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Trichinella spiralis monitoring in pigs and horses

There has been extensive surveillance for trichinellosis in New Zealand pigs and horses slaughtered for food, with consistently negative results. Nevertheless, occasional disease incidents have occurred.

Pigs become infected with *Trichinella spiralis* by ingestion of larvae encysted in muscle: through the consumption of infected rodent carcasses, foodstuffs containing infected meat (such as swill) and cannibalism of infected pig carcasses. It is probable that horses, and also pigs, can become infected through ingestion of larvae passed in rat faeces contaminating rations[1].

New Zealand authorities have undertaken various forms of surveillance for *T spiralis* in domestic farmed pigs, feral pigs and horses slaughtered and processed in abattoirs, meat export premises and game packhouses[2][3][4][5][6]. Surveillance is currently managed by the New Zealand Food Safety Authority (NZFSA). Results to date will be used to target risk properties and animals to enhance future surveillance.

Monitoring programme

Between 1969 and 1983, every culled breeding pig slaughtered was examined by trichinoscope. This involved more than 300,000 culled breeding pigs. No infected animals were detected.

In 1983, the pepsin digestion test replaced trichinoscopy. Until 1995 the pepsin digestion test was performed on about 300 randomly selected 'choppers' (culled breeding pigs) each year, providing a 95% confidence of detecting disease if present at 1% annual period prevalence.

NZFSA currently conducts routine monitoring of a proportion of choppers (about 600 each year) at slaughter, as well as:

- all exported feral pigs (none currently),
- all feral pigs heavier than 68 kg (head on) for the domestic market,
- horses slaughtered and processed at abattoirs and export premises,
- all farmed pig meat exported to Singapore.

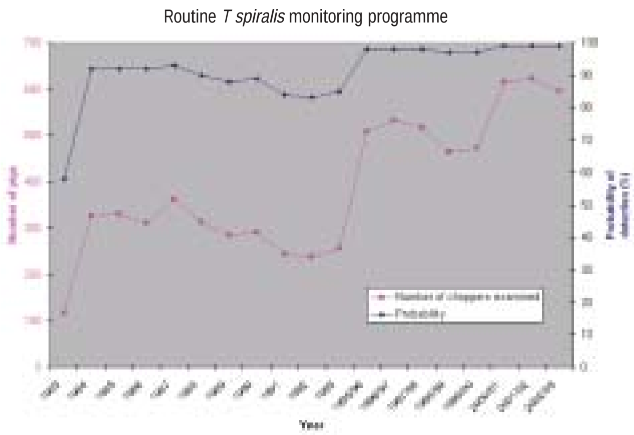
There are currently no monitoring requirements for home-killed pigs.

Testing, conducted on a specific muscle sample (diaphragmatic pillars in domestic pigs; jaw or lingual muscle in feral pigs and horses), must be performed in a MAF-approved facility. Horse meat is tested on-site and pig meat is tested off-site.

The analytical test is the magnetic stirrer method for pooled-sample artificial (pepsin) digestion (MAF Circular 93/RAM/4, 93/12/1). Each digest consists of a pool of 20 5 g samples. Samples are frozen under security until ready for batch testing.

The routine chopper monitoring programme currently run by NZFSA commenced in 1995. This class of pig was targeted because aged animals pose the greatest risk to public health, and the likelihood of detection is higher in a chronically infected animal.

Figure 1: Summary of the NZFSA domestic pig monitoring programme



A summary of all chopper monitoring from 1983 to 2003 is presented in Figure 1.

It is reasonable to assume that the approximately 600 chopper pigs currently tested annually constitutes a random sample of older breeding pigs. This monitoring therefore provides a 95% probability of detecting infected animals at an annual period prevalence of 0.5%.

Samples for analysis are submitted from all pig slaughtering premises. The rate of sampling for any one premises has been based on the chopper kill figures submitted from that premises during the previous 12 months. There have been no positive *T spiralis* findings in the monitoring programme.

Testing of all pigs for *T spiralis* is a market access requirement for Singapore. There have been more than 8,000 samples submitted from premises slaughtering pigs for this market. There have been no positive *T spiralis* findings in this targeted sample.

From 1990, all feral pigs processed through game packhouses were tested for *T spiralis.* This requirement was subsequently modified (MAF Circular 91/MP/13, 91/14/2), and now all feral pigs slaughtered for export to EU markets are tested, and only pigs heavier than 68 kg (head on) are tested for other markets and domestic consumption.

Since 1990, more than 17,500 feral pigs have been processed. Many have come from the wilderness areas of the South Island. The last testing of feral pigs for export was in April 2002, as none have been exported since then. Feral pigs heavier than 68 kg continue to be tested for the local market, but as few reach this size most are not tested. There have been no positive *T spiralis* findings in feral pig samples tested.

Figure 2: MAF 1998 ‘chopper’ survey: location of selected farms

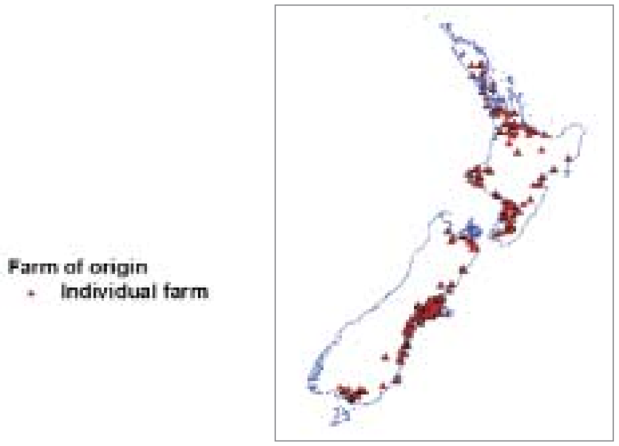
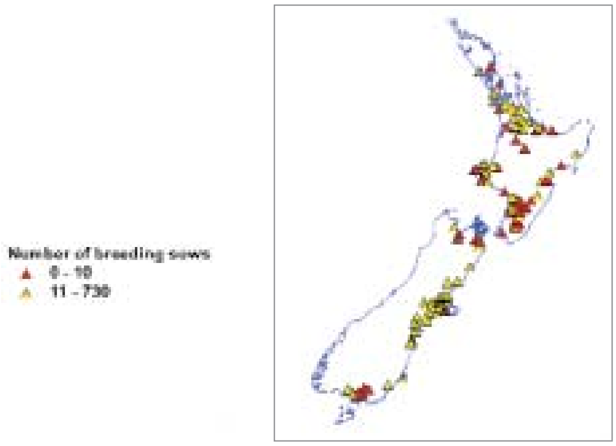


Figure 4: MAF 1998 ‘chopper’ survey: farm size



Since 1992, all horses slaughtered for export have been tested. Figures collected by MAF show more than 53,000 horses have been killed for export between 1992 and September 2003.There have been no positive *T spiralis* findings.

Trichinella spiralis survey

Trichinella spiralis is considered to be present at a very low prevalence in wildlife, and can, on rare occasions, enter the domestic pig population. It has been identified in New Zealand rats, feral cats and, very rarely, in domestic pigs.

For example, in May 1997, *T spiralis* larvae were identified in diaphragmatic muscle submitted for private testing from a sow home-killed for human consumption[6]. The sow had been purchased from a farm in the Rotorua district. All remaining pigs on this property were slaughtered and tested by MAF. Larvae were identified in four of nine sows. Examination of about 250 rats trapped in the immediate area and in the vicinity of the farm did not detect any *T spiralis* larvae. Another case, involving human infection, is reported in this issue of *Surveillance*[7].

As a result of the 1997 finding, MAF and the pork industry initiated a porcine trichinellosis survey in September 1998. Pigs were selected from a range of farming operations throughout New Zealand (Figure 2), targeting choppers most at risk (from infrequent suppliers or fed raw food waste). Suppliers of these pigs were asked to complete a survey that included questions about housing and feed practices, as well as the type of operation (small or large) and the environment. Six hundred and thirty five samples from about 250 farms were collected at slaughter and tested using the pepsin digestion method. All the samples were negative.

Figure 3: MAF 1998 ‘chopper’ survey: age of sampled pigs

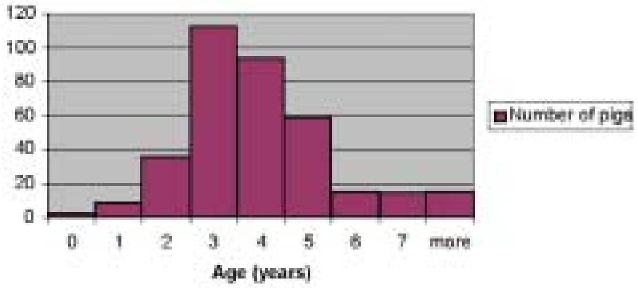
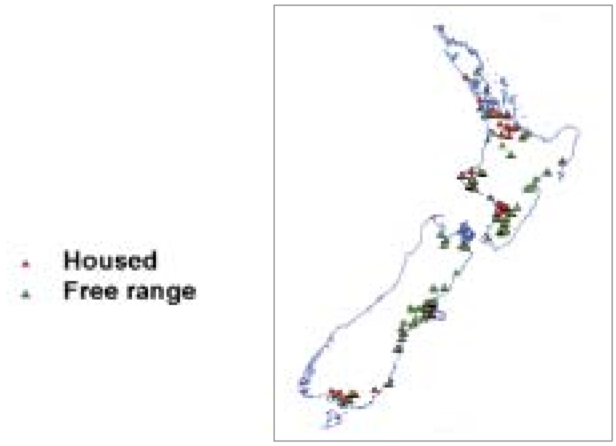


Figure 5: MAF 1998 ‘chopper’ survey: housed and free range pigs



Most of the pigs slaughtered were about three years of age, but ages ranged from less than one year to more than ten years (Figure 3).

The pigs were from a range of different farming operations. More than half were from operations with more than 50 breeding sows, and about one third from farms with fewer than ten (Figure 4).

Many of those with few breeding sows were lifestyle blocks, ‘back-yard’ breeders or were primarily farming other species such as sheep or cows. The occurrence of potential *T spiralis* risk factors (presence of rodents and rodent control, feeding of food waste and treatment before feeding, free range and proximity to public refuse sites) was assessed.

Most of the respondents had free range pigs (Figure 5). These ranged from single breeding sow units through to commercial units with more than 100 breeding sows. The type of feed supplied to pigs included meal, grass, food waste such as restaurant and household scraps, bread and cheese. Food waste was fed to about 30% of pigs and most was not cooked.

Rodent control is an important risk management tool for

T spiralis. Most farms carried out some form of rodent control. The use of poisons such as anticoagulants was the most common method, but some farms also used cats, traps or shooting. Suppliers were asked about proximity to a public refuse site, this being another source of rodents, feral cats, and food waste. The average distance from a refuse site was 13 km but the range was 0–68 km.

Comment

There has been extensive targeted surveillance for trichinellosis in New Zealand pigs and horses slaughtered for food, with consistently negative findings.

Routine monitoring has included young pigs from commercial herds, choppers from commercial and non-commercial herds, horses from all areas of the country, and feral pigs primarily from the wilderness areas on the west and north of the South Island.

A 1998/1999 survey targeted back-yard piggeries and infrequent suppliers throughout the country. It assessed the presence of potential risk factors such as rodent populations, rodent control, feeding of food waste, feed treatment, free range conditions and proximity to public refuse sites.

Some of the risk factors for the transmission of *T spiralis* exist in New Zealand, either on farm, such as access to rodents and cats and the feeding of food waste, or from close proximity to public refuse sites and feral animals. Occasional disease incidents do occur[6][7].

References

(1) Palmer S, Lord Soulsby, Simpson D. Zoonoses: Biology, Clinical Practice and Public Health Control. Pp 796-7. Oxford University Press, 1998.

(2) Elliott D. A search for infections of *Trichinella spiralis* in New Zealand pigs. New Zealand Veterinary Journal 8, 28, 1960.

(3) Cairns G. The occurrence of *Trichinella spiralis* in New Zealand pigs, rats and cats. New Zealand Veterinary Journal 14, 84-90, 1966.

(4) Liberona H, MacDiarmid S. *Trichinella spiralis* in New Zealand. Surveillance 15(1),17-8, 1988.

(5) MacDiarmid S. *Trichinella* Status of New Zealand: Ministry of Agriculture and Forestry Internal Report No156-08, 1995.

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

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

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Trichinella spiralis disease investigation

Two people contracted trichinellosis from home-killed pork from a back-yard piggery. Infected animals and meat were traced and destroyed. The source of infection appeared to have been feral cats and rats. The trypsin digest test was the gold standard but an ELISA appeared useful for survey work or valuable animals.

The manager of a farm (Farm A) in the Bay of Plenty on the east coast of the North Island contracted trichinellosis after eating infected pork from his own back-yard piggery[1]. A person who had eaten bacon gifted from the infected farm also contracted trichinellosis. Family members of the two infected people remained healthy. They had eaten infected pork but had not handled it raw as the two affected people had.

Trichinellosis is an OIE list B disease. It is endemic in New Zealand pigs but with low prevalence[1]. Feral animals appear to provide a reservoir of infection from which occasional spillovers occur into domestic pigs particularly in back-yard piggeries. The New Zealand Food Safety Authority (NZFSA) manages surveillance of pork within the regulated food chain[2].

Trichinella spiralis is a notifiable organism in New Zealand. This gives the Ministry of Agriculture and Forestry (MAF) powers of investigation and control under New Zealand's Biosecurity Act 1993 should infection be detected in pigs. MAF responded to this particular incident by confirming the diagnosis as *T spiralis*, identifying the source of infection, and removing traced infected pigs and pork from the food chain. Response activities are summarised below.

Response activities

Test methodology

Trichinella infection was diagnosed in live animals using an ELISA on serum samples and in dead animals using a pepsin digest test on diaphragmatic muscle.

Infection on Farm A

Farm A, the farm at the centre of this investigation, contained five captured feral pigs, 26 adult domestic pigs, 100 cattle, two horses and 100 sheep. It was adjacent to a refuse recycling station and native bush, and was visited by rats, feral cats and feral pigs. The farm manager shot feral cats and rats on the farm and his pigs scavenged their carcasses. His pigs also ate scraps collected from local restaurants. His dogs were fed scraps collected from a local butcher.

The manager of Farm A elected to have his remaining pigs killed,

[1] A back-yard as opposed to a commercial piggery is defined here as one that is not registered with the New Zealand Pork Industry Board

tested and buried on site. Sixteen of 31 adult pigs were positive in the pepsin digest test (Table 1). The *Trichinella* in the infected pork was identified as *T spiralis*[2].

