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 (Pairis-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.

Figure 3: Soft rope snare for examining a pig

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

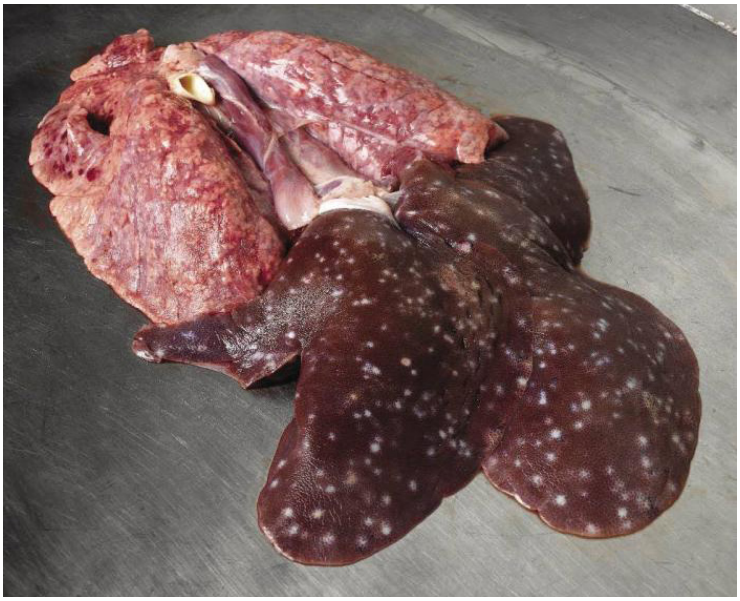

Figure 4: Haemorrhagic lesions in the cranial and apical lung lobes and "milk spots" on the liver from *Ascaris suum* larval migration (source: Jan Jourquin, Elanco)

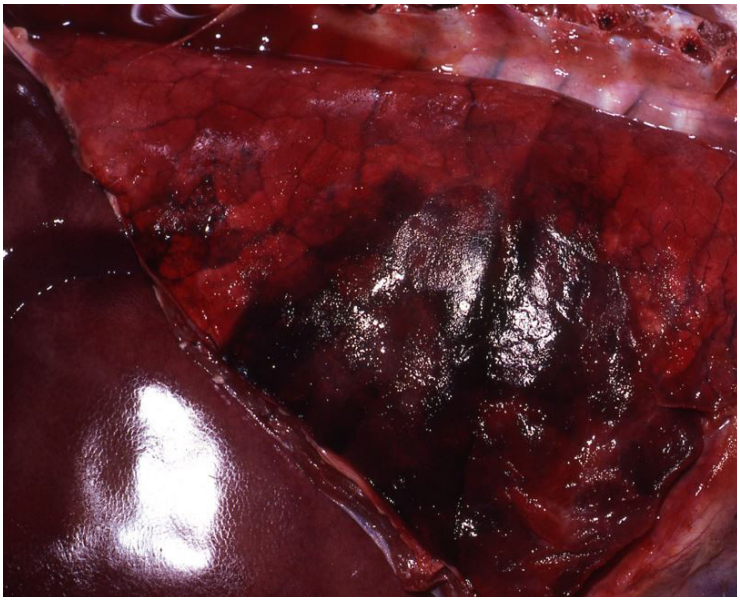

Figure 5: *Actinobacillus pleuropneumoniae* infection showing distinctive lung lesions (source: Massey University Pathobiology Department)

