REVIEW OF DISEASES OF FARMED DEER

A review commissioned by DEEResearch for the New Zealand Deer Industry

Executive Summary

This review summarises the major endemic, emerging and exotic diseases of risk to farmed deer in New Zealand. A brief description of each is provided. The review concludes with a summary of the economic impact of disease on the deer industry, the role of preventive medicine programmes, and proposed priorities for research in the short to medium term, to provide information to enhance deer health, welfare and production, and protect market access into the future.

Over the last 30 years the capturing, domesticating and farming of deer has resulted in a steep learning curve to deal with health and diseases issues. There was no precedent elsewhere in the world, so much of the world's understanding of deer diseases has evolved from New Zealand. Wild deer appeared to be very healthy, but their capture and adaptation to a farming environment resulted in wastage due to misadventure, trauma and stress related diseases such as malignant catarrhal fever (MCF) and yersiniosis. Increased stocking density and grazing on pasture soon led to parasite problems, especially lungworm, and then in the late 1970s tuberculosis (Tb) became a major problem. Trace element deficiencies, especially copper, became apparent in the early 1980s and then Johne's disease (JD) first arose in the late 1980s. Concurrently, a wide range of other herd or individual animal diseases of a more sporadic nature, similar to those seen in other domesticated livestock, have been encountered.

Research and development over the last 25 years has led to major advances in understanding of deer health, and technology for controlling and preventing most diseases. This has been achieved by the combined efforts of researchers at Invermay, Wallaceville, Massey University, Otago University, diagnostic laboratories and MAF, as well as veterinary practitioners. However, significant clinical and subclinical losses due to disease are still encountered on a large proportion New Zealand deer farms.

The development of Tb tests has led to the ability to control and eradicate Tb on deer farms. The national deer Tb control scheme was developed as a partnership between the deer industry, veterinary practitioners and MAF. Improvements in management, plus the development of Yersiniavax, have provided technology that can significantly reduced the risks of serious outbreaks of yersiniosis. Anthelmintics have been developed, trialled for efficacy and registered for deer and these, plus diagnostics tests and good management, are able to effectively control lungworm and gastro-intestinal parasites. However, continued reliance on anthelmintics may not be sustainable in the long term. Anthelmintics are not acceptable for organic farming other than for treatment of clinical illness. Improved management systems and the introduction of the Farm QA scheme has probably reduced stress and trauma of deer on farms, and improvements in pastures and provision of shelter contribute to reducing stress-related disease such as MCF, yersiniosis, parasites and ryegrass staggers. However, despite the availability of technology for the control and prevention of many diseases and subclinical losses on deer farms, data suggests that significant preventable wastage still occurs. Thus, wider application of existing technology would significantly improve deer health, and welfare, and therefore deer farm profitability. Planned animal health programmes integrated with good management have the potential to maximise productivity and minimise losses.

Despite progress in many areas, there are still a number of disease and health issues that are a serious threat to the deer industry. Tb, which is a potentially serious zoonosis,

continues to be a threat in vector risk areas. The development of new diagnostic tests, vaccines and selection for resistance will provide additional tools to limit this risk. JD is potentially the most serious threat to the NZ deer industry and is the disease for which there are the fewest tools to control it. New diagnostic tests, an effective vaccine and epidemiological information on risk factors are the highest research priorities. There is also great potential for selection for increased resistance to JD, which may be related to Tb resistance. New developments in the field of genomics may also lead to the ability to select for resistance to a raft of other disease including yersiniosis, ryegrass staggers and parasites. Leptospirosis does not appear to cause major production losses in farmed deer on a national basis, but serious outbreaks have occurred on individual farms. It is a serious zoonosis and the risk for workers in the deer industry may be reduced by vaccinating deer, as it has in the dairy cattle and pig industries, providing a means of meeting looming occupation health and safety imperatives. Research has led to improved understanding of trace element requirements of deer, and data for interpreting, diagnosing, treating and preventing trace element problems should reduce losses and result in more cost-effective production. This review has highlighted that trauma is a significant but largely overlooked cause of loss on deer farms, and research into predisposing factors and preventive measures is warranted. MCF remains an enigma, but the incidence appears to be declining.

The introduction of exotic diseases remains a major threat to deer farming in New Zealand. While Foot-and-mouth disease is a threat to all livestock enterprises, chronic wasting disease (CWD) is probably the biggest threat to deer farming. It has severely damaged the deer industry in North America and all efforts must be taken to prevent its introduction to this country. The deer industry must limit the risk of introduction of exotic disease by maintaining conservative criteria for importation of live deer and genetic material.

The financial impact of a range of clinical and subclinical diseases, and mortalities on deer farms is difficult to assess because there are insufficient survey data of their prevalence, causes or production losses. However, estimates suggest that clinical disease and current disease control measures may cost the deer industry $24 million per annum currently, and it may be speculated that at least that amount may be lost due to loss of production from subclinical diseases, resulting in potential costs approaching $50 million. Thus, investment to improve technology for disease diagnosis, control and, in some cases, eradication, should yield significant dividends for the deer industry.

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1 INTRODUCTION AND SCOPE OF THE REVIEW

This review presents summaries on a) our current state of knowledge on the main infectious or transmissible diseases and deer health issues currently affecting farmed deer in New Zealand, and trauma; b) lists “emerging” diseases (those that have the potential to become more significant); and c) highlights exotic diseases that we believe present the greatest risk to New Zealand deer and the deer industry if introduced. The health issues selected for this review are those that have the potential to affect many animals within a herd, and therefore are of greatest economic significance. Review of individual animal conditions such as neurological disorders, tumours etc. are beyond the scope of this review. Each disease summary contains some information on the relative importance of that particular disease, based on the author’s opinions, followed by discussion of their overall importance, and an estimate of their costs, their management and control, including the potential to apply technology from other species. Planned animal health programmes are discussed along with the tools required to optimise the health and productivity of farmed deer. Current research into deer disease and related programmes will be discussed, as well as summary of current research capability and expertise in New Zealand. Finally a list of research priorities will be presented and discussed.

The authors have tried to be as objective as possible. However, it is appropriate for us to acknowledge that we are involved in the majority of the current work being undertaken, and are likely to have an interest in a number of the research projects recommended.

2 PRIMARY DEER HEALTH/DISEASE ISSUES

2.1 Current

These diseases are commonly diagnosed by veterinary practitioners and/or published in laboratory surveillance reports.

Bacterial

- Bovine tuberculosis
- Johne's disease (paratuberculosis) and *M. avium*
- Yersiniosis
- Leptospirosis
- Pasteurellosis
- Colibacillosis
- Clostridial infections
- Fusobacteriosis
- Brucellosis
- Miscellaneous

Viral

- Malignant catarrhal fever
- Parapox

Parasitic

- Lungworm
- GI nematodes
- *Elaphostrongylus cervi*
- Flukes
- Ticks
- Cryptosporidiosis

Fungal

- Facial eczema
- Ryegrass staggers

Trace element deficiencies/toxicities

- Cu deficiency/toxicity
- Se deficiency/toxicity
- Iodine deficiency

Miscellaneous/Misadventure

- Trauma/injury
- Velveting and post-anaesthetic deaths

2.2 Emerging

Viral

- Cervine herpesvirus 1 (CerHV-1)
- Rotavirus and Coronavirus

Protozoal

- Suspected Theileriosis (A potentially fatal disease of, as yet, unconfirmed cause, which also causes jaundice and carcass condemnation)

2.3 Potential diseases:

BVD
Neospora
Salmonella Brandenburg
Toxoplasmosis

2.4 Exotic diseases of importance:

Viral
- Foot and mouth disease
- Bluetongue and epizootic hemorrhagic disease
- Adenovirus?

Prion
- CWD/spongiform encephalopathy

Parasitic
- Parelaphostrongylosis
- Nasal bots and warbles
- Ticks
- Protozoa
- Eleaophora
- Filaria

2.5 Disease and mortality surveys

There is limited data on disease incidence on New Zealand deer farms. A 1980 national postal survey reported health problems on only 52% of farms. Malignant catarrhal fever (MCF), lungworm and yersiniosis were most commonly diagnosed. A Canterbury survey in 1981 indicated a death rate of 2.6% with MCF diagnosed in 43% of cases. Injury and post-capture myopathy caused 18% of deaths. Progeny loss rates from birth to weaning range 6-8% and 12-15% from adult and first-calving hinds, respectively. A recent survey indicated the annual mortality of 5.7% for weaners 3-15 months of age, 1.77% of adult hinds, and 2.56 % of stags 15 months and older.

The limited data on causes of perinatal mortality show few infectious diseases. The dystocia (difficult birth) rate is approximately 1%. Foetal loss rates average approximately 1% (range 0-2.5% between farms). Data in Table 1 is the only available on seasonal mortality rates.

Table 1. Mortality rates (per 100 deer by season) on commercial deer farms (from Audigé et al. 2001a).

	Season				
Class of deer	**Autumn**	**Winter**	**Spring**	**Summer**	**Annual**
3 – 15 month-old	2.41	2.62	0.42	0.14	5.87
Females > 15 months	0.23	0.67	0.58	0.32	1.77
Males > 15 months	0.38	0.83	0.82	0.57	2.60

Data in Table 2 is the most recent available information on diagnosed disease rates on commercial deer farms. This was collected during a 2-year longitudinal observational study of 16 commercial deer farms in the southern North Island.