Also positive to the pepsin digest test were four of five feral cats tested and seven of 22 feral rats tested.

Two horses on the farm were ELISA-negative (Table 2) but these were nevertheless barred from food chains.

Table 1: *Trichinella* pepsin digest results

Farm	Species	Negative	Positive	No tested	At risk
Farm A	Pig	15	16	31[a]	54
	Feral cat	1	4	5	unknown
	Feral rat	15	7	22	unknown
	Possum	9	0	9	unknown
	Hedgehog	1	0	1	unknown
	Home kill meat[b]	5	0	5	6
Farm D	Pig	1	0	1	7
	Meat	1	0	1	1
Farm E	Meat	1	0	1	1

[a] Piglets were not tested

[b] From different pigs from the one that initiated the investigation

Table 2: *Trichinella* ELISA results

Farm	Species	Negative	Positive	No tested	At risk
Farm A	Pig	17	14	31[a]	54
	Horse	2		2	2
Farm C	Pig	3		3	3
Farm D	Pig	6		6	12
Farm F	Pig	5		5	5
Farm G	Pig	4	0	4	4
Farm H	Pig	3	1	4	4
TOTAL				**55**	

[a] Piglets were not tested

Tracing and disposal on Farm A

Potentially infected animals or meat that had entered or left the infected farm during the previous six months were traced. Risk was assessed using knowledge of the life cycle and the infectivity of *T spiralis* (Figure 1). Tracing results are discussed with reference to a symbolic network of infection (Figure 2). Farm A had received pigs from three farms during the six-month period of interest.

Farm C: Farm C was under suspicion because it had supplied pigs that had tested positive when on Farm A and had received pigs from other implicated farms. Additionally, pig meat had been received from Farm A and two people on Farm C had become ill with symptoms resembling trichinellosis (subsequently diagnosed as gastroenteritis). All three pigs on Farm C were ELISA-negative, so it was dropped from the investigation.

Farm D: Farm D had supplied pigs indirectly to Farm A, and these had tested positive. Six of 12 adult pigs, and meat from the freezer, were tested. All were ELISA-negative, so Farm D was dropped from the investigation.

Farm E: Farm E had supplied test-positive pigs indirectly to Farm A. However, there were no pigs left on Farm E so it was dropped from the investigation.

Pigs that had left Farm A were traced, and neighbouring properties were checked.

Contiguous properties: Farm A had four surrounding properties. None farmed pigs so they were dropped from the investigation.

Farm F: Five weaners had been purchased from Farm A about two months prior to butchering the sow that had produced the infected meat. All were ELISA-negative so Farm F was dropped from the investigation.

Farm G: Nine weaners had been purchased from Farm A about two months prior to butchering the sow that had provided the infected meat. Five had been on-sold and eaten but four were available for testing. All four were ELISA-negative so Farm G was dropped from the investigation.

Farm H: Four weaners had been purchased from Farm A about three weeks prior to the start of the investigation. Three were ELISA-negative but one was marginally positive. All four weaners were re-bled about one month later. During this time, the marginally positive weaner became definitely positive but the other three remained negative. The positive pig was destroyed and the other three were re-bled after another month. All three remained ELISA-negative, so Farm H was removed from the investigation.

Pig meat from Farm A had been gifted to other people and was traced. The second confirmed case of trichinellosis was in a person living on Farm B. This person had prepared and eaten meat gifted from Farm A. Other family members who had eaten the same meat had not become ill. There were 100 cows but no pigs on the property, and pork had been received only from Farm A. Farm B was therefore cleared from suspicion. The gifted meat was retrieved and tested positive to the pepsin digest test. The meat was buried.

Of five other people who had received meat from Farm A, none had contracted trichinellosis. Three lots of meat still available were returned willingly. All tested negative and were destroyed. A local butcher had processed the infected sow as home-killed meat for Farm A and had also supplied meat scraps to the infected farm for dog food. The butcher was operating legally; home-killed meat was not entering the retail food chain and cross contamination was excluded.

Comparison between the ELISA and pepsin digest test

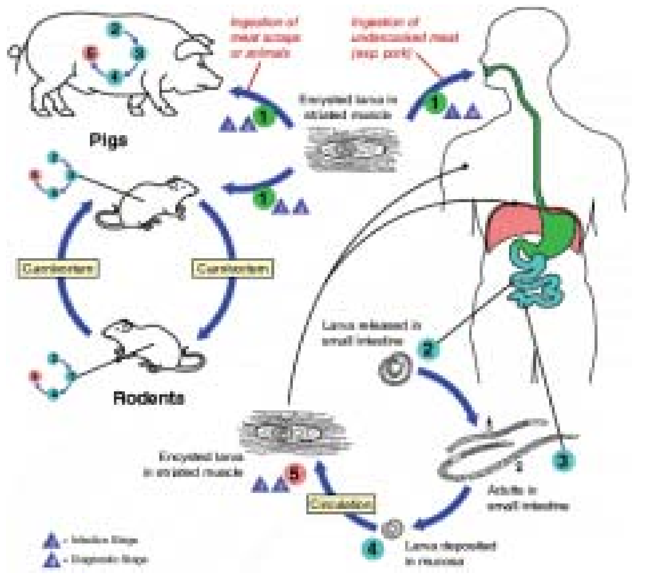
The ELISA was compared with the gold standard pepsin digest test using 31 adult pigs from Farm A (Table 3). Three of 16 ELISAs were false negative (sensitivity = 0.813) and one of 15 ELISA results was false positive (specificity = 0.933).

Discussion

Pig meat within the New Zealand regulated food chain is rarely infected with *Trichinella*[(1)(2)]. Most pork comes from commercial piggeries and these have a low risk of infection from feral animals.

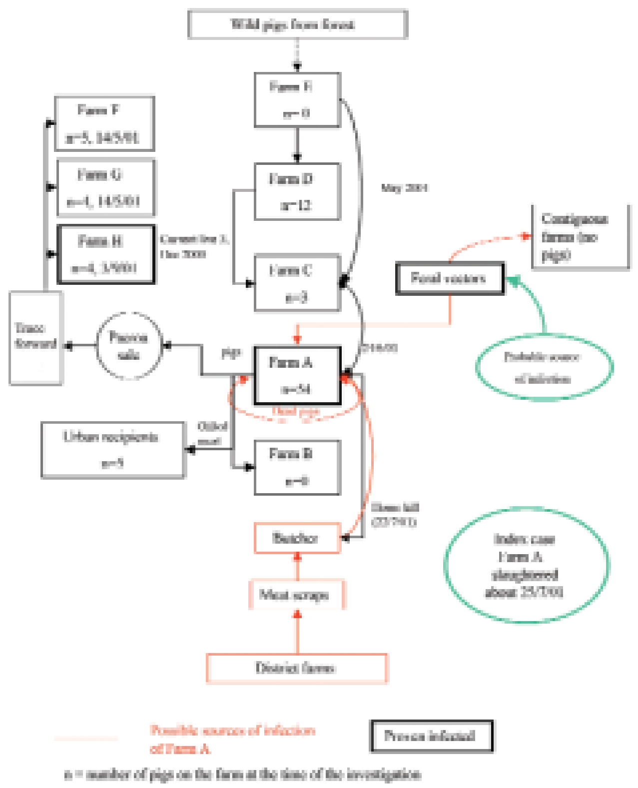
[2] Laboratory of Parasitology, Instituto Superiore di Sanita viale Regina Elena 299, 00161, Rome, Italy.

Figure 1: Life cycle of *Trichinella spiralis*



Trichinellosis is acquired by ingesting meat containing encysted larvae (1). Larvae released by exposure to digestive juices invade the small intestinal mucosa and develop into adult worms (2), (3). After one week, the females release larvae (4) that migrate and are distributed in blood to striated muscle where they encyst (5). Encystment is completed in four to five weeks and the cysts may remain viable for several years. The cycle is completed when encysted larvae are eaten. The life cycle is perpetuated mainly by rodents, but a wide variety of meat-eating mammals can be involved. The pig-to-human cycle is the obvious public health concern.

Figure 2: Tracing the source and destination of *Trichinella* infected meat



A '*Trichinella* risk mitigation programme' endorsed by the Pork Industry Board is being introduced for commercial piggeries (Burke L, personal communication).

Back-yard farm pigs usually graze outdoors and can therefore become infected by eating feral animals. Pig-to-pig cycles may also occur. Back-yard piggeries therefore present a higher *Trichinella* risk to humans than commercial piggeries. Much back-yard piggery meat enters the food chain through home-killing and is not inspected, which increases the risk further.

The spillover rate from the feral animal reservoir into back-yard herds is unknown but the rate of infection when it does occur may be high. About half of the adult pigs on Farm A were infected. A high infection rate was described in a previous similar investigation in New Zealand[3].

Human trichinellosis is nevertheless rare in New Zealand. This may partly reflect the ease with which *Trichinella* larvae are killed by freezing and cooking. The two people who contracted trichinellosis in this investigation had handled raw infected meat. Others who had consumed the same meat cooked had remained healthy.

Table 3: Comparison between the ELISA and pepsin digest tests in pigs

	Pepsin digest		
ELISA	Positive	Negative	
Positive	13	1	14
Negative	3	14	17
	16	15	31

None of the farms that had supplied pigs to Farm A during the previous six months was infected. Farm A was therefore the index farm. The high rate of infection of feral cats and rats caught on Farm A suggested that spillover from a feral reservoir had occurred.

One of four weaners received from Farm A four weeks previously by Farm H was seropositive. The time taken for seroconversion is dose-dependent but the outside limit is eight weeks[4]. The remaining three at-risk weaners on Farm H were therefore tested over a two-month period before being cleared.

The pepsin digest test is the gold standard for *Trichinella* infection. It can detect about three larvae per gram of muscle[5]. The ELISA is much less sensitive and can detect only about one larva per 100g of muscle when performed optimally. It may return false negative results early (less than 35 days) and late (more than six months) during infection because of the dynamics of the antibody response, and it may return false positive results if performed suboptimally. However, it may be more practicable as a screening test or for use on valuable animals.

During this investigation, the ELISA was compared with the pepsin digest test using 31 adult pigs from Farm A. Three of 16 ELISAs were false negative. However, these three might have seroconverted had they lived longer (as happened on Farm H), particularly as two were weaners. One of 15 ELISA results was false positive. A similarly favourable comparison has been made previously in New Zealand[3]. The ELISA would appear to be a valuable screening test.

References

(1) Liberona H, MacDiarmid S. *Trichinella spiralis* in New Zealand. Surveillance 15(1), 7-18, 1988.

(2) Clear M, Morris S. *Trichinella spiralis* monitoring in pigs and horses in New Zealand. Surveillance 31(1), 3-5, 2004.

(3) Paterson K, Black A, Reichel M. Porcine trichinellosis diagnosed in the Rotorua area. Surveillance 24(4), 6-8, 1997.

(4) Nockler K, Pozio E, Voigt WP, Heidrich J. Detection of *Trichinella* infection in food animals. Veterinary Parasitology 93, 335-50, 2000.

(5) OIE. Trichinellosis. In: Manual of Standards diagnostic tests and vaccines, Chapter 2.2.9. Office International des Epizooties, Paris, 2000.

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OIE risk analysis framework: a flexible model for pest risk analysis

In response to an incursion of the insect pest red imported fire ant (RIFA) (*Solenopsis invicta*), MAF assessed the pathways by which RIFA could enter New Zealand. The assessment was modelled on the internationally agreed framework for animal disease risk analysis, illustrating that it can be adapted to environmental pest risk analysis.

Import risk analysis in New Zealand has traditionally focused on managing those pest and disease risks of concern to the productive sectors and human health that are associated with international trade in plant and animal products. However, in recent years, as a result of greater public awareness of the importance of biosecurity, particularly with respect to biodiversity, greater emphasis has been placed on pests affecting indigenous fauna and flora. Consequently, there is an increased need for techniques to assess and manage the risks posed by such organisms. MAF Biosecurity Authority uses the standards of the Office International des Epizooties (OIE)[1] for risk analyses on animals and animal products, and those of the International Plant Protection Convention[2] for plants and plant products. This article describes the use of the OIE framework for the development of a pest risk analysis for red imported fire ant (RIFA) (*Solenopsis invicta*)[3].

Import risk analyses for plants and animals consider pests and diseases that are directly associated with plants and animals. However, there are pest species of potential environmental, or plant, animal or human health concern that, because of their life cycle, are unlikely to be introduced via, or are not exclusively associated with, animal or plant products. These organisms, often termed 'hitchhiker pests', may be transported on a variety of inanimate objects, and are therefore frequently relatively non-specific in their routes of introduction. They are also referred to as 'environmental pests' because, although they may have some consequence for, they are not principally threats to the production sector. Examples include snakes, frogs and insects.

RIFA, a native ant of South America[4], is an example of a hitchhiker pest. Although it has been associated with soil and some plant materials, it also has the potential to be transported on a variety of inanimate objects[4][5]. Its significance as a pest lies in its potential to affect agriculture, horticulture, human health and the environment.

In February 2001, a single RIFA nest was discovered at Auckland International Airport (Pascoe A, personal communication). Although the nest was destroyed, it could not be determined how the introduction had occurred. Since further incursions might be possible, a surveillance programme was initiated. MAF decided that a risk analysis should be undertaken to determine the pathways (for example, aircraft, sea vessels, containers, packaging, personal baggage and specific imported commodities such as building materials, nursery stock and used machinery) by which RIFA might be imported, and to rank them so that surveillance resources could focus on the routes of highest risk. MAF considered that the OIE risk analysis framework was appropriate for this task.

The OIE risk analysis framework consists of the following steps:

- Hazard identification
- Risk assessment
- Release assessment
- Exposure assessment
- Consequence assessment
- Risk estimation
- Risk management
- Risk communication.

Although only the hazard identification and release assessment were carried out for the RIFA assessment, the applicability of the OIE framework to pest risk analysis was evaluated. The use of the framework for the RIFA hazard identification and release assessment, and the potential to adapt the remaining sections, is summarised below.

Hazard identification

Hazard identification is the process of identifying the pathogenic agents (hazards) that could potentially be introduced in the commodity considered for importation. A hazard is defined as 'any pathogenic agent that could produce adverse consequences on the importation of a commodity'[1].

Using the OIE model, an import risk analysis for animals or animal products generally starts with a well defined commodity intended for import. For example, an analysis may consider cooked chicken meat from a particular country. All pest and disease organisms that could produce an adverse consequence and could be carried by the commodity are identified as the hazards. Alternatively, a single pest- or disease-commodity relationship may be examined, for example rabies virus in dogs.

