driver of innovation and can be seen driving innovation in biosecurity. Better Border Biosecurity (B3) is a multi-partner co-operative science collaboration that researches ways to reduce the entry and establishment of new plant pests and diseases in New Zealand. The research uses innovative technology including biogeochemistry, bioacoustics sensing and nanotechnology. Additionally, in March this year, representatives from MPI attended the inaugural biosecurity innovation exchange, held by the Department of Agriculture and Water Resources (the Australian equivalent of MPI) in Canberra. The aim of this event was to collaborate on shared problems and innovative solutions to enhance both countries' biosecurity systems.

MPI encompasses biosecurity, fisheries, food safety and forestry, and in such a large organisation it can be perceived that MPI is not innovative. However, MPI has a Research Technology and Innovation (RTI) team that focuses on novel technology and science, and how this can be used to support primary industry. Earlier this year the RTI team unveiled VAI – a robot with inbuilt artificial intelligence to answer people's queries at Auckland airport. Another example of innovative work at MPI is using 3D-printed light traps in the Arbovirus Surveillance Programme to detect *Culicoides* species blown from Australia. As a new entrant to MPI, I find it reassuring to see the value placed on innovation in an organisation that has a primary regulatory function.

Innovation is already incorporated into our biosecurity system, and continued support will nurture further growth in this area. This is important as new ideas and technologies are going to be

key to addressing the complexities in biosecurity today, and ensuring the sustainability of our biosecurity system against future challenges.



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Diseases of backyard pigs in New Zealand

Introduction

From a surveillance perspective, backyard pigs could provide the vital early warning of an exotic disease incursion. The fact that quasi-commercial pig enterprises outnumber commercial premises by as much as 50 to 1 (Neumann, 2013) reinforces the point that lapses in biosecurity in the backyard pig industry are likely to put the whole New Zealand pig industry at risk. So it is important for veterinarians 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 via 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 herd;
- depression and loss of appetite;
- increase in abortion and stillbirths;
- nervous signs;
- lameness, skin erosions, vesicles, or skin discolouration; and
- sneezing, coughing and diarrhoea.

Unlike exotic diseases in other species, morbidity and mortality are not always high in pigs, and disease outbreaks are likely to cluster in one age group or a number of production groups. If you suspect exotic disease, contact MPI via the exotic pest and disease hotline, 0800 80 99 66.

Measures to prevent the introduction of exotic disease to NZ include restrictions on feeding food waste to pigs; for further information see <http://www.mpi.govt.nz/processing/animal-feed-and-pet-food/restrictions-on-feeding-food-waste-to-pigs/>.

Like all livestock industries, the pig industry has its own terminology. Many of the procedures and diseases discussed here are age specific. It is therefore important to understand the common names used to describe pigs of various ages, weights, and uses, and some definitions are provided below. There can be considerable overlap in the usage of

these terms; the following definitions are provided only as guidance.

- Sows or breeding sows: Adult females having given birth to one or more litters of piglets.
- Boars or breeding boars: Adult males kept for the purposes of mating or semen collection.
- Choppers: Adult sows or boars destined for culling.
- Gilts: Young adult female pigs prior to giving birth to their first litter of piglets.
- Neonates, suckers, or piglets: Pigs from the time of birth (1–1.5 kg) until weaning (5–10 kg, 3–6 weeks of age).
- Weaners or nursery pigs: Pigs from the time of weaning through to 10–12 weeks of age (10–25 kg).
- Grow-finish pigs, growers, or finishers: Pigs from weaner age through to market weight at 18–26 weeks of age (65–110 kg). Pigs are commonly referred to as “growers” during the first half of the grow-finish period and “finishers” during the latter half.

Clinical examination

Clinical examination of pigs is challenging and requires a calm and patient approach and the use of appropriate restraint techniques (Jackson & Cockcroft, 2005). Examination of relaxed pigs from a distance is just as important as direct physical examination. Key observations should include behaviour (pigs are naturally inquisitive and explore their world with

their noses), body shape and condition, superficial swellings, skin abnormalities, diarrhoea, neurological signs, mammary conformation, respiratory signs and lameness (Jackson & Cockcroft, 2005).

Methods of restraint

The safe and effective restraint of pigs in a backyard setup can be a significant challenge to veterinarians. The two available methods are physical and chemical, but in order to carefully inject a pig with a sedative or anaesthetic drug you must first physically restrain it.

Although it may be possible to adapt cattle or sheep stockyards for pigs, the gapping, strength and height of the lowest rail often mean that a pig can get its snout through and escape. The two most effective physical methods of handling and restraining pigs are pig boards and pig snares.

Pig boards come in a variety of sizes, and for ease of use and cleaning the MS Schippers sort-boards are highly recommended (www.msschippers.com). These are available through Anquip NZ Ltd. (<http://anquip.co.nz/products/farm-equipment/sort-boards/>) and Shoof International Ltd (<http://shoof.co.nz/>).

Pig boards can be used to restrain pigs before snaring, sedating, injecting, or for a quick clinical examination (**Figure 1**). The key to using them is to keep the bottom edge of the board firmly in contact with the ground and to brace your knee behind it (**Figure 2**). Several people with pig boards working together can rapidly become quite proficient at moving pigs.



Figure 1: Using pig boards to move pigs



Figure 2: Using the knee to brace a pig board

Snares are the preferred method for physically restraining pigs and can be sufficient on their own for de-tusking, nose-ringing, blood-sampling, injecting, tagging and foot-trimming. The snare can be either soft rope (Shoof International Ltd 9 mm flat-braid calving rope is ideal) or wire rope; the latter can have either a short or long handle (<http://shoof.co.nz/>). The snare is placed over the top jaw with the loop behind the upper canines or tusks. The snare should be cinched down and held tightly. There should be enough room in the restraint area so that both the pig and the person holding the snare can maintain backward traction without backing into a gate, fencing, or a wall. Typically, within a few seconds of applying the snare, the holder and the pig will reach a sort of equilibrium that results in each applying a steady backward pull, without any side-to-side or back-to-front movement. Large pigs can pull with substantial force and it is important that both the holder and pig have good footing. On slippery surfaces (which are also a hazard to the holder), the pig will tend to back-

pedal its front legs, causing it to become quite agitated and not reach the pulling equilibrium that is important for good restraint. While some people experienced with pigs choose to attach the snare to a fixed object such as a gate, this is best avoided as it can be difficult to get the snare detached if a quick release from restraint is required. The holder should aim to elevate the snare just enough to keep the head elevated at about 35° to the horizontal (**Figure 3**). If the snare is too high, the pig may start lunging forward to get better purchase with its front legs. Earmuffs should be worn to prevent hearing damage when snaring pigs. When using a rope snare you must always have a pair of needle-nose pliers available to hold the loop on the snout before releasing the pig from the snare.

The preferred method of chemical restraint is the TKX mixture, known in NZ as “Eric’s mix”. A 5 ml vial of Zoletil 100 powder (250 mg tiletamine and 250 mg zolazepam; Virbac NZ) is reconstituted with 2.5 ml ketamine (100 mg/ml) and 2.5 ml xylazine 10 percent (100 mg/ml) to give a final concentration of 50 mg/ml of each (Ko *et al.*, 1993). The dose rate of the mixture is about 1 ml per 20 kg IM (or as required to get the necessary effect), which produces very heavy sedation/light anaesthesia for 30–60 minutes. Onset of sedation is rapid, with lateral recumbency achieved in about 5 minutes. The mixture can be given IV to deepen the plane of anaesthesia or increase its duration. Recovery can be quite prolonged (up to 3–4 hours) or longer in obese pigs, including NZ’s Kunekune breed; use

of yohimbine (0.2 mg/kg IV) has been reported to shorten recovery times (Parris-Garcia *et al.*, 2014). Meloxicam at 0.4 mg/kg IM is a registered analgesic for pigs in NZ and should always be considered for any painful procedure where xylazine sedation is to be reversed with yohimbine.

Endemic diseases of pigs

Respiratory system

Pigs are susceptible to several infectious respiratory diseases that are endemic in New Zealand. Backyard pigs are often predisposed to infection from substandard husbandry when kept in dusty or cold, damp and draughty housing. Inadequate dry bedding and poor ventilation will most certainly result in respiratory disease, particularly in the winter months. A pig with pneumonia is often readily identified by a moist, persistent cough, laboured breathing even at rest, and is often hairy and poorly grown.

Ascaris suum

Often pneumonia in young backyard pigs is caused by migration through the lungs of larval stages of internal parasites such as *Ascaris suum* (see page 12). Pigs will persistently cough, have poor growth and body condition, and can die acutely. If they are housed in areas that are continually in use and not adequately disinfected between cohorts, parasite loads can persist in the environment and infect naive animals. Diffuse haemorrhagic lesions in the lungs on post-mortem examination, in combination with “milk spots” on the liver (**Figure 4**, **Figure 17**), should increase suspicion of a verminous pneumonia (Johnstone, 2018).

Actinobacillus pleuropneumoniae

There are several endemic strains of this agent in the commercial pig sector (Hilbink, 1992; Reichel, 1997), but the prevalence in the backyard pig population is unknown. However, this organism is likely to be present owing to the transfer of pigs from commercial piggeries into the backyard pig population and movements of backyard-raised animals. It is transmitted both directly and indirectly, so good quarantine and biosecurity standards are essential for prevention.

A. pleuropneumoniae typically affects pigs at 8–16 weeks, when maternal immunity wanes, and causes sudden



Figure 3: Soft rope snare for examining a pig



Figure 4: Haemorrhagic lesions in the cranial and apical lung lobes and “milk spots” on the liver from *Ascaris suum* larval migration (source: Jan Jourquin, Elanco)



Figure 5: *Actinobacillus pleuropneumoniae* infection showing distinctive lung lesions (source: Massey University Pathobiology Department)

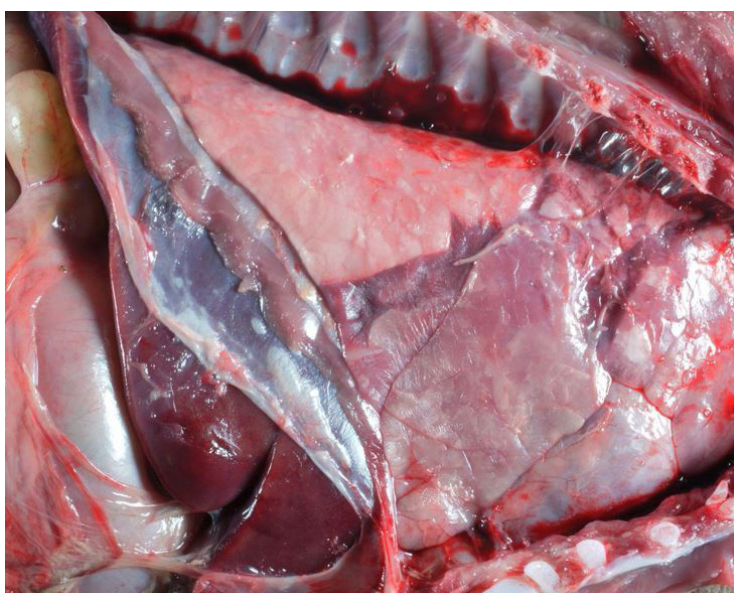


Figure 6: Enzootic pneumonia, likely including *M. hyopneumoniae* (source: Massey University Pathobiology Department)

death of varying incidence, often with no clinical signs other than blood and froth from the nostrils. The incubation period can be as short as 12 hours (The Pig Site, 2018a). At postmortem the lung lesions are distinctive (**Figure 5**) and there is evidence of pleurisy. Serological testing is available but there is no vaccine available in NZ.

***Mycoplasma hyopneumoniae* (enzootic pneumonia)**

This is one of many pig respiratory pathogens that may act as component causes in what ultimately manifests as pneumonia in a piggery (The Pig Site, 2018b). It causes enzootic pneumonia, which is present in most NZ pig populations. Vaccination is used for prevention and management (Stark *et al.*, 1998).

Enzootic pneumonia (EP) is most readily identified by a non-productive cough, typically without any nasal discharge. Post-mortem lung inspection (**Figure 6**) and laboratory testing are needed to differentiate EP from other diseases such as swine influenza (caused by *A. pleuropneumoniae*), and Glasser's disease (caused by *Haemophilus parasuis* – see below), as *M. hyopneumoniae* infection can predispose pigs to other respiratory pathogens.

***Haemophilus parasuis* (Glasser's disease)**

This is another respiratory disease in backyard pig herds (Fairley, 1997). Much like *A. pleuropneumoniae*, it is endemic in herds and most common in pigs 8–16 weeks old, at which age maternal immunity wanes (The Pig Site, 2018d). Affected animals display short bursts of coughing and typically become febrile and depressed in a short period of time. Affected pigs can also have pericarditis, peritonitis, polyarthritis, meningitis and pleurisy (**Figure 7, page 8**) (Fairley, 1997).

Dermatological

Mange

Pig mange is caused by *Sarcoptes scabiei* and is the most common parasitic skin disease of pigs in NZ (Beakenridge, 1958). The prevalence of mange was found to be highest in small herds farmed outdoors and in pigs farrowing in the summer (Christensen & Cullinane, 1990). Highly pruritic skin lesions first appear around and inside the ears (**Figure 8**) and neck 2–3 weeks after the pig becomes infested, but more generalised lesions develop as the condition becomes chronic. The scrotum of an adult boar may also become severely affected. Initially the pruritus is caused by the burrowing activities of the mite, but later on, as the initial lesions regress, a hypersensitivity reaction is responsible. Affected animals are constantly rubbing and head-shaking, which can result in aural haematomas (pillow ear). The skin can initially have a shiny appearance from released sebum, but progresses to become crusty and hyperkeratotic. Diagnosis is relatively easy, using skin scrapes from around or inside the ear.

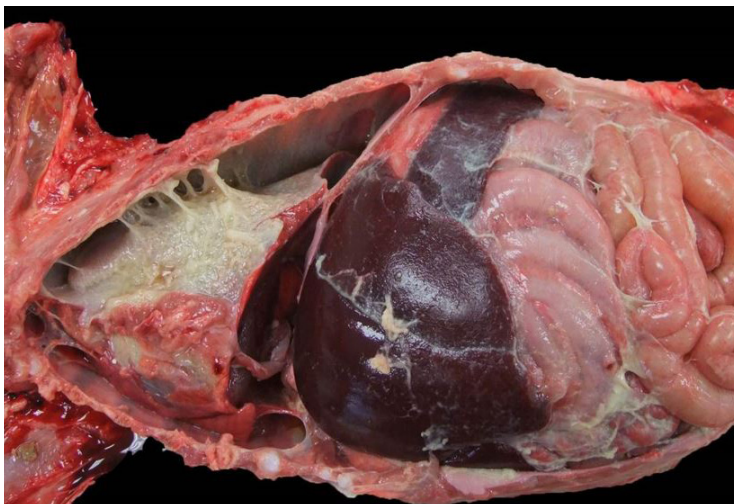


Figure 7: Glasser's disease, showing fibrin in the peritoneal cavity (*fibrinous peritonitis*) and pleural cavity (*fibrinous pleuritis*) (source: Massey University Pathobiology Department)



Figure 8: Early active *Sarcoptes scabiei* infection in the ear of a weaner pig (NADIS, 2018a) (source: NADIS & MEC White)

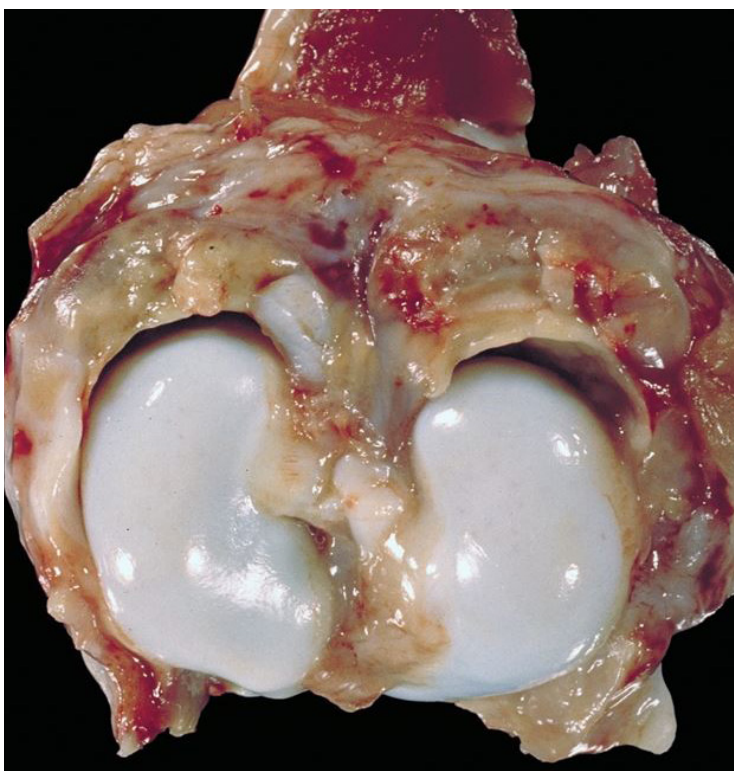


Figure 9: Septic joint associated with *Staphylococcus aureus* infection (source: Massey University Pathobiology Department)

Lice

The pig louse *Haematopinus suis* is yellowish-brown and 5 mm long, easily visible to the naked eye (Heath, 2002). Lice are easily seen on light-coloured pigs and often found around the neck, jowls, ears (inside, outside and on the base), thighs and flanks. Affected pigs may show severe irritation, which can lead to localised damage and ulceration of the pinna (where the lice like to congregate and feed).

Multi-systemic diseases and lameness

Pigs can present with generalised clinical signs such as fever, depression, lameness, skin lesions and joint swelling (**Figure 9**). These signs can be caused by viruses, bacteria, fungal toxins and even plant compounds. Febrile and depressed pigs will seek heat and avoid going out of their sheds or huts. Lamé pigs with multiple affected joints will often have a very distinct, stilted, stiff gait and arched back.

Swine erysipelas

This is a common disease with several clinical manifestations, caused by infection with *Erysipelothrix rhusiopathiae*. This bacterium is ubiquitous and a large percentage of pigs carry it in their tonsils. The disease can affect any pig on a farm, but usually it is pigs over 12 weeks of age and unvaccinated gilts that show the most severe clinical signs such as high fever, off feed, and then sudden death or abortion (The Pig Site, 2018c). When seen, the characteristic diamond-shaped skin lesions are pathognomonic for the disease (**Figure 10, page 9**).

Acutely affected pigs will be febrile, off feed and have a stiff, stilted gait with joint heat and effusion. *E. rhusiopathiae* infection can have a significant effect on breeding boars, where the fever may impact fertility and lead to small litter sizes and delayed returns to service in healthy gilts/sows. Chronic erysipelas infection can also result in joint damage, chronic arthritis, lameness (**Figure 11, page 9**) and valvular endocarditis (**Figure 12, page 9**) (Thurley, 1971).

Testing is available but the clinical signs, diamond-shaped skin lesions, and prompt response to therapy with injectable penicillin are often sufficient to make the diagnosis (NADIS, 2018b).