Table 2. Mortalities and estimated mortality rates of hinds, stags, weaners (3-15 months) and calves, from Audigé et al. (2001a).

HINDS

Diagnosis	Number	%	Mortality rates (/100 hind-years)
Unconfirmed			
Fading	13	15.7	0.28
Ataxia	1	1.2	0.02
Pneumonia	1	1.2	0.02
Enteritis	2	2.4	0.04
Rumenitis	1	1.2	0.02
Dystocia ?	2	2.4	0.04
No diagnosis	25	30.1	0.53
Total unconfirmed:	45	54.2	0.96
Dystocia	8	9.6	0.17
MCF			
Acute	7	8.4	0.15
Chronic	1	1.2	0.02
Total MCF:	8	9.6	0.17
Misadventure			
Drowned	7	8.4	0.15
Injury	1	1.2	0.02
Broken neck	5	6.0	0.11
Broken leg	3	3.6	0.06
Other	1	1.2	0.02
Total misadventure:	17	20.5	0.36
Miscellaneous			
Enzootic ataxia	3	3.6	0.06
Liver cancer	1	1.2	0.02
Volvulus	1	1.2	0.02
Total misadventure:	5	6.0	0.11
Total mortalities:	83	100.0	
Mortality rates (/100 hinds x year)			1.77
Number of hind-years at risk			4683

STAGS

Diagnosis	Number	%	Mortality rates (/100 stag-years)
Unconfirmed			
Fading	5	7.9	0.20
Interstitial nephritis	1	1.6	0.04
Black-leg	1	1.6	0.04
Johnes disease	1	1.6	0.04
No diagnosis	19	30.2	0.77
Total unconfirmed:	27	42.9	1.10
MCF			
Acute	10	15.9	0.41
Chronic	3	4.8	0.12
Total MCF:	13	20.6	0.53
Misadventure			
Handling stress	2	3.2	0.08
Broken neck	3	4.8	0.12
Broken leg	2	3.2	0.08
Stag fight	2	3.2	0.08
Injury	3	4.8	0.12
Other	8	12.7	0.33
Total misadventure:	20	31.7	0.81
Miscellaneous			
Facial abscesses	2	3.2	0.08
Gun shot	1	1.6	0.04
Total miscellaneous:	3	4.8	0.12
Total mortalities:	63	100.0	
Mortality rates (/100 stags x year)			2.56
Number of stag-years at risk			2459

WEANERS

Diagnosis	Number	%	Mortality rates (/100 weaner-years)
Unconfirmed			
Yersiniosis ?	77	41.0	2.40
Lungworm	1	0.5	0.03
Lameness	3	1.6	0.09
Swollen joint	1	0.5	0.03
Fading	1	0.5	0.03
Septicaemia	1	0.5	0.03
Hepatitis	1	0.5	0.03
Enteritis	1	0.5	0.03
No diagnosis	26	13.8	0.81
Total unconfirmed:	112	59.6	3.50
Yersiniosis	35	18.6	1.09

CALVES

Diagnosis	Number	%
Hind-calf relationship		
Dystocia	21	22.1
Stillbirth	9	9.5
Small weak fawn	2	2.1
Overmothered	1	1.1
Mismothering	6	6.3
Fawn victimised	6	6.3
Ruptured stomach	3	3.2
Total :	48	50.5
Calf diseases		
Malformation (scoliosis)	1	1.1
Cryptosporidiosis	1	1.1

Misadventure			
Broken neck	15	8.0	0.47
Broken leg	7	3.7	0.22
Injury	1	0.5	0.03
Stress at weaning	1	0.5	0.03
Other	5	2.7	0.16
Total misadventure:	29	15.4	0.91
Osteochondrosis	9	4.8	0.28
MCF*	1	0.5	0.03
Miscellaneous			
Malformation	1	0.5	0.03
Blind	1	0.5	0.03
Total miscellaneous:	2	1.1	0.06
Total mortalities :	188	100.0	
Mortality rate (/100 weaner-years)			5.87
Number of weaner-years at risk			3202

Liver abscess (navel infection)	1	1.1
Total :	3	3.2
Direct management related		
Weather stress	2	2.1
Handling stress	1	1.1
Fawn lost	1	1.1
Lost through fence	8	8.4
Left behind	2	2.1
Misadventure	2	2.1
Broken neck	1	1.1
Broken leg	1	1.1
Broken back	1	1.1
Total :	19	20.0
Unconfirmed	25	26.3
Total diagnoses :	95	100.0

* This weaner died on August 28 1993 (8-9.5 months)
? Diagnosis based on circumstantial evidence
MCF = malignant catarrhal fever

Yersiniosis in weaners, wasting in hinds, malignant catarrhal fever of stags and misadventure in all classes were the most common diagnoses in that survey.

Although these surveys help direct research to understanding these syndromes, it must be noted that they do not give a true mortality incidence rate for all diseases of deer across the country.

Mortality surveys give little indication of the impact of subclinical disease on animal performance. It is difficult to establish good data of subclinical losses. However, it is commonly believed that economic losses associated with production losses due to subclinical disease such as parasitism or trace element deficiencies of other species, and treatment of non-fatal clinical cases of disease, exceeds losses associated with death *per se*.

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3 BACTERIAL DISEASES

3.1 Bovine tuberculosis

3.1.1 Introduction

Tuberculosis (Tb), caused by infection with *Mycobacterium bovis*, has been found in farmed and wild deer world-wide. In 1978 the first cases of Tb were diagnosed in farmed deer in Canterbury in the South Island of New Zealand. Despite early individual efforts to isolate infected herds and control it, Tb spread widely throughout New Zealand as deer farming was expanding rapidly, no movement restrictions applied to infected deer herds, deer (some infected) were live-captured from the wild in large numbers and the significance of wildlife vectors was underestimated. In 1985 a national voluntary Tb control programme for deer was implemented. This became compulsory in 1989.By March 2002 the point prevalence of Tb in infected farmed deer herds in areas containing infected wildlife and in areas with no infected wildlife was 5.02% and 0.17%, respectively.

3.1.2 Transmission

Tuberculosis may be spread from infected deer by several routes, depending on the primary location within the body of lesions that are shedding organisms. It may be shed directly via aerosols passing from animal to animal by the respiratory route Spread by the oropharyngeal route (saliva) may also occur from animals sharing the same food source, through grooming or from exposure to contaminated secretions, fomites or dust particles. It is also possible for Tb to spread from discharging external or internal abscesses from many parts of the body. The route of entry to the next animal may vary, depending on the nature of the disease in individual animals within a herd, population density, management, environment and feeding patterns of animals. Evidence suggests that most deer are infected via the oral or nasal routes and infection passes through the tonsil to head lymph nodes. Others may become infected via the respiratory route, producing a primary lesion in the lung.

3.1.3 Clinical signs

Otherwise healthy deer usually show little clinical evidence of tuberculosis, except in the terminal stages of disease. Many animals may have a single caseo-calcified lesion of a lymph node and show no overt clinical signs throughout their life. Animals with bronchial disease may cough. Severely affected animals lose weight in the terminal phase of the disease and may develop peripheral lymph node enlargement, the development of sinuses tracking to the skin surface from lymph nodes (especially around the throat), loss of condition, coughing and exercise intolerance. Clinical expression of disease is often associated with a reduced immune response, which is usually associated with stresses related to poor management. The interval between the development of overt clinical signs and death is usually short.

3.1.4 Pathology

Tuberculosis lesions are most commonly found in the lymph nodes of the head and thorax. The lesions may present a range of gross and histopathological (microscopic) appearances. The infection may produce liquid pus lesions sometimes surrounded by a thick wall with a cheese-like appearance. This is known technically as liquefactive necrosis with a zone of granulomatous reactivity containing giant cells, surrounded by a fibrous capsule. Fibrous capsules may be thin and the degree of calcification may vary between different species of deer. Acute abscessation, involving white blood cells (neutrophils) and large numbers of mycobacterial organisms, seen with an acid-fast staining Tb organisms, may be found in acutely infected animals in severe outbreaks of tuberculosis in deer herds.

3.1.5 Diagnosis

The mid-cervical skin test (MCST) is the primary screening test and has an individual animal sensitivity of approximately 85%, if applied carefully and interpreted conservatively. That is, the test will be positive in approximately 85% of infected individual deer. However, the ability of this test to detect infected **herds** depends on the number of infected animals, because determination of that status requires detection of only one Tb-infected animal. Thus, if two deer in a herd are infected, there is a 97.75% probability that one will be test positive if all deer are tested, and if there are 3 infected, the probability of detecting one increases to 99.6%. Thus, the MCST is an entirely adequate test for detecting infected herds, although it has limitations for individual animal use in a test and slaughter eradication programme, because, on average, it will be negative (false negative) in 15% of infected deer.

The MCST has limited specificity, that is, its ability to distinguish between *M bovis* and other similar organisms that the animal's immune system has been exposed to.