Adapting this to a pest risk analysis, the starting point could be a defined commodity (of plant or animal origin, or inanimate) and the analysis could focus on one pest or it could identify all pests potentially associated with that commodity. In that case, the same process as an animal import risk analysis would be followed, so this type of pest risk analysis has not been discussed further here.

Alternatively, as in the RIFA assessment, a pest risk analysis can begin with a pest of interest, with no associated commodity. In this situation, there is only one hazard – the pest organism – and the pathways by which it could be introduced become the focus of the analysis.

In the RIFA assessment, the hazard identification consisted of an examination of the biology of the ant (the hazard) to identify the stages of the life cycle that could initiate its introduction. Although the different fomites and plant and animal materials capable of harbouring relevant life stages of the pest were also discussed, it would be more appropriate in future pest risk analyses for this discussion to be presented in the release assessment section of the analysis.

Release assessment

Under the OIE framework, the release assessment consists of a description of the biological pathways necessary for an importation to release hazards into an importing country and an estimate of the likelihood of the pathways occurring. For an animal and animal product risk analysis, each hazard associated with the commodity is discussed individually.

As indicated above, in pest risk analysis the release assessment should begin with a discussion of the different materials (fomites, plant and animal products) that are considered capable of harbouring the pest. Their importation channels become the potential pathways by which the pest could be introduced. As in the RIFA assessment, once the pathways have been identified, each pathway (instead of the hazard) would be discussed. This would describe the events necessary for the pathway to become infested with the pest, the ability of the pest to survive the conditions and duration of transport, and the likelihood of infestation at the time of import of the material. Where appropriate, pathways with similar features could be combined into a single discussion.

Where a large number of pathways are considered able to introduce the pest, those of greatest likelihood could be identified to allow efficient allocation of limited resources, as was attempted in the RIFA release assessment.

Exposure assessment

Exposure assessment under the OIE framework describes the biological pathway(s) necessary for exposure of animals and humans to the hazards released from the importation, and estimates the probability of that exposure occurring. Each identified hazard is considered separately for animal and animal product risk analyses. For a pest risk analysis, the exposure assessment would consist of an examination of the conditions necessary for the pest to spread or become established in the importing country, and an estimation of the likelihood of the required events occurring.

A reasoned discussion would be necessary, outlining factors such as the critical survival parameters for the pest, the availability of suitable food sources and the presence of intermediate hosts. The relationship of these factors to conditions in the importing country needs to be considered carefully to determine if establishment would be possible. Modelling, for example using tools such as the CSIRO CLIMEX model[6], might be used to determine the potential range of establishment of a pest organism.

Consequence assessment

Consequence assessment under the OIE framework describes for each hazard the potential consequences of exposure and their likelihood. Thus, a pest risk analysis should attempt to identify the potential adverse human or animal health, environmental or socio-economic consequences associated with the entry, establishment or spread of the pest, and to estimate their likelihood.

Compared with animal and animal product risk analyses, consequence assessment can be expected to be particularly difficult in pest risk analyses because of the higher likelihood of significant environmental effects. The impacts of a pest on unique fauna and flora are usually difficult to predict and, for this reason, a worst case scenario is often assumed. In addition, society has a range of views on the value of the environment, and there is no agreed measure by which to evaluate different environmental impacts.

Risk estimation

Risk estimation consists of integrating the results of the release, exposure and consequence assessments with a view to determining whether risk management measures are warranted for each of the identified hazards.

In a pest risk analysis, the risk estimate would clearly focus on the pest. A summary would state whether the pest constitutes a hazard that warrants risk management measures. Where the analysis has discussed a number of potential pathways for introduction, those pathways considered capable of introducing the pest should be identified.

Risk management

The OIE framework defines risk management as the process of selecting and implementing measures that achieve an importing country's appropriate level of protection, at the same time minimising negative effects on trade.

This concept applies readily to pest risk analysis. To minimise the likelihood of importing the pest, risk management measures would be chosen and adopted for each of the pathways the risk estimation identifies as requiring management. Measures could be developed for each pathway individually or a range of measures known to be effective could be stated as options for the identified pathways.

In the RIFA release assessment, the management measures already being used during the importation of a commodity were considered when assessing the likelihood that a pathway would introduce RIFA. Thus, the assessment was implicitly aimed at determining the adequacy of existing measures rather than determining what measures were necessary to achieve a stated level of protection. For the latter approach, it would be necessary to estimate the likelihood of introducing the pest in the absence of any measures, and then to recommend appropriate risk management measures.

A comparison of the current measures with the recommended measures would then follow. Considering 'current mitigation measures' in the release assessment may result in pathways being classified as not able to introduce the pest, but if the measure is subsequently changed or removed the pathway may become a viable route of introduction that has not been identified.

Conclusion

The use of the OIE risk analysis framework in pest risk analysis should be guided by the same principles as those for animal import risk analysis, including transparency, consistency and use of the best science available. The RIFA assessment was affected by the same limitations as an animal risk analysis, notably uncertainty as a result of incomplete scientific information. It was particularly difficult to estimate the likelihood that a particular pathway would become infested before export, as infestations are essentially random events that could be influenced by numerous factors including the biology of the ant and the density of infestations in the exporting country. However, the framework was an effective tool to allow the presentation of current scientific knowledge, and it allowed consistent and transparent decision-making.

Although only the hazard identification and release assessment steps were carried out for RIFA, the process demonstrated the applicability of the OIE animal/animal product import risk analysis framework to pest risk analysis. The general format followed for RIFA could be further developed and refined to design a generic pest risk analysis model within the framework presented in the OIE Terrestrial Animal Health Code.

References

(1) OIE. Terrestrial Animal Health Code. 23-30. Office International des Epizooties, Paris, 2002.

(2) International Plant Protection Convention. International standards for phytosanitary measures no.11: Pest risk analysis for quarantine pests. Food and Agriculture Organization of the United Nations, Rome, 2001.

(3) MAF Biosecurity Authority. Hazard identification and import release assessment: The introduction of red imported fire ant into New Zealand via the importation of goods and arrival of craft from Australia, the Caribbean, South America, and the USA. Ministry of Agriculture and Forestry, Wellington, 2002.

(4) Bradleigh Vinson S, Sorensen AA. Imported fire ants: Life history and impact. Department of Entomology, Texas A&M University, College Station, Texas, and Agricultural & Environmental Sciences, Texas Department of Agriculture, Texas, 1986.

(5) California Department of Food and Agriculture. Red imported fire ant statewide detection survey. In: California action plan - red imported fire ant executive summary. http://pi.cdfa.ca.gov/rifa_plan/rifa_ltp.htm. Date downloaded, 06 September 2001.

(6) Sutherst RW, Maywald G, Yonow T, Stevens P. CLIMEX. Predicting the effects on climate on plants and animals. CD-ROM and User guide. CSIRO Publishing, Melbourne 90pp, 1999.

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Investigation of post-weaning multisystemic wasting syndrome

Suspicion of post-weaning multisystemic wasting syndrome (PMWS) on a farm in the Waikato region of the North Island was officially reported to MAF in 2003. This article reports the investigations undertaken on this and three other farms on which PMWS has now been confirmed.

Post-weaning multisystemic wasting syndrome (PMWS) is an emerging disease first reported in Canada in 1996[1]. The syndrome has now been described in most countries worldwide but not in Australia. The only factor known to be necessary for the syndrome is porcine circovirus type 2 (PCV2)[2][3]. However, while necessary, PCV2 alone does not appear to be sufficient to cause PMWS in commercially managed pigs. It has been argued that other causal components are necessary for PCV2 to cause disease[4][5][6]. Porcine circovirus type 2 appears to be ubiquitous in pig populations worldwide and has been recovered from most healthy, slaughter-age pigs sampled in a recent New Zealand study[7][8].

PMWS generally affects eight- to 14-week-old weaned pigs, although it has been described in both older and younger pigs. Affected pigs are generally unthrifty and pale and may develop dyspnoea in the later stages because of severe lung pathology.

Less frequently, they may also have diarrhoea, gastric ulceration, icterus and lymph node enlargement. As these signs are not specific to PMWS, other diagnoses must be considered. Morbidity and mortality are most severe in acute outbreaks, in which usually 4-40% of pigs are affected, of which 70-80% usually die. As with other diseases, the severity depends on farm-specific factors such as management, environment, nutrition and health status[9][10][11].

Materials and methods

PMWS was confirmed using the diagnostic criteria described by Sorden, with minor modifications (Table 1)[9][10]. Testing to exclude differential diagnoses and to assess the involvement of endemic agents was also undertaken on Case Farm One (Table 2).

After confirmation of PMWS on Case Farm One, a delimiting survey was undertaken. Farms were selected through tracing of risk conveyors from infected properties, through report cases to the central MAF exotic disease reporting freephone and by targeting herds that used semen sourced from outside Australasia.

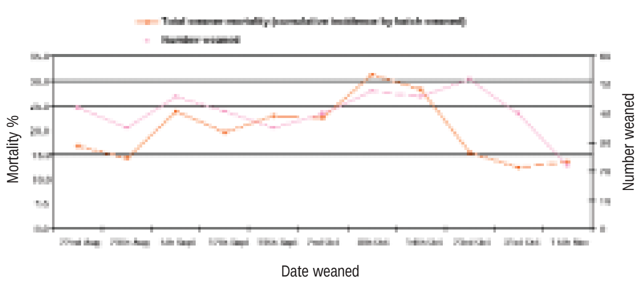
Table 1: Criteria for diagnosis of PMWS

1. *Clinical signs*
Pigs aged four to12 weeks affected by a wasting syndrome with high mortality. Other signs may be present, including a respiratory component characterised by terminal dyspnoea, enlarged lymph nodes, diarrhoea, pallor and jaundice.
2. *Histopathological changes*
Granulomatous inflammatory lesions in various tissues and organs including lymph nodes, spleen, tonsil, lung, liver, kidney and ileum. In the lymphoid tissues the following were considered indicative of PMWS: loss of cortico-medullary architecture, depletion of lymphoid cells, infiltration of follicular and paracortical regions by histiocytes, presence of multinucleated histiocytes and the finding of botryoid cytoplasmic inclusion bodies in areas of lymphocyte depletion or histiocyte infiltration.
3. *Immunohistochemistry*
Demonstration of abundant PCV2 antigen within characteristic histological lesions.

The investigative protocol for traced farms involved clinical assessment and necropsy by two veterinarians: an experienced pig veterinarian and an Exotic Disease Investigator from MAF's National Centre for Disease Investigation (NCDI). Where suspicious signs were observed, at least six pigs were euthanased and tissue samples collected into buffered 10% formalin for histological examination by the MAF reference pathologist. If any pig had histological lesions consistent with or suspicious of PMWS, selected tissues from all pigs sampled on that farm were examined by immunohistochemistry (IHC) for PCV2 antigen at the Veterinary Laboratories Agency, Weybridge, UK. For a herd to be declared PMWS-infected, all three criteria listed in Table 1 had to be met.

Quarantine measures on infected premises and movement controls between the North and South Island were legally enforced to limit the spread of disease.

Figure 1: Case Farm One: weaner (4-12 week) mortality by batch weaned



Results

Case Farm One

This property is a 150-sow farrow-to-finish herd that purchased boars from only two nucleus herds. High mortality in the post-weaning period appeared to have had an abrupt onset in August 2002, preceded by a three- to four-month period of infertility in the sow herd. Disease onset was usually two to three weeks after weaning. Affected animals wasted rapidly and many also had diarrhoea. Dyspnoea was a terminal sign and icterus was seen occasionally. Most of those affected were non-responsive to antibiotic therapy.

The farmer reported weaner mortality of 20-50% in most batches, with a case fatality around 80%. Weaner mortality by batch is summarised in Figure 1. Since August 2002, many endemic diseases complicated by management factors had been suspected,

Table 2: Case Farm One: Summary of investigation to exclude differential diagnoses and assess involvement of endemic agents and management factors

	Status +/- (number +ve)	Laboratory test used	Number tested	Animal group tested
Viral infectious agents				
Porcine Respiratory and Reproductive Syndrome virus	–	Antibody ELISA	40	22-24 wk slaughter pigs
Porcine Parvovirus	+ (3)	Antibody ELISA	40	22-24 wk slaughter pig
	+ (7)		7	Sows – historic laboratory report
Aujeszky's Disease Virus	–	Antibody ELISA	39	22-24 wk slaughter pigs
		VNT antibody detection	10	
Porcine Respiratory Coronavirus (including TGVE)	–	Antibody ELISA	10	22-24 wk slaughter pigs
Encephalomyocarditis Virus	+ (4)	VNT antibody detection	30	22-24 wk slaughter pigs
Pestivirus	–	Antigen ELISA	30	22-24 wk slaughter pigs
Bacterial infectious agents				
Haemophilus parasuis		Histopathology		
	+	Historic laboratory reports		Weaner age pigs
Pasteurella species	+	Bacterial culture		Weaner age pigs
		Historic laboratory reports		
Bordetella bronchiseptica	+	Bacterial culture		Weaner age pigs
		Historic laboratory reports		
Actinobacillus pleuropneumoniae	+	Bacterial culture		Weaner age pigs
		Historic laboratory reports		
Salmonella Typhimurium phage type 193	+	Bacterial culture		Weaner age pigs
Leptospira pomona	+ (3)	MAT antibody detection	10	22-24 wk slaughter pigs
Other infectious agents				
Ascaris suum		Gross pathology		
	+	PigCheck® records		
Toxoplasma gondii	+ (8)	LAT antibody detection	30	22-24 wk slaughter pigs
Mycoplasma hyopneumonia	+	PCR		
		Gross pathology		
		PigCheck® records		
Management factors				
Air quality	+	Pig veterinary specialist		
Water quality	+	Chemical and bacterial screening		
Feed quality	+	Pig veterinary specialist		
		Mycotoxin and bacterial screening		
Temperature regulation	+	Pig veterinary specialist		
Enzootic pneumonia vaccination programme	+	Farm history		
Management stressors (mixing, single air-space, other)	+	Pig veterinary specialist		

Figure 2: Weaners from Case Farm One (green dot indicates previous antibiotic therapy)

investigated and confirmed. Transient but significant decreases in weaner mortality followed various farmer and veterinary initiatives, which included improved nutrition, medication, management and environmental changes.

Clinical assessment by a specialist pig veterinarian confirmed the presence of a herd-level syndrome, non-responsive to therapy and characterised by wasting and illthrift (Figure 2).

The condition was considered consistent with PMWS, although it could have resulted from a combination of disease, environmental and feed-related factors. Nine affected animals, aged from seven to ten weeks, were purposively selected for clinical examination, and for targeted necropsy, histological and PCV2 antigen examinations.