Arthritis caused by *Mycoplasma hyorhinis* and *M. hyosynoviae*

Both these mycoplasmas are associated with lameness and (rarely) pneumonia in pigs (Davenport *et al.*, 1970; The Pig Site, 2018e). While *M. hyorhinis* typically affects pigs 3–10 weeks old, *M. hyosynoviae* tends to cause disease in older animals (12–24 weeks). Both organisms are widespread and can be identified in healthy and diseased pigs, but triggers of clinical disease are poorly understood. Importantly, these organisms should be considered quite separately



Figure 10: Skin lesions associated with *Erysipelothrix rhusiopathiae* infection (source: Massey University Pathobiology Department)



Figure 11: Crippling, irreversible arthritis in a weaner pig (NADIS (2018b) (source: WJ Smith)

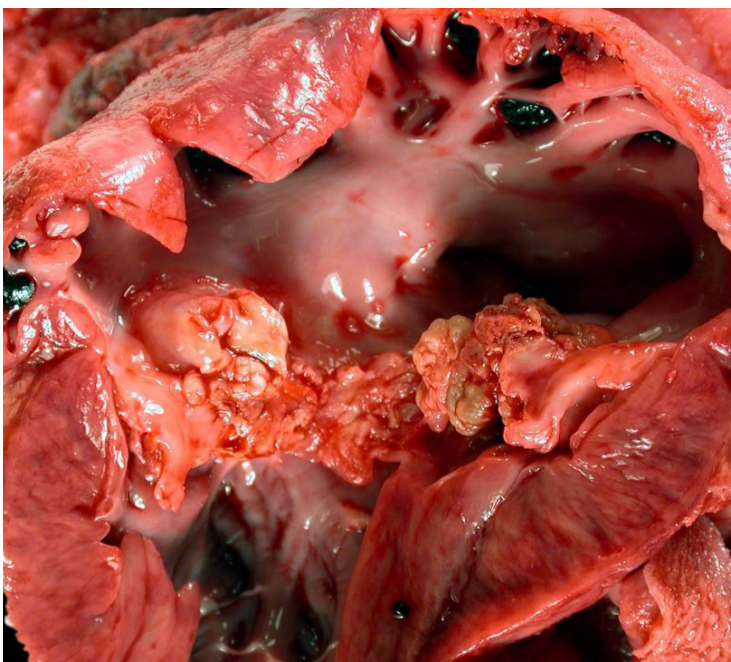


Figure 12: Valvular endocarditis secondary to *Erysipelothrix rhusiopathiae* infection (source: Massey University Pathobiology Department)

from *M. hyopneumoniae* (the cause of enzootic pneumonia), which is only found in the lungs and is never associated with lameness.

Acutely affected pigs are visibly ill, lame, and often have fever. Along with causing an infectious arthritis, these agents can cause fibrinous peritonitis that may produce abdominal pain and a preference to lie in sternal recumbency. Arthritis, often severe, may be found in one or more joints and as the disease progresses will be accompanied by villous hyperplasia in affected joints. Affected pigs should be treated with a combination of antibiotics and pain relief. Vaccines are not available for either organism.

Mycotoxicosis

Pigs in backyard settings may show signs of mycotoxicosis, caused by exposure to stored grains or other foods fed as a component of waste food or swill. In particular, bakery and brewery waste products, if poorly stored, can be a dangerous source of mycotoxins. Though mouldy or rotten waste foods are an obvious risk, many mycotoxins (as distinct from the mould organisms themselves) may not be detected by visual inspection or smell. Pigs of all ages are susceptible to mycotoxins and grow-finish pigs will often have poor growth, a rough hair coat, and are frequently lethargic. In breeding pigs, numerous clinical signs are possible including infertility, anoestrous, abortion, swollen and reddened vulva, and others depending on the specific toxin that is present (Table 1, page 10) (The Pig Site, 2017). It should be noted that each of these fungal genera has several species, not all of which are toxic (The Pig Site, 2017). Pigs can be discerning eaters and one of the first hints of the presence of mycotoxins in feed is refusal of food.

Incidents of bullae and vesicles on the snouts, feet, ventrum and udders of white-skinned pigs in NZ have been linked to contact with green vegetable material containing parsnips, celery or giant hogweed (Montgomery *et al.*, 1987a). Parsnips and celery are known to contain furocoumarins, which are potent phototoxins. It has been suggested that lesions are caused by absorption of furocoumarins from these plants into the skin of the snout and feet, followed by exposure to ultraviolet light (Montgomery *et al.*, 1987b). Furocoumarin toxicity is a very important differential for exotic vesicular diseases of pigs (including foot-and-mouth disease) and because many pigs in New Zealand are outdoors or semi-outdoors and fed food scraps, this should be considered whenever vesicles or blisters are present.

Table 1: Common mycotoxins and related clinical signs (The Pig Site, 2017 – MR Moorhead & TJL Alexander)

Fungus	Toxins	Level of no clinical effect	Toxic level	Clinical signs
<i>Aspergillus</i> spp.	Aflatoxins	< 100 ppb	300–2 000 ppb	Poor growth, liver damage, jaundice, immunosuppression
<i>Aspergillus</i> spp. & <i>Penicillium</i> spp.	Ochratoxin & citrinin	< 100 ppb	200–4 000 ppb	Reduced growth, thirst, kidney damage
<i>Fusarium</i> spp.	Deoxynivalenol, diacetoxyscirpenol, T-2 toxin	< 2 ppm	4–20 ppm	Reduced feed intake, immunosuppression, vomiting
<i>Fusarium</i> spp.	Zearalenone	< 0.05 ppm	1–30 ppm	Infertility, anoestrous, prolapse, pseudopregnancy
<i>Fusarium</i> spp.	Fumonisin	< 10 ppm	20–175 ppm	Reduced feed intake, respiratory signs, fluid in lungs, abortion
Ergot	Ergotoxin	< 0.05%	0.1–1% ergot bodies (sclerotia) by weight	Reduced feed intake, gangrene of the extremities, agalactia caused by mammary-gland failure



Figure 13: PMWS infection in a 3-month-old Saddleback pig. The superficial lymph node is oedematous and enlarged (source: Massey University Pathobiology Department)



Figure 14: PMWS infection in a 3-month-old Saddleback pig. The lungs are diffusely firm and mottled dark red, with interlobular septae expanded by clear fluid (source: Massey University Pathobiology Department).

Post-weaning multisystemic wasting syndrome (PMWS)

This comparatively new disease of New Zealand pigs was first diagnosed in October 2003 (Rawdon *et al.*, 2004; Stone, 2004). It is associated with porcine circovirus type 2 (PCV2) and presents as wasting, mainly in weaned pigs 8–14 weeks of age (Jaros, 2007). Although still known as PMWS in NZ, worldwide there is a growing preference to call it porcine circovirus associated disease (PCVAD), to indicate the clear association between PCV2 and the clinical disease. Clinical signs in weaner pigs usually include ill-thrift, dyspnoea, visibly enlarged lymph nodes and, less frequently, diarrhoea and icterus. In acute outbreaks 4–10 percent of pigs may be affected (range 1–60), of which 70–80 percent usually die (range 50–100) (Bryce, 2002). No treatment is successful and severely affected pigs should be euthanased. Prevention is by vaccinating piglets before 3 weeks of age (highly effective vaccines are available) and diagnosis is confirmed by post-mortem examination. Typical signs seen at postmortem include cachexia, pale and jaundiced skin, enlarged oedematous superficial lymph nodes (Figure 13), non-collapsed rubbery lungs with interstitial oedema (Figure 14), gastric ulceration and fluid-filled lower intestine (Jaros, 2007).

Leptospirosis

Pigs are the recognised maintenance hosts in NZ for *Leptospira* serovars Pomona and Tarassovi (Marshall & Manktelow, 2002). The organism may circulate at very low levels in commercial grow-finish and adult pigs, where the disease is rare thanks to good hygiene, routine use of vaccines, rodent control and a predominance of indoor production. In backyard settings, however, where risk factors are not tightly controlled, the organism may be more common and

clinical disease is more likely to occur. In these circumstances, the two groups most likely to show clinical signs are young weaner piglets and pregnant sows. The clinical signs of acute infection in young pigs are mild and very non-specific, with affected animals showing anorexia, pyrexia and listlessness. Chronically infected pregnant sows may suffer from abortion and low litter sizes, and give birth to non-viable piglets.

Concerns about the zoonotic risks of leptospirosis have led to the introduction of a voluntary leptospirosis control programme by the NZ Pork Industry Board, which requires vaccination of the breeding herd at least every 6 months, and certification of the grow-finish herd as free of leptospirosis at least once every 12 months. Grow-finish herd certification is based on the results of serological testing of at least 10 grow-finish pigs, either within 2 weeks of slaughter or at slaughter, using the microagglutination test (MAT) for *Leptospira Pomona*. Based on the results of this serological monitoring, exposure of pigs to *Leptospira* spp. on commercial pig farms is confirmed as low, as is the likelihood of the organism being present in slaughtered pigs. Likewise, pigs are likely to have a negligible role as a maintenance host for those *Leptospira* spp. that are more commonly found in NZ cattle or sheep. However, the extent of vaccine use in backyard pig farms is unknown (though likely low), so these pigs cannot be assumed to have similar low infection rates to commercial herds and as such represent a potential health risk to veterinarians, pig owners and anyone involved in butchering.

Trichinella spiralis

This is the causal agent of trichinellosis, which may be present in feral pigs (although never proven) and backyard piggeries, but at extremely low prevalence; no recent data are available to document its occurrence. When infection does occur, it is likely the result of the pig's having eaten the carcass of infected feral animals such as Norway or brown rats (*Rattus norvegicus*), cats and mustelids. By the same pathway, it is possible that non-commercial pigs could become infected. Since these rats are common in urban areas and on farms, poorly maintained backyard pig setups could be at risk. Interestingly, the ship rat (*Rattus rattus*), which is the common

rat in New Zealand forests, may not be a suitable intermediate host for *Trichinella spiralis* (W. Pomroy, pers. comm.). However, there is no ongoing systematic surveillance in feral, non-commercial or commercial pigs. Previous targeted surveillance testing in commercial abattoirs has never detected *T. spiralis* in a commercially raised pig (Richardson, 2006) and the parasite is not considered a significant cause of zoonotic disease here (Liberona & MacDiarmid, 1988).

The four confirmed reports of human trichinellosis have all been linked to backyard pig production (Paterson *et al.*, 1997; Thornton & King, 2004; Richardson, 2006). Trichinellosis in humans is transmitted by eating infected pig meat that has not been properly cooked (> 60°C) or frozen for long enough (-15°C for > 20 days) to kill the parasitic cysts present in the muscles. Symptoms of the disease in humans include fever, nausea, diarrhoea, vomiting and abdominal pain. These may be followed by headaches, fever, chills, coughs, aversion to bright light, swollen or puffy eyes, aching muscles and sometimes a rash or skin irritation. Abdominal symptoms can occur 1–2 days after infection. Further symptoms usually start 2–8 weeks after eating contaminated meat (Ministry for Primary Industries, 2018).

Affected pigs do not show clinical signs. Veterinarians should help clients with backyard piggeries to minimise the risk of exposure by encouraging good vermin control, storing feed in vermin-proof containers, fencing to avoid contact with feral pigs, and eliminating exposure to uncooked meat and the carcasses

of deceased pen-mates (or any other mammalian carcass).

Neurological Meningitis

Streptococcus suis is a Gram-positive coccus and a common cause of bacterial infection of pigs; the Type 2 strain is known to be present in New Zealand and is among the most common causes of meningitis in weaner and grow-finish pigs. Affected pigs show signs of inco-ordination, staggering, dog-sitting, lateral recumbency, paddling, opisthotonus and tetanic convulsions or sudden death. *S. suis* can also be associated with bronchopneumonia, epicarditis (**Figure 15**), septicaemia and polyarthritis. The organisms can be isolated from tonsils, and healthy tonsillar carriers play an important role in the spread of the disease (Robertson, 1985). *S. suis* is zoonotic (Hughes *et al.*, 2009), with infection acquired through handling pigs or fresh meat, and has been the cause of meningitis and an associated permanent vestibular and auditory disturbance in humans (Zanen & Engel, 1975; James *et al.*, 2009).

Salt poisoning

Pigs have high daily requirements for clean fresh water, which must be supplied at all times as very little of the water found in a pig wallow is actually consumed. Grow-finish pigs require up to 10 litres per day (1 litre/10 kg LW/day) and lactating sows require 30–45 litres daily, depending on weather. Failure to provide adequate water can rapidly lead to salt poisoning, a common problem in backyard pigs (Herriot, 1981). Clinical signs start within 48 hours of

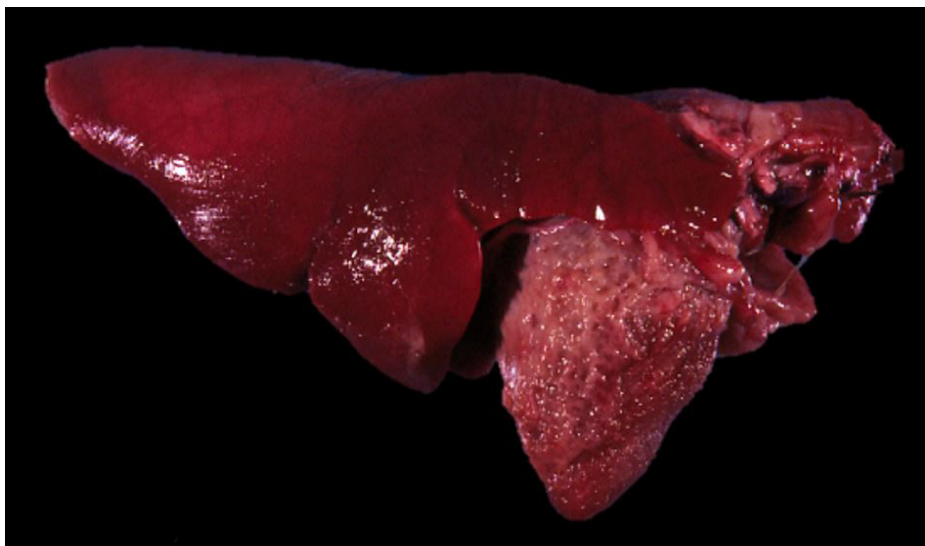


Figure 15: Epicarditis associated with *Streptococcus suis* infection (source: Massey University Pathobiology Department)

water supply failure. Initially affected pigs appear deaf, blind and oblivious to their surroundings, causing them to wander aimlessly, bump into objects and pivot on one leg, before progressing to seizures, convulsions, coma and death. Practitioners should always consider the quality and supply of water on backyard pig properties. Whenever pigs are fed whey or other high-salt waste-food products, a separate supply of clean fresh water must be available.

Gastrointestinal tract

Piglet diarrhoea

One of the most common diseases in a backyard pig setup is piglet diarrhoea. It can occur pre- or post-weaning and in the former case it is important to rule out whether the dam is suffering from mastitis, metritis and agalactia (MMA) since the piglets from a sow with MMA may also present with diarrhoea. In the backyard situation, rotavirus, enterotoxigenic *Escherichia coli* (K88) and coccidiosis (*Isospora suis* and *Eimeria* spp.) are probably the most common causes of pre-weaning diarrhoea. *Isospora suis* is associated with early onset diarrhoea and *Eimeria* with later-onset diarrhoea, close to weaning (W. Pomroy, pers. comm.). Diarrhoea caused by *E. coli* can occur at any time after 24 hours of age. However, diarrhoea caused by coccidiosis will not occur until at least 4–5 days of age as it takes that long for the parasite to reach the point in its life cycle where diarrhoea is produced. In addition to *E. coli*, neonatal diarrhoea at < 3 days of age can also be due to *Clostridium perfringens* type A or rotavirus. Whereas *E. coli*, rotavirus and *C. perfringens* type A never produce haemorrhagic diarrhoea, *C. perfringens* type C can produce acute, hemorrhagic and highly fatal diarrhoea in this age group, though this organism appears to be very uncommon in New Zealand. When encountering an outbreak of piglet diarrhoea with extremely high morbidity or mortality rates (80–100 percent), the practitioner should always consider contacting MPI via the exotic disease hotline.

Post-weaning diarrhoea is often associated with poor formulation and feeding of post-weaning diets (Hampson & Beban, 1985). The provision of clean *ad lib* water is also important. The most common cause of post-weaning diarrhoea is *E. coli* (colibacillosis), which

presents within 10 days of weaning as a watery diarrhoea of any colour. Oedema disease is also caused by *E. coli*, but instead of diarrhoea the affected piglets develop neurological signs, eyelid oedema, and have changed vocalisation.

Grower diarrhoea

Classic swine dysentery or bloody dysentery is caused by *Brachyspira hyodysenteriae* and is considered to be uncommon in NZ (Pearce & Smith, 1975). Disease usually presents in grower and finisher pigs with pyrexia, anorexia and greyish-yellow faeces flecked with blood (Figure 16). The diarrhoea progresses to a muco-haemorrhagic colitis with high morbidity and low mortality. A more typical diarrhoea, spirochaetal colitis, is caused by a related organism, *Brachyspira pilosicoli*. However, aside from causing intermittent diarrhoea (very soft faeces, of normal to grey colour, without blood), this disease appears to have a very minor effect on the overall health or productivity of affected pigs.

Porcine proliferative enteropathy, also commonly known as ileitis, is an enteric disease of pigs worldwide. It is characterised by thickening of the small intestine that particularly affects the terminal ileum. After extensive confusion about the cause of ileitis, the agent is now known to be a novel intracellular enteropathogen called *Lawsonia intracellularis*. Ileitis occurs as subclinical disease, in chronic forms (necrotic enteritis or NE and porcine intestinal adenopathy or PIA), and as an acute haemorrhagic form (porcine haemorrhagic enteropathy). Ileitis

generally does not resolve without antibiotic therapy.

Hyoststrongylus rubidus

Also known as the red stomach worm, this is a member of the family Trichostrongyloidea. The worm is red, hairlike, about 10 mm long and found unattached on the surface of the stomach. The life cycle is direct and can be a problem in backyard pig setups. Ingested larvae invade the gastric glands for two moults before emerging as immature adult nematodes. They share a similar life cycle to *Ostertagia/Teladorsagia* in ruminants and many of the pathological changes are similar. These include mucosal hyperplasia and metaplasia. In addition, infection has been associated with gastric ulceration and anaemia, with weaner and early grower pigs showing ill-thrift, weight loss and diarrhoea, and breeding sows showing weight loss and intermittent melena following gastric haemorrhage (Dodd, 1960). This is an extension of the pathological changes with disrupted cell junctions causing ulcers to form. Treatment with broad-spectrum anthelmintics is effective, and implementing a control programme for *Ascaris suum* (see below) will also prevent this worm from establishing.

Trichuris suis and *Ascaris suum*

Infection by both *Trichuris suis* (pig whipworm) and *Ascaris suum* (pig roundworm) is transmitted by infective eggs rather than infective larvae such as occurs with *Hyoststrongylus rubidus*. This makes parasite control in backyard pigs difficult because the infective eggs are long-lived (2 years for *T. suis*, 10 years for *A. suum*; Roepstorff & Murrell, 1997).