This is especially when deer are in an environment in which high levels of non- pathogenic environmental (saprophytic) mycobacteria are present. The specificity is about 99.5%, which means that if 1000 non-infected randomly selected deer are tested, five would be expected to be test positive (false positive). However, in practice, false positives occur at a higher incidence on some farms than others. This has required the introduction of follow-up (ancillary) tests, including the comparative cervical skin test (CCT) and blood test for Tb (BTB), to improve the precision of Tb diagnosis in deer. The BTB is a composite test that measures the reactivity of circulating lymphocytes and antibody levels in blood samples. It has consistently high levels of sensitivity (>90%) and specificity (>98%). A modified ELISA, which measures IgG1 antibodies, has been evaluated recently for tuberculosis diagnosis in deer. The test has high sensitivity (87%) for tuberculosis diagnosis, and acceptable levels of specificity (97%).

Surveillance during routine inspection of carcasses at Deer Slaughter Premises (DSP) is an extremely important method of diagnosis, and is the only method in some finishing herds. Histopathological examination of suspect lesions in a range of tissues is routinely used to confirm a diagnosis of tuberculosis *post mortem*, usually from specimens collected from DSPs. Culture and/or polymerase chain reaction (PCR, a DNA test) are used to confirm *M. bovis* infection in tissue from suspect lesions. Unfortunately there seems to be a lack of knowledge or employment of the PCR by DSP veterinarians and inspectors, thus limiting the opportunity to get a rapid diagnosis, and therefore confirmation of status of the herd in question. Strain typing of *M. bovis* is performed using restriction enzyme analysis, or other DNA-based techniques.

3.1.6 Treatment

Treatment is impractical for Tb in farmed ruminants. Indeed, it is contra-indicated because treatment does not necessarily ensure the organism is eliminated from an animal. The organism may merely be suppressed, and re-appear later.

3.1.7 Control

The control and eradication of Tb in farmed deer is strongly influenced by the presence or absence of wildlife vectors of tuberculosis. In the absence of infected wildlife, the most effective strategy for eradication is to use a comprehensive test-and-slaughter policy, surveillance of animals sent for slaughter and rigorous control of the movement of livestock from infected properties. In addition, herd management strategies aimed a minimising risk of further spread, and of identifying test negative infected deer (False negative) will accelerate eradication from a herd. Herd depopulation may be invoked if there is a severe outbreak and the prevalence of infection is above 40%. However, the currently available diagnostic tests for tuberculosis allow eradication from infected herds cost-effectively in a

short period if managed properly, using the available test protocols, combined with selective slaughter.

The presence of infected wildlife vectors, brush-tailed possums (*Trichosurus vulpecula*) and ferrets (*Mustella furo*), in areas known as "vector-risk areas", makes national eradication impossible at present. Indeed, the relative risk of infection of deer herds in vector-risk areas is 14 times that in vector-free areas. However, Tb can be eradicated from individual infected herds in vector risk areas provided the appropriate combination of vector control, testing and management practices are implemented. The problem in these situations is that if continued vector control is not practised, the risk of re-infection is high. Reduction of wildlife vector numbers may greatly assist in reducing the number of herd breakdowns. Vaccination of deer, although demonstrated to be efficacious, may interfere with routine diagnostic methods and compromise disease surveillance programmes. The selection of deer (and cattle) for increased resistance, and the culling of highly susceptible animals may also assist in control of Tb.

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3.2 Johne's disease and *Mycobacterium avium* infection

3.2.1 Introduction

Since the mid 1980s Johne's disease (JD) or paratuberculosis, caused by *Mycobacterium paratuberculosis* (*M. ptb*), has emerged as a problem on deer farms in the United Kingdom, Germany, New Zealand, Canada, Ireland, USA, Argentina and France. In New Zealand the first confirmed case of JD in deer was reported in the mid-80s. Passive surveillance, principally by the examination of suspect 'tuberculous' lesions identified in deer slaughter plants has resulted in *M. ptb* being identified in over 600 farmed deer on 300 properties. The herd prevalence based on this information is ~6% (300/~5000), but the true prevalence of infection amongst New Zealand's 2.5 million deer is expected to be higher than this figure.

M. avium infection can occasionally cause a disease syndrome very similar to JD.

3.2.2 Transmission

JD is spread exclusively by the oro-faecal route from animals excreting *M. ptb* in faeces onto soil or pasture that other deer then ingest. Faecal contamination of water or udder teats may act as a source of infection for young animals. Infected dams may excrete bacteria in milk and infect the suckling offspring. Intrauterine transmission from dam to foetus has been demonstrated to occur in cattle, but whether this occurs in deer is not known. Neonatal animals are considered to be most at risk from infection, and adults appear to be more resistant. Thus it is believed that the most serious challenge is to young deer.

Farmed red deer in New Zealand have been shown to be susceptible to both sheep and cattle strains of *M. ptb*. Infected sheep and cattle herds may act as sources of infection for deer if cross-grazing or contamination of food or water occur. While JD occurs in wild

ruminant populations, limited opportunities for co-mixing between wild and farmed animals suggest that the risk from wild ruminant reservoirs is not of major significance.

M. avium is primarily carried by birds, which excrete large numbers of organisms in their faeces, contaminating water and food. The majority of deer are exposed to *M. avium* and develop subclinical infections, but a few develop clinical disease. It appears that certain strains of *M. avium* may be more pathogenic for deer and that concurrent stress may make them more susceptible to developing lesions and clinical disease, especially in young deer.

3.2.3 Clinical signs

Typically animals with JD may develop chronic scouring, weight loss and death. Two clinical syndromes have been recognised in farmed red deer:

- Sporadic cases in mixed age deer, with an incidence of 1 – 3% per annum;
- Outbreaks in 8 – 15 month old deer, that may involve up to 20% of a group.

Sporadic cases of clinical JD, characterised by chronic destruction and thickening of the intestinal wall (granulomatous enteritis), occur in all ages and classes of farmed deer. Affected animals typically lose weight over a period of a few months, and the majority develop continuous or intermittent diarrhoea. There is usually low morbidity (<1% of animals actually get sick) but high mortality (~100% of animals that get sick end up dying of the disease) with little or no response to symptomatic treatment. This is very similar to paratuberculosis in cattle and sheep.

Outbreaks of JD in young red deer, involving 5 - 20 % of the 8 – 15 month-old animals, have occurred on 5 to 10 farms a year for the last 6 years in New Zealand. The affected animals initially “fail to thrive”, stop growing and then rapidly start to lose weight and condition. They invariably develop diarrhoea and became soiled with green faecal material around the tail, hindquarters and hocks. In spring they only partially moult their winter coats and take on a patchy or “moth-eaten” appearance. The clinical disease has a course of a few weeks, and it appears that the younger the animal, the quicker the progression to emaciation and death. The differential diagnosis includes yersiniosis (in weaners in winter), abomasal parasitism, avian tuberculosis and chronic malignant catarrhal fever.

The prevalence of subclinical *M. ptb* infections in farmed deer is not known, but up to 10% of animals in some lines of apparently normal deer from infected properties have had macroscopic lesions in mesenteric lymph nodes at slaughter.

3.2.4 Pathology

Necropsy examination of JD cases typically reveals enlarged intestinal (jejunal and ileo-caecal) lymph nodes, often with white or cream caseous (cheesy) lesions. Unlike sheep and cattle, there may not be any gross thickening of the terminal small intestine (ileum), but there are often prominent lymphatic drainage vessels from the mid small intestine) (jejunum) to the adjacent lymph nodes. There is usually no fat in the tissues around the intestines (omentum), which is often oedematous and may be adherent to the affected jejunum and lymph nodes in severe cases. Lesions, which may grossly appear typical of bovine Tb may be found in the retropharyngeal lymph nodes of the head

Histopathological examination of lesions typically reveals extensive areas of invasion of affected lymph nodes by large white blood cells (macrophages), often with pus with foci of calcification and numerous small acid-fast organisms (stained *M. ptb)* present in the macrophages. The ileo-caecal valve may show loss of structure of the lining with mixed cellular infiltrate, and contain numerous acid-fast organisms..It appears that primary lesions may be found further up the ileum and/or jejunum in deer than in other ruminants.

Subclinical paratuberculosis infection in deer is often detected at slaughter and typically a single tuberculoid lesion is detected in the jejunal lymph node without any macroscopic evidence of enteric lesions. Occasionally there is a generalised inflammation of the lymph nodes (lymphadenitis) with normal lymph node constituents replaced by bizarre giant cells. This condition is sometimes misdiagnosed as a lymphoid neoplasm. Lesions associated with *M. avium* infections are very similar to those of JD.

3.2.5 Diagnosis

A trial conducted in New Zealand showed that, of the tests available, the most sensitive and specific serological test for confirming a diagnosis of clinical JD in red deer is the agar gel immuno-diffusion (AGID or GD) test. However, none of the currently available serological or white blood cell-based (cell-mediated) immunological tests is sufficiently sensitive or specific for detecting subclinical *M. ptb* infections in deer to be useful for control of JD on infected properties. Serological tests suffer from lack of sensitivity because antibody is only produced late in the disease. Cell-mediated tests, such as the intra-dermal skin test, the lymphocyte transformation test, suffer from poor specificity because of widespread sensitisation of deer exposed to closely related *M. avium* complex organisms. Further, none of the currently available antigens are specific enough to differentiate between them.