The pigs selected were either thin or emaciated. Rectal temperatures varied between 37.6 and 40.1°C. Respiratory distress was evident in four individuals. Superficial lymph nodes of five were marginally enlarged and on palpation were better demarcated than normal with a 'rubbery' consistency. Diarrhoea was present in five. Pallor was evident in four and one weaner had hyperaemic mucous membranes. None were icteric.

Gross postmortem findings included pneumonic change involving anteroventral and diaphragmatic lung (10-60%) consolidation in seven pigs, with incomplete lung collapse evident in two animals. Fibrinopurulent peritonitis, pleuritis and pericarditis were present in two pigs from which *Salmonella* Typhimurium phage type 193 was isolated. Localised lymphadenopathy, usually relating to nodes draining areas of suppurative inflammation, was noted in three pigs, but enlargement of lymph nodes was not otherwise obvious.

One pig had moderate thickening of the keratinised mucosa of the pars oesophageal region of the gastric mucosa. Eight had excessively fluid contents in the small and large intestines, and four

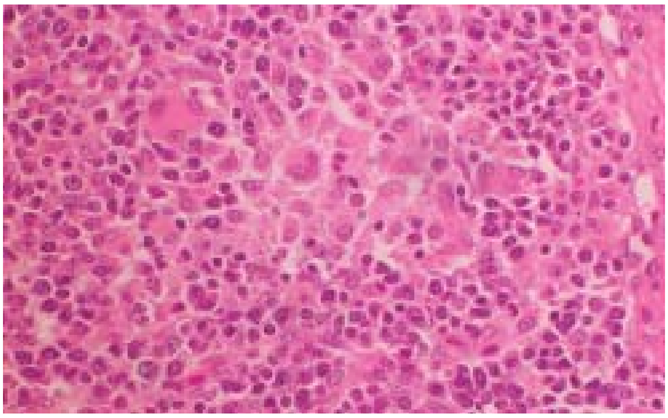


Figure 3: Histiocytic infiltration of lymph node cortex of a pig with PMWS (Case Farm One). Many of the histiocytes are bi- or multi-nucleate. (H&E, x 150)

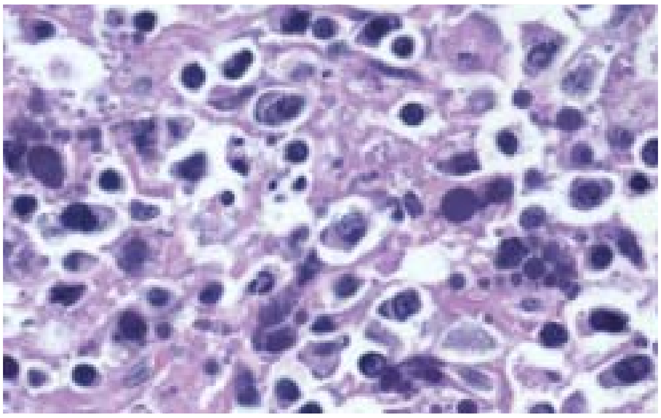


Figure 4: Multiple botryoid inclusion bodies of circovirus 2 in the lymph node of a pig with PMWS (Case Farm One). (H&E, x 220)

of these had mesocolic oedema. Two of these animals had fibrinonecrotic plaques on the mucosal surface of the ileum and spiral colon. Liver and kidneys were grossly normal. These changes were consistent with those observed in earlier postmortem examinations performed on this farm.

Histological changes varied considerably from animal to animal, and often between tissues of individuals. Five animals demonstrated the full spectrum of lymphoid lesions and associated 2-5 micron botryoid inclusion bodies consistent with PMWS (Figures 3, 4, 6). In the remaining pigs, not all of the features were identified. Eight had moderate to severe diffuse lesions of histiocytic interstitial pneumonia that was frequently complicated by suppurative to fibrinosuppurative broncho- to broncho-interstitial pneumonia. There was mild to moderate focal granulomatous interstitial nephritis in three pigs, and seven had granulomatous hepatitis with mild sinusoidal, portal and perilobular fibrosis.

Immunohistochemistry confirmed abundant PCV2 associated with characteristic histological lesions in all nine pigs sampled (Figure 5).

The investigation into endemic disease agents, differential diagnoses and management factors is summarised in Table 2.

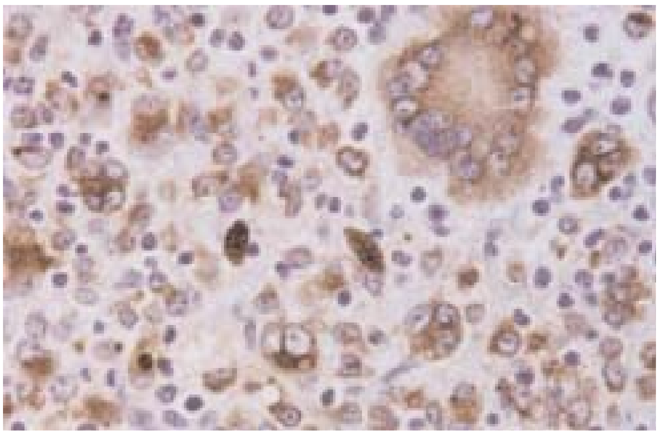


Figure 5: Immunohistochemistry for PCV2 antigen in lymph node (Case Farm One). Intense cytoplasmic staining (brown) of syncytial cells and macrophages visible. Acknowledgement to Merial UK for providing anti-PCV2 antibody

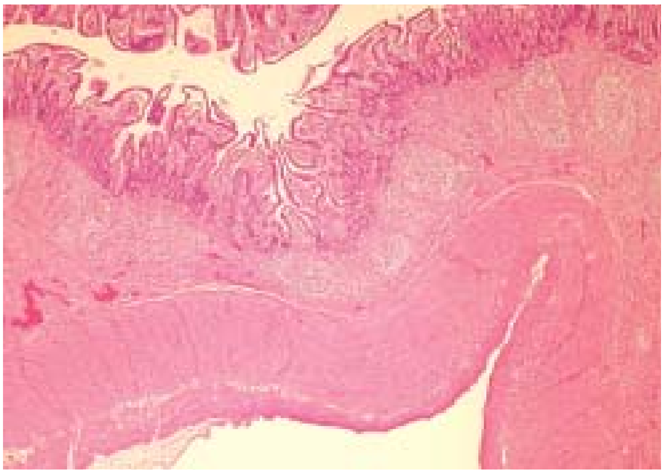


Figure 6: Ileum of a pig with PMWS, showing severe diffuse atrophy of the lymphoid tissue of Peyer's patches. (H&E, x 15)

Further investigations

By the middle of February 2004, 40 further farms in the North Island identified by tracing or reports had been visited and subjected to the same investigation procedure. Of these, infection has been confirmed on three (all three criteria in Table 1 fulfilled), while a further 13 returned suspicious clinical and histological findings and are presently awaiting confirmatory IHC testing. Findings on the confirmed infected farms are summarised below.

Case Farm Two is a 47-sow herd that feeds byproducts milled on Case Farm One. The farm sells progeny between ten and 22 weeks of age, depending on demand. Weaners and replacements are purchased from many sources including markets. The farm was identified through tracing of risk conveyors from Farm One. The farmer reported two periods of high weaner mortality typified by wasting weaner-aged pigs, generally non-responsive to therapy, in August 2001 and February/March 2003. Mortality in affected batches reached about 50% during these episodes.

At the investigation, it was difficult to evaluate PMWS as a clinical syndrome because of the rapid turnover of stock. Around 2% of weaners were in suboptimal condition. There was evidence of respiratory disease but not of respiratory distress. Two weaners had slightly enlarged superficial lymph nodes, although these animals turned out to be IHC-negative. No diarrhoea, jaundice or pallor was seen in the sampled group. Histological examination demonstrated the full spectrum of lesions consistent with PMWS in two pigs from the purposive sample of six. IHC confirmed these two cases as PMWS-positive.

Case Farm Three is a 300-sow farrow-to-finish herd fed byproducts. The farm has purchased replacement boars and gilts from two nucleus herds and on occasions has bought in weaners. The farm was the subject of a MAF-led investigation into a grower mortality event in June 2003. Re-evaluation of tissue samples from this previous investigation by the MAF reference pathologist, in consultation with visiting Professor Chanhee Chae (Seoul National University, South Korea), raised suspicions of PMWS. This led to further sampling using the standard protocol. Farm records showed fluctuations in post-weaning mortality of 2-15% over the period 2000 to 2003. Weaner cohort mortality was approximately 8%, and 3% of weaners were in suboptimal condition. Three of eight sampled pigs had signs of respiratory distress, severe in two animals. Lymph nodes were marginally enlarged in three weaners (two were confirmed PMWS-positive by IHC), and no diarrhoea, jaundice or pallor was seen in the sampled group. Histological examination demonstrated the full spectrum of lesions consistent with PMWS in two pigs. IHC confirmed these two and one further weaner as PMWS-positive.

Case Farm Four is a 160-sow farrow-to-finish herd fed byproducts. It bought in weaners from a single source and replacement boars from one nucleus herd. The herd was reported to the MAF exotic disease freephone by a veterinary pathologist who had identified suspicious lesions in samples submitted by a veterinary practitioner. Weaner health deteriorated in August 2003 coinciding with the purchase of pigs from a weaner source.

Mortality amongst weaners subsequently increased to a maximum of 40%. At the time of investigation, about 20% of the weaners were in suboptimal condition. Respiratory distress was evident in two and lymph node enlargement was marginal in three of six weaners sampled. Diarrhoea was a feature in one weaner in the sampled group. Jaundice was evident in one, one had cyanotic mucous membranes and two were pale. Histological examination demonstrated lesions highly suspicious for PMWS in four of the six pigs, although no inclusion bodies could be identified. IHC confirmed all six weaners as PMWS-positive.

Discussion

PMWS is a high mortality, variable morbidity, wasting syndrome affecting weaner pigs. Variable morbidity within and between herds has been noted in the confirmed positive farms in New Zealand. It is possible that this may reflect differences in management, nutrition and environment, or time since infection[9][11][12]. After an initial period of high mortality that may continue for six to 24 months, herds are believed to enter a recovery phase. Although weaner mortality in herds affected with PMWS in England reached an average peak of 19.9%, mortality before infection was 3.7% and during perceived recovery was 9.8%[12]. Historical mortality on Case Farm Three suggests that it may have been infected before or during 2000, which could explain its current relatively low mortality rates.

Variability in intra- and inter-herd morbidity, the non-specific nature of the clinical and gross postmortem findings, and the often favourable responses to management and veterinary interventions, will make the clinical diagnosis of PMWS difficult in some herds. As a result, PMWS should always be considered when there is an unexplained and sustained increase in post-weaning mortality (more than 4% during the weaner stage or more than 6% total post-weaning mortality). In such cases, affected animals between eight and 12 weeks of age should be sacrificed for necropsy and fresh samples of lymph nodes (minimum two), tonsil, lung, liver, kidney, spleen and small intestine (including Peyer's patches) collected into buffered 10% formalin and submitted for histopathology.

Botryoid inclusion bodies are an excellent indicator of PMWS when seen in combination with lymphoid depletion and histiocyte infiltration. Inclusion bodies were seen in 45% (13/29; range by farm 25% [2/6] to 67% [4/6]) of weaners from farms confirmed positive in New Zealand. In a large study of 455 Spanish pigs, the detection rate was 32%[9]. Overseas findings and experience gained during this investigation suggest that at least six clinically affected pigs should be sampled to ensure adequate sensitivity. The majority of clinical signs observed in pigs on the described farms are typical for PMWS. However, superficial lymph node enlargement is commonly reported overseas[9][11] but was infrequently observed here. In this investigation there was mild enlargement of superficial lymph nodes, which on palpation were firmer and more demarcated. This finding was not confined to PMWS-positive animals. In an assessment of 38 weaners from five farms, the 11 IHC-positive pigs had a mean superficial lymph node volume (calculated as a cylinder) of 6.8 cm^3 (sd 4.9; 95% CI 3.5-10.1) whereas the mean for 27 negative weaners was 5.6 cm^3 (sd 3.4; 95% CI 4.3-6.9). This 21% difference was not statistically significant and would not appear to be important as a guide for raising clinical suspicion of PMWS in New Zealand.

The four known positive and 13 suspect positive herds detected have all been in the North Island. All are located in the upper half of the island and can be linked to one another through known contacts, including a common feed source, or live pig or other conveyor movements. Information about these epidemiological links is still accumulating and will be reported fully at a later stage.

PMWS has not been detected in any nucleus farms or farms importing semen. A survey of pig farms has provided no evidence of PMWS in the South Island. This apparently limited distribution of PMWS suggests that the disease has been recently introduced, perhaps within the last five years.

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References

(1) Harding JCS, Clark EG, Strokappe JH, Willson PI, Ellis JA. Postweaning multisystemic wasting syndrome: epidemiology and clinical presentation. Swine Health and Production 6, 249-54, 1998.

(2) Kennedy S, Moffett D, McNeilly F, Meehan B, Ellis JA, Krakowka S, Allan GM Reproduction of lesions of postweaning multisystemic wasting syndrome by infection of conventional pigs with porcine circovirus type 2 alone or in combination with porcine parvovirus. Journal of Comparative Pathology 122, 9-24, 2000.

(3) Bolin SR, Stoffregen WC, Nayar GPS, Hamel AL. Postweaning multisystemic wasting syndrome induced after experimental inoculation of cesarean-derived, colostrum-deprived piglets with type 2 porcine circovirus. Journal of Veterinary Diagnostic Investigation 13, 185-94, 2001.

(4) Morris RS, Davies PR, Lawton DE. Evolution of Diseases in the World's Pig Industry. 17th International Pig Veterinary Conference Proceedings, Ames, Iowa, 2002.

(5) Chadd SA. The characteristics and attempted control of post-weaning multisystemic wasting syndrome (PMWS) and porcine dermatitis and nephropathy syndrome (PDNS) in European pig herds - A review. Pig Journal 51,13-25, 2003.

(6) Quintana J, Segales J, Rosell C, Calsamiglia M, Rodriguez Arrioja GM, Chianini F, Folch JM, Maldonado J, Canal M, Plana Duran J, Domingo M. Clinical and pathological observations on pigs with postweaning multisystemic wasting syndrome. Veterinary Record 149, 357-61, 2001.

(7) Krakowka S, Ellis JA, McNeilly F, Gilpin D, Meehan B, McCullough K, Allan G. Immunologic features of porcine circovirus type 2 infection. Viral Immunology 15, 567-82, 2002.