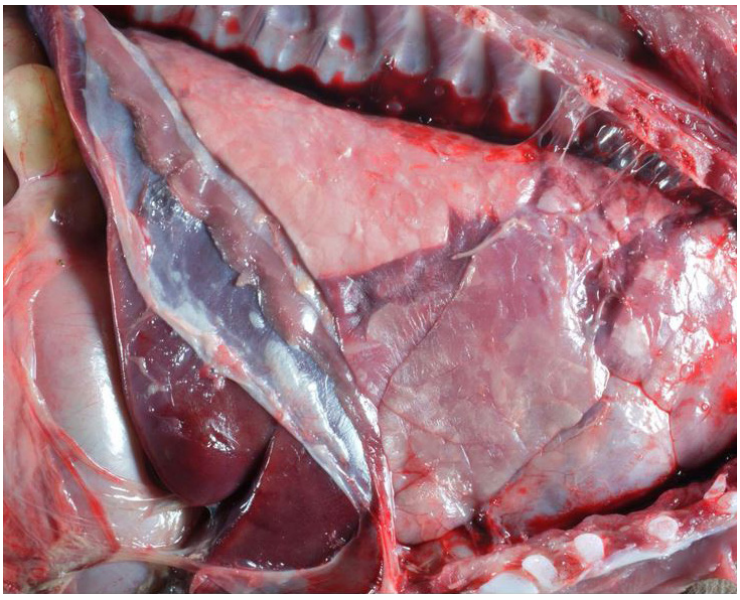

Figure 6: Enzootic pneumonia, likely including *M. hyopneumoniae* (source: Massey University Pathobiology Department)

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.

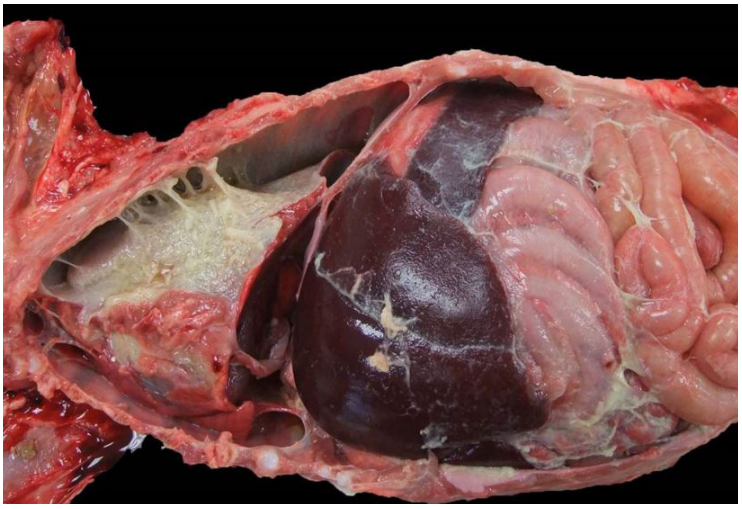

Figure 7: Glasser's disease, showing fibrin in the peritoneal cavity (*fibrinous peritonitis*) and pleural cavity (*fibrinous pleuritis*) (source: Massey University Pathobiology Department)

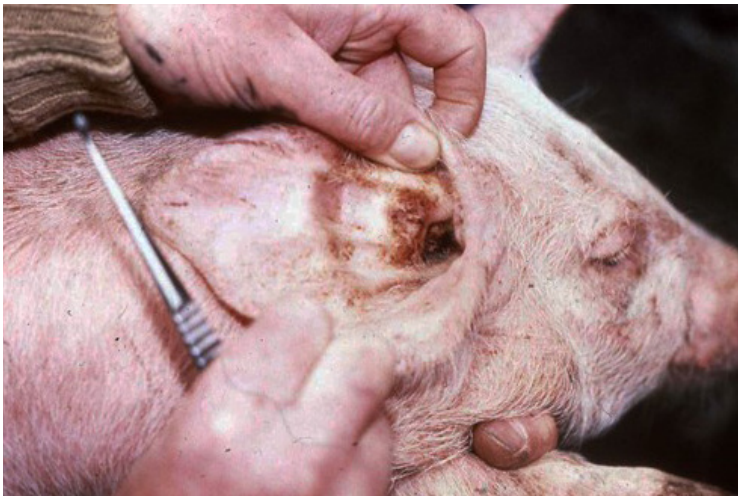

Figure 8: Early active *Sarcoptes scabiei* infection in the ear of a weaner pig (NADIS, 2018a) (source: NADIS & MEC White)