Faecal smears, culture or PCR may be used to detect infected individuals. Bulk faecal cultures, whereby faecal pellets from 50 – 100 animals are blended together and cultured, have been used successfully to detect infected sheep flocks in Australia. If this technique is shown to be as sensitive for deer as sheep, it could be used to screen deer herds for infection in a cost-effective manner.

At commercial DSPs in New Zealand, the discovery of lesions in the intestinal (mesenteric or ileo-caecal) lymph nodes causes considerable problems because of the similarity between lesions due to *M. bovis*, *M. avium* and *M. ptb* when examined grossly and histopathologically. Further, there are a number of other incidental bacterial infections of lymph nodes, such as *Arcanobacterium pyogenes* and *Rhodococcus equi,* which can cause lesions that to the naked eye look like mycobacterial lesions. The differentiation of the three mycobacterial diseases is technically challenging and is made even more difficult by the requirement for a quick, definitive diagnosis to allow the status of the herd of origin to be determined without delay. The development and use of a PCR test has markedly sped up this process, but it seems this is not widely known or employed by DSP veterinarians or inspectors. In addition, the PCR test can equally well detect the bovine and ovine strains of *M. ptb*. However, culture remains the “gold standard” for differentiating *M. bovis*, *M. avium* and *M. ptb* because it is the most sensitive, and isolates can be characterised and typed to show strain differences in order to provide epidemiological information. Apart from distinguishing them from bovine Tb, it is extremely important to differentiate avian Tb and JD because of the epidemiological implications and the different control and prevention measures required. The introduction of the liquid culture-based BACTEC system has increased the sensitivity and also sped up the process of culturing mycobacteria from DSP samples. Strain typing of *M. paratuberculosis* can be performed using restriction enzyme analysis (REA) or strain-specific probes (insertion sequences IS*900* and IS*1311*) and PCR tests.

3.2.6 Treatment

Treatment is impractical for Johne’s disease in farmed ruminants.

3.2.7 Prevention

There is currently no restriction on the movement of animals from JD infected herds and therefore it is “buyer beware”. If JD has never been diagnosed in a deer herd and the

farmer has no reason to suspect that his herd is infected, it would be wise to take all sensible precautions to prevent its introduction. This means selecting appropriate options which may include:

a) Keep a closed herd, avoid buying in animals and use AI to bring in new blood lines.
b) Only purchase animals from “low risk” herds.
c) Quarantine
d) Avoid grazing sheep or cattle on the deer farm unless they are known to come from flocks or herds that are low risk.

Farmers who are in this situation are advised to seek professional advice on risk analysis and management. A market assurance programme, which screens herds for infection, would provide a mechanism for assessing risk and provide a premium for replacements from low risk herds.

3.2.8 Control

Control options on infected deer farms are currently limited to culling affected stock, culling test-positive animals, changing from a breeding operation to a weaner finishing or velvet operation, or depopulation and restocking after two years. The hand-rearing of fawns (applied as a measure in dairy herds) in isolation is impractical for the majority of deer farmers, but may be cost-effective with valuable stud stock. All these options should be subjected to professional advice and a rigorous cost/benefit analysis to determine the most economic and practical alternative.

It is hoped that vaccination may provide an alternative means of control if a vaccine can be developed that a) is efficacious, b) does not interfere with the national Tb control programme and c) does not cause carcass blemishes or downgrading. A live attenuated oil-adjuvanted vaccine has been used successfully to control JD in deer in the United Kingdom, but recent trials in New Zealand suggest that oil-adjuvanted vaccines are unlikely to be acceptable due to interference with Tb testing.

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3.3 Yersiniosis

3.3.1 Introduction

Yersiniosis, caused by *Yersinia pseudotuberculosis* (*Y. pstb*) was first diagnosed in outbreaks of diarrhoea among deer herds in 1978. The disease rapidly became regarded as one of the most serious and common infectious diseases of farmed deer in New Zealand, and remains one of the most prevalent causes of mortality in young deer. Yersiniosis is common in young farmed deer during their first autumn/winter or in newly captured deer. Predisposing factors include stressors such as weaning, transport, bad weather and underfeeding. The disease also occurs in farmed deer in Australia, North America and Europe. *Y. pstb* can cause zoonotic disease.

3.3.2 Transmission

The epidemiology of yersiniosis in New Zealand has been reviewed by Mackintosh (1992). Although at least six serotypes of *Y. pstb* exist world-wide, only serotypes I, II and III are found in New Zealand. *Y. pstb* is carried in the intestines by a wide range of wild and domestic animals including birds, rodents, rabbits, hares, pigs, cattle, sheep and household pets. Carrier animals tend to excrete more *Y. pstb* in the faeces at times of stress, especially in winter, and the organisms survive in the environment for long periods in cold, wet conditions. The majority of farmed deer are exposed to contaminated food and water during their first autumn/winter and experience subclinical infections. Outbreaks of disease occur when animals are stressed and experience a heavy challenge. Genetic factors also appear to influence susceptibility.

3.3.3 Clinical signs

Affected deer, which are usually four to eight months old, tend to be depressed, anorexic, to separate off from the herd and stand back from feed. Signs of green watery diarrhoea staining of the hocks and tail hairs are usually present. The diarrhoea generally turns dark or bloody. Elevated temperature (pyrexia) is frequently noted early in the course of the disease. Affected deer rapidly become dehydrated and weak. Acute cases may be found dead without any obvious clinical signs.

3.3.4 Pathology

Yersiniosis is characterised by an acute bloody inflammation of the intestines (haemorrhagic enteropathy), especially involving the lower small intestine (jejunum and terminal ileum). The caecum and colon are sometimes affected, and occasionally changes extend to the upper small intestine and abomasums (fourth stomach). Oedema of the outside (serosal) surface of the alimentary tract frequently occurs. The contents of the intestines are usually watery and bloody. In some cases, the intestinal wall is thickened and is covered by a false lining if tissue debris (fibrino-necrotic pseudomembrane). The mesenteric lymph nodes are frequently swollen, oedematous, haemorrhagic and necrotic (dead tissue). The small multiple abscess lesions of liver, spleen and mesenteric lymph nodes seen in other species are seldom seen in deer.

3.3.5 Diagnosis

A number of causes of rapid death in weaners during the autumn/winter period can be confused with yersiniosis, including malignant catarrhal fever, leptospirosis, salmonellosis, colibacillosis, pasteurellosis, stress enteropathy, stress/duodenal ulceration (and subsequent fungal infection), lungworm, fusobacterial abscesses of rumen/liver/lung as well as misadventure, poisoning and polioencephalomalacia. As the majority of healthy deer experience subclinical infection with *Y. pstb* and can carry and excrete the organism for some weeks or months, the organism may be isolated, with *Yersinia*-specific bacterial culture media, from faeces and intestinal contents of normal animals and of animals dying

from other diseases. In cases of clinical yersiniosis, *Y. pstb* organisms cross the wall of the intestine and spread to the mesenteric lymph nodes via the lymphatic drainage, and isolation of *Y. pstb* from the mesenteric lymph nodes should be possible in these cases. Diseased animals suffering from other conditions may also experience a recrudescence of *Y. pstb* infection when terminally ill, or overgrowth of *Y. pstb* after death.

A confirmed diagnosis of yersiniosis should include typical clinical signs, the isolation of *Y. pstb* from mesenteric lymph nodes, and a characteristic histopathological (microscopic) picture of pseudomembranous enteritis with colonies of the bacterium seen as Gram-negative stained coccobacilli.

3.3.6 Treatment

Intensive care treatment with antibiotics, such as tetracyclines or potentiated sulphonamides, and oral or parenteral fluids is usually effective if administered early in the course of the disease. Isolation and housing and other nursing care is essential to achieving the best outcome of treatment. During an outbreak, yarding should be undertaken for detection of new cases, and treatment. Although some believe that this is contra-indicated, as a source of stress that may exacerbate the outbreak, this has not been proven. Indeed, failure to do this has resulted in perpetuation of serious explosive outbreaks in some situations Treatment of all in-contact animals with oral or injectable antibiotic is essential in the face of an outbreak.

3.3.7 Control

The recommended strategy for prevention of yersiniosis in farmed deer in New Zealand involves the use of good management aimed at reducing the animal's response to stressful situations, such as conditioning deer to handling and interactions with people, optimal feeding, provision of shelter and minimising stress. These are combined with the use of a locally developed killed vaccine. The vaccine contains serotypes I, II and III together with diethylaminoethyl (DEAE) dextran adjuvant and has proved a valuable adjunct to good management for disease prevention. The vaccine was shown to have significant efficacy for protection against yersiniosis in experimental challenge trials and in field trials where it reduced morbidity and mortality by more than 65%. The vaccine has been used commonly in New Zealand since 1994 and has demonstrated good protection against outbreaks of disease in most circumstances. However, there have been some outbreaks despite vaccination, and this highlights the importance of management factors in reducing the risk of disease outbreaks. In those situations, research data suggests that mortalities would have been significantly worse had vaccination not been done., Vaccination does not prevent clinical disease in every individual, since occasional sporadic losses occur in vaccinated herds, due to factors peculiar to individuals within those herds.