(8) Tham KM, Hansen M. Detection of porcine circovirus types 1 and 2 in abattoir-slaughtered pigs in New Zealand. Surveillance 30(1), 3-5, 2003.

(9) Segales J, Domingo M. Postweaning multisystemic wasting syndrome (PMWS) in pigs. A review. Veterinary Quarterly 24, 109-24, 2002.

(10) Sorden SD. Update on porcine circovirus and postweaning multisystemic wasting syndrome (PMWS). Swine Health and Production 8, 133-6, 2000.

(11) Allan GM, Ellis JA. Porcine circoviruses: a review. Journal of Veterinary Diagnostic Investigation 12, 3-14, 2000.

(12) Gresham A, Cook AJC, Thomson JR, Kennedy S. Survey of veterinary practitioners on PMWS and PDNS in the UK. Veterinary Record 153, 400-3, 2003.

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Health assessment of wallabies from Kawau Island

The health status of 30 live-trapped wallabies on Kawau Island was assessed for export purposes. Tests included gross pathology, histopathology, bacteriology, serology, virology and parasitology. There was no evidence of leptospirosis, brucellosis, Johne's disease, chlamydiosis, tuberculosis or significant viral infection. *Yersinia enterocolitica* was present in low numbers in faeces from four animals. Coccidiosis and capture myopathy were noted.

There are four free-ranging species of wallaby on Kawau Island: tammar (*Macropus eugenii*), parma (*M parma*), brushtailed rock (*Petrogale penicillata penicillata*) and swamp (*Wallabia bicolor*). They are descended from stock originally released on the island in the 1870s[1]. Kawau Island is in the Hauraki Gulf north of Auckland, about 8 km offshore.

Few disease investigations have been carried out on free-ranging wallabies in New Zealand and there have been no significant parasites or infectious diseases reported[2]. The only parasite recorded for tammars and parmas on Kawau Island is an unidentified louse-like ectoparasite (Vujcich, 1979, cited in reference 2). There is no evidence that wallabies in New Zealand act as vectors for bovine tuberculosis.

From wallabies maintained in captivity in New Zealand, there is one report of tuberculous lymphadenitis in an animal that died from toxoplasmosis[3]. Toxoplasmosis has caused several outbreaks of acute mortality with interstitial pneumonia and non-suppurative encephalitis at both zoos and research facilities[3][4]. Intestinal coccidiosis was also recorded from a captive wallaby in Hamilton[5].

The purpose of this investigation was to assess the health status of tammar and parma wallabies that are free-ranging on Kawau Island and in contact with animals to be repatriated to South Australia. The diseases of greatest concern were bovine tuberculosis and Johne's disease. Thirty animals were selected for examination. This would provide a 95% confidence of detecting a disease at a prevalence of 10% in a random sample.

Methods

Thirty wallabies (ten tammar and 20 parma) were trapped and held in pens on the island for a variable length of time before transportation by road to Massey University. They were individually crated and delivered in three shipments of 11, ten, and nine animals each. Upon arrival they were sedated using ketamine (10 mg/kg, i/m), and up to 10 ml blood was collected from the lateral caudal vein into untreated Vacutainers. They were then euthanased by intracardiac injection of sodium pentobarbital (Pentobarb 500). Pouches of females were checked for joeys and, if present, they were euthanased using the same procedure. Postmortem examinations were conducted within an hour of death.

Bacteriology samples (lung, faeces) from seven wallabies were collected aseptically into sterile sample containers and chilled overnight before plating. Virology samples (lung, tonsil) from six wallabies were collected aseptically into cryovials (Nunc) and frozen in liquid nitrogen for shipment to MAF's National Centre for Disease Investigation (NCDI). Postmortem examination of all 30 wallabies included dissection and examination of all lymph nodes including submandibular, prescapular, axillary, mediastinal, gastric, hepatic, pancreatic, mesenteric, inguinal, lumbar, colonic and mammary. The nodes were sectioned longitudinally to inspect for granulomas and then fixed in 10% neutral buffered formalin. Each organ system was examined systematically and appropriate samples were fixed in formalin. Histopathology was performed on samples from the first six tammar wallabies.

Samples for bacteriology were cultured for aerobic organisms and selective media used for *Salmonella* sp, *Campylobacter* sp, and *Yersinia enterocolitica*. For virus isolation, lung and tonsil samples were pooled and homogenised and passaged twice in tissue culture using possum kidney cells. The monolayers were then examined for cytopathic effects.

Nematode egg counts were performed on faecal samples, from ten wallabies, following addition to 33% $ZnSO_4$. One animal had a particularly high egg burden so a complete intestinal washing was carried out to recover adult parasites. Nematodes were washed in saline and then fixed in 70% ethanol for identification.

Serology was conducted on ten animals (nine tammars and one parma) for the following: *Brucella abortus* (CFT and ELISA), *Chlamydia* (CFT), *Mycobacterium paratuberculosis* (CFT and AGID), *Leptospira copenhagenii* (MAT), *L hardjo* (MAT), *L pomona* (MAT) and macropod (wallaby) herpesvirus type 1 (VNT).

Some of the sera showed anticomplementary reactions with the CFT and therefore their serological status could not be established with this test. The VNT for macropod herpesvirus type 1 was carried out using a possum kidney cell line.

Results

Gross pathology: The only gross lesion noted in the tammar wallabies was diffuse thickening of the oesophageal wall from one adult male animal (wallaby D). The mucosa of the oesophagus was hyperplastic and appeared to have numerous cyst-like structures (up to 1 mm diameter) within the thickened epithelium. The remainder of the gastrointestinal tract was unremarkable.

One parma wallaby that was dead on arrival (wallaby K) had a subcutaneous haematoma on the left side of its face surrounding the parotid salivary gland. It also had several white foci (up to 5 mm diameter) scattered throughout the liver.

Five male parmas had external trauma lesions ranging from bite wounds on the limbs or face and ears to focally extensive areas of alopecia and epidermal erythema over the crown of the head. They also had skeletal myopathy affecting the ileopsoas muscles in the lumbar region and the adductor and gracilis muscles.

In all animals the lymph nodes were small and had normal colour and architecture. Apart from the one animal mentioned above, grossly the gastrointestinal tract was unremarkable in all animals and there was no evidence of enteritis or diarrhoea.

Histopathology: The results of histopathological examination of six tammar wallabies are summarised in the table. No acid-fast bacteria were detected in lymph nodes using Ziehl-Neelsen staining.

Histological diagnoses in six tammar wallabies	
Wallaby A	Mild acute myopathy in diaphragmatic muscle.
Wallaby B	Mild acute myopathy in skeletal muscle.
Wallaby C	Mild small intestinal coccidiosis without significant associated tissue changes (see Figure 1). A small chronic hepatic granuloma of uncertain cause but possibly parasitic.
Wallaby D	Subacute diffuse oesophagitis with hyperkeratosis, ballooning degeneration, cyst formation and focal haemorrhage (see Figure 2). Possibly caused by reflux acidosis or ingestion of a toxin.
Wallaby E	Mild small intestinal coccidiosis without significant associated tissue changes.
Wallaby F	Mild subacute myopathy in skeletal muscle. Mild acute subcapsular hepatitis with neutrophil infiltration.

Bacteriology: Yersinia enterocolitica was cultured from faeces of wallabies D, E, F and G. No other bacteria were isolated. There was no viral cytopathic effect detected. The results of all serology tests were negative. Some sera (six of ten) were anticomplementary in the chlamydiosis CFT and as no alternative test was available their status could not be assessed.

Parasitology: Two types of strongyloid nematode eggs were recovered in low to moderate numbers from faeces. A new species of *Austrostrongylus* was identified from an adult parma (Beveridge I, personal communication).

Virology: No cytopathic or haemagglutinating agents were isolated.

Discussion

The wallabies were in moderate to good body condition without overt signs of illness. Trauma and skeletal myopathy lesions in males were acute and associated with capture and holding in wire pens before shipping. All animals had reduced internal fat reserves but six of 12 females had pouch young suggesting that their body condition was sufficient to allow normal reproduction.

No ectoparasites were observed on the wallabies although there is a previous report of lice on wallabies from Kawau Island (Vujcich 1979, cited in reference 2), and lice occur on free-living wallabies in Australia[6].

A subcutaneous nematode, *Pelecitus* (*Dirofilaria*) *roemeri*, described for macropods in Australia was not observed in these animals[7].

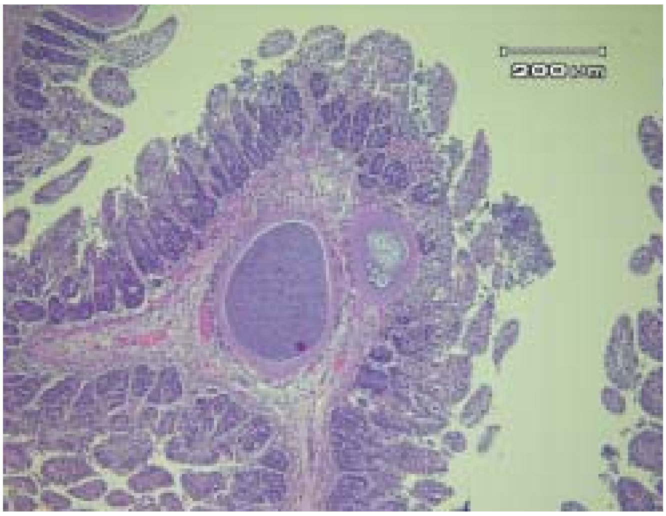

Figure 1: Tammar wallaby C. Small intestine H&E.
Coccidian megaloschizont and macrogametocyte in the lamina propria.
Note the absence of an inflammatory response

Other endoparasites encountered included the new species of *Austrostrongylus.* There was no evidence that these worms were clinically significant in any of the animals examined.

Two of the tammar wallabies had coccidian parasite life stages in the lamina propria and epithelium of the small intestine. These were present without associated pathology suggesting that they are host-adapted. Coccidial oocysts, identified as *Eimeria macropodis,* were recently recovered from faeces of a tammar captive at Hamilton Zoo and are the first record of coccidiosis in wallabies in New Zealand[5].

Coccidiosis is apparently a common finding in tammars in Australia and rarely causes disease although it has the potential to do so in young animals (Rose K, Beveridge I, personal communication). Overall, some 35 species of diprotodonts (macropods and koalas) have coccidian parasites with some 50 species of *Eimeria* described[8]. In larger species of macropods in Australia coccidiosis frequently takes a peracute form, with the animal found dead without premonitory signs. At necropsy there is

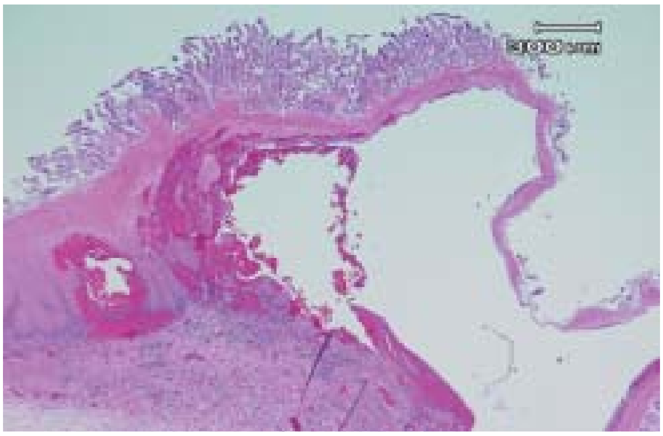

Figure 2: Tammar wallaby D. Oesophagus H&E. This section shows the surface of the mucosa lined by numerous ciliated protozoa and bacterial colonies. There is a large haemorrhagic vesicle formed within the squamous epithelium with ballooning degeneration of keratinocytes towards the left of the image. The lamina propria is infiltrated by large numbers of granulocytes and there is submucosal oedema

severe haemorrhagic enteritis with blood throughout the small intestine but normal contents in the large intestine[9]. Beveridge has described hepatic coccidiosis in tammar wallabies in Australia[9]. This was not detected in the limited number of animals sampled here but one wallaby did have a focal hepatic granuloma suggesting a resolving parasitic infection while a second had evidence of an acute suppurative hepatitis of unknown aetiology.

There was no evidence of brucellosis, chlamydiosis, leptospirosis, salmonellosis, Johne's disease or tuberculosis. The only bacterium of concern was *Yersinia enterocolitica* isolated from faeces of four of seven animals tested. This bacterium can cause zoonotic infection and is regarded as an emerging disease[10]. It has previously been isolated from marsupials, among other zoo and wild mammals[11]. It is of little consequence in free-living animals and rarely causes clinical disease[12]. There was no evidence of gastroenteritis in the wallabies examined and the number of bacteria was small, isolation requiring up to 14 days enrichment culture.

Among the viral diseases of wallabies, macropodid herpesviruses (macropod herpesvirus I and II) are probably of greatest concern because of their potential to cause disease outbreaks in stressed animals. Epidemics have previously been recorded in captive and free-ranging parma and tammar wallabies in Australia, and serologic surveys have shown high seroprevalence in several macropod species[13][14][15][16][17][18][19]. Herpesviruses were not isolated from the tammar wallabies from Kawau Island and all animals tested for antibodies were negative, suggesting that the viruses do not occur on Kawau Island. Thus, the tammar wallabies shipped to Australia could be at risk when they come in contact with endemically infected animals. Other viruses that do not occur in New Zealand, and to which tammars may be exposed in Australia, are the arthropod borne orbiviruses. Wallal and Warrego viruses have usually been associated with epidemics in the larger macropods[20]. However, an as yet uncharacterised tammar orbivirus has caused epidemics of sudden death in captive animals at zoos and research facilities that began in October 1998[21]. This could pose a significant risk to newly arrived naïve animals.

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References

(1) Wodzicki KA, Flux JEC. Guide to introduced wallabies in New Zealand. Tuatara 15, 47-59, 1967.

(2) Warburton B, Sadleir RMF. Dama wallaby. In: King CM (ed). The Handbook of New Zealand Mammals. Pp 35-44. Oxford University Press, Auckland, 1990.

(3) MAF. Toxoplasmosis in wallabies. Surveillance 2, 14, 1974.

(4) MAF. Toxoplasmosis in wallabies. Surveillance 4, 19, 1980.

(5) McKenna PB. Register of new host-parasite records: Coccidia in a wallaby. Surveillance 30(1), 12-3, 2003.

(6) Durden LA. Ectoparasites: Lice. In: Samuel WM, Pybus MJ, Kocan AA (eds). Parasitic diseases of wild mammals. Pp 3-17. Iowa State University Press, Ames, Iowa, 2001.