Figure 16: Swine dysentery (source: Massey University Pathobiology Department)

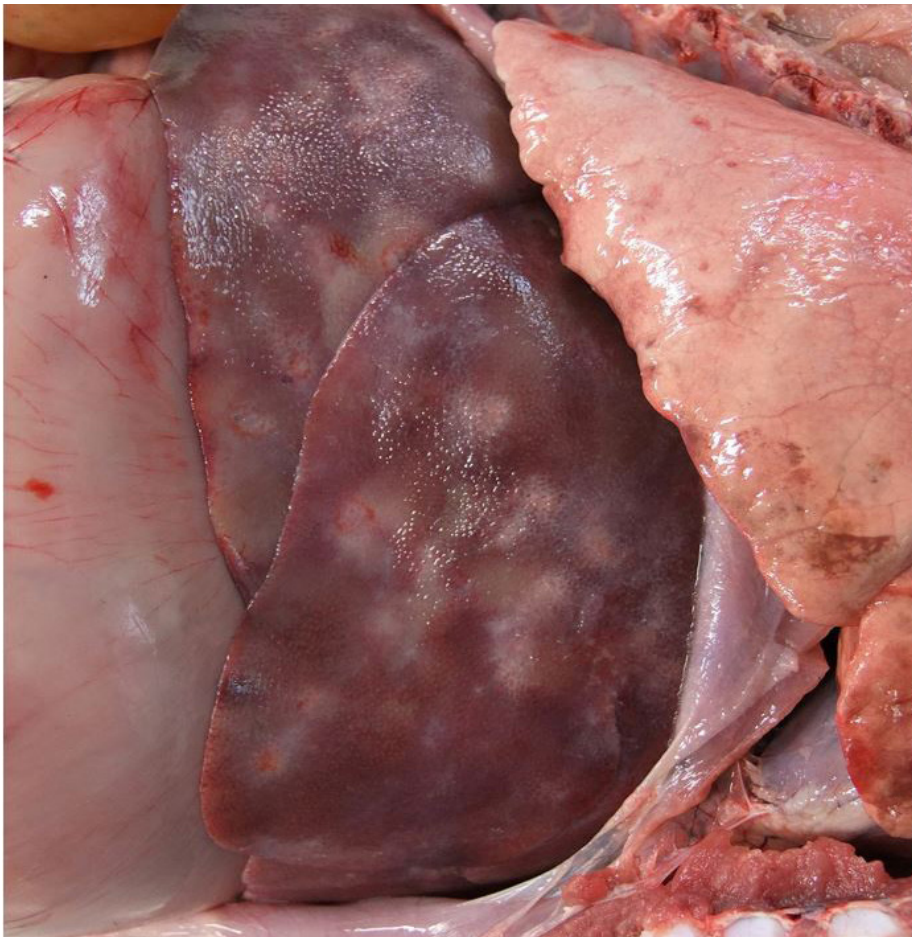


Figure 17: Severe milk spot lesions on liver, associated with migration of *Ascaris suum* larvae (source: Massey University Pathobiology Department)

Heavy *T. suis* and *A. suum* infections cause gastrointestinal disturbances and ill-thrift in young pigs (Elliott & Robinson, 1972). *T. suis* parasitises the caecum and the life cycle is direct. Eggs are passed in the faeces; the L1 larvae develop inside the egg and are released when ingested. *A. suum* parasitises the small intestine and the life cycle is also direct, although on ingestion the larvae migrate through the liver and then the lungs, finally establishing in the small intestine after being coughed up and swallowed. This migration causes considerable damage, sometimes resulting in reduced growth rates and illness, and can be seen as fibrosis of the liver, known as milk spot (**Figure 4**, **Figure 17**). Results from scoring the presence and severity of milk spot on post-mortem inspection at abattoirs are used by commercial piggeries to monitor the effectiveness of their *A. suum* control strategies. Without this option, levels of infection and pasture contamination in backyard pig setups can reach very high levels (Nejsum *et al.*, 2012). Injected macrocytic lactones are highly effective against *A. suum* but not against *T. suis* (Schillhorn & Gibson, 1983); control of

T. suis is better achieved with in-feed anthelmintics. A good option, with good broad-spectrum efficacy for the control of all parasites in backyard pigs, is flubendazole administered in the feed. Flubendazole is a benzimidazole and the dose rate is 5 mg/kg orally twice a year.

Concluding remarks

For a veterinarian approaching disease in backyard pigs in NZ it is important to look at the wider context, including the source of the pigs, the food being fed, housing and the breed and age of pigs on the property and surrounding properties. Once a broad picture is obtained of the herd and the particular risks to which it is exposed, any clinical signs seen can be assessed in relation to the most common likely causes as outlined in this article. Disease testing is often expensive and infrequently done in the backyard pig setting. Instead, in many cases, post-mortem examination becomes the default method for diagnosis. Many vaccines are also difficult to source in quantities that are economic for small producers, and many medications are off-label for pigs. Despite these challenges it is possible to obtain information and help from the

MPI Incursion Investigation team as part of routine surveillance.

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Quarterly report of diagnostic cases: January to March 2018

Gribbles Veterinary Pathology Bovine

A 3-month-old Friesian cross bull calf from Northland lost weight progressively and died. At postmortem there were scattered abomasal ulcers, but little else was reported. The main findings on histopathology were serous atrophy of fat, severe enteritis with villous blunting or loss, and multiple protozoan life stages, consistent with severe coccidiosis.

A group of yearling Murray Grey bulls from Northland lost condition over several weeks and became very weak. Three died, two during stock movement. A very high tick burden was identified on all the bulls and post-mortem examination of one animal revealed pale, slightly icteric mucous membranes and nutmeg liver. Subsequent histological examination of the liver revealed centrilobular necrosis, consistent with ischaemic damage caused by anaemia. Three live bulls had a PCV of 0.14–0.18 when tested by the referring veterinarian and these were confirmed to be between 0.13 and 0.19 (reference range 0.24–0.4) when the samples were received at the laboratory, with concurrent low haemoglobin (51–64 g/L, reference range 90–150) and *Theileria* sp. was seen on erythrocytes. The diagnosis was theileriosis, possibly exacerbated by the heavy tick burden.

Five of 15 six-week-old Friesian cross calves from the Rodney district became recumbent and died over several days. Post-mortem examination of one calf revealed ascites, hydrothorax, haemorrhage in the perirenal fat, and apparent abomasitis. Acorn toxicity was suspected on clinical grounds. Fragments of acorn were found in the abomasum. Histologically, the kidney showed multifocal acute tubular necrosis, mild interstitial nephritis and oedema, consistent with nephrosis caused by **acorn toxicity**.

Three dairy cows from the Auckland region aborted in the last trimester (6 weeks from the expected date of parturition). PCR of pooled serum from

two cows was negative for bovine viral diarrhoea (BVD) virus and IFA tests for *Neospora* were also negative. A post-mortem examination and histopathology on one calf revealed no significant findings, but subsequent histopathology of placenta from two calves revealed multifocal necrosis, neutrophilic infiltrates and infiltrating fungal hyphae, suggestive of **mycotic abortion**. Most fungal abortions in cattle are sporadic and related to feeding mouldy hay or poor-quality silage.

Ten out of 30 rising-2-year-old bulls and heifers presented with multifocal ulcerated cutaneous nodules on the flank, neck and leg regions. Histopathology was consistent with a pyogranulomatous dermatitis with intralesional bacteria and Splendore-Hoeppli material. Culture of one of the nodules yielded an *Actinobacillus* species. A diagnosis of **cutaneous actinobacillosis** was made. Although this disease is classically known for causing oral lesions (woody tongue), it can also cause cutaneous lesions after traumatic injury.

Formalin-fixed tissues were received from a rising-2-year-old Angus heifer, one of 60 on a South Canterbury beef farm. It had been noticed to be light in condition for a week and then became blind as the result of corneal oedema. There were areas of erosion and ulceration under the tail and around the anus and vulva. There were no oral ulcers and no ocular or nasal discharge. The animal was euthanased and a range of fixed tissues were submitted for histological examination. Lesions of lymphocytic vasculitis typical of **malignant catarrhal fever** caused by a sheep virus, **ovine herpesvirus-2**, were prominent in the brain, abomasum, intestine, and kidney.

A South Canterbury veterinarian was called to investigate illness in a group of 30 two-year-old cattle that had been grazing a paddock of burnt stubble. Eight were sick and four had died. The affected animals were observed to be depressed and in sternal recumbency with melaena before they died. A variety of tissues were collected and fixed in formalin.

The tissues were in a very poor state of preservation with marked putrefaction but the kidney was preserved sufficiently to allow identification of acute tubular necrosis. The animals had not eaten acorns and the paddock they were in had been grazed clean. It was suspected, but could not be proven, that they had been poisoned by *Amaranthus retroflexus* (redroot), a weed that grows among crops and on wasteland. This is a known but uncommon cause of renal tubular necrosis of cattle in New Zealand.

When pregnancy-scanning a group of 650 milking heifers, a mid-Canterbury veterinarian noticed several animals with masses at the junction of the vulval mucosa and the skin. Older cows were not affected and the masses did not appear to have any influence on the reproductive performance of the affected heifers. A biopsy from one animal revealed histological lesions typical of a **fibropapilloma**. This is a papillomavirus-induced lesion and it is likely that it was transmitted to the heifers by a bull.

A hundred mixed-age dairy cows due to calve in 4 weeks were grazing on dryland in the Central Hawke's Bay district when about 14 of them were noted to have lost condition. Nine of the cows aborted and five died. Some that aborted were jaundiced or anaemic on clinical examination and others were dehydrated. Several cows had ticks on them. *Theileria orientalis* was detected by PCR from serum taken from 5/5 cows. In addition, 3/6 cows had titres of 1:800 to > 1:1 600 to *Leptospira Hardjo* and 6/6 had titres of 1:50 to 1:800 to *L. Tarassovi*. The most significant histological findings were hepatic periportal necrosis and cholestasis, compatible with severe haemolytic anaemia. There was also multifocal interstitial nephritis. The final diagnosis was **theileriosis**, causing anaemia, deaths and abortions. An additional role for **leptospirosis** could not be excluded.

A 3-year-old Friesian cow on a Franklin district dairy farm developed neurological dysfunction characterised by decreased menace reflex, head tremor, hind-limb paresis and a droopy

ear. The clinical condition deteriorated over the ensuing 3 days so the cow was euthanased and the brain removed and processed for TSE surveillance. Multifocally, arterioles and venules throughout the forebrain, midbrain and brain stem were surrounded by eosinophilic hyaline material (indicating protein-rich oedema), erythrocytes, eosinophilic globules or basophilic material. There was rarefaction of the neuropil around some of the affected vessels. The lesions were considered characteristic of **clostridial enterotoxaemia**. While more commonly recognised in lambs, similar lesions are occasionally seen in cattle of various ages (Fairley, 2005). The detection of *Clostridium perfringens* type D in a subset of cases suggested a similar pathogenesis to that in sheep, i.e., proliferation of bacteria in the presence of carbohydrate-rich substrate in the intestine, production of toxin, and vascular damage (Jones *et al.*, 2015).

Nine 2–3-year-old cows from an Otorohanga dairy herd aborted early-to-mid-term fetuses over a few days. The herd was on rations of green feed maize, palm kernel expeller and grass. Leptospirosis vaccination was complete. Samples were collected from two fetuses and histology showed focal inflammatory lesions in the brain, heart, muscle and kidney. Culture of stomach contents produced a mixed bacterial growth, while PCR of stomach contents was positive for *Neospora caninum* and negative for bovine viral diarrhoea virus and *Ureaplasma diversum*. These findings confirmed a diagnosis of **neosporosis**.

Four 4-month-old dairy beef calves in a group of 468 on an Otago dairy farm showed neurological signs that included ataxia, blindness and recumbency over a 10-day period in January. One was euthanased and necropsied. No gross lesions were apparent so the brain was removed, fixed in formalin and examined at the laboratory. Histopathological findings were consistent with a diagnosis of **polioencephalomalacia**. Another affected animal was treated with thiamine by injection and recovered.

A group of 600 milking cows on a Southland dairy farm broke into a paddock of HT (herbicide-tolerant) swedes overnight in late March. They were removed from the paddock the next day. Three days later a number of the cows had clinical signs of

photosensitivity. Their udders were most severely affected and their legs were also swollen. Nine of the most severely affected cows were blood-tested and their liver enzymes were measured. Bilirubin varied from 13 to 134 $\mu\text{mol/L}$ (reference range < 8), GGT results were 458–2 123 IU/L (reference range 3–47) and GLDH results were 107–829 IU/L (reference range 5–35), confirming severe liver damage and supporting the clinical diagnosis of **hepatogenous photosensitivity**. In 2014 and 2015 a particular variety of HT swede was associated with a new syndrome in stock that was characterised by ill-thrift, photosensitisation, recumbency, death and periparturient problems, so the recent ingestion of swedes was thought to be relevant in this case.

Four 6-month-old dairy calves out of a group of 200 on a Central Otago dairy farm died over a period of 4 days. They were observed to have increased respiration, depression and pyrexia before dying within a few hours of showing the clinical signs. Necropsy of one recently dead calf showed a large amount of fibrinous peritoneal fluid and adhesions throughout the abdominal cavity. *Pasteurella multocida* was cultured from a sample of the peritoneal fluid. Histopathology revealed a fibrinosuppurative peritonitis with intralesional bacteria, consistent with a diagnosis of **septicaemic pasteurellosis**. There were several outbreaks of **salmonellosis** in milking cows on Southland dairy farms during this period. In one case an unspecified number of cows were affected with diarrhoea. Culture of samples of faeces from three affected cows all yielded *Salmonella* **Typhimurium** **phage type 101**. In another outbreak *S. Typhimurium* **phage type 9** was cultured from the faeces of one of a number of cows that developed a severe, watery diarrhoea after a dietary change from grass silage to whole-crop silage. There was a marked drop in milk production at the same time in this herd. In a third outbreak, 30 percent of 350 milking cows showed pyrexia, diarrhoea and a marked drop in milk production. *S. Typhimurium* **phage type 101** was cultured from the faeces of two of the affected cows.

Ovine

A group of 500 seven-month-old lambs in the Auckland region were moved from a ryegrass-dominant pasture to a

plantain/clover mix pasture. Within 48 hours six had died. All were in good to fat body condition (body condition score 4.5–5). They had received clostridial 5-in-1 vaccinations when they were a few weeks old but their booster vaccinations had been delayed. A post-mortem examination of one lamb revealed a fibrinous peritonitis and perirenal petechiae. Histopathological examination of the kidney and small intestine revealed a fibrinosuppurative peritonitis with intralesional Gram-negative coccobacilli, consistent with **histophilosis**. *Histophilus somni* can cause acute sepsis in lambs when associated with high levels of feeding.

Several mixed-age ewes were found dead over a 2-month period in the Tararua district. Histopathology on one revealed a neutrophilic and lymphohistiocytic meningoencephalitis of the brain stem, consistent with the nervous form of **listeriosis**. Listeriosis can be sporadic or occur in outbreaks associated with feeding silage. It is thought to arise from an initial infection in the nasal or oral cavity, which then enables the bacteria to ascend the cranial nerves to the brain. In another case, several two-tooth ewes died in the Wairarapa district. Clinical signs included opisthotonus and fever. Examination of the brains of two animals showed histological lesions consistent with **listeriosis**.

About 10–15 percent of an unknown number of sheep drenched orally with a seaweed extract developed swelling around the ventral mandibular and neck region within 2–3 hours and seven died within the following 24 hours. There was no obvious trauma induced by the drench guns, but histopathology of the affected head and neck tissues was consistent with an **acute neutrophilic cellulitis and myositis**. It was suspected that these injuries were chemically induced and could have developed after drench leaked into surrounding tissue through micro-abrasions in the oral cavity.

An outbreak of **salmonellosis** was diagnosed on a Central Hawke's Bay farm in January. The farmer reported that 14 ewes had died out of 1 100 over a period of a few days. Samples were taken from two ewes and submitted for histopathology and culture. Histological changes in one ewe were characteristic, comprising fibrinonecrotising enteritis and lymphadenitis associated with bacterial colonies. A moderate growth

of *Salmonella Hindmarsh* was cultured from the small intestine of both ewes.

About 40 out of 400 five-month-old lambs on a Hastings district farm developed lethargy, ataxia and blindness while grazing a greenfeed sorghum crop. Only one death was reported but the other affected lambs did not improve with isolation and preferential treatment. No significant gross post-mortem lesions were seen. The brain was removed and submitted for histopathology. Microscopic examination revealed numerous eosinophilic granular spheroids within nuclei in the medulla, cerebellar peduncles and midbrain. Scattered dilated myelin sheaths containing gitter cells were also observed. These lesions were considered consistent with **sorghum toxicity**. Similar lesions have been noted in previous NZ laboratory submissions from sheep grazing sorghum, and have been described in the literature, albeit infrequently (Bradley *et al.*, 1995).

A Gisborne District farmer reported about 40 sudden deaths among 1 000 four-to-six-month-old lambs grazing a chicory crop. The lambs had been fully vaccinated against clostridial diseases with a six-strain vaccine. Post-mortem examination of two lambs revealed gross and histological findings compatible with **acute pneumonia**.

In March, a veterinarian was called to a Southland farm to investigate a large percentage of lambs that were unthrifty and exhibiting diarrhoea. Anthelmintic treatment containing minerals had been given recently without any observable beneficial effect. An affected lamb was killed and necropsied and there were no gross lesions. Histopathological examination of the gastrointestinal tract showed severe changes consistent with **nematode parasite** damage. The liver selenium concentration was very high (70 790 nmol/kg; toxic level > 30 000). The farmer had been giving the lambs selenium in the worm drench and had also topdressed the lamb paddocks with selenium prills. The cause of the ill-thrift and diarrhoea was considered to be a combination of **parasitism and selenium toxicity**.

Cervine

Twenty adult hinds and 18 fawns aged 2–3-months out of a group of 350 deer were found dead on an Otago deer farm. They had died 24 hours after

being moved from a hill block onto a new paddock that was very exposed to the weather. They were being fed a grain-and-silage mixture as there was minimal grass cover on the paddock. Shortly after they were placed on the paddock there was a severe storm and the dead deer were found the next day. A number were necropsied. There were no obvious gross lesions, post-mortem changes were minimal and their rumens were full of normal ingesta. Histopathological examination of a range of tissues from two of the dead deer showed no significant changes. Lightning strike was ruled out as the dead animals were scattered throughout the paddock. At first it was thought that the deer had died from exposure, but a closer examination of the paddock revealed scattered lumps of diammonium phosphate that had been spread on the paddock 3–4 months before. The lumps were still present because there had been little rain during the intervening period. Given this finding, **ammonia toxicity** was considered likely. Rumen pH levels in two deer were alkaline at 7.5 (normal range 6–7), which was considered to support that diagnosis, but it was not possible to measure ammonia concentration in the blood, rumen contents or eye fluid so a definitive diagnosis could not be made.

Caprine

A five-year-old milking goat from the Hauraki-Coromandel region lost weight, was lethargic and anorexic, and had reduced milk production over a 1-month period. The doe had no lameness, pyrexia or other abnormalities on physical examination, was serologically negative for caprine arthritis encephalitis virus by ELISA, and had been recently treated for endoparasites. Johne's PCR on a faecal sample showed a high number of MAP genomes, consistent with **Johne's disease**.

A 2-year-old goat, one of a group of 20 on a Canterbury property, developed an abscess in her udder. A swab from the abscess was cultured and a moderately heavy, pure growth of *Staphylococcus aureus* was isolated, confirming a **staphylococcal udder abscess**. The isolate was resistant to penicillin but sensitive to all other antibiotics tested (amoxicillin/clavulanic acid, cephalothin, enrofloxacin, tetracycline and trimethoprim/sulfamethoxazole) using the disc-diffusion method. It was also sensitive to ceftiofur (which is used

as a screen for methicillin/meticillin resistance).

Camelid

A three-year-old female alpaca from the Nelson region had persistent skin lesions. Skin scrapings and a biopsy were collected by the examining veterinarian. Two other alpacas on the property had similar lesions, but they were unrelated animals that did not share the same paddock. The lesions had been present for some time and were thought initially to be the result of mite infestation but had responded only partially to doramectin treatment. No *Dermatophilus* spp. organisms were seen on a methylene-blue-stained preparation of the skin scraping, and no fungal elements were seen in a KOH preparation. Histopathological examination of the biopsy revealed mild eosinophilic dermatitis with moderate acanthosis and marked compact orthokeratosis as well as focal suppurative folliculitis and focal pustular dermatitis. The eosinophilic dermatitis and thick, compact nature of the stratum corneum suggested that the skin had been subjected to pressure such as occurs with rubbing from pruritus. *Chorioptes* sp. mites were seen in the skin scrapings in a KOH preparation, and this, along with the histological changes, supported a diagnosis of **ectoparasitic infestation**.