3.3.8 References

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3.4 Leptospirosis

3.4.1 Introduction

Leptospirosis is caused by serovars (equivalent to strains) of the bacterium *Leptospira interrogans*. Throughout the world, a large number of serovars are each carried by a restricted range of maintenance hosts (those that get infected and maintain the infection in populations of that species), which are often rodents, but also include other wildlife species and domestic animals. Infection can "spill-over" into a wide range of mammalian species, including deer. Infection is often inapparent, but can also result in serious disease, including redwater (haemoglobinurea), hepatitis, jaundice and kidney disease (nephritis). There is suspicion that it may cause aborting in deer in NZ, but this has yet to be proven conclusively. Leptospirosis is also a potentially serious disease of humans (zoonosis).

In New Zealand, serovars *L. pomona*, *L. hardjo* and *L. copenhageni* have been isolated from clinically affected farmed red deer. Blood test results in one survey have been positive for other serovars, but the significance of those results is not yet certain.

3.4.2 Transmission

Maintenance hosts for particular leptospiral serovars develop chronic interstitial nephritis and shed leptospires in their urine for long periods of time. Accidental or spill-over hosts shed the organism in their urine for shorter periods. Red deer have been shown to shed leptospires in urine for as long as eight months. The organism can persist for long periods in the environment, especially under cool, wet conditions.

Animals are primarily infected by exposure to leptospires in urine, contaminated water or food and the organisms enter across mucous membranes, especially conjunctiva of the eye, and membranes of the mouth or nose, and through skin cuts or abrasions. Spread via the blood leads to localisation in various organs, especially the liver, kidneys and placenta.

3.4.3 Clinical signs

Young animals are generally more susceptible to clinical disease than adults. Clinical signs may range from sudden death in peracute cases to general malaise, pyrexia and loss of appetite (anorexia), as well as haemoglobinurea in some cases. Haemolytic disease (breakdown of red blood cells), severe nephritis, and jaundice have been observed in young red deer infected with *L. pomona*. In contrast, infections with *L. hardjo* are usually

subclinical. There is clinical evidence of one outbreak of redwater and jaundice associated with serovar *copenhageni.* Leptospirosis has been implicated in late-term abortions in farmed fallow deer in New Zealand, by association with blood test positive animals. However, this does not necessarily prove causation, since the positive blood results may have been coincidental. Proof of cause of abortion requires culture and other tests on aborted foetuses.

3.4.4 Pathology

Post-mortem signs are highly variable, and depend upon the severity of the infection. In farm animals. The acute form is characterised by anaemia, jaundice, haemoglobinuria and widespread haemorrhages. Acute nephritis may also occur and most animals that recover develop chronic interstitial nephritis. In DSPs, a relatively high proportion of deer have white spotted kidneys, and evidence suggests that many of these are due to leptospirosis.

3.4.5 Diagnosis

Diagnosis on clinical grounds is difficult because other diseases cause similar symptoms. A combination of dark ground microscopy of urine, culture of urine or kidneys, and fluorescent antibody techniques, especially on kidney tissue, is important for confirmation. In freshly dead deer, including aborted late-term foetuses, dark ground examination of aqueous humour may reveal leptospires. Serology on a herd basis, particularly of paired samples taken at an interval of fourteen days or more, can also help establish evidence of infection. The microscopic agglutination test is the most common serological test and is especially useful on a herd basis. However, serology on individuals is not completely reliable because some infected animals can have negligible titres.

3.4.6 Treatment

Streptomycin at 25 mg/kg daily for four days appears to be effective at treating clinically-affected animals in the early stage of disease, and eliminating the carrier state. Tetracyclines may also be used during the acute phase, but may not prevent kidney colonisation. Young deer suffering from a haemolytic crisis may be treated by blood transfusion. As foetal erythrocytes are less resistant to haemolysis, adult deer should be used as donors. Electrolyte therapy should be considered, especially if the kidneys are damaged.

3.4.7 Prevention

A number of practical measures can be taken to reduce the risk of exposure of deer to infection. Vaccination of all herd animals and replacement stock against the prevailing serovars is the most common. However, while there have been data published showing antibody response to a commercial vaccine used in cattle, and now licensed for use in deer, there has been no evidence presented that deer are indeed protected by the vaccine. Further, there have been no studies demonstrating the most effective vaccination regime, including the optimal frequency of boosters vaccinations.

Purchased animals may be treated with streptomycin before entry into a herd and then vaccinated. Carrier animals may be detected by examination or culture of urine, and treated. Wildlife sources of infection should be controlled or eliminated. Clean drinking water should be supplied and natural water sources fenced off. Deer should not be grazed with other livestock, especially cattle and pigs, unless known to be free of infection.

An important consideration for control and preventions is that pigs and cattle are the primary risks for serovar *pomona* and *hardjo*, respectively. To reduce risk, deer should not come into direct or indirect contact with unvaccinated cattle, or be exposed to effluent or run-off from pig or cattle farms.

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3.5 Pasteurellosis

3.5.1 Introduction

Sporadic outbreaks of pasteurellosis, an acute bacterial infection of the lung causing pneumonia, due to the organism *Pasteurella multocida* have been reported in farmed, park, zoo and wild deer worldwide. High mortality in fallow deer as a result of *Pasteurella septica* has also been reported in a park in the UK. Sporadic losses occur in farmed deer of all ages in New Zealand, but occasional outbreaks in recently weaned deer calves can cause multiple deaths.

3.5.2 Transmission

Pasteurellosis, also known as haemorrhagic septicaemia or shipping fever, is an infectious disease usually caused by *P. multocida*, which is thought to be part of the normal flora of the upper respiratory tract of a wide range of animals and has a world-wide distribution. Transmission is by aerosol, droplets and faeces. Disease appears to be precipitated by a number of stressors including transport, underfeeding, overcrowding, inter-current viral infections, inclement weather (especially high temperatures, heavy rain or strong winds), and high parasite burdens, especially lungworms. Similar disease may also be caused by *P. haemolytica* (now *Manhiemia haemolytica)* and *P. septica*.

3.5.3 Clinical signs

This infection causes acute disease. During outbreaks of pasteurellosis most deer are found dead or show severe depression and respiratory distress for less than 24 h before death. The animals may be panting with open mouth, heaving for breath, hold their heads low with the ears drooping, and show excess salivation. A soft cough may be observed. In the pneumonic form of the disease, acute death may occur, but signs of severe respiratory distress are often observed.

3.5.4 Pathology

The septicaemic (known commonly as blood poisoning) form of pasteurellosis results in acute haemorrhagic disease with engorged blood vessels and widespread spotted (petechial) haemorrhages on tissue surfaces. Gross post-mortem signs include enlarged congested lymph nodes, enlarged spleen, and haemorrhages in the endocardium, lungs, coronary fat and on the surface of the diaphragm. Swelling of the head has been reported

in fallow deer. Degeneration changes may be seen in the kidney capillaries. All of these signs are characteristic symptoms of endotoxic shock caused by bacterial toxins and damaged tissue.

The thoracic or respiratory syndrome results in pneumonia with haemorrhages in the lungs, trachea, and on occasions, the nasal mucosa. Cases in wapiti have shown inflamed nasal turbinates and trachea, retropharyngeal lymph node oedema, excess thoracic fluid containing fibrous clots, fibrinous adhesions in the thorax and red consolidated lungs.

3.5.5 Diagnosis

The diagnosis of pasteurellosis is based upon a combination of post-mortem signs, and the isolation and identification of the bacteria involved.

3.5.6 Treatment

Antibiotic treatment may be attempted if infection is detected before the animal becomes moribund, Oxytetracycline (either 10 mg/kg intravenously for three days, or 20 mg/kg of the long-acting preparation intramuscularly), trimethoprim/sulphonamide (3-5 ml/45 kg daily for three days), and penicillin (20,000-30,000 international units [IU] daily for three days) have been shown to be effective. In an outbreak situation, treatment of all in-contact animals may be worthwhile. Sulphamethazine in water, oxytetracycline in feed or long-acting injection may be used.

3.5.7 Control and prevention

Given that *P. multocida* is ubiquitous and carried by a wide range of animals in the respiratory tract, disease prevention should be aimed at improving management as well as minimising stressors and predisposing factors.

3.5.8 References

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3.6 Colibacillosis

3.6.1 Introduction

Colibacillosis is caused by *Escherichia coli*, a common pathogen of most species of domestic livestock, and is involved in a variety of clinical conditions, especially in young animals. In wapiti and red deer calves, infection with this bacterium may cause neonatal diarrhoea, meningitis and pneumonia, sometimes in association with other pathogens. The organism is ubiquitous and is a normal inhabitant of the gastrointestinal tract of all animals. Numerous serotypes are non-pathogenic, and among pathogenic serotypes some are specific for septicaemia and some for enteric (gut) disease. The organism is one of the first encountered by a new-born animal, and poor hygiene, lack of colostrum and overcrowding are important factors in development of neonatal diarrhoea. Septicaemic colibacillosis occurs when invasive strains of the bacterium enter the systemic circulation via the intestinal tract, mucosal surfaces or umbilical cord. Systemic spread may lead to localisation of the organism in almost any tissue, and arthritis and meningitis have been reported in cervids. There are no published reports of this disease in deer from New Zealand, but this is probably because it is such a common disease, that diagnosis does not warrant reporting. It is also probable that most cases of this disease in newborn deer on

New Zealand farms remain un-noticed and therefore undiagnosed. While it is likely to occur in deer at pasture it is more likely to occur in artificially reared calves.