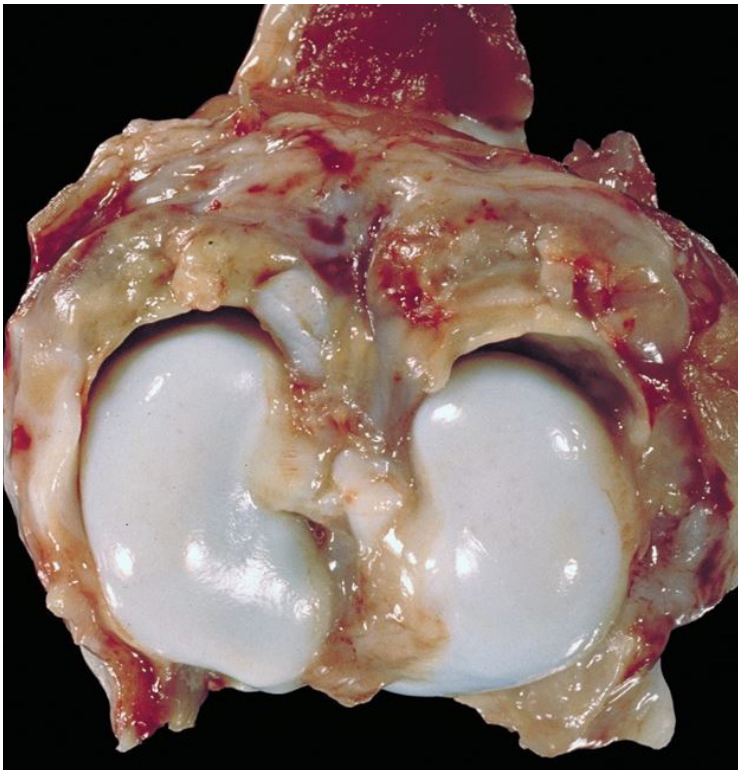

Figure 9: Septic joint associated with *Staphylococcus aureus* infection (source: Massey University Pathobiology Department)

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. Lame 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

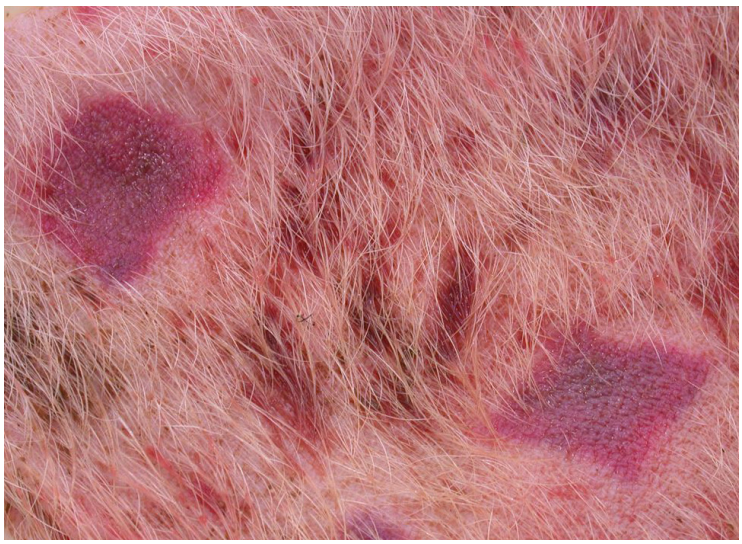

Figure 10: Skin lesions associated with *Erysipelothrix rhusiopathiae* infection (source: Massey University Pathobiology Department)