Equine

A faecal sample from a 2-year-old Thoroughbred mare from Canterbury was found to contain 650 **strongyle** eggs per gram, as well as small numbers of *Anoplocephala* spp. eggs. The latter is an uncommon finding in this region. This equine tapeworm is generally considered to be of low pathogenicity and mild infections may be subclinical, but heavier burdens may be associated with poor thrift, colic and diarrhoea. No *Anoplocephala* spp. eggs were seen in a subsequent sample from this horse after anthelmintic treatment was administered.

A swab taken from the site where a surgical plate had been removed from a 6-month-old male Gypsy horse from Otago was cultured for bacteria. There was a heavy growth of mixed bacteria including *Pseudomonas stutzeri* and *Staphylococcus aureus*. The *S. aureus* isolate was resistant to dosage with ceftiofur, which is the method used to screen for **meticillin/methicillin resistant Staphylococcus**

aureus (MRSA). This isolate was also resistant to tetracycline and enrofloxacin but was sensitive to cephalothin and trimethoprim/sulfamethoxazole using the disc-diffusion method. Although MRSA is occasionally seen in companion animals (mainly dogs), but finding it in a horse is very unusual.

A 2.5-year-old male Thoroughbred horse in Hawke's Bay District had white mucus in the trachea on endoscopy post exercise. A tracheal wash was carried out and numerous germinating fungal spores with fine hyphae, and some bacteria, were seen cytologically. Culture yielded *Escherichia coli* and a fungal species most closely resembling a *Cladosporium* sp. *Cladosporium* spp. are environmental organisms found in decaying plant matter and have occasionally been isolated from the respiratory tract of horses (Nardoni, 2005). It is not known whether this was a primary *Cladosporium* and *E. coli* respiratory infection or whether these organisms were causing secondary infections concurrent with other, underlying respiratory disease.

Donkey

A 20-year-old female donkey from the Wairarapa district had a doughnut-shaped swelling on the ventral midline. An aspirate of the lesion was sent to the laboratory for cytological evaluation. The sample consisted only of lipid, consistent with a diagnosis of **lipoma**. Lipomas have been occasionally diagnosed in donkeys (Mozaffari, 2011), but are usually intra-abdominal.

Avian

Several rosella parrots (*Platycercus* sp.) in a large Northland aviary died. Post-mortem examination revealed intestinal ulceration, yellow foci in the liver, coelomitis and myocardial changes. Histopathology showed a necrotising and heterophilic enteritis and multifocal necrotising hepatitis with intralesional Gram-negative bacilli consistent with a Gram-negative infection and suggestive of **salmonellosis**. This infection can be associated with rodent infestation in aviaries.

Faecal swabs from three tuturuatu or New Zealand shore plovers (*Thinornis novaeseelandiae*) from a wildlife breeding facility were received at the laboratory for bacterial culture. No history was available. A *Salmonella* sp. was isolated from the samples from two of the three

birds. Typing was performed on one of these isolates, which was identified as ***Salmonella* Worthington**, an uncommon isolate at this laboratory.

A neonatal hoiho or yellow-eyed penguin chick (*Megadyptes antipodes*) from Banks Peninsula with a suspected leg injury and an infestation of ticks and fleas, was found to be anaemic on clinical examination. A blood smear was received for examination. The total white cell count was estimated to be $48\text{--}53 \times 10^9/\text{L}$. No reference range was available for this species but for rockhopper penguins mean white cell counts are reported to be $8.6 \times 10^9/\text{L}$ with a standard deviation of 4.6×10^9 , so the count in this chick was considered to be significantly increased, consistent with inflammatory demand. There was moderate polychromasia of the erythrocytes, consistent with a regenerative response to the clinically apparent anaemia. Small numbers of the erythrocytes contained intracytoplasmic elongated structures consistent with a protozoan blood parasite. The morphology was most consistent with a *Haemoproteus* species. These insect-transmitted parasites are seen in many avian species but thought to be of low pathogenicity in healthy birds. However, they are found more commonly or in increased numbers in sick birds, and **haemolytic anaemia** has sometimes been associated with infection. The significance in this case was unclear and the anaemia may have been multifactorial, involving blood loss from injury, ticks and fleas, with or without haemolysis associated with the *Hemoproteus* infection.

Seven out of 20 two-year-old laying hens in a backyard flock lost weight and died over a 6-month period. Necropsy of one sick bird revealed emaciation and an enlarged liver containing multiple large pale foci. **Avian tuberculosis** was confirmed on histopathological examination, which revealed multiple granulomas composed of clusters of large epithelioid cells that often contained clumps of acid-fast bacilli consistent with a *Mycobacterium* species. Avian tuberculosis had been diagnosed on the same property 3 years before.

A free-range poultry farm lost 1 000 young birds over a 1-month period. The farm was running large groups of about 8 000 laying hens that had access to a large grass paddock during the day

and were shut in a large barn with nest boxes at night. Affected birds were found moribund in the paddock during the day and died over the next few hours. Multiple antibiotic treatments via the water supply only temporarily slowed the death rate. Older birds in adjacent paddocks were not affected. Five birds were necropsied and the most consistent findings were traumatic feather loss, an enlarged friable liver, an enlarged but empty oviduct and an ovary containing many developing ova, a few of which contained necrotic yolk. ***Pasteurella multocida*** (the agent of fowl cholera) was cultured from various tissues in all five birds. This isolate was typed by PCR as LPS group 3, which corresponds to Heddleston types 3 and 4.

Feline

A 7-year-old Siamese cross cat from Auckland had intermittent diarrhoea and was losing weight despite a good appetite. Pooled faecal samples were positive for *Giardia* spp. on antigen ELISA and also positive for *Tritrichomonas foetus* on PCR, suggesting combined **giardiasis and trichomoniasis**. The patient improved clinically on ronidazole and fenbendazole, and repeated post-treatment tests were negative.

A 3–4-month-old rescued female Domestic Shorthair kitten at an Auckland shelter developed respiratory distress after being desexed. Radiographs revealed diffuse, patchy lung densities. Treatment with topical imidacloprid and moxidectin and oral doxycycline for 14 days failed to resolve the clinical signs or radiographic changes and euthanasia was chosen. Significant gross post-mortem findings were limited to the lungs, which were diffusely consolidated and had patchy, light brown mottling. Histologically, normal pulmonary architecture was extensively disrupted by numerous **nematode** larvae and eggs filling the airways, and these were associated with chronic inflammatory and hyperplastic changes. Many arteries had marked medial hypertrophy, subintimal infiltrates of eosinophils and intimal proliferation. These findings were consistent with severe **verminous pneumonia** caused by *Aelurostrongylus abstrusus*. While the majority of lungworm infections in cats may be relatively mild, severe infestations can develop in young, old and immunocompromised cats. In addition, there are reports of anaesthetic deaths

associated with lungworm infections. Cats are infected by ingesting L3 larvae in intermediate hosts (snails, slugs) or paratenic hosts (birds, rodents, lizards, frogs). Heavy infestations can result in severe clinical signs in the pre-patent period (generally 4–6 weeks).

Lagomorph

Four 8–10-week-old rabbits from Auckland died suddenly. Post-mortem examination of two revealed multiple abscess-like lesions throughout the livers. Histopathology of these lesions revealed marked bile-duct distension by coccidia, in association with biliary hyperplasia, cholangitis, multifocal hepatic necrosis and thrombosis. **Hepatic coccidiosis** caused by infection with *Eimeria stiedae* was diagnosed. This infection can be incidental, but was probably the cause of death in this case.

Thirty 3-month-old New Zealand White rabbits from a property in the Rangitikei district died. Histopathology of one of the affected rabbits revealed a necrotising and heterophilic enteritis and typhlitis with numerous intralesional coccidial organisms. This was consistent with **enteric coccidiosis**, an important cause of clinical and subclinical disease in rabbits, especially young rabbits housed in large numbers. The severity of the disease can be affected by factors such as immune status, overcrowding, inadequate nutrition, the coccidial species involved and overgrowth of other enteric pathogens (especially *E. coli* and rotavirus).

Rodent

A 2-year-old male pet rat from the New Plymouth district was presented to a veterinarian with two skin masses in the inguinal region. The masses were surgically removed and submitted for pathological examination. Grossly they consisted of protruding cream-coloured warty skin masses. Histologically one mass was a sebaceous adenoma and the other was a granuloma involving the dermis and subcutis. A Gram stain revealed Gram-positive pleomorphic bacteria within macrophages. The nature of the lesions and bacterial morphology were considered consistent with *Corynebacterium kitcheri* infection. This is a recognised pathogen of rats, causing focal or disseminated granulomatous lesions.

Piscine

An Eastern rainbowfish (*Melanotaenia splendida splendida*) from a zoological collection developed nodules on its back. Other fish in the display group had died recently. Histopathology of the skin and muscle showed regionally extensive areas of necrosis, and infiltrates of granulocytes and macrophages. The kidneys, hepatopancreas, heart and gills had multifocal areas of necrosis and macrophages (granulomas) containing acid-fast bacilli, consistent with **mycobacteriosis**. Mycobacterial diseases of fish are common, particularly in intensive aquaculture systems, display aquaria and zoological collections. This disease is of concern in recirculating systems, and once established can be difficult to eradicate.

SVS Laboratories Bovine

Out of a mob of seventy 18-month-old dairy heifers on a run-off block in the Whakatane district, 10 died without premonitory signs and two more were found recumbent. Investigation of the grazing area showed poor pasture and paddock management, with copious discarded plastic silage wrapping, an old abandoned car and a large number of weeds including a heavy infestation of fathen (*Chenopodium album*). Post-mortem tissues of one euthanased heifer revealed marked diffuse renal tubular nephrosis with distal tubular crystalline deposits, suggestive of renal oxalate toxicity, which can be caused by the high oxalate levels in fathen. Sudden death caused by a high ingestion of fathen is more commonly seen in grazing sheep, whereas in cattle a hypocalcaemic effect is more usual. This probably reflected a high ingestion of fathen over a short period. The dangers of grazing replacement heifers away from the main farm, on poorly managed runoff blocks, was highlighted.

A dairy cow in the Waikato district presented with weakness, lethargy and pale mucous membranes. Haematology results (limited to a *Theileria* panel) confirmed a severe anaemia with a haematocrit of 0.10 (range 0.24–0.40) and haemoglobin was 30 g/L (reference range 85–130), while *Theileria* spp. seen on blood-smear examination suggested a haemolytic anaemia caused by theileriosis. However, since the cow had

been administered a zinc bolus 2 weeks previously, serum zinc was also tested and was at toxic levels (110 umol/L; reference range for facial eczema prophylaxis is 20–35). At this level, excess zinc can cause haemolytic anaemia (corresponding with the clinical signs); it can also cause sudden death, particularly in young stock.

Throughout this quarter there was a significant incidence of **Johne's disease** in dairy cows in the Waikato, particularly during periods of hot weather. Cows presented with dehydration and diarrhoea, haematology on some blood samples showed haematocrits of up to 0.51 (reference range 0.26–0.48) and *Mycobacterium avium* ssp. *paratuberculosis* infection was diagnosed on positive serology in clinical cases. It was considered likely that subclinical or early clinical cases were exacerbated by the heat and lack of water troughs around milking-sheds that meant affected cows walking to the sheds in the heat were unable to drink until returning to the paddock after milking. It was notable around the region that dairy cows were seen queueing at paddock water troughs after milking.

A mob of dairy cows in south Waikato that had access to turnips each afternoon, presented with skin lesions on the white areas and swelling of legs. Biochemistry revealed marked increases in liver enzymes with GLDH up to 637 U/L (reference range 8–41), indicating hepatocellular damage, and GGT up to 795 U/L (reference range 1–36), indicating cholestasis/biliary epithelial damage. These results could indicate either facial eczema (FE) or **glucosinolate toxicity**, but the latter was considered more likely, owing to the grazing history and the fact that January was early for FE. It was considered necessary to review the grazing period on turnips and the variety grown, and to provide concurrent supplementary forage.

A Friesian dairy cow in the Rotorua district presented with an ocular lesion at the scleral conjunctival junction. Histopathology of the excised mass revealed a highly infiltrative **ocular squamous cell carcinoma**, most commonly seen on non-pigmented eyes predisposed to damaging effects of solar irradiation (particularly on farms with insufficient shade). Owing to the

tendency for progressive local infiltration and recurrence, enucleation would be required for treatment, or culling on welfare grounds.

Nineteen out of 70 R2 bulls in a cell-grazing system in the Taupo district were affected with acute diarrhoea and two died. Clinical pathology on samples from several affected bulls revealed marked protein and electrolyte losses in one bull, with albumin 14 g/L (reference range 25–40), Cl 63 mmol/L (reference range 96–104) and Na 120 mmol/L (reference range 132–152). There was evidence of dehydration causing renal impairment, with HCT 0.50 (reference range 0.26–0.48), urea 37.6 mmol/L (reference range 2.7–12.3) and an acute inflammatory leukogram. These findings indicated mucosal damage and probable bacterial enteritis, although faecal cultures from several bulls did not yield *Salmonella* or *Yersinia*. Faecal examination revealed a moderate strongyle count of 750 epg in the worst-affected bull, indicating that **parastitic gastroenteritis** was a likely contributing factor (lower counts were found in other bulls.) A review of the cell-grazing management showed that the water system consisted of a modern nipple-drinking unit in each cell, with clean water flowing on demand. However, since the dead bulls were found nearby, it seemed possible that the system did not provide sufficient flow on hot days. These large bulls were so dependent on water for cooling (there being no shade in the grazing cells) that heat stress and dehydration would have exacerbated any underlying mild enteropathies or predisposed the bulls to bacterial overgrowth and enteritis.

A mob of 74 six-month-old dairy calves in the Buller district presented with diarrhoea and ill-thrift, with six calves dying. Faecal examination for worm eggs revealed counts as high as 4,050 strongyle epg, confirming **parasitic gastroenteritis**. Post-mortem tissues from one calf were submitted and histopathology showed marked gastrointestinal tract pathology, including confirmation of the **enteric helminthiasis**. In addition, signs consistent with **bovine adenovirus infection** were present including abomasitis, multifocal vasculitis and diffuse oedema within the abomasum, duodenum and jejunum sections. Bovine adenovirus infection causes disease in immunosuppressed young stock.

A **compound odontoma** was diagnosed in a gingival wedge biopsy in a rising-2-year-old Hereford cross heifer in the Waipa district. Examination of the oral cavity revealed a lumpy ventral mandible and tooth displacement. Odontomas are considered congenital or developmental hamartomatous anomalies that include well-differentiated dental tissues such as dentin, enamel, cementum, dental pulp, dental papilla and denticles. While this lesion is benign, it can be locally destructive owing to expansion.

Caprine

A **mammary papillary adenoma** was diagnosed in a mammary wedge biopsy from an adult Saanan doe goat in the Whangarei district. Mammary tumours are rare in goats and most that have been described in the literature are adenocarcinomas.

In the Matamata-Piako district, about 20 female Saanen goats aged 6–8 months died on a dairy-goat farm within a couple of hours after feeding. **Urea toxicity** was suspected. Biochemistry on samples from two acutely affected survivors revealed uremia, with urea 17.3 mmol/L (reference range 4.2–10.1). Since there was no evidence of dehydration or other evidence of renal impairment, excessive ingestion of urea was a likely cause. Although blood ammonia measurement is the key test for urea toxicity, the volatile nature of ammonia means the test is often impractical and hence rarely carried out.

Acute scours in 3-year-old buck goats was seen 7 days after joining with the does. Eight bucks out of a total of 72 died and 12 were acutely ill with scours and were treated. Histopathology of submitted necropsy tissues revealed a heavy infestation of nematodes within the duodenum, giving a diagnosis of **parasitic enteritis** but there was also a severe enterocolitis with multifocal crypt microabscesses, consistent with **yersiniosis**. However, cultures were negative, owing to recent treatment. Stress factors on the young bucks at joining, and hence reduced water intake, were likely to have exacerbated the underlying enteropathies.

Equine

A 5-year-old Appaloosa mare presented with several 1-cm-diameter raised skin nodules located at the base of the ear. Histopathology of

excised samples revealed multifocal **collagenolytic granulomas** with a prominent eosinophilic inflammatory component. These are common skin nodules of unknown aetiology, although the eosinophilic component suggests reaction to insect hypersensitivity in some locations (Pilsworth *et al.*, 2005). Other possible causes include skin trauma, autoimmune response to physical collagen damage, and fibroblast disruption. Despite the presence of eosinophils, however, the lesions are usually non-pruritic. Histopathology of punch biopsies is warranted to distinguish these benign nodules from skin neoplasia, dermoid cysts and fungal granuloma.

Ultrasound examination of a 3-month-old weanling Thoroughbred colt with chronic diarrhoea showed oedema of the large-colon wall and increased peritoneal fluid (modified transudate). A faecal PCR test for *Lawsonia intracellularis* was positive, confirming the diagnosis of **equine proliferative enteropathy**. This disease in horses differs from other species in that diarrhoea is not always present (if the colon is not involved) but the key signs are hypoalbuminaemia and its effects on plasma colloid osmotic pressure (leading to the modified transudate and oedema). Weanlings are particularly prone to this infection, owing to the stresses of weaning and dietary changes.

Poultry

Coccidiosis was diagnosed twice in poultry from two entirely different management systems. In the first case, five chickens in a small backyard poultry flock in the Matamata-Piako district died. Histopathology of submitted post-mortem samples revealed severe enteritis and coccidial infection throughout the small intestines, giving a diagnosis of **coccidiosis** (infection with *Eimeria* spp). In addition, urate topi were visible in the kidney and heart, which were secondary to enteritis-related dehydration. Coccidiosis is a common disease in domestic fowl and other birds, and is characterised by enteritis and bloody diarrhoea (Schmidt, 2018). The second case involved depression and loose faeces in 28-day-old birds from the parent stock of a large commercial poultry breeding unit. **Coccidiosis** was suspected clinically, and was confirmed on histopathology of submitted post-

mortem tissues. Although the exact species could not be determined histologically, the presentation and location of infection were consistent with *Eimeria tenella*.

Companion animals

A 5-year-old crossbred entire female dog was diagnosed clinically with pericarditis. Histopathology of a biopsy of the pericardial sac revealed a severe diffuse transmural **granulomatous pericarditis** with healing granulation tissue. Tests with special stains for bacterial, fungal and acid-fast organisms were negative. This is an unusual lesion in a dog and possible underlying causes include progression from a suppurative pneumonia or extension from an intrathoracic lesion (such as oesophageal trauma). An underlying cause in this case was not found.

A 1-year-old castrated male Domestic Shorthaired cat from Hamilton presented with multiple skin nodules around the head, some of which were ulcerated.

Feline leprosy was suspected clinically and confirmed on histopathology of biopsy samples: microscopic examination revealed granulomatous dermatitis with plentiful intracellular acid-fast organisms consistent with *Mycobacterium* sp. Feline leprosy usually presents with multiple nodules on the head and limbs, with abundant intrahistiocytic acid-fast organisms, as seen in this case.

This contrasted with a case of **atypical mycobacterial** skin infection in another young castrated male cat, in the Bay of Plenty district, which had nodular lesions around the perineum. Histopathology revealed granulomatous dermatitis with only occasional acid-fast organisms, characteristic of skin infection with atypical mycobacterial infection. These opportunist bacteria are ubiquitous in the environment (soil, water) and can contaminate skin lesions such as bite wounds and other trauma.

A 3-year-old male Labrador dog in the Kawerau district was anorexic and had icteric mucous membranes. Biochemistry showed marked renal compromise, with a severe azotaemia (creatinine 784 $\mu\text{mol/L}$, reference range 45–435; urea 59.3 mmol/L , reference range 2.6–10.2; phosphate 5.10 mmol/L , reference range 1–3). There was also liver disease (ALP 401 U/L, reference range 0–185; ALT 182 U/L, reference range 0–75; and bilirubin was 96.4 $\mu\text{mol/L}$, reference

range 0–6). Serology was positive for *Leptospira* IgM antibodies, giving a diagnosis of acute **leptospirosis**. The owner was reminded to wear gloves when handling the dog, owing to the zoonotic risk.