(7) Spratt DM. Aspects of the life history of *Dirofilaria roemeri* in naturally and experimentally infected kangaroos, wallaroos, and wallabies. International Journal for Parasitology 2, 139-56, 1972.

(8) Duszynski DW, Upton SJ. Enteric protozoans: *Cyclospora, Eimeria, Isospora,* and *Cryptosporidium* spp. In: Samuel WM, Pybus MJ, Kocan AA (eds). Parasitic diseases of wild mammals. Pp 416-59. Iowa State University Press, Ames, Iowa, 2001.

(9) Beveridge I. Marsupial parasitic diseases. In: Fowler ME (ed). Zoo and Wild Animal Medicine, 3rd ed. Pp 288-93. WB Saunders Company, Philadelphia, 1993.

(10) Bottone EJ. *Yersinia enterocolitica:* The charisma continues. Clinical Microbiology Reviews 10, 257-76, 1997.

(11) Mingrone MG, Fantasia M. Characteristics of *Yersinia* spp. isolated from wild and zoo animals. Journal of Wildlife Diseases 24, 25-9, 1988.

(12) Gasper PW, Watson RP. Plague and yersiniosis. In: Williams ES, Barker IK (eds). Infectious Diseases of Wild Mammals, 3rd ed. Pp 313-29. Iowa State University Press, Ames, Iowa, 2001.

(13) Finnie EP, Littlejohns IR, Acland HM. Mortalities in parma wallabies (*Macropus parma*) associated with probable herpesvirus. Australian Veterinary Journal 52, 294, 1976.

(14) Webber CE, Whalley JM. Widespread occurrence in Australian marsupials of neutralizing antibodies to a herpesvirus from parma wallabies. Australian Journal of Experimental Biology and Medical Science 56, 351-7, 1978.

(15) Acland HM. Parma wallaby herpesvirus infection. Journal of Wildlife Diseases 17, 471-7, 1981.

(16) Callinan RB, Kefford B. Mortalities associated with herpesvirus infection in captive macropods. Journal of Wildlife Diseases 17, 311-317, 1981.

(17) Kerr A, Whalley JM, Poole WE. Herpesvirus neutralizing antibody in grey kangaroos. Australian Veterinary Journal 57, 347-8, 1981.

(18) Wilks CR, Kefford B, Callinan RB. Herpesvirus as a cause of fatal disease in Australian wallabies. Journal of Comparative Pathology 91, 461-5, 1981.

(19) Britt JO, Frost DF, Cockrill JM. Fatal herpesviral hepatitis in red kangaroo (*Macropus rufus*). Journal of Zoo and Wildlife Medicine 25, 580-84, 1994.

(20) Durham PJK, Finney JW, Lawrence DA, Alexander P. Blindness in South Australian kangaroos. Australian Veterinary Journal 73, 111-2, 1996.

(21) Rose KA, Kirkland PD, Davis RJ, Cooper DW, Blumstein DT. An epizootic of sudden death in tammar wallabies (*Macropus eugenii*). Proceedings of the American Association of Zoo Veterinarians, New Orleans, September 2000.

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Do chain springs on leghold traps reduce injuries to captured possums?

A field trial was conducted to assess the effectiveness of the addition of chain-springs to two models of leghold traps for reducing injuries to captured possums. Injuries were reduced but were still more frequent and severe than those caused by a third (smaller) type of trap.

Brushtail possums (*Trichosurus vulpecula*) were introduced into New Zealand from Australia between 1850 and 1900 to establish a fur trade, and they have now spread and occupied most of New Zealand. In indigenous forests they have become a significant conservation pest by reducing the biodiversity of the vegetation and by preying on bird eggs and chicks. On farmland where they share grazing areas with cattle and deer, they have become the major wildlife vector of bovine tuberculosis, which continues to pose a significant threat to New Zealand's exports of beef, dairy and venison products. To reduce the impact of this species on both the conservation and farming estate, in excess of $NZ50 million is spent annually on control using both traps and a range of vertebrate pesticides. Even though there is strong justification for controlling possums in New Zealand, the animal welfare and environmental impacts of the methods used are continually under review to ensure there is a constant improvement in our control technology. One set of control tools that has recently been the focus of attention is the use of leghold traps.

Figure 1: Possum captured in a Lanes-Ace long-spring trap

Steel-jawed leghold traps such as the Lanes-Ace or 'gin' trap have been used extensively for trapping possums in New Zealand since the 1920s (see Figure 1). The Lanes-Ace trap is powered by a flat-metal spring, and is called a 'long-spring' trap, in contrast to the now more commonly used Victor-type traps that are powered by two coil springs, and hence called 'double-coil' traps (see Figure 2).

Since the late 1970s there has been considerable public opposition to the use of leghold traps, especially the serrated jawed and larger models, because of concerns for the welfare of the captured animals. In 2001, the National Animal Welfare Advisory Committee (NAWAC) recommended that two models of leghold trap, the Lanes-Ace and the Victor No 1½ (and their equivalents), be prohibited or restricted in their use.

Before the Minister of Agriculture and Forestry can recommend an Order in Council to prohibit or restrict the use of traps, he or she must assess whether the traps can be modified to address the welfare concerns.

Figure 2: The traps tested: Lanes-Ace (top), Victor No 1½ (middle), and Victor No 1 (bottom)

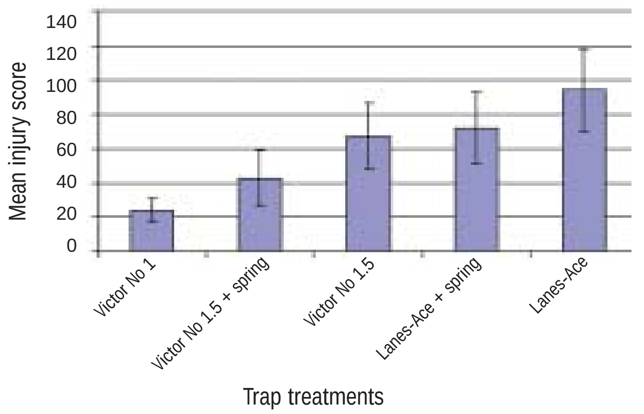
One simple and potentially effective modification for reducing injuries caused by leghold traps is to attach an appropriately sized coil spring between two links of the trap chain to cushion the impact resulting from the animal's struggles (see Figure 3). Because some experienced possum trappers recommended such modification, MAF requested a trial be undertaken to assess whether the modification was effective at reducing the injuries to captured animals.

Figure 3: Victor No 1½ double-coil spring trap with chain-spring modification

Frequency of injuries in four categories of severity that resulted from possums being captured in five trap treatments

Trap type	Number of possums	Mild	Moderate	Moderately severe	Severe
Victor No 1	30	10	13	7	0
Victor No 1½ + spring	30	10	6	9	5
Victor No 1½	32	4	7	14	7
Lanes-Ace + spring	31	5	5	12	9
Lanes-Ace	31	3	5	7	16

Figure 4: The mean cumulative injury scores (± 95% CL) of the five trap-treatments tested



At present the most popular leghold traps in New Zealand are the various brands of No 1 sized traps, especially the Victor No 1. Trappers favour this trap, introduced during the 1980s, because it is compact, light and has an acceptable capture efficiency. The traps also cause less frequent and less severe injuries than the larger traps recommended for prohibition[(1)]. For the addition of chain-springs to be an acceptable modification, the injuries caused by the larger traps must be reduced to at least the level recorded for possums captured in the smaller No 1 sized traps.

Field trials

To assess the effectiveness of chain-springs for reducing injuries, a field trial was carried out using five trap treatments:

- unmodified Lanes-Ace
- Lanes-Ace with chain-springs
- unmodified Victor No 1½
- Victor No 1½ with chain springs
- unmodified Victor No 1

Once a trap site had been selected along a trap line, the trap-treatment to be set at the site was selected randomly. A sample of 30 possums was trapped for each of the five trap treatments, and captured possums were killed quickly with a sharp blow to the head, and their captured limb removed for later inspection by a pathologist (LabWorks Animal Health Ltd, Lincoln) who did not know from which trap the limb had been sampled. The pathologist identified any capture-related traumas, and each separate injury was given a score based on its perceived severity[(2)]. The scores were then summed to give a cumulative trauma score for each animal. Additionally, each trauma was rated as mild, moderate, moderately severe or severe[(2)].

Results

Mean injury scores for each trap treatment varied from a high of 94 for possums captured in unmodified Lanes-Ace traps to 24 for possums captured in Victor No 1 traps (Figure 4).

The differences in the mean injury scores of possums captured in modified and unmodified Lanes-Ace traps (22 points) and Victor No 1½ traps (24 points) were significant (P = 0.03 for both treatments). However, although addition of chain-springs significantly reduced injuries in these two trap models, the resultant mean injury score for possums captured in the modified Lane-Ace traps (72 points) was still significantly higher than that obtained from possums captured in Victor No 1 traps (24 points), (P < 0.001). The mean injury score for possums captured in the modified Victor No 1½ traps (43 points) was not significantly different from that for those captured in Victor No 1 traps (24 points), (P = 0.15), although the Victor No 1½ traps did cause more injuries that were classified as severe in comparison to the Victor No 1 traps (see table).

As an alternative to using mean injury scores for measuring welfare impact of the traps, comparison of the traumas, when rated as mild, moderate, moderately severe or severe, also showed that the addition of chain-springs to both Lanes-Ace and Victor No 1½ traps reduced the number of more severe classes of injury. This provided some evidence of a spring effect for both trap types (P = 0.08). As with the injury scores, although the addition of a chain-spring reduced the severity of injury, the traps still caused more of the severe classes of injuries than the Victor No 1 traps. For example, the modified Lanes-Ace caused three times as many moderate-severe or severe injuries (P = 0.0005), and the modified Victor No 1½ twice as many (P = 0.1) (see table).

Discussion

The addition of chain-springs had a significant effect on reducing the severity of injuries to possums captured in Lanes-Ace and Victor No 1½ traps, but these reductions were not sufficient to enable chain-spring modifications to be accepted as an effective method for making these traps acceptable. Consequently, the recommended prohibitions for these larger traps should still be supported.

The Victor No 1 traps have a capture efficiency similar to that of Lanes-Ace and Victor No 1½ traps[(1)], and therefore prohibiting Lanes-Ace and Victor No 1½ traps (in standard and chain-spring modified form) and allowing the continued use of Victor No 1 traps will still leave trappers with an effective leghold trap, but one that causes the least injuries. The trial did not include a test of No 1 sized traps with chain-springs and so there is the potential that these smaller traps could be similarly modified to cause even fewer injuries than currently accepted.

References

(1) Warburton B. Victor foot-hold traps for catching Australian brushtail possums in New Zealand: capture efficiency and injuries. Wildlife Society Bulletin 20, 67–73, 1992.

(2) ISO. Animal (mammal) traps – Part 5: Methods for testing restraining traps. ISO 10990-5, International Organisation for Standardisation, Geneva. 20pp, 1999

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Quarterly review of diagnostic cases – October to December 2003

Each quarter, Surveillance publishes a review of selected diagnostic cases handled by New Zealand's veterinary diagnostic laboratories, all of which are owned by Gribbles Veterinary Pathology. These cases do not necessarily reflect the national disease profile but they do represent diseases of interest to the livestock industry or of significance to wildlife.

Cattle

Enteric yersiniosis was diagnosed in a group of 112 three-month-old calves in Otago. Most were unthrifty with diarrhoea, and about 14 were particularly badly affected. *Yersinia pseudotuberculosis* was isolated from the faeces of eight of 12 faecal samples cultured.

There was a small outbreak of the **'jejunal haemorrhage syndrome'** on a Southland farm milking Jersey cows. Two cows, healthy and milking well the previous day, were found affected in the morning before milking. One cow was dead, and the other was down with melaena, anaemic and with a low temperature, and was killed in extremis. A necropsy on the first cow showed most of the jejunum contained haemorrhagic contents and a 30-40 cm distal segment was occluded by a blood clot. About two weeks later a third cow was found lethargic and anaemic and in obvious abdominal pain. As she was still standing an exploratory laparotomy was performed and again a blood clot was obstructing the jejunum, and associated mesentery was oedematous. The cow survived an intestinal resection and anastomosis. The jejunal haemorrhage syndrome was first reported in the USA about six years ago, apparently associated with grain feeding and possibly a *Clostridium perfringens* Type A infection. An identical condition occurring in this country in three grass-fed milking cows may rule out a feed initiated cause. As few cows dying on dairy farms are necropsied there may be more cases in this country than recognised.

Severe **abomasal ulcerations** with perforations and peritonitis caused the deaths of 12 of 100 two- to three-month-old calves over a three-week period on a Southland dairy farm. The calves were being fed only pasture from a clean paddock. Although the occasional sporadic death from perforating abomasal ulcerations occurs in calves about this age, the number on this farm suggested a toxic agent. The farmer had built two large mobile sheds to house newborn calves for the first ten days of life. The sheds were made from recently tanalised timber that was still wet. Chronic arsenic poisoning from exposure to tanalised timber was suspected but liver arsenic concentration was negative in the one calf tested. However, as arsenic is rapidly eliminated from soft tissues like liver, and as there was a lag period between exposure and the signs of toxicity, this negative result may not rule out arsenic poisoning.

Monensin toxicity caused the sudden deaths of a number of two- to three-month-old calves on at least three Otago farms where farmers were mixing this compound with meal as a preventative for coccidiosis. The most consistent necropsy finding was a severe pulmonary oedema, often associated with a severe hydrothorax. In one case from which sections of the heart were examined histologically, a severe cardiomyopathy typical of **ionophore toxicity** was seen.

Another case of suspected **ionophore toxicity** involved ten-week-old calves from Taranaki. Over a three-week period, three had died, one a week, with clinical signs of elevated heart and respiratory rates. At necropsy the lungs were diffusely congested and the trachea filled with stable froth. Histologically the lungs were diffusely oedematous and congested. Throughout the heart were multifocal regions of myofibre degeneration adjacent to zones of nuclei proliferation, findings suggestive of a cardiac toxin such as ionophore, oleander or avocado. It appeared the farmer had been adding monensin to the milk each day. The dose was not accurately measured.

Each year several outbreaks of **polioencephalomalacia** occur in calves and this year individual outbreaks in Canterbury and the West Coast have lasted several weeks. In some cases the calves have had severe diarrhoea (of unknown cause). Overseas, this disease is often seen in feedlot cattle on concentrate diets and increasing the roughage in the diet is apparently beneficial. However, the outbreaks in New Zealand seem to coincide with increasing roughage in the diet as the pasture becomes mature.