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

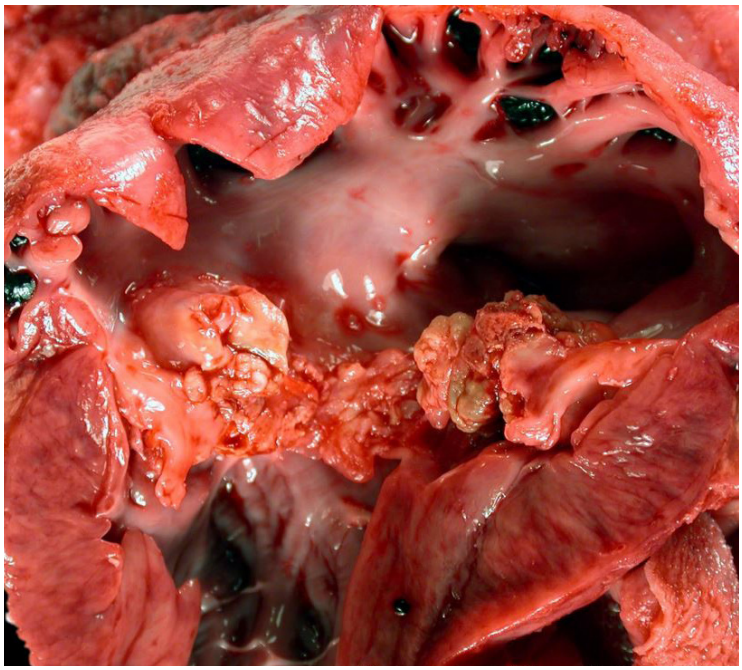

Figure 12: Valvular endocarditis secondary to *Erysipelothrix rhusiopathiae* infection (source: Massey University Pathobiology Department)

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**
Aspergillus spp.	Aflatoxins	< 100 ppb	300–2 000 ppb	Poor growth, liver damage, jaundice, immunosuppression
Aspergillus spp. & *Penicillium* spp.	Ochratoxin & citrinin	< 100 ppb	200–4 000 ppb	Reduced growth, thirst, kidney damage
Fusarium spp.	Deoxynivalenol, diacetoxyscirpenol, T-2 toxin	< 2 ppm	4–20 ppm	Reduced feed intake, immunosuppression, vomiting
Fusarium spp.	Zearalenone	< 0.05 ppm	1–30 ppm	Infertility, anoestrous, prolapse, pseudopregnancy
Fusarium 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

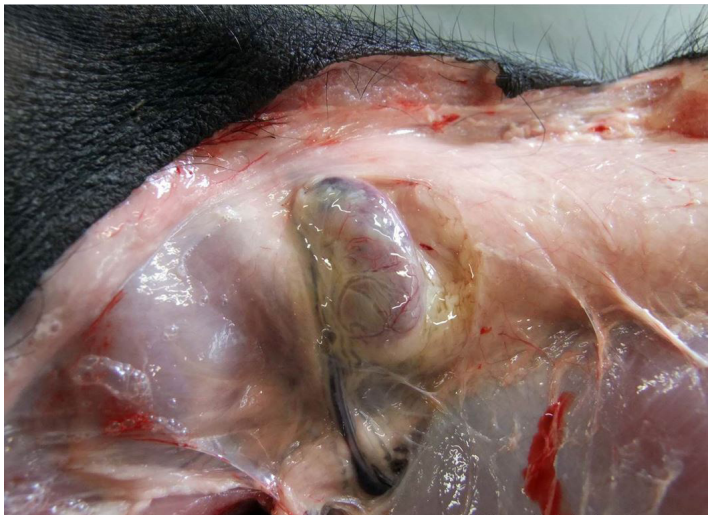

Figure 13: PMWS infection in a 3-month-old Saddleback pig. The superficial lymph node is oedematous and enlarged (source: Massey University Pathobiology Department)

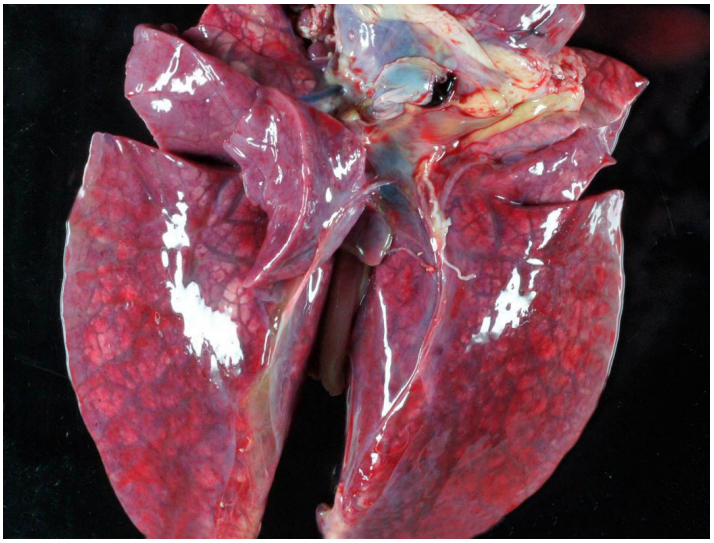

Figure 14: PMWS infection in a 3-month-old Saddleback pig. The lungs are diffusely firm and mottled dark red, with interlobular septae expanded by clear fluid (source: Massey University Pathobiology Department).