3.6.2 Transmission

Infection is by the faecal-oral route. Overcrowding, poor hygiene and inadequate colostral protection are important predisposing factors.

3.6.3 Clinical signs

Clinical signs vary according to the age of the affected animal and the strain of the bacterium. These can range from sudden death due to endotoxic shock in very young animals, to acute severe watery diarrhoea, with or without haemorrhage, to nervous signs and collapse in cases of meningitis. A less severe form of enteritis, known as white scour, occurs in red deer in New Zealand and Australia, and is usually associated with bottle-feeding. This syndrome may be caused by a strain of the bacterium that is not enterotoxigenic. White scour can occur in slightly older calves up to three weeks of age and is characterised by a watery or pasty yellowish scour, occasionally streaked with blood. Affected animals are dull, anorexic, show signs of abdominal pain and become dehydrated.

3.6.4 Pathology

In peracute cases, few gross post-mortem signs are present. In cases of septic shock, cardiovascular collapse occurs with intravascular coagulation. Haemorrhage and necrotic changes may be found in the lungs, kidney or liver. In acute colibacillosis, subserosal and submucosal petechial haemorrhages occur in the gastro-enteric tract in association with gut inflammation and dehydration.

3.6.5 Diagnosis

Specific diagnosis requires the isolation and typing of the bacterium. In septicaemic cases, this will involve sampling heart blood and abdominal viscera. Identification of specific pathogens in cases of enteritis is complicated by the fact that one or more pathogens may be involved. Sudden death and diarrhoea in newborn wapiti and red deer calves have several potential causes which must be distinguished. These include trauma, maternal rejection and infection by other pathogens. In the case of elk calves, a number of pathogens have been implicated, including rotavirus, coronavirus, *Cryptosporidium* spp., *Clostridium perfringens* type D and *Salmonella* spp.

3.6.6 Treatment

Escherichia coli is generally susceptible to several different antibacterial agents including ampicillin, trimethoprim-sulphonamide, enrofloxacin and tetracyclines, but these are usually only successful if administered early in the course of the disease. Efficacy in the treatment of enteritis is the subject of debate, but such antibacterials are useful for cases of septicaemia if administered early. Appropriate electrolyte therapy must also be provided to control dehydration.

3.6.7 Control and prevention

Where farmed deer are managed in paddocks, and not subjected to intense husbandry practices, *E. coli* is unlikely to become a serious pathogen affecting multiple deer calves, although hand-raised animals are at risk. Vaccination of hinds with *E. coli* vaccines to promote higher levels of specific colostral antibodies may be of some benefit. Good husbandry and minimising stress of females around parturition may improve the colostral uptake by calves. When hand-raising calves, good hygiene and frequent small feeds of colostrum during the first 24 h of life are essential components of control of neonatal

enteritis including colibacillosis. In addition, the use of 20 ml-30 ml of colostrum in every feed for one to two weeks has a beneficial local immunoglobulin (IgA) effect in the gut.

3.6.8 References

Mackintosh, C.G., Haigh, J.C., Griffin, F. 2002. Bacterial diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 249-263.

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3.7 Clostridial diseases

3.7.1 Introduction

Clostridial organisms cause a wide range of diseases in domestic livestock and wildlife. *Clostridium* spp. have a world-wide distribution and are commonly found in soil, as well as in the gastrointestinal tract of healthy animals. The organisms are spore-forming anaerobes (living in the absence of oxygen) which are highly resistant to a wide range of environmental conditions. Disease is caused by the production of potent specific toxins excreted by the organisms (exotoxins).

Over the last thirty years, since the beginning of deer farming in New Zealand, Australia, North America and Europe, numerous sporadic cases of clostridial disease have been reported in deer. Most have been cases of pulpy kidney (*Clostridium perfringens* type D) in young red and fallow deer. Blackleg or malignant oedema associated with trauma, darting or injection sites, occurs in adult red stags and wapiti. These animals are prone to injury from fighting during the breeding season and often need intramuscular injections for chemical restraint during handling and velvet antler removal. Tetanus (*C. tetani*) has also been recorded in fallow bucks in Australia after castration with rubber rings, and is seen occasionally by veterinary practitioners on deer farms in New Zealand.

3.7.2 Pulpy Kidney (Enterotoxaemia)

Enterotoxaemia due to *C. perfringens* type D has been recorded in young red and fallow deer, but is not commonly diagnosed, possibly because it can be confused with other common causes of sudden death. The disease usually has a per-acute (very sudden) or acute course. Animals may be found dead, or may die in convulsions within 24 h of first showing signs of anorexia, diarrhoea and depression. Few post-mortem signs of disease are observed in per-acute cases. The carcass is usually in good condition, and an excess of clear straw-coloured fluid is often noted in the pericardial sac. Petechial haemorrhages may be found in the heart. In acute cases, the contents of the small intestine have a watery consistency, and areas of congestion of the abomasal and intestinal mucosa may be observed. Usually the liver is congested and the kidneys are soft and gelatinous. Confirmatory diagnosis requires identification of typical bacteria or toxin in intestinal contents.

Given the very rapid onset and short course of the disease, treatment is usually impractical.

3.7.3 Malignant oedema and blackleg

Malignant oedema, also known as gas gangrene, is an acute, rapidly fatal wound infection caused by several different members of the genus, including *C. septicum*, *C. chauvoei*, *C. perfringens*, *C. sordelli* and *C. novyi.* Blackleg, caused by *C. chauvoei*, develops when the spores which lodge in normal animals in skeletal muscle without symptoms, proliferate under anaerobic conditions such as in deep bruising or wounds, either of which may be associated with handling or fighting.

Malignant oedema and blackleg are usually associated with deep puncture wounds, but have also been observed after surgery, accidental wounding and parturition. In most cases, few signs are present other than peracute death. At necropsy, malignant oedema is characterised by gangrene of the skin and oedema of the subcutaneous and connective tissue in the surrounding areas. Affected wapiti had subcutaneous and intramuscular haemorrhage, with emphysema in the extremities, oedematous internal organs, tarry blood and bloody fluid exudate from body orifices. Swelling and crepitus (bubbly gas in tissues, felt when pressure is applied) of affected muscles are typical manifestations of blackleg.

Diagnosis requires the isolation of the organism from lesions or identification of the organism using a fluorescent antibody test.

In New Zealand, a number of cases resembling of severe septic oedema of the head and face of stags after velvet antler removal have occurred. Some were associated with local anaesthetic containing adrenalin, which reduces blood flow, possibly creating the anaerobic tissue environment that stimulates clostridial spores to multiply. *Clostridium septicum* has been isolated from at least one case.

Treatment is usually impractical because of the very rapid onset and short course of the disease.

3.7.4 Tetanus

Tetanus, caused by *C. tetani*, has been occasionally reported in red and fallow deer.

3.7.5 Prevention of clostridial diseases

Clostridial diseases are usually too acute to permit treatment of affected animals. Therefore, apart from good husbandry, vaccination is the only practical means of protecting animals at risk. Although not licensed for deer, multivalent clostridial vaccines are used in New Zealand, Australia, North America and the United Kingdom (UK). The effectiveness of these vaccines is difficult to evaluate. Generally, pregnant females are vaccinated annually in late gestation, and fawns are vaccinated at weaning. Adult stags are often boosted annually to optimise protection against clostridial complications of wounds or bruising associated with fighting during the breeding season. Cattle 7- or 8-way clostridial vaccines are commonly used in deer in Canada.

3.7.6 References

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3.8 Fusobacteriosis (necrobacillosis)

3.8.1 Introduction

Fusobacteriosis (necrobacillosis), caused by *Fusobacterium necrophorum*, commonly affects wild and farmed deer causing purulent necrotic lesions of the mouth, throat, umbilicus, feet, liver and lungs. The organism is a pleomorphic Gram-negative filamentous rod, a strict anaerobe with a world-wide distribution. Infection commonly causes foot abscesses in farmed red, wapiti and fallow deer, and mouth lesions in fallow deer in New

Zealand, Australia, Europe and North America, and has also been associated with die-offs of wapiti on winter feeding grounds in the USA, and white-tailed deer in Canada.

3.8.2 Transmission

Fusobacterium necrophorum is part of normal intestinal flora and is an opportunistic pathogen. The organism is present in faeces and survives well in soil and mud. Entry is usually through damaged skin or mucous membranes and infection often occurs in animals that are debilitated or suffering stress such as overcrowding. Abrasions or injuries of the oral mucosa, due to thistles, foreign objects or teething, can lead to infections of the mouth and throat, while cuts and abrasions to the feet or lower legs can lead to infected limbs, especially in cold, muddy conditions or in animals kept in feedlots. Mixed infections involving other pyogenic (pus-forming) organisms are common. Grain overload, which damages the lining of the stomachs, may predispose to fusobacteriosis of the wall of the rumen or reticulum, the first two stomachs. Spread via the blood can lead to abscesses in the liver, lungs and brain. Toxins produced by the bacterium kill cells in the surrounding tissue, disrupting the blood supply and providing the anaerobic conditions suitable for multiplication.

3.8.3 Clinical signs

Infections of the oral mucosa, which usually affect young fallow deer under three months of age, result in swelling of the head and/or tongue, submandibular oedema , salivation, fever and an inability to suckle or feed. Infections of feet and lower limbs lead to interdigital dermatitis, infected joints ligaments and tendons and lameness. Systemic spread to the liver and lungs leads to fever, loss of appetite, respiratory distress and death.