Blood samples were submitted from an 8-year-old Huntaway bitch from a cattle station in Wairoa. There was a history of anaemia, no access to rat bait and an immune-mediated anaemia was suspected clinically. Haematology confirmed severe anaemia: HCT was 0.13 (reference range 0.35–0.55), showing regeneration (reticulocytes $398.4 \times 10^9/\text{L}$; reference range 0.0–60.0), mean cell volume was 54 fL (reference range 60–77) and there was no RBC agglutination. These results suggested chronic blood loss. Further discussions with the veterinarian revealed that the dog's kennel was covered in fleas, leading to chronic low-grade blood loss caused by **chronic flea infestation**.

A 6-year-old male American Bulldog cross with diarrhoea of 2 weeks' duration showed frequent tenesmus and occasional frank blood in the faeces. A worm egg count revealed 3,800 *Trichuris* sp. eggs and 50 *Strongyle* sp. eggs, giving a diagnosis of **parasitic enteritis**. The veterinarian highlighted the public-health aspects of the case and the importance of regular worming to the owner.

Salmonellosis was diagnosed in two small-animal cases. In one, a litter of eight 3-month-old Bull Terrier puppies in the Bay of Plenty district presented with intermittent diarrhoea. Faecal cultures yielded *Salmonella* Typhimurium phage type 101. In another case, in the Matamata-Piako district, *Salmonella* Bovismorbificans was isolated from the faeces of a 10-week-old female kitten with diarrhoea. The kitten was otherwise bright and alert, indicating the infection was localised to the intestinal tract.

New Zealand Veterinary Pathology Bovine

Three out of a mob of 34 well-grown 7-month-old Jersey/Friesian cross dairy heifer calves in the Waikato were found to be in respiratory distress when moved. Clinical examination revealed markedly pale mucous membranes with a yellow tinge. Zinc boluses had been administered a few days previously and

there was some clinical concern about acute zinc toxicity. One calf died and post-mortem examination revealed pale mucous membranes, mild, generalised jaundice and a pale yellow, swollen liver. The zinc bolus was *in situ* and undamaged. Haematuria was not evident. Bloods submitted from two affected calves confirmed marked anaemia, with both calves having haematocrits of 0.11 (reference range 0.24–0.4). Organisms consistent with *Theileria* sp. were visible on blood-smear examination. Further testing was performed to investigate possible causes of death other than *Theileria* infection. Serum zinc levels were good, at 30 and 22 $\mu\text{mol/L}$ (20–35 is the target range for facial eczema protection). GDH was elevated in one calf and hyperbilirubinaemia was evident in both. Another calf subsequently died, with post-mortem findings similar to those seen previously. The liver copper level was normal at 760 $\mu\text{mol/kg}$ (reference range 95–2 000). Histology of the liver revealed a moderately severe cholestasis. In the absence of findings consistent with zinc toxicity, copper toxicity or facial eczema this case was regarded as a severe manifestation of **theileriosis**.

A Hereford cow in the Waikato was lagging behind the mob and presented with pale mucous membranes. Five weeks previously there had been difficulty administering a zinc bolus and it was thought to have lodged in the oesophagus. Serum copper was within normal limits and a blood smear for *Theileria* was negative. However, GHD was markedly elevated at > 250 IU/L (reference range 8–41), as was the serum zinc at 370 $\mu\text{mol/L}$ (20–35 is the target range for facial eczema protection). The cow died 2 days later and an oesophageal diverticulum (presumably traumatic) extending 400 mm ventrally to the thoracic inlet was discovered at postmortem. Histological examination of the pancreas and kidney revealed exocrine cellular degeneration with fibrosis and severe haemoglobinuric nephrosis, consistent with **zinc toxicity** and associated haemolysis.

A single mature Jersey cow in the Waikato presented with acute-onset bilateral hyphaema with petechiae on the vulvar mucosa. The pupillary light reflex was present, but there was no menace reflex and the cow appeared blind. The cow was in calf, between 45 and 55 days

gestation. Serology for leptospirosis serovars Pomona and Hardjo was negative. Blood smear examination revealed markedly decreased platelet numbers, indicating **thrombocytopenia** was the likely cause of haemorrhage. There was also hyperfibrinogenaemia of 9.1 g/L (reference range 2.0–7.0), and in the absence of hyperproteinaemia this was considered likely to be due to inflammation rather than dehydration. In the absence of exposure to bracken fern, **immune-mediated thrombocytopenia** was considered the most probable explanation, possibly triggered by underlying infectious disease.

In the Raglan area, a single 5-month-old female Jersey calf from a mob of 60 was noted to be off-colour for 3 days, then was found dead the next morning. The body was severely dehydrated but the faeces appeared normal. Post-mortem examination revealed a segmental area of reddened fluid within the small intestine. Faecal examination subjectively revealed light to moderate numbers of coccidial oocysts, and no *Salmonella* sp. or *Yersinia* sp. were isolated on culture of intestinal contents. **Coccidiosis** was considered to be the most likely cause of enteropathy, given the appearance of the intestine, but possibly an undetected concurrent disease contributed to debilitation and death in this case. Numbers of oocysts shed in a single faecal sample do not necessarily reflect the severity of coccidial infection, and multiple animals are usually infected within a mob. Clinical disease is most common in stock 3–8 months of age, but can occur in all ages.

Five 6-month-old dairy calves from a mob of 85 showed loss of condition despite supplementation with vitamin B12 and selenium, and anthelmintic drenching. One became recumbent and was euthanased for post-mortem examination, which revealed gross evidence of enteritis. Haematology from two animals showed polycythaemia (RBC 12.8 and 14.4 x 10⁹/L; reference range 6.5–11.9 x 10⁹), which was most likely due to dehydration. Another animal showed hyponatraemia of 121 mmol/L (reference range 132–152), hypochloraemia of 81 mmol/L (reference range 96–104) and decreased bicarbonate of 23 mmol/L (reference range 26–34 mmol/L), all most likely due to intestinal loss. An elevated β -hydroxybutyrate level of 1.4 mmol/L

(reference range 0.2–1.0) indicated concurrent ketoacidosis, probably caused by negative energy balance secondary to enteropathy. *Yersinia pseudotuberculosis* was isolated from two out of three faecal samples from affected animals.

Two out of a group of 236 mature dairy cows in the Manawatu showed signs of dermatopathy after grazing a rape crop, with a provisional clinical diagnosis of **rape scald**. Both animals revealed serum biochemical evidence of hepatopathy, with GGT 987 and 1 495 IU/L (reference range 0–3) and GLDH 308 and 854 IU/L (reference range 8–41). Eleven additional non-clinical animals were evaluated for evidence of subclinical hepatopathy; of these only one showed increased liver enzymes (GGT 660 IU/L and GLDH 110 IU/L). Some brassica forage crops including rape, turnips, swede, kale and their hybrids have the potential to induce a variable degree of **hepatogenous photosensitisation** that is clinically similar to facial eczema (Collett, 2014). Cutaneous lesions are restricted to non-pigmented areas, which initially become leathery and wrinkled and may slough. In some cases lesions may be confined to the teats.

In the Far North district, a neonatal calf (breed not specified) presented with cutaneous/mucosal defects involving the muzzle and mouth. Histopathology revealed extensive subepidermal clefting and vesicle formation, sometimes containing proteinaceous material and occasional neutrophils. There was mild inflammation in the underlying dermis. A diagnosis of **epidermolysis bullosa** was made. A variety of forms of this genetic disorder exist, with both dominant and recessive modes reported. The epidermis is easily separated from the dermis, and initial lesions most commonly develop in the mouth and over limb joints as a result of trauma/friction at these sites.

A single adult Friesian cow from a mob of 450 in the New Plymouth district presented with an interdigital lesion on the left hind foot. On histopathology a sample of the lesion was mainly composed of hyperkeratotic epithelium with areas of necrosis and suppurative inflammation within the epidermis. Colonies of filamentous bacteria were visible. A tentative diagnosis of **bovine digital dermatitis** was proposed, but Warthin-Starry stain failed to

demonstrate the presence of *Treponema* sp. PCR testing can be used to confirm the diagnosis, but was not undertaken in this case.

Six 18-month-old Friesian steers from a mob of 200 in the Manawatu showed a variety of oral and cutaneous proliferative/granulomatous lesions. One case had a firm subcutaneous swelling caudal to the stifle, which did not respond to a prolonged course of tetracycline treatment. A sample collected for histopathology revealed the swelling was composed of multiple granulomas with multinucleated giant cells and bacterial club colonies with morphology typical of *Actinomyces* sp. or *Actinobacillus* sp. infection. Ultrasound examination of the lesion revealed a heterogenous appearance with fluid pockets. Cytology of the aspirated fluid revealed suppurative to pyogranulomatous inflammation without visible infectious agents. Anaerobic culture for *Actinomyces* sp. failed to isolate any organisms, perhaps because the bacterial agent was aerobic or a result of the antibiotic treatment.

Two cows from a mob of 300 in the lower North Island presented with late-term abortion followed 2 days later by severe respiratory signs with rapid deterioration and death. Samples of lung for histopathology revealed multifocal acute-to-subacute necrotising pneumonia with intralesional vasculitis. The blood vessel walls contained areas of necrosis and associated thrombus formation, with intralesional fungal hyphae. Although the agent involved could not be confirmed without fungal culture, the clinical history of recent abortion followed by pneumonia with vasculitis was typical of *Mortierella wolfii* infection. This presentation typically occurs sporadically, but outbreaks can be associated with poorly fermented or spoiled silage.

Four six-month-old calves from a group of 150 became ataxic, with one dying suddenly. Brain was submitted for histopathology, and transverse sections showed laminar regions of fluorescence within the cerebral cortex. Histology confirmed corresponding areas of necrotic neuron cell bodies and spongiosis of the neuropil. A diagnosis of **polioencephalomalacia** was made, with the likely cause considered to be **thiamine/vitamin B1 deficiency**. Similar lesions may also be seen with

salt poisoning, lead poisoning and high sulphur intake.

Six cows aborted in a mob of 450 in the Taranaki region. Blood samples were collected from three of the affected cows, and twin aborted fetuses were also submitted for post-mortem examination and histopathology. Serology from the three samples was positive for *Neospora* by IFAT, with all results $\geq 1:2\ 000$ providing strong evidence that **neosporosis** (either recent infection or reactivation of a latent infection) was the cause. Although the fetal samples for histopathology were autolysed, there were foci of inflammation in the heart and lungs compatible with neosporosis.

In the Waikato region, four from a group of seven 6-month-old crossbred calves showed scouring and dehydration, with a low body-condition score and some degree of pyrexia. One animal had died. Faecal samples from the other three had variable numbers of **strongyle eggs** (350–1 200 epg). All samples also contained low numbers of **coccidial oocysts**, although this is not necessarily a good indication of the severity of infection. No *Salmonella* sp. or *Yersinia* sp. were isolated on faecal cultures. Mixed **endoparasitism** was likely the cause of signs in these animals.

Two mature cows from a group of 250 in the lower North Island presented with severe loss of condition despite grazing well and being otherwise bright and responsive. They had diarrhoea, and serology from both was positive for **Johne's disease** on ELISA. *Mycobacterium avium* ssp. *paratuberculosis* was considered the probable cause of diarrhoea and condition loss in these animals.

A single 3-year-old cow from a group of 900 at-risk animals suffered an oblique, comminuted fracture of the left humerus. Evaluation of a liver sample revealed a copper level of $< 45\ \mu\text{mol/kg}$ (reference range 95–2 000). **Copper deficiency** was diagnosed, which can cause pathological fractures in cattle through osteoporosis and decreased bone-matrix strength. Low dietary intake of copper or excessive pasture molybdenum and sulphur can lead to copper deficiency.

Ovine

Recumbency and sudden deaths in Romney lambs 3–5 months of age occurred in the central North Island,

with 30 dead from a flock of about 600. The lambs were on pure lucerne but inappetent and had last been drenched 5 weeks previously. Clostridial vaccination was up to date, but selenium and vitamin B12 supplementation was not provided. A very high faecal egg count was noted in-house (numbers not provided) and trichostrongyles were visible in abomasal fluid. Routine haematology and biochemistry on one of the lambs only revealed mild, non-specific changes, but liver samples from four lambs revealed evidence of **selenium deficiency**, with all selenium levels $< 100\ \text{nmol/kg}$ (reference range 450–15 000). Selenium deficiency in soil is very common in New Zealand, although lucerne may accumulate higher levels than other pasture species. Most lambs improved clinically after anthelmintic drenching, indicating that **endoparasitism** was a major contributing factor to the deaths, but some lambs continued to be lethargic and prone to falling over. While histopathology of heart and skeletal muscle did not reveal evidence of myopathy, the muscular lesions may have been localised and the possibility that lesions of delayed-onset white muscle disease were present in other muscle groups cannot be excluded.

From a group of 40 six-month-old ram lambs in the Waikato region, two were identified as lethargic and recumbent and one died overnight. Serum biochemistry revealed evidence of hepatopathy, with AST 2 976 and 6 718 IU/L (reference range 64–225), GLDH 44 and 6 IU/L (reference range 0–20), GGT 732 and 356 IU/L (reference range 32–70), and bilirubin 127 and 56 $\mu\text{mol/L}$ (reference range 0–9). Serum copper in both animals was elevated at 48.0 and 75.0 $\mu\text{mol/L}$ (reference range 11.0–25.0). Post-mortem examination of the lamb that died revealed friable kidney and liver, petechial haemorrhages on epicardial and pericardial surfaces, and cranial lung lobe consolidation. Histological evaluation of the liver and kidney was limited by autolysis, with suspected underlying necrosis. The liver copper was 5 600 $\mu\text{mol/kg}$ and kidney copper was 1 400 $\mu\text{mol/kg}$ in this animal (reference ranges 95–2 000 and 0–157 respectively). These findings confirmed a primary diagnosis of **copper toxicity**. Suppurative bronchopneumonia was also evident. Lung culture yielded heavy growths of *Mannheimia haemolytica* and *Pasteurella multocida*, confirming

concurrent bacterial pneumonia in the lamb that died.

On a Manawatu farm, 30 two-year-old hoggets from a group of 400 died suddenly. The sheep had been fasted prior to dipping the day before and then left overnight on a new ryegrass crop that had recently been fertilised. Aqueous humour sampled from three of the dead animals and serum from two surviving animals all had nitrate levels $> 25\ \text{mg/L}$, consistent with **nitrate toxicity**. Ingested nitrate is converted to nitrite in the rumen, and once absorbed oxidises haemoglobin to methaemoglobin, which cannot transport oxygen. Rapid absorption of a large amount of nitrite can result in sudden death, caused by the combined action of reduced oxygen-carrying capacity of the blood and concurrent vasodilation causing collapse of the circulatory system.

A single 2-year-old ewe from a group of four presented with acute-onset scouring, weight loss and depression. Coccidiosis and roundworm infection were ruled out by in-house testing. Faecal culture isolated *Salmonella* **Typhimurium**. Enteric salmonellosis is often associated with stresses such as sudden feed changes, yarding and transport, but identifiable stress factors do not always precede outbreaks.

Canine

A 3-year-old female Toy Poodle presented with intermittent colitis, with mucoid faeces and occasional haematochezia. Pyrexia and inappetence were also present. Clinical signs responded temporarily to metronidazole treatment but recurred on withdrawal of the antibiotic. Faecal examination for ova or parasites was negative, but a heavy growth of *Salmonella* sp. was isolated on culture. Serotyping by ESR Enteric Reference Laboratory identified *Salmonella* **Thompson**, a potentially zoonotic organism that has in the past been associated with outbreaks of food poisoning in humans. It is also reportedly the second most commonly isolated serovar in captive exotic reptiles in New Zealand (Kikillus, Gartrell & Motion, 2011).

Equine

A 4-year-old Thoroughbred filly from the Waikato presented with initially mild colic that increased in severity, and had a prior history of 1–2 months

of intermittent pyrexia. About 48 hours after colic signs were first noticed, surgical exploration of the abdomen was performed. At least 40 mass lesions were found throughout the small intestine, and the mesenteric lymph nodes were enlarged. Histopathology of mass lesions and lymph node showed effacement of the tissue by medium-sized lymphoid cells, and a diagnosis of **intestinal lymphoma** was made.

A 6-year-old mare that presented with acute colic initially responded to NSAIDs, but when the colic recurred an exploratory laparotomy was performed. An impacted area of the small intestine was identified, associated with gross thickening of the intestinal wall and several yellowish-white plaques about 5 mm in diameter on the jejunal serosa, accompanied by petechial haemorrhages. Histopathology of the thickened area of jejunum revealed marked infiltrates of eosinophils in the submucosa accompanied by fibroplasia, with eosinophilic inflammation extending through the muscle layers to the serosa. A diagnosis of **idiopathic focal eosinophilic enteritis** was made. This is considered to be an emerging disease of unknown aetiology (Archer *et al.*, 2014), and does not appear to have been previously reported in New Zealand.

A 4-month-old Thoroughbred foal from the Waikato presented with ill-thrift and scouring for the previous 5 days. Severe panhypoproteinaemia was present, with albumin 11 g/L (reference range 19–32) and globulin 15 g/L (reference range 24–47). These findings suggested severe **protein-losing enteropathy**. Faecal egg count was negative, but PCR was positive for *Lawsonia intracellularis*, the agent associated with **equine proliferative enteropathy**, which mainly affects weanling foals 4–7 months of age.

A 5-week-old Thoroughbred filly died following a long history of liver disease. Histopathology of the liver revealed evidence of a congenital lesion characterised by marked **hepatic fibrosis and bile ductule proliferation**. The findings resembled an autosomal recessive syndrome described in Swiss Freiberger horses (Haechler *et al.*, 2000).

A single 2-year-old Thoroughbred gelding from the Waikato presented with a submandibular swelling suggestive of a bacterial abscess. Culture from the

lesion was performed to investigate possible strangles, and *Streptococcus equi ssp. zooepidemicus* was isolated. This generally opportunistic pathogen in horses can mimic the abscesses seen with strangles, and is also a zoonotic agent that can cause severe disease in humans.

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Quarterly report of investigations of suspected exotic diseases

MPI Incursion Investigators are at present intensively involved in the *Mycoplasma bovis* response. Incursion Investigators are continuing to respond to notifications as normal, but the demands of this particular event mean that the writing up of case reports, as presented in *Surveillance*, has been a lower priority. Consequently, in this edition there are fewer case histories than usual. All investigations will be published as they become available.

Canine influenza excluded

Two cats recently imported from Canada into an Auckland quarantine centre were noted to be sneezing. The disease was assumed to be cat flu, caused by feline herpesvirus or feline calicivirus, or both. However, owing to the emerging epidemic of canine influenza (H3N2) in North America, it was deemed necessary to rule out this disease before releasing the cats or any other animals in the quarantine centre. Nasal swabs were obtained from both cats and transported in viral transport medium to the MPI Animal Health Laboratory (AHL), Wallaceville, where PCR found no evidence of canine influenza virus. The investigation was closed and animals were allowed to be released.

Anthrax excluded

A veterinarian contacted MPI via the exotic pest and disease hotline to report that two cows had died suddenly, with no clinical signs. One cow had died a few days prior to the notification, and the other just before. Both had blood running from at least one orifice; the most recently dead cow had blood running from the vulva, rectum, nose and eyes. A blood smear tested negative by staining and by PCR for *Bacillus anthracis*, the causative agent of anthrax. Culture for other significant bacteria was negative, with only mixed unremarkable growth reported. Other possible causes of sudden haemorrhagic death in cattle include bacterial septicaemia, intoxication (e.g., with bracken fern and aplastic anaemia) and underlying conditions affecting haemostasis. Because anthrax is a zoonotic disease,

Exotic disease investigations are managed and reported by MPI Diagnostic and Surveillance Services (DSS), Wallaceville. The following is a summary of investigations of suspected exotic disease during the period from January to March 2018.

the carcasses could not be examined and no cause of death was identified, though exotic disease was ruled out. The investigation was closed.