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 (> 60ºC) or frozen for long enough (-15ºC 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

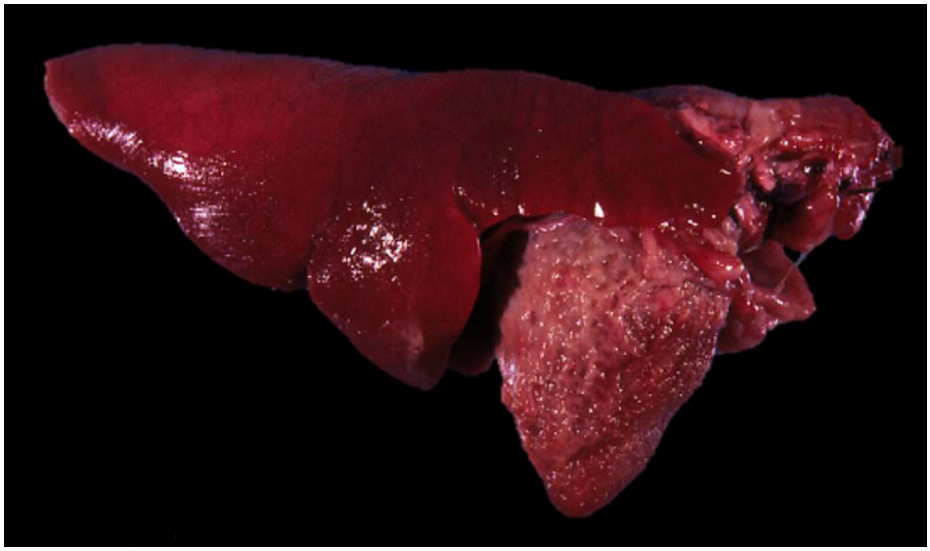

Figure 15: Epicarditis associated with Streptococcus suis infection (source: Massey University Pathobiology Department)

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 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.

Hyostrongylus 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 *Hyostrongylus 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).

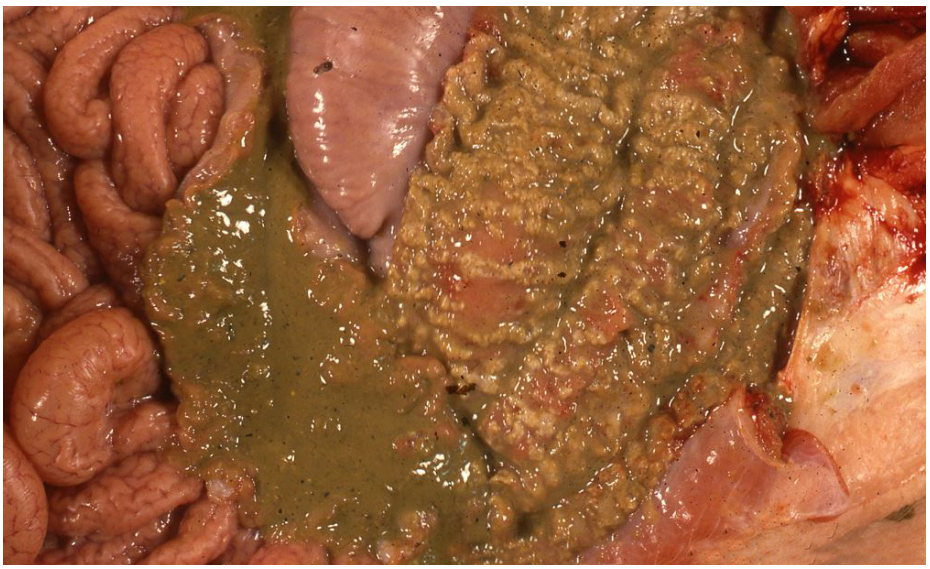

Figure 16: Swine dysentery (source: Massey University Pathobiology Department)