A recently weaned Friesian bull calf from Hawke's Bay developed a stagger, incoordination and eventually became recumbent, before euthanasia. Neuropil necrosis, neurone degeneration and hypertrophy of blood vessel endothelium confirmed **polioencephalomalacia.**

***Salmonella* Typhimurium** was cultured from the faeces from a group of five-week-old Friesian cross calves from Hawke's Bay that had severe diarrhoea, and from a two-year-old Taranaki dairy cow with severe bloody diarrhoea that contained flecks of mucosal lining.

Two calf-rearing properties in the Hawke's Bay and one in the Manawatu experienced severe respiratory disease and deaths in calves. In the first case 200 12-week-old calves had been purchased three weeks earlier. Seven calves died after developing depression, lethargy, anorexia, condition loss and a cough. In the second case, eight-week-old calves were found dead. In the third case, ten-week-old calves had developed a chronic respiratory syndrome unresponsive to antibiotics. Necropsy showed marked pulmonary consolidation and abscessation. Histologically there was multifocal abscessation and extension of inflammation into surrounding alveoli and bronchi. A heavy growth of ***Haemophilus somnus*** was isolated from lung in each case.

In an autumn-calving herd of Friesian cows from Wanganui, ten cows began cycling when they should have been six months pregnant. One partially autolysed foetus was found. Multifocal regions of neuropil and cotyledonary coagulative necrosis were visible on microscopy, consistent with a diagnosis of **neosporosis.**

Clinical signs of epistaxis, illthrift and pneumonia were reported in two-year-old bull beef rearing units in the Wanganui region. Detection of abscessation of liver and lung at slaughter prompted an investigation in a Waikato bull beef unit. In both cases, and in other cases seen over the past few years, the lesions and clinical signs reflect the pathogenesis of **portocaval syndrome:** a trail of pathology through the rumen, liver and lung. Initiated by acidosis of the rumen (linked to the consumption of high carbohydrate feeds) the first lesion is rumenitis. Once bacteria penetrate the damaged mucosa, they embolise to the liver. Here they may be phagocytosed and neutralised, or multiply and induce necrosis. Fibrosis walls off the necrotic material to create abscesses. If the abscess can be contained the process may stop here and the liver is condemned at slaughter. If not, necrosis of the liver parenchyma and blood vessel walls allows bacteria-laden necrotic material to embolise to the lungs where the process begins again. Abscesses may be walled off, or necrosis may destroy blood vessel walls, resulting in haemorrhage and epistaxis. Once believed to be an overseas feedlot phenomenon linked to grain or concentrate feeding, this syndrome is seen frequently in pasture-fed cattle. Improved and new variety grass species are often fed ad lib. Presumably their high carbohydrate levels cause rumenal acidosis. The syndrome has also been seen in dairy cattle fed silage and concentrate.

Six 15-month-old well grown Friesian dairy heifers from a mob of 40 died suddenly. Postmortem showed only congestion of the abomasum and small intestine. A few leaves were seen in the rumen. Histopathology revealed a subtle myofibre necrosis in the heart. A careful search of the field uncovered browsed, dried oleander clippings discarded in one corner. The heifers had abundant pasture, but inquisitiveness probably led to ingestion of the leaves. As few as six leaves are fatal for an adult horse. **Oleander toxicity** often leads to cardiac myonecrosis.

A three-week-old Hereford Charolais cross calf from Northland lost condition and was euthanased on welfare grounds. It was one of six affected from about 100 running with their dams. Histopathology of skeletal muscle and heart revealed a **skeletal and cardio-myopathy**. The skeletal muscle changes included hyaline degeneration, myofibre regeneration and atrophy. Insufficient DNA was available from the hair samples to establish whether this animal was affected by the myophosphorylase gene deletion myopathy (glycogen storage disease type IV). The cause of the disease was not established.

A Hereford heifer from Kumeu with crusty lesions of the coronet of all feet was provisionally diagnosed with *Dermatophilus* and treated with antibiotics. Despite a partial recovery, within weeks the lesions had extended up the legs and become more severe, and appeared on the brisket adjacent to where the hooves rested when the animal was in sternal recumbency. The animal became hypoproteinaemic and weak, recumbent and was eventually euthanased. Histopathologic examination of the skin revealed a severe dermatitis, with marked hyperkeratosis and superficial bacterial colonisation, ulceration and oedema. A skin culture yielded a heavy mixed growth of *Corynebacterium* spp, a moderate mixed growth of Gram-negative bacilli, and a scant growth of *Aspergillus fumigatus.* The animal tested positive to BVD antigen. The extensive and severe dermatitis is considered a reflection of immunosuppression related to presumed **persistent infection with BVD virus.**

Deer

A one-year-old red deer stag from Hawke's Bay was paralysed in the hindquarters. Serum copper concentrations were 2.9 µmol/l (normal 8-18.5) and liver copper 32 µmol/kg (normal 100-2000) confirming copper deficiency and suggesting **enzootic ataxia.**

Sheep

Every year on a sheep farm running Coopworths, a number of crossbred lambs have been born without wool – only a fine down. Many die from exposure shortly after birth but those that survive grow normally. As an iodine deficiency was suspected, a long-acting iodine injection was given to all pregnant ewes last winter. This reduced the number of perinatal deaths but about the same number of lambs continued to be born without wool. Tests for hairy shaker disease were negative and histology of skin sections from one lamb showed small empty primary follicles and reduced follicle numbers, characteristic of **hypotrichosis.** Recent publications have confirmed a genetic aetiology (autosomal recessive) that appears to have a variable phenotypic expression, from complete lack of wool to a symmetrical lack of wool on only parts of the body.

Hairy shaker disease (HSD) was implicated in abortions, perinatal deaths and the birth of hairy lambs on a Southland sheep farm. Tests on foetal spleens were positive. The farmer estimated that 75% of his ewe flock of 800 ewes had affected lambs. Congenital infection was also suspected as the cause of death of a small number of four-week-old lambs yarded with their mothers on an Otago farm. Affected lambs became recumbent and died shortly after first showing weakness and stiff limbs. Some also had diarrhoea. The brain of one showed a severe distension of the lateral ventricles and histologically a marked hydranencephaly. Sections of its distal ileum showed changes similar to those seen in cattle dying from acute mucosal disease. Possibly the CNS lesions were caused by HSD during gestation and were incidental, and the cause of death was the severe enteritis that may have resulted from superinfection of a persistently infected lamb with a homologous strain of HSDV. No more deaths occurred so samples were not available to test this hypothesis.

This year, deaths from **abomasal bloat** have been a problem on several farms in Otago where sometimes large numbers of lambs (six to eight weeks of age) were being artificially reared on whole milk or milk replacer. At necropsy enlarged abomasums were seen, often associated with rupture or gas filled walls. Bacteria resembling *Sarcina* sp (an anaerobic, packet-forming coccus) were often seen in histological sections of abomasal wall although attempts to culture this organism, which proliferates in an acidic, lactose-rich environment, were unsuccessful. In one case, sections of the abomasum also had large numbers of a large spore-forming Gram-positive bacillus resembling *Bacillus* sp, suggesting poor hygiene or inadequate storage of the milk may have contributed. Reduction of the amount provided at each feed appeared to effect a cure.

On an extensive Central Otago farm, each year after shearing small numbers of Merino hoggets develop a severe hind limb ataxia. No gross evidence of spinal cord damage was seen and liver copper was adequate but histology of the thoracic spinal cord revealed a severe, symmetric Wallerian degeneration of ventral and dorsal white matter tracts confirming a **degenerative spinal myelopathy.** The histological changes resembled a genetic ataxia reported in Australian Merinos by Harper et al (Australian Veterinary Journal 68, 357-8, 1991).

Investigation of diarrhoea and faecal staining around the rump of a mob of 600 one-year-old sheep from Hawke's Bay revealed 60 were ill and seven dead. The intestine had thick catarrhal membranes of necrotic debris, fibrin and bacteria lining the mucosa. Abundant necrotic debris filled gland crypts. Numerous microabscesses were observed in the liver. ***Salmonella* Hindmarsh** was cultured from the faeces of two animals.

A mob of 675 one-year-old sheep from the Wairarapa were yarded overnight. The sheep were in light condition on poor feed. Once released from the yards and returned to the field, 27 died within 12 hours. Mild enzootic pneumonia detected at postmortem and histological examination was not severe enough to cause death. Further investigation revealed browsed poroporo (*Solanum ariculare*) plants growing in the yards. **Poroporo toxicity** has previously been blamed for deaths in hungry sheep and hoggets. (Holloway I. Sudden death in hoggets while grazing a crop, Proceedings of Society of Sheep and Beef Cattle Veterinarians of the NZVA, 32, 31-5, 2002.)

Leg fractures and hind limb ataxia (sway back) were reported in a mob of lambs from the Wairarapa. Histopathology of bone and spinal cord revealed ostopenia and ventral neuropil chromatolysis. There was also significant pulmonary and gastrointestinal parasitism. Liver copper concentrations were 46 to 160 µmol/kg (normal 300-3000), confirming copper deficiency and **enzootic ataxia.**

Alpacas

Mycobacterium bovis was recovered from the lung of a Nelson area alpaca that had granulomatous lesions in the lung, liver and mediastinal lymph node. Acid-fast bacteria were plentiful and many were present in airway exudate.

A three-month-old Helensville cria running with its mother was found down, gasping for breath, and it died shortly after. A necropsy showed wet and consolidated lungs. Histology of the myocardium revealed patchy floccular myodegeneration, disorganisation, atrophy and regeneration of myofibres, and early fibrosis. A subacute interstitial pneumonia was consistent with cardiac failure. The animal had access to the dam's concentrate feed containing monensin, suggesting **ionophore toxicity.**

Pigs

Weaner piglets in a small 12-15 sow unit of Duroc pigs in the Te Awamutu region were not thriving, lost weight and appeared anaemic. Two of 40 died. At necropsy the piglets were anaemic and one also had multiple cutaneous and serosal haemorrhages, pulmonary oedema and enlargement, oedema and swelling of the mediastinal and mesenteric lymph nodes. Serum from the first and liver in the second piglet had low copper levels; serum copper 2.6 µmol/l (normal range 11-31) and liver 22 µmol/kg (normal range 80-130). Histology of tissues from the second piglet revealed lymph nodal and splenic lymphoid depletion and necrosis and mild reticular activation, splenic lymph nodal and pulmonary haemosiderosis and a diffuse interstitial pneumonia. Numerous loose rounded to ovoid organisms consistent with *Pneumocystis carinii* were present throughout the lung sample. No lesions suggestive of copper deficiency were evident in the aortic wall but the multisystemic haemosiderosis can be attributed to the anaemic effects of copper deficiency. ***Pneumocystis* pneumonia** is a rare disease in pigs. In humans, foals and dogs it occurs in association with immunodeficiency diseases. In one report in piglets, the farm had underlying enteric problems. The lymphoid tissue histology in this case implies an underlying immunogenic problem but its cause was not determined. The copper deficiency may have played a role but there may have been other underlying predisposing conditions on the farm.

A large number of 12-week-old pigs on an Otago pig farm showed illthrift and many had diarrhoea but there were no deaths. A necropsy showed a haemorrhagic mucosa with yellow necrotic material over the surface of some areas of the caecum and coiled colon. Histological examination of affected colon showed a marked goblet cell hyperplasia and shallow ulcerations of the superficial mucosa associated with a moderate neutrophil infiltration. A thick pseudomembrane composed of fibrin, neutrophils and bacterial colonies often covered the ulcerated mucosa. These findings are consistent with **swine dysentery,** caused by the spirochete *Serpulina hyodysenteriae.* Outbreaks are usually initiated by overcrowding and poor hygiene.

Horses

Three cases with similar signs were submitted from different areas of the country. Mature horses showed multiple nodules, variously described as wheals or hives, which were crusty and/or oozing. Distribution was mostly generalised. All cases showed similar histological features of superficial vesicular to spongiotic dermatitis with varying amounts of superficial crust and small numbers of free keratinocytes characteristic of acantholytic cells. The diagnosis was tentatively given as **spongiotic vesicular dermatitis** as described by Hargis et al (Veterinary Dermatology 12, 291-6, 2001). This morphological diagnosis is putatively associated with allergies including feed, vaccine and contact. The main differential is pemphigus foliaceous. Feedback from one case reported the horse recovered uneventfully when moved to a different region.

A mandibular mass present since birth was biopsied in a two-month-old foal from the Waikato. The histology showed immature fibro-osseous tissue with spicules of woven bone and clusters of osteoblasts. There were also widespread blood vessels of varying diameter and scattered siderocytes and inflammatory cells. The histology, history and location are typical of a relatively rare condition known as **juvenile equine ossifying fibroma** of the mandible. This is a benign non-neoplastic lesion of bone in young foals that is curable by hemi-mandibulectomy but otherwise tends to be recurrent (Veterinary Pathology 25, 415-9, 1988).

A slightly lame six-year-old Thoroughbred cross gelding from the Nelson region presented with ulcerative coronitis and sloughing of chestnuts and ergots. Faeces were soft and formless. Haematology revealed a mild eosinophilia. A mild hyperglobulinaemia with a marked elevation of gamma glutamyl transferase and glutamate dehydrogenase were the main changes evident on serum biochemistry. The horse deteriorated over a three-week period, the dermatitis becoming generalised and the diarrhoea severe. The horse was euthanased. At necropsy, the pancreas was pale, multinodular and firm. The liver had a lobular appearance and there was generalised thickening of the descending colon. Histologically, there was a marked, predominantly eosinophilic infiltrate of the pancreas, liver and colon. Dermatitis of the coronet was characterised by dense infiltrates of eosinophils associated with marked acanthosis and hyperkeratosis. The combination of clinical findings, haematology, serum biochemistry, gross changes and histopathology confirmed **multisystemic eosinophilic epitheliotropic disease.** The aetiology has not been established. (Schumacher J et al. Chronic Idiopathic Inflammatory Bowel Diseases of the Horse. Journal of Veterinary Internal Medicine 14, 258-65, 2000.)

A five-week-old Arabian foal from Marlborough had been poorly since birth with a chronic nasal discharge and a suppurative infection of one joint. A joint aspirate grew both *Enterococcus faecalis* and *Staphylococcus aureus.* Antibiotic treatment gave no improvement and the foal was destroyed. At necropsy it had an extensive pneumonia and suppurative arthritis, and *Enterococcus faecalis* and *Staphylococcus aureus* were isolated from the lung. Histologically, there was a chronic **proliferative bronchiolitis** and suppurative pneumonia, and adenoviral inclusions were present in the bronchiolar epithelium. Although not confirmed by DNA testing, it is likely that this foal had a severe underlying immunodeficiency.