3.8.4 Pathology

Oral lesions result in destruction of tissue of the tongue, gums and the roof of the mouth. Liver and lung abscesses are pus-filled, foul smelling and often have a greenish tinge. Ulcerative inflammation of the rumen is often accompanied by peritonitis and abdominal adhesions. In cases of foot abscess, the digit above the claw is swollen, and there may be a sinus that discharges pus.

3.8.5 Diagnosis

The history and appearance of the lesions are fairly characteristic, but culture and isolation of the organism must be attempted to confirm the causative organism. As the bacterium is difficult to grow, a fluorescent antibody technique may be used for confirmation. An ELISA (blood test) may be used to detect antibodies to *F. necrophorum*.

3.8.6 Treatment

Early cases of foot abscess may be treated with injectable antibiotics together with cleansing and dressing of the affected foot. Hydrogen peroxide is useful for washing wounds and abscesses, and the application of oily antibiotic mastitis preparation has been successful in treating deep-seated abscesses in red deer feet. In severe or chronic cases, digital amputation may be required. Treatment of early cases of necrotic stomatitis (severe mouth lesions) involves extensive débridement of lesions and vigorous antibiotic therapy. High doses of penicillin, tetracyclines and sulphonamides have been used successfully. If an outbreak is occurring in a herd, preventive treatment of unaffected deer has proven to be useful. Concurrent vaccination against tetanus and clostridial diseases may be indicated. Once generalised lesions have developed, treatment is usually futile.

3.8.7 Control and Prevention

Prevention of necrobacillosis depends on the removal of predisposing factors such as sharp or protruding surfaces, which may lead to abrasion of the lower limbs. Access to thistles or other plants with prickles, which may injure the oral mucosa, should be prevented. Grain overload should be avoided, and in feedlots, clean dry bedding should be provided.

In the past, deer have been vaccinated against *F. necrophorum.* Veterinarians and deer farmers in New Zealand and Australia claim that vaccination of fallow does with a killed *F. necrophorum* vaccine significantly reduced the incidence of necrotic stomatitis in young fawns. Weaners that are at risk were vaccinated twice in the autumn and pregnant does given an annual booster three weeks prior to fawning. However, the vaccine is no longer available in New Zealand.

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3.9 Brucellosis

3.9.1 Introduction

Sporadic outbreaks of epididymitis (infection of the epididymus attached to the testis), due to *Brucella ovis* have been recorded in farmed red deer stags in New Zealand since 1996. Prior to then, *Brucella ovis* had been diagnosed only in sheep, in which it is quite common, and for which there is a national control and flock accreditation programme.

3.9.2 Transmission

Infection can occur via any mucous membrane including the reproductive tract, rectum, mouth and eye. It can be transmitted between stags in direct contact during the mating season and between infected rams and stags grazing in the same paddock. Transmission has not observed to occur by indirect contact with successive grazing of paddocks by infected and then uninfected stags, or by indirect contact through fences. Evidence suggests that the primary source of infection is infected rams, although the method of transmission from rams to stags remains unclear. Observations suggest that transmission is by direct homosexual activity and licking or sniffing of infected semen, as has been suggested for transmission in rams.

Research in deer has shown that hind can carry the infection in the vagina and pass it on to uninfected stags during mating.

3.9.3 Clinical signs

B. ovis causes epididymitis in red deer and, although there may not be any overt clinical signs of disease, there may be swelling and hardening of the epididymus in some affected animals, and infection is associated with reduced semen quality and lowered fertility or complete infertility. Experimental infection of hinds produced no clinical signs or abortion.

3.9.4 Diagnosis

Lesions may be detected by palpation, although some are very subtle and may not be obvious even when the testes are examined at necropsy or meat inspection. *B. ovis* may be recovered from affected testes, semen or accessory glands in the reproductive tract, and prepuce by culture.

Serological (blood) tests, including the complement fixation test (CFT), ELISA and gel diffusion (GD) tests provide good sensitivity and specificity early in infection. However, titres decline more rapidly over time than in rams, which means that blood tests become less reliable with time.

3.9.5 Treatment

Treatment of farmed deer is usually not attempted, as known infected animals are generally culled. Recent experimental evidence shows that a significant proportion of infected stags self-cure without treatment, over a 12 month period.

3.9.6 Control and eradication

Several options exist for control and eradication of this disease from infected deer herds. Choices depend on the nature of the farming operation and the occurrence of the disease.

This disease is only of direct consequence to breeding stags, causing infertility or sub-fertility. Therefore, if infection is found only in velvet-producing stags on a property, which are separate from breeding stags, then no action may be necessary other than to keep them separated. In that situation, it would be advisable to test blood samples and culture semen from breeding stags to confirm absence of infection prior to the breeding season. Infected stags should be kept away from hinds during the rut.

If infection is found in breeding stags, semen culture and/or blood test positive stags should not be used for mating. Those stags should be separated from others immediately, and either slaughtered, or left until the next breeding season for re-testing of blood and semen culture, as well as semen quality evaluation. This is because self-cure occurs in many animals. Ideally, if more than one infected stag is present on the farm, they should be grazed separately, with other deer that are not at risk, such as young hinds, and away from other stags. This could present management problems, but may permit salvage of a valuable stag if it is subsequently shown to self-cure. Thus, the option of retaining stags will depend on the value and genetic worth of the stag, and other management imperatives. This option may result in continuation of infection on the property if self-cure does not occur in an individual. Those animals that self-cure should only be mixed with uninfected stags after extensive testing over a prolonged period. Other stags on the property should also undergo regular blood testing to detect infection.

It is also essential that infected stags are kept away from direct contact with rams.

3.9.7 Prevention

Stags must not be grazed with infected rams, and ideally not with any ram. Rams on a deer farm should undergo a *B. ovis* testing programme, and any infected ram slaughtered immediately (rams do not self-cure).

It is essential to introduce stags only from *B. ovis*-free herds. This status is best achieved by blood testing all breeding stags or stags in the sale group on the property of origin, as a minimum, but preferably testing all stags on the property. To be absolutely sure, a second blood test should be done at a later date. If only one test can be performed, it should be done shortly after the rut, when it is believed most of the transmission occurs.

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3.10 Salmonellosis

3.10.1 Occurrence

Salmonella enterica enterica serovar Typhimurium (abbreviated as *S*. Typhimurium) was isolated from liver, lung, intestines and spleen of a three week old deer calf which died after depression and nervous signs, and with pale creamy intestinal contents. This organism was cultured from similar cases suggestive of septicaemia where the organism was isolated from lung, liver, intestine and thyroid. There have been further isolates of *S.* Typhimurium and *S.* Bovismorbificans from laboratory cultures but few from clinical cases. One clinical case of diarrhoea from which *S.* Typhimurium was isolated from spleen, liver, intestine and colon was reported in 1983. That report suggested that salmonellosis in deer in New Zealand was rare, with three isolates from clinical cases and 12 as incidental findings. *S.* Typhimurium has been isolated from healthy stags. In another study, culture for salmonella species from 3,810 faeces from apparently healthy deer from 122 farms were all negative.

There have been no reports of *S.* Typhimurium or *S.* Bovismorbificans associated with clinical disease in New Zealand since 1983. This could indicate that the disease is uncommon or isolations have remained unpublished. It is also possible that clinical diagnosis could be confused with yersiniosis if bacterial culture confirmation is not undertaken. One author (PRW) has confirmed one unreported case resembling yersiniosis and diagnosed by the farmer as such, from which *S.* Typhimurium and not *Yersinia pseudotuberculosis* was cultured.

In 1999, *S.* Brandenburg was identified in three hinds from a group of 20 that presented without gross pathology but with eosinophilic gastroenteritis suggestive of parasitism. More recently, *S.* Brandenburg has been isolated from three separate post-mortem cases involving adult stags with haemorrhagic enteritis and diarrhoea. One case had histological lesions of malignant catarrhal fever, so *S.* Brandenburg may have been a secondary infection.

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3.11 Other bacterial infections

Rhodococcus equi

This bacterium is commonly isolated from small lesions in the retropharyngeal lymph nodes at the back of the throat of cattle and deer. Infections with *Rhodococcus equi* do not appear to cause clinical disease in deer or cattle, but the lesions are a nuisance in slaughter plants because of their gross similarity to Tb lesions It is also suspected of causing cattle to react non-specifically to the Tb skin test.

Arcanobacterium infections

The bacterium *Arcanobacterium pyogenes* (formerly *Corynebacterium pypgenes* and latterly *Actinomyces pyogenes*), is isolated commonly from abscesses in various parts of the body. These may arise from penetrating wounds such as grass seeds in the mouth, external abrasions or lacerations, or other tissue damage. The organism is occasionally cultured from DSP samples of lymph node lesions that grossly resemble tuberculosis. This organism is commonly cultured from foot lesions in association with *Fusobacterium necrophorum,* and also from lung lesions secondary to foot problems.

Streptococcus species are also commonly isolated from abscesses in deer.

4 VIRAL DISEASES

4.1 Malignant catarrhal fever

4.1.1 Introduction

Malignant catarrhal fever is the most important viral diseases of farmed deer. The disease, as a clinical entity, is known from all continents and most countries. At least three types of the virus have been implicated in the disease.