Hydatids excluded

An MPI Verification Services veterinarian called the exotic pest and disease hotline to report multiple cysts in the liver of one of a consignment of 20 mixed-age red deer hinds at a Southland slaughterhouse. Hydatid cysts could not be ruled out on gross examination. Hydatid cysts are the intermediate stage of the cestode *Echinococcus granulosus* and can be found in the liver and lungs of a number of species including deer. However, New Zealand was declared hydatid-free in 2002. The liver was submitted to a veterinary pathologist. Histopathological examination identified intrahepatic congenital cysts, believed to be derived from embryonic bile ducts. Exotic disease was ruled out and the investigation was closed.

Exotic bees excluded

AnASUREQuality Apiculture Officer called the exotic pest and disease hotline to report that an apiary inspector had found unusual-looking bees during routine inspection of an Auckland apiary. A completely black bee was found that had a very pointed abdomen and was half the size of a normal honey bee (*Apis mellifera*). This description was consistent with the exotic Cape bee (*Apis mellifera capensis*). In another hive, bees were seen behaving aggressively, which could potentially indicate an incursion of the exotic African honey bee (*Apis mellifera scutellata*). Bees were submitted to the MPI Plant Health and Environment Laboratory (PHEL), Tamaki. After examination and molecular assessment the bees were confirmed to be the normal honey bee, *Apis mellifera*. An

exotic bee incursion was ruled out and the investigation closed.

Leptospiral abortions in alpacas

A Massey University veterinarian called the MPI exotic pest and disease hotline to report abortions in alpaca. The farm had 50 pregnant alpacas, seven of which had aborted in the previous 2 weeks, with up to two abortions per day. The season had been wet and rainy, with flooding in several pastures. In addition, two young alpacas had developed disease: one had a systemic inflammatory illness and pneumonia, and the other had renal insufficiency. Exotic disease was considered unlikely, and no exotic diseases were considered as differential diagnoses for this syndrome. However, very little is known about the cause of abortion in New Zealand alpacas and the case was investigated further. Post-mortem and histopathology findings were unremarkable and did not suggest that bacterial placentitis was present. A mild but consistent vasculitis was present in the placenta, which suggested a possible viral cause including herpesviruses. Suspected viral causes of alpaca abortion include bovine viral diarrhoea virus (BVDV), equine herpesviruses (EHVs) and potentially also generic coronavirus. Testing for BVDV was repeatedly negative, so this was ruled out. Two sets of tissues were sent to the AHL (Wallaceville). The first set included multiple fetal and placental tissues from four separate cases, which were tested by general bacterial culture and by PCR for generic equine herpesviruses, EHV-1 and EHV-4. Bacterial culture produced mixed growth but no consistent species and was negative for *Salmonella* spp. Tests of the second set, which included stomach contents and placenta from two aborted

fetuses, were negative by PCR for EHV-1, EHV-4, and generic coronavirus, and were negative by virus-isolation using four cell types (BL12, BHK, Vero and RK-13). Leptospirosis testing was also included, as there had been an increase in cases in other species. *Leptospira* panels were run on sera from a large group of aborted dams with positive titres for *Leptospira* serovar Pomona, and titres were 1:3 200 in all but one case. PCR testing by New Zealand Veterinary Pathology was positive for *Leptospira* spp. in four aborted fetuses. The abortion storm was considered to be due to *Leptospira* Pomona infection, perhaps associated with the wet conditions during calving. There are no reports in the international literature or from within New Zealand of *Leptospira* abortion in alpacas. This adds to the very short list of endemic causes of abortion in alpacas.

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Quarterly report of investigations of suspected exotic marine and freshwater pests and diseases

Exotic mosquitoes found in Kaipara Harbour

MPI implements the National Saltmarsh Mosquito Surveillance Programme (NSP), aimed at detecting the presence of exotic saltmarsh mosquitoes. During the latest surveillance survey, two larvae and six pupae of an exotic mosquito were found in the Kaipara Harbour, near Helensville. MPI's Plant Health and Environment Laboratory (PHEL) confirmed the identification as the exotic mosquito species *Culex sitiens*. This is a coastal species, widespread in Australia, the Pacific Islands, Southeast Asia, Micronesia and the Middle East. Adult females are nocturnal biters and will readily feed on humans, birds and mammals. Laboratory studies have indicated that *Cx. sitiens* is a potential vector of Ross River virus and Japanese encephalitis, but there is minimal evidence that it can transmit either of these diseases in the field. In New Zealand, *Cx. sitiens* poses a very low risk to humans as the diseases it may be able to transmit are not present. If it were to establish in New Zealand then it would likely become a nuisance biting pest. The Incursion Investigator instigated a delimiting survey deploying CO₂ light traps and dipping of suitable habitat to determine the extent of the population. No adults were found but two more larvae were detected, indicating the likelihood of a breeding population, so the MPI Response and Readiness Directorate intervened to undertake an eradication programme. The detection of exotic mosquitoes at such a low prevalence demonstrates the power of the surveillance programme to intercept exotic species at the early stages of establishment.

Range extensions for ascidians

A member of the public contacted MPI via the exotic pest and disease hotline to report an unidentified marine organism that he found attached to his mooring line in Kenepuru Sound. The notifier reported that he not seen anything like this before, and claimed that the clusters

Exotic marine and freshwater pest and aquatic disease investigations are managed and reported by MPI Diagnostic & Surveillance Services, Wallaceville. The following is a summary of investigations of suspected exotic marine and freshwater diseases and pests during the period from January to March 2018.

of the organism had displaced all other life on the mooring line. Photos were sent to the MPI Incursion Investigator, who recognised that it was an ascidian but was unsure of the species. The NIWA Marine Invasive Taxonomic Service (MITS) identified three species of ascidian – *Ciona intestinalis*, *Asciidiella aspersa* (both introduced species) and *Asterocarpa humilis* (a cryptogenic species). All three species had previously been found in New Zealand, but not from Kenepuru Sound, so they have been recorded as range extensions.

Mantis shrimp range extension

A member of the public called the MPI exotic pest and disease hotline to report a mantis shrimp from a fishing net in the southeastern area of Whangaroa Harbour, Northland. The specimen was dead when found and was 15 cm long. It was preserved in methylated spirits and submitted to MITS for identification. MITS confirmed the specimen was *Oratosquilla oratoria*. This is a range expansion for this marine pest, which had only previously been found as far north as Kerikeri. The Northland Regional Council was notified and the investigation stood down.

Pacific oyster mortality in Whangaroa Harbour

A farmer of Pacific oysters (*Crassostrea gigas*) in Whangaroa Harbour, Northland, notified MPI of 80 percent mortality in oysters 20–50 mm in length. This area had previously tested positive for ostreid herpesvirus-1 microvariant (OsHV-1). Specimens from two different family lines were submitted for testing for OsHV-1 at the MPI Animal Health Laboratory. Nine specimens (two from the sensitive family and seven from the

more resilient family) were tested, and real-time PCR confirmed the presence of OsHV-1. The notifier was informed, and as this virus is already established in New Zealand, the investigation was stood down.

Dead pipi and other shellfish in Whangarei Harbour

A researcher contacted MPI via the exotic pest and disease hotline to report a number of recently dead and dying (i.e., gaping) pipi (*Paphies australis*) and oblong venus clams (*Venerupis largillierti*) in Whangarei Harbour. The mortalities had occurred within several high-density patches of shellfish in the northern part of a study area on Mair Bank. A distinct smell of decay in the area was also reported.

Samples from three locations were submitted to the MPI Animal Health Laboratory. Sick, healthy and control groups showed similar histopathology results: there were sporocysts (initial infection phase) of a parasitic digenean trematode (flatworm) and bacterial infiltrate in all three groups, although there was a higher prevalence in the sick group. Whether this occurred pre- or post-mortem is unclear. Digenean sporocysts can cause castration and a reduction in the tolerance of environmental stress; the high temperatures recently experienced in the area may therefore have been the cause of the mortalities. Interestingly, all samples showed signs of gonad resorption (effectively castration), potentially caused by the sporocysts, and *Rickettsia*-like inclusions were also common. All three groups showed similar bacterial growth, although the sick group showed a higher prevalence of *Vibrio harveyi*, a common marine bacterium. None of the bacteria detected were new to New Zealand.

The cause remains unclear, but as no further mortalities have been observed since the original notification, it was possibly due to higher than normal temperatures during the 2017–2018 summer, and the increased susceptibility of the pipis to environmental change as a result of the sporocysts. MPI will continue to monitor the pipi population in consultation with the Northland Regional Council, and has closed this investigation.

White growth on bullies, Waituna Creek, Southland

Environment Southland contacted MPI via the exotic pest and disease hotline to report common bullies (*Gobiomorphus cotidianus*) with white growths under the pectoral fins, from Waituna Creek, Southland. Several affected fish were seen but they were not dead or dying. A single specimen was collected and submitted to the MPI Animal Health Laboratory (AHL). This sampling had resulted from an earlier notification from Environment Southland of white growths on eels. The AHL carried out a biopsy of the white lumps and identified them as encysted metacercariae (late-stage larvae) of a parasitic trematode (flatworm), probably *Telogaster opisthorchis* or *Stegodexamene anguillae*, both of which are common parasites of New Zealand freshwater fish. Environment Southland was informed of the results and since no mortalities were reported the investigation was closed. However, Environment Southland will continue to monitor the health of the fish in Waituna Creek and will alert MPI if there are any mortalities.

Seaweed on an anchor, Paihia

A vessel owner notified MPI via the exotic pest and disease hotline of a mass of dense seaweed two metres long that he had pulled up with his anchor, at Roberton (Motuarohia) Island, Paihia. The notifier knows the area very well and had never seen the weed before so he collected samples and sent them to MITS. Molecular taxonomy found the species was *Chaetomorpha linum*, which has been previously recorded from New Zealand and is not thought to be invasive, so the investigation was closed.

Mantis shrimp, Nelson

The Nelson Marina (Nelson City Council) called the pest and disease

hotline to report a floating invertebrate in the visitor pontoon area of the marina. It was described as 7 cm long with “scales” and many legs, “similar to a centipede”, with nine segments on its back. The specimen was frozen and submitted to MITS, where it was identified as the indigenous mantis shrimp, *Heterosquilla tricarinata*. The caller was informed and the investigation stood down.

Caprellid shrimp, Tauranga

MPI received a report from a biosecurity officer at the Bay of Plenty Regional Council (BOPRC) of a suspect *Caprella mutica* on the hull of a boat in Tauranga Harbour. The specimen was submitted to MITS, where it was identified as *Caprella* cf. *equilibra*, which is cryptogenic, i.e., previously recorded in New Zealand (Webber *et al.*, 2010) but whether native or exotic is unknown. Also identified was *Jassa* cf. *marmorata* (Ischyroceridae), a cryptogenic tube-building amphipod, and *Stenothoe* cf. *moe* (Stenothoidae), an indigenous amphipod. As these specimens were obtained from the hull of a boat, they were not considered range extensions. BOPRC was notified of the results.

Greenlipped mussel mortality, Nelson

MPI received a call via the exotic pest and disease hotline about greenlip mussels (*Perna canaliculus*) in the Marlborough Sounds that had high levels of mortality in juvenile populations (large mussels showed no mortality.) Eighteen specimens were submitted to the AHL for bacteriology and histopathology testing, with specific testing for *Perkinsus*. It was noted at the time that the area was experiencing higher water temperatures than usual and little rain, and upwelling in the region could have caused functional starvation.

Results were negative for all *Perkinsus* sp. hypnospores. Histopathology showed severe multifocal lesions of the gills and the intestinal and digestive epithelium. The lesions were associated with autolysis as well as sloughing of the cells into the digestive lumen, often exposing the underlying basement membrane. No pathogens were found and the observed lesions were directly adjacent to healthy digestive epithelium. Collectively these observations suggest that toxicological or environmental events were the cause of the mortalities.

With no evidence of an infectious disease, the investigation was stood down.

Fish affected with fungus, Lake Benmore

A family camping at Lake Benmore over the Christmas holidays called the exotic pest and disease hotline to report a native galaxiid fish that looked as though it had a fungal disease. There appeared to be a kind of rot over the rear half of the body. The fish was still alive when caught, but appeared to be in poor condition and died shortly afterwards. The sample was frozen and submitted to the AHL. The notifier reported having seen a few other fish in the lake with the same problem. A skin scrape from the frozen specimen was examined for the presence of myxosporidian parasites and the fungal disease *Saprolegnia*, with negative results. No fungi were able to be cultured. The frozen sample was unsuitable for histology so the AHL was unable to conclude what the cause was.

Sick rainbow trout, Canterbury

Fish & Game North Canterbury called the exotic pest and disease hotline to report a number of sick and lethargic fish in Lakes Grassmere and Hawdon, inland Canterbury. Photos showed a rainbow trout with extensive reddening of the skin. A number of trout and bullies were submitted to the AHL to rule out an infectious disease. As a precaution, Fish & Game North Canterbury also ceased all transfer from its hatcheries (one in Christchurch and one on the Rakaia River), closed its hatcheries to visitors, and postponed its annual public fishing events that utilise hatchery-reared fish. Diagnostic results indicated that a non-infectious condition caused the mortality. All fish submitted had full digestive tracts, indicating that they were actively feeding. There was a lack of consistency in necrosis which, combined with the low severity of lesions, negative or insignificant bacterial growth, and negative virology results, suggested an environmental cause. From these results (and after consultation with Fish & Game North Canterbury) the investigation was stood down, though there was continued monitoring of the lakes over the remainder of the 2017–2018 summer.

Mortalities in geoduck larvae, Nelson

A researcher from the Cawthron Institute contacted MPI via the exotic pest and disease hotline to report high larval mortality in an experimental stock of geoduck (*Panopea zelandica*). The larvae were about 18 days old, which is a critical stage for settlement. Two million larvae were being used to investigate rearing at different temperatures. It was noted that the health of the larvae was likely to have been compromised by accidental underfeeding, as broodstock in the same water appeared healthy. Prior to this notification, bacteriology samples had been independently submitted to Nelson Hospital for analysis. Results from MALDI-TOF mass spectrometry (commonly used to identify bacterial isolates) showed no evidence of any significant pathogen. MPI and the Cawthron agreed that the mortalities were likely due to accidental underfeeding, and the investigation was stood down.

Porcupinefish mass mortality, Waihi

The MPI exotic pest and disease hotline received a number of calls from the public over a 3-day period regarding a large number of dead seabirds, porcupinefish (*Allomycterus jaculiferus*), tuatua, sponges and kelp washed up on the northeastern shores of the North Island, including Waihi Beach, Papamoa Beach, Coromandel and beaches north of Auckland. The first notification made was of dead porcupinefish on Waihi Beach, so two specimens were submitted to the AHL to rule out an infectious disease. The fish were very decayed and had no visible lesions other than small haemorrhages in the retina and around the mouth. Internal inspection was not possible owing to severe decomposition of the organs. All subsequent notifications in relation to this event came more than 24 hours after the first notification, so any specimens would have been too degraded for further analysis.

A large storm event bringing strong onshore winds and a king tide had occurred before the mortality event. Considering the large number of birds, fish, and other marine organisms that were found over an extended section of the North Island eastern coastline, they

were more likely to have been casualties of this storm event than a disease or toxic event. Porcupinefish are a frequently discarded by-catch of coastal trawlers in northern NZ and tend to be washed ashore undamaged, as they are buoyant and (perhaps owing to their dense body covering of spines and the toxicity of their organs) are not broken down by marine scavengers. Based on this storm event, and given the lack of fresh samples for bacteriological testing, this investigation was stood down.

Shellfish washed up on Peka Peka Beach

A member of the public called the exotic pest and disease hotline to report many dead pipi washed up on Peka Peka Beach, Horowhenua. Two separate samples were submitted to the AHL to rule out any exotic disease agents. However, both submissions were already autolysed on arrival at the laboratory, suggesting the shellfish had been washed up for some time, and it was not possible to perform diagnostics on them. There had been numerous adverse storm and extreme heat events prior to this time and it is likely that these shellfish mortalities were primarily due to environmental factors.

Very thin trout, Lake Taupo

A member of the public called the MPI exotic pest and disease hotline after he caught a 40-cm trout in Lake Taupo that looked healthy but was very thin. The notifier reported that upon gutting the fish he noticed there was nothing in the digestive tract and that the entire length of the back of the body cavity was black. There was also a 20-mm-wide area of what was described as “black congealed blood” under the entire length of the membrane between the gut cavity and the backbone, but this is an accurate description of both the location and appearance of normal kidney in fishes. The fish was submitted to the AHL, but because it had been cleanly gutted there were no remains of any internal organs from which cell culture of bacteria or virus could be performed. The sample had also been frozen, destroying the cellular structure and rendering it unsuitable for histology. This investigation was closed as MPI was unable to provide any diagnostic information.

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Clarifying the pest status of some exotic termite species for New Zealand

Exotic termites of pest significance pose a risk to New Zealand's biosecurity and our nation's trade if they become established. New Zealand has experience of exotic termites arriving here via imported commodities and other pathways. Fortunately the vast majority of these arrivals have either been dealt with at our borders or, if found within our borders, they have been contained, treated and eradicated.

Only three exotic termite species are considered to have become established in New Zealand – *Kalotermes banksiae* and *Glyptotermes brevicornis* (Kalotermitidae), and *Porotermes adamsoni* (Stolotermitidae), evidenced by repeated collections over time from local-origin host material. While none of these established species are considered of great economic significance, other exotic termite species found within New Zealand's borders certainly are significant pests. Of these, the most important are the Australian subterranean termites *Coptotermes acinaciformis* and *Coptotermes frenchi* (Rhinotermitidae), and the West Indian drywood termite *Cryptotermes brevis* (Kalotermitidae). Owing to the economic significance of these three species, whenever they have been found in New Zealand the response objective has been total eradication.

Here we provide a summary of the detections of these species, present up-to-date information on the eradication measures that have been undertaken in response to them, and propose that the status of all three species for New Zealand can be considered "absent: eradicated".

The need for clarification

It is crucial to be clear about which species are established and which are not. There has been some confusion regarding the presence or absence in New Zealand of the three exotic termite species *Coptotermes acinaciformis* and *Coptotermes frenchi* (Rhinotermitidae), and *Cryptotermes brevis* (Kalotermitidae). This stems from a number of factors. There has not been

a great deal of attention given to termites as a taxonomic group generally in New Zealand, perhaps owing to our very small native fauna. There has also been little discussion of exotic species in the New Zealand context. What publications do exist have been rather sporadic, resulting in long periods of time during which the status of some exotic species may not have been clear. Further, there has been a lack of published information regarding any detections of exotic termites and actions undertaken by agencies that hold this information (i.e., MPI and its predecessors). On top of all this, confusion arises from the use of different terms, definitions, or interpretations of terms, by different authors and agencies.

The field of invasion biology has created many terms to describe the status of an organism in a particular geographic area, both fundamentally (i.e., is it present or absent) as well as the nature of any presence. MPI developed operational protocols for responding to detections of *Coptotermes*, which have used the term "eliminated" to designate the stage of operational activities when all known activity of the termite at a location had ceased, and this was evidenced by a series of consecutive inspections that found no activity. Previously MPI only used the term "eradicated" at the end of a subsequent period of continued surveillance at the location, in most cases up to 5 years after all termite activity actually ceased.