An aged pony mare showed clinical signs typical of **Cushings syndrome,** including a long silky coat. Routine biochemistry showed no significant changes. The overnight dexamethasone suppression test showed no suppression by 19 hours, confirming the condition. The baseline cortisol was 114 mmol/l reaching 118 mmol/l at 19 hours (normal baseline cortisol of about 138 mmol/l reaches about 14 mmol/l at 19 hours).

A 13-year-old female donkey was showing unusual nervous signs. It appeared demented, was pressing into people, had a wobbly gait, was biting and started champing at its food. Haematology showed no significant abnormalities. Biochemistry showed marked increases in GGT (924 U/L), mild increases in GDH (111 U/L), hypoalbuminaemia (23 g/l) and hyperglobulinaemia (56 g/l). These are consistent with a cholestatic hepatopathy, resulting in **hepatic encephalopathy.** A biopsy is needed for a definitive diagnosis.

Dogs

A pink fleshy 5 x 8 mm nodule was removed surgically from the temporal limbus of the cornea of a five-year-old Border Collie. The histopathology of the nodule has been described as **nodular granulomatous episclerokeratitis,** nodular episcleritis or ocular nodular fasciitis. The exact aetiology is not known but it usually affects young to middle-aged Collie type dogs and develops at the limbus of the cornea. The nodules often regress with immunosuppressive therapy such as cortisone and/or azathioprine. (Paulsen ME et al. Nodular granulomatous episclerokeratitis in dogs: 19 cases (1973-1985). JAVMA, 190, 1581-7, 1987.)

Infectious canine hepatitis is rarely seen in the laboratory but two cases were seen within the same month, in unvaccinated dogs from Marlborough and North Canterbury. There were typical inclusions in hepatocytes, endothelial cells in hepatic vessels, glomeruli and, in one animal from which brain was submitted, there was a typical vasculitis with inclusions.

An entire litter of pups became sick between three and six days of age and died. A necropsy on two revealed red splotchy kidneys and pulmonary oedema. The kidneys, liver and lung had histological lesions typical of **herpesvirus** infection. This disease is uncommonly seen in the laboratory.

An emergency caesarean section was performed on an aborting bitch. Six puppies were dead and three alive. *Listeria monocytogenes* was cultured from the stomach contents of one dead foetus, indicating **listerial abortion.**

A several-week-old German Shepherd Bull Mastiff cross pup from an Auckland animal shelter was presented dead with a history of severe diarrhoea. The pup was representative of a serious problem of diarrhoea in puppies in the colony. Histopathological examination of the colon revealed an ulcerative colitis with numerous microabscesses in the superficial mucosa, many containing monomorphic bipolar staining cocco-bacilli. Culture of the colonic contents produced a pure growth of *Yersinia* spp, supporting a diagnosis of **yersiniosis.**

A 12-week-old Dalmatian puppy from Auckland was presented for routine vaccination and appeared in good health. Eight hours after vaccination with a commercial multivalent vaccine, the animal became lethargic and appeared unwell. The following day it had an elevated temperature, began vocalising and vomited. It died that night. At necropsy there was extensive petechiation of the carcase, free blood in the abdomen, and melaena. Histological examination of the liver revealed multifocal hepatocytic necrosis associated with large numbers of intranuclear basophilic inclusions in hepatocytes. A litter mate, released by the breeder at the same time, was unaffected. The cause of the **hepatitis** was not established, but its acute nature and severity suggests immunosuppression at the time of vaccination, or coincidental incubation of the disease.

Cats

A one-year-old Burmese cat showed signs of anorexia and lethargy for two weeks and appeared to have a sore back. Ataxia and progressive weakness led to ventral flexion of the head. There was a marked hypokalaemia of 2.5 mmol/l (reference range 4.2-5.6), marked elevation in CK of 18972 U/l (reference range 105-674), increased AST of 389 U/l (reference range 22-66) and ALT of 332 U/l (reference range 28-123). These changes are consistent with a **polymyopathy** reported in Burmese cats related to **hypokalaemia,** and believed to be familial or inherited.

Other

A two-month-old **rabbit** in poor condition with loose faeces was euthanased. At necropsy the liver was enlarged with multifocal to confluent 1-3 mm white foci almost obliterating normal parenchyma. Impression smears of the liver revealed large numbers of coccidia (*Eimeria stediae*) confirming a diagnosis of **biliary coccidiosis.**

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Quarterly report of investigations of suspected exotic diseases

Exotic disease investigations are managed and reported by MAF's National Centre for Disease Investigation (NCDI). The following is a summary of investigations of suspected exotic disease during the quarter from October to December 2003.

Vesicular diseases ruled out

A veterinarian noted severe mouth lesions in a three-year-old Friesian dairy cow. An exotic disease investigator decided the lesions were consistent with actinobacillosis (woody tongue). Their severity was probably the result of the delay between onset of clinical signs and the veterinary examination. No other cows on the property were affected. The cow responded to streptomycin injection.

Transmissible spongiform encephalopathy ruled out

The cases reported in this section are the more significant suspected transmissible spongiform encephalopathy investigations for the quarter.

A six-year-old crossbred Friesian cow had been ataxic, anorexic and circling for some days before euthanasia. On histopathological examination of the brain there was moderate perivascular and multifocal to random infiltrates of neutrophils, lymphocytes and plasma cells, admixed with increased numbers of glial cells (gliosis) amongst neuropil necrosis in the medulla oblongata and midbrain, consistent with a diagnosis of listeriosis.

Brains were examined from 56 cattle, older than two years and with a history of chronic neurological disease. All were dairy cattle between five and seven years old. Three had vascular inflammatory lesions typical of malignant catarrhal fever, one had necrotising lesions in the hindbrain typical of listeriosis, and two had no lesions. Bovine spongiform encephalopathy was ruled out in each case.

Numerous deer brains were examined, mostly from animals with illthrift. Despite no clinical history of neurological disease, two cases of malignant catarrhal fever, one case of listeriosis, one case of a hepatic encephalopathy, and an abscess in the frontal lobe were detected. Transmissible spongiform encephalopathy was ruled out in each case.

A seven-year-old Friesian heifer from Ruawai in Northland developed a head tilt, circling, ataxia and opisthotonos. She was treated with penicillin twice over four days with no response and was found dead in an electric fence on the fourth day. Histopathologic examination of the brain revealed perivascular mononuclear cuffs and microabscesses in the neuropil of the hindbrain with focal malacia. The findings are consistent with a diagnosis of listeriosis.

Exotic bovine theileriosis ruled out

A clinical pathologist reported a case of suspected acute theileriosis in a Jersey heifer. The animal had a moderate regenerative anaemia. A polymerase chain reaction (PCR) test was positive for *Theileria.* Further DNA sequencing tests identified the blood parasite as

Theileria buffeli, a benign endemic species from the *Theileria orientalis/buffeli* family. The heifer made a full recovery.

A veterinarian and a pathologist both reported an anaemic dairy cow with suspected haemoparasites. The cow had been in good body condition but pale and weak, with a thready pulse and dry scant faeces. The practitioner considered the cow unlikely to survive. Blood samples returned a haematocrit of 10% with a high level of intra-erythrocytic parasites. Theileriosis was presumptively diagnosed. PCR confirmed the infecting species as *T buffeli*. The cow responded well to antibiotic therapy, and no herd mates appeared affected. The farmer had recently purchased the cow. Ticks were often seen on the farm, and the farm had no tick treatment programme.

Post-weaning multisystemic wasting syndrome diagnosed

Post-weaning multisystemic wasting syndrome (PMWS) was diagnosed by a combination of clinical, histopathological and immunohistochemical evidence in pigs in four herds in Waikato and Auckland. Epidemiological investigations are continuing, and final confirmation by immunohistochemistry is pending for a further ten suspect places. It appears the disease may have been present on some farms for two to three years. All suspect and positive farms are under statutory movement control pending decisions on long term management. Detailed reports of investigations, including delimiting surveillance in the North Island and a survey to determine status of the South Island, will be published in due course.

Aujeszky's disease ruled out

An extensive piggery suffered a severe perinatal piglet mortality over a four- to six-week period. Approximately 200 piglets were either stillborn or died during the first week of life. NCDI requested samples for postmortem examination to gather baseline diagnostic information. Only two dead piglets were submitted as the mortality event abruptly ceased. Neither piglet had postmortem signs suggestive of infectious disease. The farmer changed his management system at that time and to date there has been no recurrence of the mortalities.

Equine herpesvirus abortion ruled out

Abortions and birth deformities were reported in miniature horses on a property in Nelson with 13 horses. In 2002 there had been two mid-term abortions. A full-term foal was stillborn with congenital deformities including an overshot jaw, malformed head and arthrogryposis. A fourth foal, born normally, died at three days. In 2003, during July a mare aborted at six months gestation, and in August another deformed foal was aborted at eight months gestation, prompting reporting to MAF. Later in August a further horse aborted, became septicaemic and was destroyed. The investigation considered infectious and non-infectious causes. Paired serology indicated prior exposure to equine herpesvirus (EHV) type 4 but not type 1, no significant rise in leptospirosis titres, and ruled out equine viral arteritis and equine infectious anaemia. Bacteriology on various foetal tissues returned mixed growth of Gram-negative rods, Gram-positive rods and cocci, but no *Salmonella* sp or other significant isolates. Histology on foetal tissues gave no indication of an inflammatory or necrotising process. The owner reported poor quality pasture, with emergence of many ornamental plants previously grown on the property, including Calla lily and English iris. The National Poisons Centre lists both as toxic plants. All parts of the Calla lily plant are poisonous, containing oxalates and other toxins. Signs of toxicity include irritation of mucous membranes, nausea, vomiting, diarrhoea, salivation, and rare systemic effects, caused by the corrosive nature of oxalic acid, and calcium binding effects of oxalate complexes. Birth defects and abortions are not specifically recorded. Calla lily was considered an unlikely cause of poisoning in this case. English iris bulbs and leaves are toxic. Ingestion by livestock has been recorded as causing bloody diarrhoea, and abortions and deaths have been reported in pigs. Hyperthermia has been reported in cattle. The toxins are glycosides: they include iridene, iricene and lysteric acid. Although no definitive diagnosis was established in this case, exotic infectious causes were ruled out. Plant toxicosis was considered a strong possibility.

Brucella canis ruled out

Brucella canis was ruled out by card test in a nine-year-old German Shepherd dog. The dog was initially presented with a urinary track infection (UTI). It subsequently collapsed and had a suppurative orchitis and epididymitis, possibly an extension of the UTI.

A dog was castrated as a treatment for orchitis. Testicular tissues with purulent exudates were submitted to a diagnostic laboratory with a request to test for *Brucella canis*. NCDI requested blood samples and the card agglutination test gave a negative result. Non-exotic bacterial orchitis was diagnosed.

A veterinary practitioner castrated an 18-month-old pit bull terrier because of severe orchitis. The dog was born in New Zealand and had not been used for stud work. Serum was requested and a card test for *Brucella canis* gave a negative result.

Spirometra sp cestode diagnosed in a cat

A Massey University parasitologist reported a previously unreported cestode belonging to the genus *Spirometra* in a feral cat trapped at a local rubbish disposal site. Import health standards guard against the introduction of cestodes with imported carnivores. Historic importation in primary or intermediate hosts (rainbow skinks, possums) appears possible. It is likely that this tapeworm has remained undetected for some time.

Newcastle disease and avian influenza ruled out

A ship heading for New Zealand encountered a heavy storm off the coast of Taiwan and many pigeons landed on the vessel looking for shelter. Five birds were still with the ship three weeks later on arrival in Nelson. The birds took flight when approached by a MAF Quarantine Service Officer. Six days later a pigeon taken to a local veterinarian was identified by leg bands as a Taiwanese racing pigeon. The bird was healthy, although hungry.

A MAF virologist examined, euthanased and autopsied the bird, collecting samples for histology and serology. The ship continued its journey via Bluff to Port Chalmers, Dunedin. Two days after arrival in Dunedin, two further Taiwanese racing pigeons were delivered to MAF Quarantine Service in Dunedin. The same MAF virologist examined and sampled these birds. The vessel was inspected again but no more pigeons were seen. Antemortem, postmortem and histological examination revealed no significant abnormalities. Serology was negative for Newcastle disease, avian influenza and avian adenovirus.

Babesiosis in ostriches ruled out

Sixteen birds from an ostrich farm in Hawke's Bay were condemned when sent for processing because of haemosiderosis causing carcass discoloration. Birds on the farm were blood sampled, and what were believed to be haemoparasites were seen. Light microscopy was unable to identify them definitively, but they were believed to be *Babesia* or *Theileria* species. The farm was visited and 20 birds sampled for transmission electron microscopy (TEM). Light microscopy confirmed that the structures were still present, but TEM demonstrated they were not haemoparasites. A *Theileria* and *Babesia* PCR test was negative. All birds on the farm were fed large quantities of onion in their diet and this may have resulted in the red blood cell abnormalities.

European foulbrood ruled out

European foulbrood (EFB) was ruled out by PCR and bacterial culture of specimens from a hive in Wellington with suspicious clinical signs. The beekeeper had reported a brood problem.

EFB was ruled out by PCR and bacterial culture of specimens from another hive with suspicious clinical signs. The hive was under treatment for *Varroa* mites.

A Hamilton beekeeper reported brood damage in a recently split hive. A frame examined had damage involving the periphery, where areas of black discoloration and some multiple egg clusters were evident. Samples of affected brood tested for EFB by culture and PCR were negative. It was concluded that 'chill brood' was the most likely cause of the brood damage.

Exotic bee mites ruled out

An AgriQuality apiaries officer reported a swarm of bees caught in a trap at the Port of Nelson. Testing of a swarm trap sample at National Plant Pest Reference Laboratory, Lincoln, ruled out the presence of varroa mite *Varroa destructor* and *Tropilaelaps clareae,* tracheal mite (*Acarapis woodi*) and small hive beetle (*Aethina tumida*). The bees were very dark black but wing length measurements confirmed they were not Africanised honeybees.

A local beekeeper collected and hived a swarm of bees discovered on the Port of Picton wharf, and reported the find to an AgriQuality apiaries officer. Sampling and testing for *Varroa destructor, Tropilaelaps,* tracheal mite and small hive beetle all gave negative results. Wing length measurements were not consistent with Africanised bees.

A swarm found on waste ground close to the Port of Picton during routine surveillance was collected. Samples tested for varroa mite, *Tropilaelaps,* tracheal mite and European foulbrood were all negative.

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MAF

Ministry of Agriculture and Forestry
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Animal Disease Emergency

To report suspected exotic diseases in animals, please phone toll free, all hours 0800 809 966

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