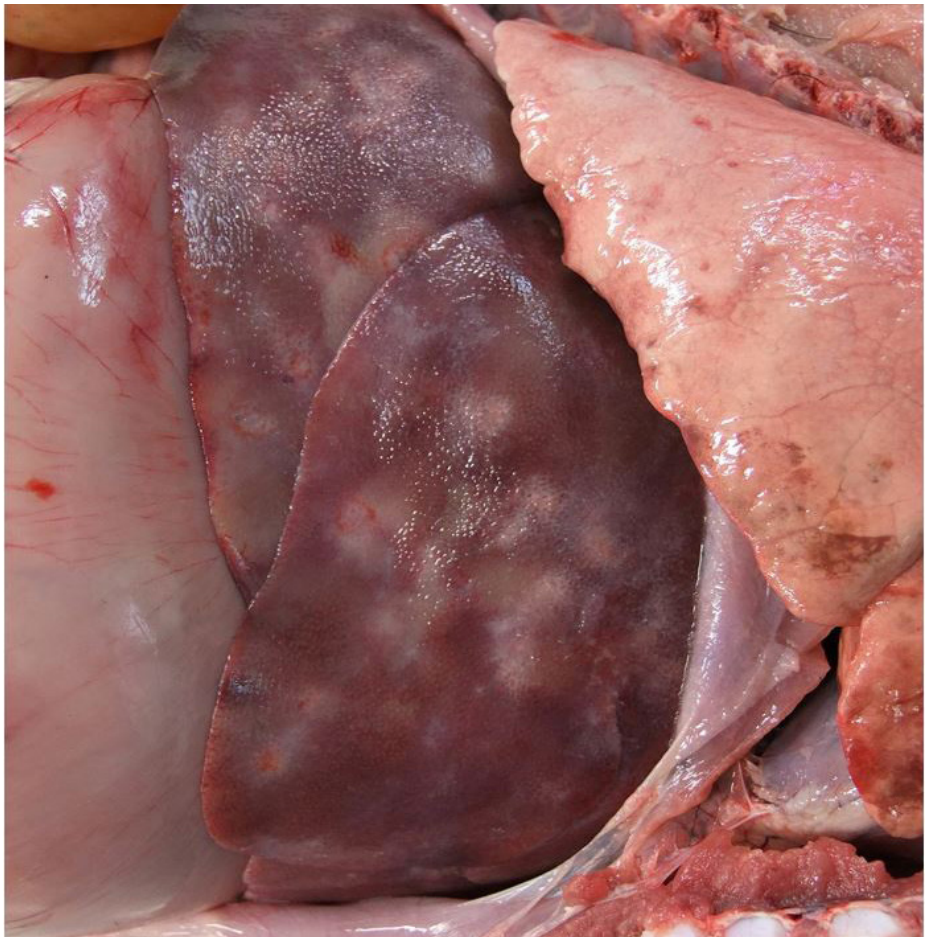

Figure 17: Severe milk spot lesions on liver, associated with migration of *Ascaris suum* larvae (source: Massey University Pathobiology Department)

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.

Kevin Lawrence
Senior Lecturer in Pastoral Livestock Health
School of Veterinary Science
Massey University
K.Lawrence@massey.ac.nz

Eric Neumann
Owner
Epi-Insight Limited
e.neumann@epi-insight.com

Naya Brangenberg
Veterinarian and Farm Manager
Longbush Free Range Pork
longbushpork@gmail.com

References

Beakenridge DT (1958). Mange in pigs: A survey. *New Zealand Veterinary Journal* 6, 166–167. doi: 10.1080/00480169.1958.33314.

Bolt I, Marshall R (1995). The epidemiology of *Leptospira interrogans* serovar Pomona in grower pig herds. *New Zealand Veterinary Journal* 43, 10–15.

Bryce AC (2002). Post-weaning multisystemic wasting syndrome – PMWS. *Vetscript* 15, 4–5.

Christensen NH, Cullinane LC (1990). Monitoring the health of pigs in New Zealand abattoirs. *New Zealand Veterinary Journal* 38, 136–141. doi:10.1080/00480169.1990.35639.