The International Plant Protection Convention (IPPC) International Standards for Phytosanitary Measures (IPPC 2017a,b) define specific terms and pest status categories. The IPPC does not use the term "eliminated", but IPPC (2017a) does define "eradication" as "application of phytosanitary measures to eliminate a pest from an area [formerly eradicate]". Thus it is clear that "eradication" and "eradicated" mean the same thing as "eliminate" and "eliminated". Therefore we propose to use the term "eradicated" rather than "eliminated" wherever the activity of an exotic termite is deemed to have ceased.

The national pest status of the termite species from the time when all cases have been eradicated can be considered as "absent: eradicated" (IPPC 2017b). With this in mind, we can review the known detections of *C. acinaciformis*, *C. frenchi* and *C. brevis* in New Zealand and consider the eradication measures undertaken and outcomes achieved to date for these species.

Coptotermes acinaciformis and *Coptotermes frenchi*

Bain & Jenkin (1983) reviewed a range of exotic termite species for New Zealand, including *C. acinaciformis* and *C. frenchi*. The terms they saw as best applying to records of exotic termite species were "established", "recorded from" and "intercepted". They considered both *C. acinaciformis* and *C. frenchi* to be "established", which they defined as meaning that there was "evidence of these species spreading from host material in which they were not imported". The designation "established" was based on known activity of the two species in New Zealand at the time: *C. acinaciformis* was still active at one site in Te Puke and another in Auckland, and *C. frenchi* was still active at one site in Hamilton and another in Auckland. It is important to note that these locations were under treatment at the time, and that Bain & Jenkin (1983) also reported other locations of historical activity for both species where they had been successfully eradicated.

In the more than 20 years since, further detections were made of both *C. acinaciformis* and *C. frenchi* in New Zealand (MPI, unpublished data). All detections, both prior and subsequent to Bain & Jenkin (1983), have been treated by relevant government agencies, and official programmes continued through the 1980s and 1990s to eradicate them. Eventually in 2005 both species were officially declared to be "eradicated" following a period of surveillance after activity had actually ceased (Ross, 2005a,b). It is important to revisit our earlier comments about the terms used at the time by MPI's predecessor, the

Ministry of Agriculture and Forestry (MAF), which used the term “eliminated” rather than “eradicated” when all *Coptotermes* activity actually ceased in 2001 (Ross, 2005a). We proposed that it was at this point that eradication, as per the IPPC (2017a) definition, was actually achieved. Following this, MAF ran a surveillance programme until 2005, during which no further termite activity was found.

After the widespread efforts up to 2001 to eradicate *Coptotermes* from New Zealand, two new detections of *C. acinaciformis* were made in January 2006 at Richmond (near Nelson), and in January 2007 at Coatesville (near Auckland) (Ross, 2006; Pearson & Bennett 2008; Philip *et al.*, 2008). As before, official eradication programmes were implemented for both locations. Eradication (as per IPPC definition) was quickly achieved at both locations, with all activity having ceased at Richmond by June 2007 and Coatesville by April 2008 (Pearson & Bennett, 2008). MacFarlane *et al.* (2010) included both *Coptotermes* species in their contribution to the *New Zealand Inventory of Biodiversity* as “adventive (naturalised alien)”. The inclusion of *C. acinaciformis* can be understood, considering that MAF continued to use the term “eliminated” rather than “eradicated” at the time (e.g., Pearson & Bennett, 2008). Similarly, Evans (2011) reviewed invasive termites of the world, and he too designated *C. acinaciformis* as “established” in New Zealand, citing Bain & Jenkin (1983), Philip *et al.*, (2008) and Ross (2005a,b).

It is unclear why MacFarlane *et al.* (2010) included *C. frenchi* in their list. Unfortunately their information has in turn been repeated in several online resources (Anon., n.d., a, b). In contrast, Evans (2011) correctly postulated that

C. frenchi had “possibly been eliminated from New Zealand” because the species had been under active management at the time of Bain & Jenkin (1983), and had not been mentioned from New Zealand sources since. Indeed, no new detections have been made of *C. frenchi* in New Zealand since 1998 (MPI, unpublished data).

Despite the successful eradication again of *C. acinaciformis* from known sites in New Zealand by April 2008, still more detections of this species have been made in subsequent years. The location, date of detection, date of eradication, and actual or proposed end of surveillance for these recent responses to *C. acinaciformis* are shown in **Table 1**. At the time of their detection all these could be considered as fitting with the IPPC definition of “establishment”: “perpetuation, for the foreseeable future, of a pest within an area after entry” (IPPC, 2017a). However, at present they are all at a stage where they should be considered to be eradicated (as per IPPC definition). Five locations are still under post-eradication surveillance.

Cryptotermes brevis

Cryptotermes brevis (Kalotermitidae) is not included in any of the publications about termites in New Zealand mentioned so far. Alate termites were noted by residents of a house in February 2011, and they reported this to MAF who identified them as *C. brevis*. The first public report of this termite was by Bain (2011), who summarised the detection and the actions being undertaken by MAF, and Bennett (2012) provided more details. A thorough inspection of the house found multiple imported wooden items with signs of drywood termite. Several items were destructively sampled and were confirmed as infested with

live *C. brevis*. The official eradication programme involved removal of wooden items from the house for fumigation, and an entire house-fumigation using sulphuryl fluoride, in October 2011. MPI is still conducting extended post-eradication surveillance for *C. brevis*, similar to the eradication protocols for *Coptotermes* but in this case for 10 years. MPI therefore expects to end response activities for *C. brevis* by 2021. To date there has been no activity of *C. brevis* found since 2011. We therefore conclude that this species should also be considered “absent: eradicated” from New Zealand.

Summary

It is clear that *Coptotermes acinaciformis*, *Coptotermes frenchi* and *Cryptotermes brevis* have been found in New Zealand in the past, and that many of these finds could have been justifiably considered as an “establishment” (as defined by IPPC) at the locations where they were found. However, MPI has implemented measures to eradicate all detections of these species and there is no evidence of any activity by any of them in New Zealand at present. Also, these species have never been found without a direct association or link with imported timbers. For example, all incursions of *C. acinaciformis* since 1999 have been directly associated with imported Australian hardwood. Therefore we propose that all three species can be considered “absent: eradicated” from New Zealand.

To comply with operational protocols that MPI has adopted for these pests, MPI will continue post-eradication surveillance at some of these locations for a number of years. However, this should not be taken to suggest these pests are not yet eradicated, but to show that MPI

Table 1: Responses to <i>Coptotermes acinaciformis</i> detected in New Zealand since 2001 (source: MPI, unpublished data). Locations numbered 1 to 9 are also shown in Figure 1.			
Location	Date detected	Date eradicated	End of post-eradication surveillance
1 Richmond (Nelson)	Jan 2006	Jun 2007	Jul 2011
2 Coatesville (Auckland)	Jan 2007	Apr 2008	Mar 2013
3 Nelson	Nov 2009	Mar 2011	Aug 2016
4 Pukekohe/Patumahoe (Auckland)	Nov 2010	Dec 2011	Dec 2016
5 Point Wells (Auckland)	Jan 2012	Nov 2013	Pending – set for Nov 2018
6 Drury (Auckland)	Nov 2012	Nov 2013	Pending – set for Nov 2018
7 Mangere (Auckland)	Sep 2013	Dec 2014	Pending – set for Dec 2019
8 Walton (Waikato)	Sep 2013	Dec 2014	Pending – set for Dec 2019
9 Omaha (Auckland)	Sep 2014	Nov 2015	Pending – set for Nov 2020

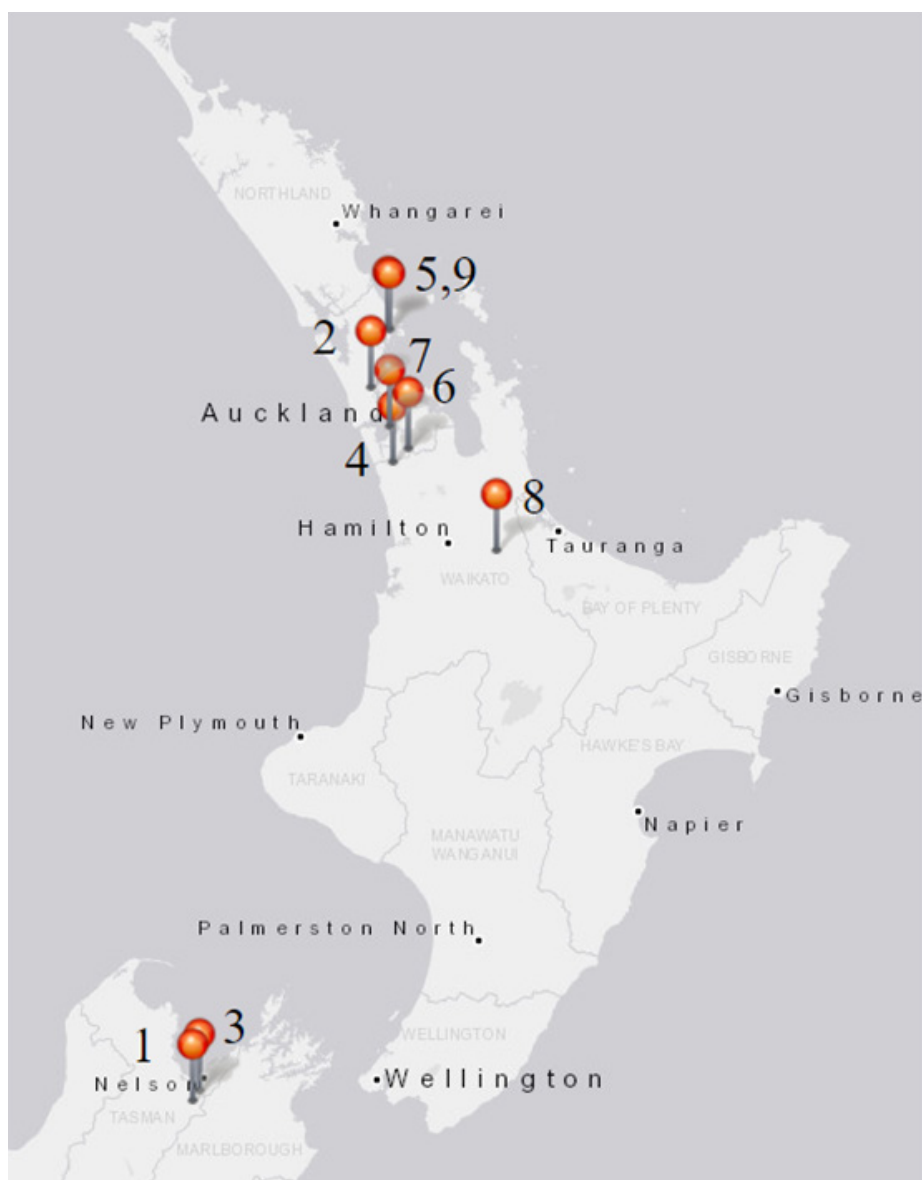


Figure 1: Locations of responses to *Coptotermes acinaciformis* detected in New Zealand since 2001. See Table 1 for details of each location.

is continuing to take a precautionary approach. Notably, there have been five successful eradication programmes for *C. acinaciformis* at different sites in New Zealand since 1999, none of which has subsequently seen a renewal of activity. With all responses since 2005 following similar protocols, we have very high confidence in each eradication, and so New Zealand should be considered free from *Coptotermes* and *Cryptotermes brevis* as defined by ISPM (IPPC, 2017a).

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3D printing and biosecurity

Three-dimensional (3D) printing is an exciting and innovative technology. It is relatively easy to use, is available to anyone and has applications in biosecurity. It enables the user to create a model of just about any object, save that model as a piece of code and then turn it into a three-dimensional copy. This technology is becoming increasingly common and is just beginning to be used in biosecurity.

While 3D printing may seem complicated, it is actually quite straightforward. It is basically like two-dimensional printing, but with multiple layers that are built up sequentially – in contrast with the usual processes of cutting, machining, casting and moulding.

Printing starts with a model drawn in a 3D-creation software (**Figure 1**). These programmes allow the user to create

almost any shape and then export it as an .stl file. Alternatively, individual model .stl files can be downloaded from online databases. One of these databases (thingiverse) has over a million available models. The selected .stl file is then loaded into a slicing software, which converts the model into a .gcode (**Figure 2**) that contains the design of the model and the information to drive the printer. Once the file is received, the printer heats up the printing material until it can be extruded out of the nozzle and the model is printed in a series of horizontal layers, working from the bottom up.

The most common materials used for printing are plastics such as acrylonitrile butadiene styrene (ABS), polylactic acid or polylactide (PLA), polyethylene terephthalate (PET), polyvinyl alcohol (PVA) and aliphatic or semi-aromatic polyamides (nylon). Printing

can also use plastic combined with other materials, such as wood, metals or carbon fibre, to alter the look and features of the final product. Not all printers are capable of printing with all these materials, and it is the final use of the object that will determine the printer and material used.

PLA plastic is the most common material used as it is cheap, easy to use and produces medium-to-high-quality objects that are adequate for most purposes. Printing using pure metal is becoming increasingly popular at the industrial level, but is not yet available in printers producing objects for retail markets.

The speed of printing largely depends on the size of the object, but is also affected by the speed at which the nozzle is programmed to move, the thickness of the object shell and the percentage of infill: a hollow model (0 percent infill) is much quicker to print than a solid model (100 percent infill). These settings can be changed to faster travel speeds and lower infill densities for faster printing, but this may reduce the quality. Objects can take anything from a few minutes to several hours to print. For example, a regular coffee mug at 20 percent infill takes 8–9 hours to print.

The software programs generate the code to communicate with the printer, but interpreting the software programs and troubleshooting when printing goes wrong can be challenging. Settings in the slicing software must be customised for the printer. Small changes to the settings and the characteristics of the material being used for each printing job can make the difference between success and a pile of melted plastic. It is important to tap into the knowledge of people who have experience in 3D printing and use the extensive online resources available, but lots of trial and error may still be required. While some programmes and printers are more user-friendly than others, the sheer number of functions available for modelling and slicing software can be daunting. This can make the whole process frustrating and time-consuming at the outset, but will ultimately provide worthwhile benefits.

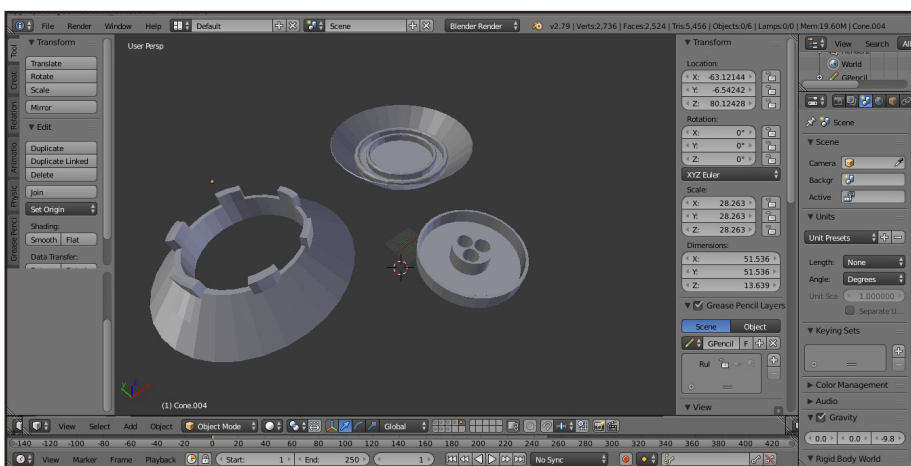


Figure 1: The model of the ant trap in the 3D-modelling software Blender™

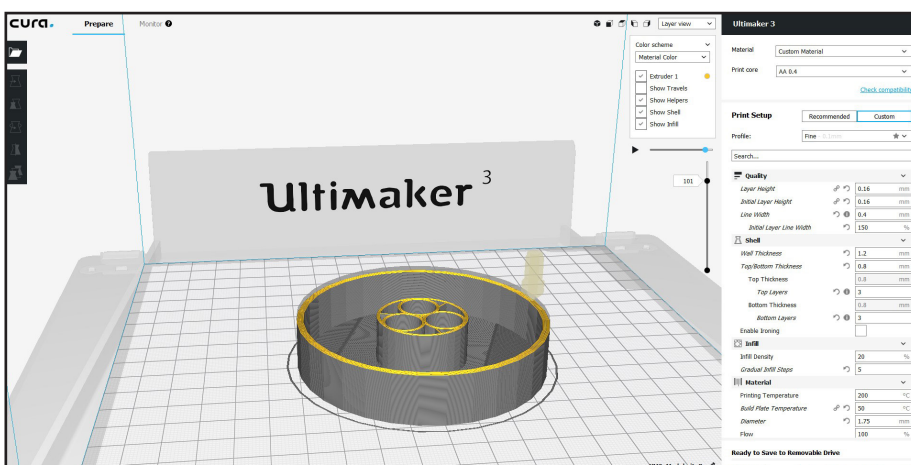


Figure 2: The bait container for the ant trap in the slicing software Cura™. The software takes the model in Figure 1 and slices it into horizontal layers before printing.

The real value of 3D printing comes from empowering the user to cheaply design, make and alter niche-use objects. Rather than having to improvise or out-source expensive traditional manufacturing, 3D printing enables objects to be designed, refined and produced in-house. Many 3D printers cost less than \$1,500, and as they become even more readily available they will become commonplace.

MPI is using a 3D printer to make ant surveillance traps for the National Invasive Ant Survey programme (NIAS). The programme, which has been running since 2003, uses targeted surveillance at high-risk sites to detect incursions of exotic ants. One of the tools used by NIAS is a dome trap (**Figure 3**), which provides surveillance for up to 2 weeks at high-risk sites. Dome traps complement pottle traps, which are used at higher densities for shorter periods (2 hours) because the attractant in them dries out within this time. In trials in 2017 at Ports of Auckland, four dome traps each detected an exotic ant incursion that other methods had failed to find. In 2018, two dome traps each caught an exotic ant species. It was noted, however, that the large size of the dome traps, and the time required to put attractant into the baiting system, limited their practical use.

The specialised purpose of dome traps and lack of a commercial market means that outsourcing for continued trap development would be costly and time-consuming. Instead, MPI purchased a 3D printer and modified the original dome trap design in-house. Scale models have been produced to ensure that the modifications work (**Figure 4**). The next stage is to print out full-sized traps for testing in the field.

In summary, 3D printing is readily available, relatively cheap and does not require any particular skill to operate, just the desire to learn and ability to persist. There are many potential applications of 3D printing in biosecurity – all it takes is some innovative ideas and the tenacity to develop practical applications for them.

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Figure 3: Original dome trap in the field



Figure 4: 3D-printed ant trap made up of three components

Exotic plant and environment investigations report: January to March 2018

On-line trading, imported goods and seeds

Growth of imports from on-line trading has increased New Zealand's exposure to unauthorised seed importations, with accompanying risks of introducing new exotic invasive plants and seed-borne diseases. There were three detections over the last quarter involving unauthorised seed importations: two from on-line purchases and one from a retail purchase. The first case involved a small parcel containing six packets of seeds purchased on-line from an e-commerce store in China and submitted to MPI by someone who had received a mislabelled package. A note in the package listed the seeds as raspberry (*Rubus* sp.), chilli (*Capsicum* sp.), rose (*Rosa* sp.), ginger (*Zingiber officinale*), ginseng (*Panax ginseng*) and multi-coloured grass seeds (unknown species), but the declaration stated that the contents were "Toys". The investigation established the recipient's address in Wainuiomata, and that parcels of seed destined for this place had been intercepted on six other occasions in November 2017. The case was referred to the MPI Intelligence Targeting Team to follow up the possibility of intentional importation and false declaration, and MPI staff at the International Mail Centre sent information sheets to the importer after the initial seizures.