Davenport PG, Shortridge EH, Voyle B (1970). Polyserositis in pigs caused by infection with Mycoplasma. *New Zealand Veterinary Journal* 18, 165–167.

Dodd DC (1960). Hyostrongylosis and gastric ulceration in the pig. *New Zealand Veterinary Journal* 8, 100–103.

Elliott DC, Robinson AJ (1972). Trichuris infections in pigs: A treatment trial in the field. *New Zealand Veterinary Journal* 20, 98–101. doi: 10.1080/00480169.1972.34023.

Fairley RA (1997). Infectious diseases of pigs in New Zealand. *Surveillance* 24(2), 5–7.

Hampson D, Beban R (1985). Diet and control of porcine post-weaning diarrhoea. *New Zealand Veterinary Journal* 33, 190.

Heath ACG (2002). Ectoparasites of livestock and companion animals in New Zealand. *New Zealand Veterinary Journal* 50, 48. doi:10.1080/00480169.2002.36266.

Herriot J (1981). *The Lord God Made Them All.* Michael Joseph, London.

Hilbink F, de Lisle GW, Penrose M, Aldersley B, Carman M (1992). *Actinobacillus pleuropneumoniae* in New Zealand pigs.

New Zealand Veterinary Journal 40, 173–175.

Hughes JM, Wilson ME, Wertheim HF, Nghia HDT, Taylor W, Schultsz C (2009). *Streptococcus suis*: an emerging human pathogen. *Clinical Infectious Diseases* 48, 617–625.

Jackson P, Cockcroft P (2005). Clinical examination of the pig. *In practice* 27, 93–102.

Jaros P (2007). An investigation of the aetiology and natural transmission of postweaning multisystemic wasting syndrome in pigs: a thesis presented in partial fulfilment of the requirements for the degree of Master of Veterinary Studies in Epidemiology at Massey University, Palmerston North, New Zealand. Massey University.

Johnstone C (2018). Introduction to Parasitology. The Importance of Life Cycles. http://cal.vet.upenn.edu/projects/merial/introduction/intro_4.htm. Accessed 7 June 2018.

Ko JC, Williams BL, Smith VL, McGrath CJ, Jacobson JD (1993). Comparison of Telazol, Telazol-ketamine, Telazol-xylazine, and Telazol-ketamine-xylazine as chemical restraint and anesthetic induction combination in swine. *Laboratory Animal Science* 43, 476–480.

Liberona HE, MacDiarmid SC (1988). *Trichinella spiralis* in New Zealand. *Surveillance* 15(1), 17–18.

Marshall R, Manktelow B (2002). Fifty years of leptospirosis research in New Zealand: a perspective. *New Zealand Veterinary Journal* 50, 61–63.

Mason PC (1978). *Trichinella spiralis* in New Zealand. *New Zealand Veterinary Journal* 26, 215–216. doi:10.1080/00480169.1978.34545.

Ministry for Primary Industries (2018). *Trichinella spiralis*. https://www.mpi.govt.nz/dmsdocument/11069-trichinella-spiralis. Accessed 30 May 2018.

Montgomery J, Oliver R, Poole W (1987a). A vesiculo-bullous disease in pigs resembling foot and mouth disease I. Field cases. *New Zealand Veterinary Journal* 35, 21–26.

Montgomery J, Oliver R, Poole W, Julian A (1987b). A vesiculo-bullous disease in pigs resembling foot and mouth disease II. Experimental reproduction of the lesion. *New Zealand Veterinary Journal* 35, 27–30.

NADIS (2018a). Pig Health – Sarcoptic Mange. http://www.nadis.org.uk/bulletins/sarcoptic-mange.aspx. Accessed 30 May 2018

NADIS (2018b). Pig Health – Erysipelas. http://www.nadis.org.uk/bulletins/erysipelas.aspx. Accessed 30 May 2018.

Nejsum P, Betson M, Bendall R, Thamsborg SM, Stothard J (2012). Assessing the zoonotic potential of *Ascaris suum* and *Trichuris suis*: looking to the future from an analysis of the past. *Journal of Helminthology* 86, 148–155.