The second case also involved seeds from the same retailer, which were intercepted by MPI Quarantine Officers at the International Mail Centre. Investigations determined that several other unauthorised packages had previously been received by the person in question, and some imported cherry seeds had been scattered throughout a yard on a rural lifestyle section. Seeds retrieved by MPI were destroyed and, after allowing time for the seeds to germinate, the yard was inspected and no seedlings were found. Information was provided to the residents about the risks of on-line seed purchases, with a request to call MPI if they saw any plants that had germinated from the previous illegal online purchase. The Biosecurity Act 1993 and the Hazardous Substances and

The Ministry for Primary Industries Incursion Investigation team and Plant Health and Environment Laboratory (PHEL) investigate and diagnose suspect exotic pests and diseases in the plant and environment sectors. Investigators and scientists are based in Auckland, Wellington, Rotorua and Christchurch. These teams provide field investigation, diagnostic testing and technical expertise to detect and report new pests and diseases affecting plants and the environment. They support surveillance and response functions, including carrying out research and development. The following report summarises investigations during the first quarter of 2018, covering the late summer period.

New Organisms Act 1996 apply.

The final case involved seeds found in a loofah back-scrubber purchased from a homeware retail chain. It was likely that the seeds had been left behind when the loofah was processed after harvest. The owner of the loofah collected the seeds and sent them to MPI for destruction.

Brown marmorated stink bugs

There were eight post-border detections of brown marmorated stink bug (*Halyomorpha halys*, BMSB) during this quarter, all confirmed by PHEL entomologists, and control measures were applied.

The first occurred at a container facility in Auckland, where an adult flew into a fork lift and was caught by the driver, who reported it to MPI. The specimen was an unmated, non-reproductive female. Three Incursion Investigators and two PHEL entomologists visited the site and found no further specimens. BMSB pheromone traps were deployed and routinely visited for 8 weeks, and no further BMSB were found. Fact sheets and posters were provided and staff were informed and asked to report any further sightings.

The second case involved an adult that was found by a MPI Quarantine Officer at a freight-forwarding Transitional Facility, on a pallet destined for export. The facility had received imported consignments from China, the US and Italy. The specimen was confirmed as an unmated non-reproductive female. A MPI Incursion Investigator and PHEL entomologists inspected the site

and placed two BMSB traps that were monitored for 12 weeks with no further detections.

The third detection involved a saddle from the US at the Auckland International Mail Centre, where an MPI inspector found an adult inside a securely wrapped, well-taped package. The saddle was fumigated and inspected again before release. The specimen was confirmed as an unmated, non-reproductive female.

In the fourth case, a Nelson company found live adults in a containerised consignment of machinery parts air-freighted from Germany, which included conveyer-belt parts from Italy. An MPI Quarantine Officer undertook a site visit and assisted with fumigation of the sealed consignment, which had arrived shrinkwrapped.

The fifth occurred inside a house in Auckland. A live adult was found on an interior wall by a person who had recently arrived from Seattle, USA. Twenty boxes of personal effects had been received the day before and were being unpacked. An MPI Incursion Investigator and PHEL entomologist inspected and supervised unpacking of the remaining boxes. One further squashed adult BMSB was found in the folds of a rug. Six boxes of crumpled paper used as packing material were destroyed by incineration as a precaution.

In the sixth case, a dried-out adult was found in a box of hydraulic fittings on stock shelves in an Auckland storeroom. The stock had arrived from Italy in December 2017 and undergone mandatory fumigation on arrival. Staff

conducted a thorough search of the storeroom and found no further insects.

The seventh detection occurred at the Auckland International Mail Centre, when the NZ Post manager caught a live bug on the phone in her office and gave it to an MPI Quarantine Officer. The office was in an internal room with no external openings. The office was inspected by MPI staff and no further insects were found. MPI staff also inspected the building and deployed two UV light traps at the site.

The eighth detection, in Christchurch, involved a pre-cleared vehicle from Japan; one dead adult was found in the vehicle, together with specimens of yellow spotted stink bug (see below).

Yellow-spotted stink bugs

There were four detections of yellow-spotted stink bug (*Erthesina fullo*, YSSB), all in imported vehicles from Japan. The identifications were confirmed by PHEL entomologists, control measures were applied and each investigation was closed without any further sightings.

The first case involved one adult that was caught as it emerged from a car in a sandblasting bay in Christchurch, when the exhaust cover was removed. The vehicle had been recently imported from Japan. It was fumigated and no further insects were found after a post-treatment inspection.

In the second case, staff at a compliance yard in Christchurch found a bug on the windscreen of a second-hand vehicle that had been pre-cleared in Japan. An MPI Incursion Investigator checked the site and collected the bug, together with three more dead specimens found inside the vehicle. The vehicle was fumigated and no further insects were found. PHEL identified the live specimen and two of the dead bugs as YSSB; the other dead specimen was a BMSB. Detection of more than one species of stink bug in one event occurs periodically. Staff at the compliance yard were asked to watch for further bugs, and MPI provided posters and specimen jars. It was also established that the vehicle had been stored for almost 2 months at an overflow car yard in Wigram. MPI staff inspected the yard and placed awareness posters in the staff area. There were no further sightings.

The third case also occurred in Christchurch, when a panelbeater working in a vehicle compliance yard

found and squashed an adult YSSB after removing a panel. MPI was notified and an Incursion Investigator visited the workshop and collected the remains of the insect, which was identified by PHEL. The vehicle was fumigated and no further insects were found.

The fourth case occurred in Dunedin, where a live YSSB was found inside a back bumper when it was removed as part of a vehicle-compliance check. The vehicle was fumigated and re-inspected, and no further bugs were found.

Exotic ants

There were four detections of exotic ants, which were identified by PHEL entomologists. Three different species were involved, control measures were applied and each investigation was closed with no further sightings.

The first case involved carpenter ants, *Camponotus* sp., which were found at a Transitional Facility in Albany, when unusually large ants were seen in a shipment of milkshake syrup from Australia at a food-import business. Carpenter ants are a regulated pest. Some species damage wood, and the soldiers may bite. A thorough inspection was conducted and no further ants were found. The pallets were sent for incineration as a precaution.

The second detection was in Wellington, where an ant nest was found among a shipment of boots made in Indonesia. The stock was treated with insecticide and inspected by shop staff. No further ants were found. The ants were identified as *Trichomyrmex destructor*, the Singapore ant, an exotic species. Further investigation determined that the consignment had arrived several weeks prior and had been stored at a cargo depot in Auckland before distribution. The depot staff were contacted and agreed to check remaining stock. However, only one pair of the boots remained and no more ants were found. There have been no subsequent reports of ants in boots from the public. The depot has been added to the National Invasive Ants Surveillance (NIAS) programme for 2019.

In the third case, ghost ants (*Tapinoma melanocephalum*) were found at Ports of Auckland during the NIAS programme. A contractor treated the site with insecticide and laid toxic bait stations. The site was re-visited and no more ants

were found, but it was re-treated.

The fourth case also resulted from the NIAS programme, when Singapore ants, *Trichomyrmex destructor*, were found at Centreport, Wellington. An ant-control contractor was despatched to the site and searched for further ants using attractant bait in suitable habitat close to the detection site. No ants were found. Toxic baits were laid and the site was treated with a residual insecticide. Follow-up visits yielded no further ants. The residual treatments used would have eliminated any cryptic population of ants present.

Agriculture

There were two detections of velvetleaf (*Abutilon theophrasti*), an Unwanted Organism and serious pest of agricultural crops (also known as Indian mallow).

The first was on a farm on the Awhitu Peninsula, near Auckland, where a single plant was identified by PHEL. The MPI velvetleaf response team was notified for referral to Auckland Council to undertake initial removal and treatment action. The MPI Long Term Management Team has arranged for ongoing management and control.

The second case was in the Wintergardens, Auckland Domain, where several plants were found among sunflowers in a glasshouse. Some had set seed. The New Zealand-sourced sunflower seeds were considered a possible source, so the remaining packets of these seeds were inspected but found to be free of contamination. The supplier also inspected a sample of the bulk seed lot and found no contaminants. After further on-site inspection no further plants were found. The glasshouse had been scheduled for demolition, and Auckland Council agreed to monitor the site.

At Leeston in Canterbury, chaff, seeds and soil were found in a combine harvester imported from Austria, and MPI was notified. The harvester was dismantled, cleaned and inspected in a Transitional Facility. Seed samples were sent to theASUREQuality Seed Testing Laboratory for identification and viability testing. More than a thousand seeds of 19 species were found in the chaff samples, and seeds of 12 of these species were found to be viable. MPI staff concluded that there was demonstrable risk and non-compliant seed associated with this

case, and has forwarded details to MPI's Intelligence and Targeting Team with recommendations for assessment and action.

Forestry

There were six reports involving wood-boring insects. The first occurred in Rotorua, where a motelier found insect frass coming out of small holes in a luggage rack. Several racks had recently been purchased from a supplier who had imported the racks from China. Photos of the damage and a description were consistent with a live infestation of wood-boring insects. The racks were heat-treated as a precaution. The importer advised MPI that the racks had been widely distributed and no other reports of problems had been received. A fumigation certificate stated that the racks had been fumigated in China before export.

The second case involved a live beetle found in a wooden coaster from Thailand, and was reported from Gore. The beetle was identified as the powderpost beetle (*Apoleon edax*), a species not considered present in New Zealand. The set of coasters had been purchased from a homeware store about 18 months previously. The coasters were frozen to kill any remaining insects.

In the third case, in Wellington, borer holes were found in a baseball-bat set bought at a chain store and imported from India. Four adult male and female African powderpost beetles (*Lyctus africanus*), were identified from the bat. This exotic species is present throughout Africa, Madagascar and the Orient, and has been introduced to Europe. It is regarded as one of the most destructive pests of timber and timber products, including plywood, and can also infest dried roots and tubers. The Australian supplier was notified by MPI and collected the bats for inspection and destruction. MPI staff also arranged for treatment of the property where the borer was first found.

The fourth case, in Hastings, involved a wood-boring insect found in a wooden brick mould imported from India and sold as a rustic ornamental item. A customer had returned it to a Napier homeware store, and when the store owner soaked it in water a larva emerged, which he killed in boiling water before calling MPI. The larva was

identified by PHEL as the Kulsi teak borer, *Stromatium barbatum* (a regulated species not established in New Zealand). Destructive testing revealed two further live *S. barbatum* larvae. The remaining stock of 12 moulds was inspected for insect damage; two had signs of damage and were destructively inspected, with no insects found. The remaining 10 were externally inspected and found to be free of any sign of insect infestation, but were frozen for 7 days to kill any insects present and returned. Kulsi teak borers are native to a number of Asian countries with hotter climates than New Zealand. A certificate indicated that the moulds had been fumigated in India prior to shipment, but the presence of live larvae demonstrated that this had failed. The MPI Intelligence and Targeting Team was notified.

The fifth case involved borer in a wooden serving platter purchased from a Tauranga chain store. The platter was thought to contain live borer, and photos received by the MPI Incursion Investigator confirmed this. The item was destroyed and staff at the store removed the remaining stock from the shelves for inspection. No further damage was found. The consignment that had included the infested item was examined and there was no evidence of a widespread problem. There have been no further reports of damage.

The sixth case involved borer in cricket sets imported from India. Borer holes were found in the sets by an Approved Person at a Transitional Facility in Auckland. From the consignment of 378 sets, all but 41 had been sold from stores around the country. Further investigation by the Approved Person found five more sets with borer damage at several sports stores. These were recalled to the Transitional Facility and one set was destructively inspected by PHEL entomologists. Specimens were identified as the false powder post beetle (*Sinoxylon anale*), which is not present in New Zealand. The 40 remaining cricket sets were sent for destruction.

Sawn timber imported from Bali for manufacture into furniture, souvenirs and handicrafts was found to be covered in mould and showing signs of fungal decay. The timber had been imported in a container along with other goods. Spiders were found in the container during MPI inspection in Auckland

and it was fumigated. After release, the container was transported to Waiharara, Northland, but the wood was found to be very damp on arrival. Moisture could have built up in transit, accelerating the growth of mould. The timber was contained inside a tarpaulin and MPI staff supervised its destruction through burning on-site.

Items described and declared as cat and dog toys, purchased online by a person in Whanganui, were found to consist of bark-covered twigs, and MPI was notified. The items were purchased through a Facebook listing and the notifier believed the origin was the US, but the parcel arrived from China via China Post. The twigs were destroyed by MPI.

Horticulture

MPI was told by a person in Palmerston North that an imported consignment of tulip bulbs (*Tulipa* sp.) had not received the proper border clearance. The bulbs had been purchased in Amsterdam by the notifier's friend and sent by mail as a Christmas gift from Germany. MPI Quarantine Officers collected the bulbs and arranged destruction.

An Australian mango purchased from a supermarket in Nelson had several live weevils inside the seed, which PHEL entomologists identified as mango seed weevils, *Sternonchetus mangiferae*. Commercial consignments of Australian mangoes undergo irradiation before export, and therefore any live insects would have been rendered sterile. Untreated *S. mangiferae* would be unlikely to establish in New Zealand as they are host-specific, requiring mangoes to complete the life cycle, and they also have a tropical distribution.

Environment

A person informed MPI that they had collected hornwort (*Ceratophyllum demersum*) plants from Lake Rotoehu for their indoor aquarium and had subsequently propagated and advertised them for sale on Trade Me. They then became aware that hornwort was being eradicated from the lake, and, since it was an Unwanted Organism, propagation and distribution was illegal. Trade Me was contacted by an MPI Incursion Investigator and the listing was immediately withdrawn. MPI provided further information about the regulatory

status of hornwort, and all remaining plants were destroyed.

There were four reports of exotic spiders during this quarter. The first was of a huntsman spider found in a Rotorua motel room while cleaning. PHEL staff identified *Neosparassus calligaster*, an Australian species not present in New Zealand. It was likely that the spider arrived in the luggage of an international traveller.

The second occurred at Hanmer Springs when a red-backed jumping spider (*Phidippus johnsoni*) was found in Californian grapes at a grocery store and reported to the produce manager. The grapes had arrived from California about 10 days previously and most had already been distributed. Remaining stock was checked for spiders and webbing at the distribution centre and the store, and found to be clean. The spider, which was initially identified from photos and subsequently confirmed by a PHEL entomologist, was likely a lone hitchhiker.

The third interception involved a banana spider (*Heteropoda venatoria*) found in Christchurch by an Approved Person at a Transitional Facility, on the floor in the container devanning area. It was not directly associated with any imported goods but this is a commonly intercepted species that has not become established in New Zealand. It occurs in tropical and some subtropical regions including Asia, South America, the southeastern US and Australia.

The fourth involved a packaging business where an unusual spider was found in the bathroom and reported to MPI. It was identified by PHEL as the Victorian huntsman, *Isopedeella victorialis*. It was possible that the spider was an isolated hitchhiker from one of the neighbouring sites. An Incursion Investigator contacted Transitional Facilities in the vicinity and asked staff to report any further detections to MPI. While several specimens were found in Christchurch in 2005 and 2006, this species is not known to be established in New Zealand.

In Nelson, a variable shield bug (*Kapunda troughi*) was found in the wrapping around oven trays from an oven imported from Adelaide, Australia. The oven was wrapped in plastic and polystyrene and the bug was therefore likely packed into the oven at the factory.

Storage pests

In Napier, a person reported the presence of live black beetles in chickpeas purchased from a café store. PHEL entomologists identified them as *Callosobruchus maculatus*, a regulated stored-product pest not known to be present in New Zealand. MPI's investigation determined that the chickpeas were a containerised 4,000-kg shipment from Turkey, which had been inspected, released, distributed to retailers nationwide and sold. The chickpeas at the café were in a sack, which was frozen for 10 days after which no further insects were found on re-inspection. *C. maculatus* is intercepted periodically, and while it is damaging to some stored pulses, it is not considered likely to establish in New Zealand because of our temperate climate and absence of living hosts. Infestations in storage areas can be eliminated by freezing infested pulses and keeping them in sealed containers, and thorough cleaning including in cracks and crevices.

In Auckland, insects in spices from Sri Lanka were identified as adult drugstore beetles (*Stegobium paniceum*, which are present in New Zealand) and two adult female Singapore ants (*Trichomyrmex destructor*, a regulated species not known to be present here). Also found was *Tyrophagus tropicus*, a stored product mite not known to be present in New Zealand. The spices were frozen to kill the pests.

Also in Auckland, live moth caterpillars were found in cinnamon sticks and dried chillies purchased from an ethnic food store. They had dispersed through the pantry where the spices were stored. Specimens were identified as the dried fruit or date moth (*Cadra calidella*), which is not known to be present in New Zealand. All contaminated items were frozen for 48 hours and the pantry was cleaned.

New to New Zealand species Cricket found in house in Northland

A notifier in the Far North, who was an experienced entomologist, reported a suspect new cricket species, and stated that for the past 10 years the crickets had arrived every year on the same date in February and stayed only for a couple of days. The notifier had lived in the house

for more than 12 years. The cricket was provisionally identified as *Pteronemobius truncatus* group (Orthoptera: Gryllidae). This was confirmed by a specialist on Orthoptera at the Australian National Insect Collection. Male specimens of this species group can be distinguished only by their song or call, so it was not possible to identify it and thus determine whether it is a newly discovered native species or an introduction from Australia. *Pteronemobius* is a very large genus with worldwide distribution and is very common in tropical regions. It is difficult to assess the potential biosecurity risk for this organism because it does not have a species name yet. There are no molecular tools available for this group of insects, so no comparisons can be made for the sequences extracted from this specimen.

A literature search did not reveal any information about any *Pteronemobius* species being potential pests, but any risk can be conclusively evaluated once an identification is made to species level. The cricket appears to be well established in the area, and it is considered important to have a final identification to a species level. Therefore the notifier was asked to record the call of the crickets when they appear again (presumably in February 2019) and to provide live individuals for rearing by PHEL. A PPIN report was generated to record this detection.

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PEST WATCH: 27 February – 23 May 2018

Biosecurity is about managing risks: protecting New Zealand from exotic pests and diseases that could harm our natural resources and primary industries. MPI's Diagnostic and Surveillance Services (DSS) 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 27 February 2018 to 23 May 2018. 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	<i>Pteronemobius</i> sp. cricket	Found in house	Northland	PHEL (General Surveillance)	Noted each summer over several years.

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



Veterinary Diagnostic Laboratories

GRIBBLES VETERINARY PATHOLOGY

- **AUCKLAND**
Courier: 37–41 Carbine Road, Mount Wellington, Auckland 1060
Postal: PO Box 12049, Penrose, Auckland 1642
Tel: 09 574 4701 Fax: 09 574 5304
- **HAMILTON**
Courier: 57 Sunshine Ave, Hamilton 3240
Postal: PO Box 195, Hamilton 3240
Tel: 07 850 0777 Fax: 07 850 0770
- **PALMERSTON NORTH**
Courier: 840 Tremaine Avenue, Palmerston North 4440
Postal: PO Box 536, Palmerston North 4440
Tel: 06 356 7100 Fax: 06 357 1904
- **CHRISTCHURCH**
Courier: 7 Halkett Street, Christchurch 8140
Postal: PO Box 3866, Christchurch 8140
Tel: 03 379 9484 Fax: 03 379 9485
- **DUNEDIN**
Courier: Invermay Research Centre, Block A, Puddle Alley, Mosgiel, Dunedin 9053
Postal: PO Box 371, Dunedin 9053
Tel: 03 489 4600 Fax: 03 489 8576

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
St Johns
Auckland
Tel: 09 909 3568

Investigation and Diagnostic Centre –
Christchurch
14 Sir William Pickering Drive
Christchurch
Tel: 03 943 3209

NEW ZEALAND VETERINARY PATHOLOGY

- **AUCKLAND**
Courier: NZCCM, Gate 2, Auckland Zoo, Motions Road, Western Springs, Auckland 1022
Postal: PO Box 44 422, Point Chevalier, Auckland 1246
- **HAMILTON**
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