Neumann EJ (2013). Biosecurity and exotic disease surveillance in the New Zealand pig industry: a thesis presented in partial fulfilment of the requirements for the degree of Doctor of Philosophy at Massey University, New Zealand. Massey University.

Pairis-Garcia MD, Johnson AK, Millman ST, Stalder KJ, Karriker LA (2014). Effects of yohimbine, an alpha 2-antagonistic reversal agent, on physiological recovery parameters of anesthetized sows. *Journal of Swine Health and Production* 22, 16–23.

Paterson K, Black A, Reichel MP (1997). Porcine trichinosis diagnosed in the Rotorua area. *Surveillance* 24(4), 6–8.

Pearce H, Smith C (1975). Swine dysentery. *New Zealand Veterinary Journal* 23, 183–184.

Rawdon T, Lawton D, Johnstone A, Stone M, Spencer Y, Johnson L (2004). Investigation of post-weaning multisystemic wasting syndrome. *Surveillance* 31(1), 11–15.

Reichel MP (1997). Serovars of *Actinobacillus pleuropneumonae* isolated from New Zealand pigs. *Surveillance* 24(1), 28–29.

Richardson EKB (2006). Aspects of *Trichinella spiralis* in New Zealand: a thesis presented in partial fulfilment of the requirements for the degree of Master of Veterinary Studies in Epidemiology at Massey University. Massey University.

Robertson ID (1985). The isolation of *Streptococcus suis* types 1 and 2 from pigs in New Zealand. *New Zealand Veterinary Journal* 33, 148. doi:10.1080/00480169.1985.35201.

Roepstorff A, Murrell K (1997). Transmission dynamics of helminth parasites of pigs on continuous pasture: *Ascaris suum* and *Trichuris suis*. *International Journal for Parasitology* 27, 563–572.

Schillhorn TW, Gibson CD (1983). Anthelmintic activity of ivermectin in pigs naturally infected with *Ascaris* and *Trichuris*. *American Journal of Veterinary Research* 44, 1732–1733.

Stärk K, Pfeiffer D, Morris R (1998). Risk factors for respiratory diseases in New Zealand pig herds. *New Zealand Veterinary Journal* 46, 3–10.

Stone M (2004). Response to first diagnosis of post-weaning multisystemic wasting syndrome. *Surveillance* 31(4), 8–14.

The Pig Site (2017). Mycotoxins. http://www.thepigsite.com/pighealth/article/482/mycotoxins/. Accessed 24 Oct 2017.

The Pig Site (2018a). *Actinobacillus Pleuropneumonia* (App). http://www.thepigsite.com/diseaseinfo/4/actinobacillus-pleuropneumonia-app/. Accessed 17 January 2018.

The Pig Site (2018b). Enzootic Pneumonia (EP) or *Mycoplasma Hyopneumoniae* Infection. http://www.thepigsite.com/pighealth/article/323/enzootic-pneumonia-ep-or-mycoplasma-hyopneumoniae-infection/. Accessed 17 January 2018.

The Pig Site (2018c). Erysipelas. http://www.thepigsite.com/diseaseinfo/41/erysipelas/. Accessed 14 February 2018.

The Pig Site (2018d). Glässers Disease (*Haemophilus Parasuis* Hps). http://www.thepigsite.com/pighealth/article/329/glssers-disease-haemophilus-parasuis-hps/. Accessed 17 January 2018.

The Pig Site (2018e) Mycoplasma Arthritis (*Mycoplasma hyosynoviae* infection). http://www.thepigsite.com/pighealth/article/196/mycoplasma-arthritis-mycoplasma-hyosynoviae-infection/. Accessed 14 February 2018.

Thornton R, King C (2004). *Trichinella spiralis* disease investigation. *Surveillance* 31(1), 5–8.

Thurley D (1971). Locomotor diseases of pigs. *New Zealand Veterinary Journal* 19, 121–126.

Zanen H, Engel H (1975). Porcine streptococci causing meningitis and septicaemia in man. *The Lancet* 305, 1286–1288.

Diseases of backyard pigs in New Zealand

Lawrence K

2018-06-01