The disease is caused by members of the gammaherpes viruses group. Alcelaphine herpes virus-1 (AHV-1), carried asymptomatically by the wildebeest in Africa has been diagnosed in disease outbreaks in zoos in North America. Ovine herpes virus-2 (OHV-2) is carried by sheep without showing disease. A third, as yet unnamed virus, has been detected in an outbreak of MCF in white-tailed deer.

There are at least 25 herpesviruses serologically related to AHV-1 that have been detected in a variety of ruminants, but only AHV-1, OHV-2 and a new virus in white-tailed deer appear to be pathogenic.

Ovine herpesvirus-2 is the virus of MCF in deer.

4.1.2 Transmission

OHV-2 is thought to be endemic in most sheep flocks worldwide and sheep-associated MCF (SA-MCF) occurs in deer where there is direct or indirect contact with sheep. Its epidemiology in deer is not yet well understood. Ewes experience a recrudescense of infection in late pregnancy and shed virus in lacrimal (eye), nasal, oral and vaginal secretions, infecting the next generation of lambs soon after birth. This may also explain the seasonal increase in cases in deer in late winter and early spring. The disease has also occurred in other circumstances, and direct contact is not necessarily required. Wind-borne infections have been reported and deer have become affected after being carried in a truck that had earlier been used for sheep transport..

Stress plays a major role in the development of MCF. It is well known from studies in the UK, Australia and New Zealand that the incidence of the disease rises in winter/spring, at a time when conditions are harsh and deer may be in poor condition and disease incidence rises in crowded conditions. MCF occurs more frequently on intensively managed properties than on extensive operations. Clinical disease due to MCF is rare in deer less than one year of age. Stags are three times more susceptible to MCF than hinds. Injections of dexamethasone have caused adult wildebeest to excrete virus, and a sika deer that had recovered from an initial infection, redeveloped clinical signs when injected with dexamethasone.

The disease only affects a small number of susceptible animals in a group, although occasionally a higher morbidity can occur. Some deer species, including Père David's, rusa, sika, axis and white-tailed deer are highly susceptible, while some, especially fallow deer, appear highly resistant to MCF.

4.1.3 Clinical signs

MCF can occur in forms ranging from peracute to chronic disease. The outcome is invariably fatal. In the peracute form, infected animals may die with no prior clinical signs. The most common manifestation of MCF in deer is a very rapid onset of bloody diarrhoea, dark stained urine, marked depression, and death within 48 hours. Chronic cases last from a few days up to 5 weeks and may develop the so-called 'head and eye' form of the disease, which is the type normally seen in cattle, and in the early stages is characterised by congestion of the mucous membranes, excessive salivation, lacrymation and nasal

discharge. Bilateral corneal opacity develops, there may be haemorrhage into the anterior chamber of the eye and as cases become more chronic the eyelids may swell and eventually become closed with a mat of catarrhal exudate. The muzzle, and often the vulva, develops a dry crusty appearance and erosions of the mucous membranes of the mouth are evident. Lymph nodes are swollen and the superficial ones can readily be palpated. Nervous signs are also sometimes seen, particularly an initial dullness followed by incoordination and hyperesthesia.

The clinical pathology most often involves an initial rise in white blood cells followed by a marked leucopenia (reduction of the number of circulating leucocytes, a type of white blood cell). An increase in immature circulating white blood cells also occurs, acute inflammatory proteins are raised and, if diarrhoea is present, the ensuing dehydration gives rise to increases in haemoconcentration (measured by packed cell volume and haemoglobin concentration).

4.1.4 Pathology

The lesions seen at post-mortem vary somewhat according to the clinical course of the disease. In peracute forms gross changes may be limited. In less rapidly fatal forms lesions may be seen throughout the entire gastrointestinal tract. These include necrotic erosions, congestion, oedema and frank blood in the intestinal lumen. Lymph nodes are usually swollen, and the mesenteric nodes may be grossly enlarged, with much oedematous fluid around them. Cross section of these and other nodes shows necrosis and inflammation.

Congestion and reddening of the mucous membranes may be seen throughout he respiratory tract and there may be extensive ulceration and necrosis. Both the bladder and kidney are often covered in haemorrhagic foci, and in the kidney characteristic lesions may be seen under the capsule and in the cortex. These appear as raised white foci 1-4 mm in diameter, sometimes surrounded by a thin red zone of haemorrhage. The vaginal mucosa may also contain haemorrhagic lesions. Other sites where lesions may be seen are the joints, in which swelling, reddening and excess fluid may be evident, and the brain, which may be congested.

The most characteristic histological lesion is a lymphocytic vasculitis affecting the arteries, arterioles, veins and venules. In peracute cases they are characterised by invasion of lymphoid cells into all levels of the vessels, and in the most severe cases they may even occlude the lumen. Vascular haemorrhages epithelial degeneration and lymphoid hyperplasia with invasion of lymphoid cells into non-lymphoid tissues also occur, especially in chronic cases. These infiltrations may be visible macroscopically in the kidney.

In chronic cases, there may be numerous nodules around major blood vessels, known as peri-arteritis nodosa. These may be found in many organs, but are most common around the blood vessels to the uterus and intestines, and in the kidney.

4.1.5 Diagnosis

Clinical and epidemiological history and post-mortem diagnosis are important means of confirming a case of MCF. There are two tests currently available for the diagnosis of clinical MCF. A competitive inhibition ELISA test has been developed which detects specific antibodies to an antigenic epitope, which is conserved across all viruses in this group. The sensitivity of this test is 95 to 100% and the specificity is 91 to 100% when the test is used to diagnose clinical cases of MCF. PCR tests are available, but they are specific to each virus within the group. PCR test for OHV-2 viral DNA has a sensitivity of 95-97% and a specificity of 94 to 100% when used on fresh tissues or peripheral blood lymphocytes.

4.1.6 Treatment and control

Treatment, even of chronic cases, is considered hopeless. There is no vaccine available. Prevention requires that deer do not have direct or indirect contact with sheep.

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4.2 Deer Parapox

4.2.1 Introduction

A new species-specific parapoxvirus infection of red deer was identified in New Zealand in 1985. The virus is a member of the parapox genus of the poxvirus family that has specific characteristics that distinguish it from other poxviruses and parapoxviruses. Restriction endonuclease analysis of viral DNA and PCR has shown it to be different from parapoxviruses of sheep or cattle.

Pox viruses in the parapox group have a worldwide distribution, but the specific parapoxvirus of deer has not been reported outside New Zealand. The three situations where severe infections and deaths have occurred are infection in stags in velvet, hinds

and calves close to calving, and recently stressed or transported animals. As all deer in New Zealand originated from other countries it is possible that the virus is also present elsewhere but has not been either seen or reported.

Transmission of parapoxvirus occurs by direct contact with active lesions on infected animals, or with exudates or scab material in the environment. It is probable that parapoxvirus infection in deer spreads in a similar manner. In severe outbreaks in New Zealand the presence of thistles in paddocks was considered to play a major role in the development of lesions on the limbs. The parapox viruses are extremely resistant to most environmental factors such as sunlight and cold, and can exist outside the body for long periods of time.

Note, this virus will affect people, causing scabby lesions. Gloves should be worn when handling infected Affected animals and velvet antler. The risk of transmission of infection to humans via the consumption of infected velvet antler is not known, but some methods of processing velvet antler do not destroy the virus. Affected velvet antler should be destroyed.

4.2.2 Clinical signs

In New Zealand the most common clinical signs in stags are scabs on the velvet antler, ears, muzzle, face and inside the lips. In more severe cases the lesions develop over widespread parts of the body and in one outbreak 21 of 55 recently captured animals were severely affected and died. The disease in young animals results in scabby lesions about the lips, and sometimes, blisters on the tongue and in the mouth. There may also be pox lesions on the coronary band, the line of tissue immediately above the horn of the hoof, and between the hooves. Sometimes these lesions may bleed. This form of the disease is similar to Foot and Mouth Disease, although there is rarely a fever, as seen in that disease. Lesions on the feet may spread up the leg causing lesions with crusty scabs that also may bleed if damaged. In females, it is common to observe scabby lesions about the vulva and perineum. Secondary bacterial infection of the damaged skin may result in severe infection and death. This form of the disease has been seen in deer as young as a few days of age, up to 6-8 months, and because of secondary bacterial infection, can be fatal. Occasionally hinds may show a mild form of the disease with scabs about the mouth and udder. There is evidence that deer can carry this disease without showing clinical signs.

4.2.3 Pathology

Lesions appear as severe proliferative dermatitis with extensive hair loss and scab formation. Removal of the scabs leaves a red raw surface. The lesions on antler velvet appear as multiple raised vesicopustules, which are like small blisters with pus.. The lesions on the lips are typical of a proliferative viral dermatitis and closely resemble those seen in sheep with orf.

4.2.4 Diagnosis

Diagnosis is based upon the clinical picture and the nature of the disease. It must be confirmed by histological examination as well as electron microscopy for identification of the typical appearance of the parapox virus in cells. DNA identification of the virus can be used for confirmation and a PCR that can distinguish among the four known members of the genus has been reported. Secondary bacterial invasion of lesions is likely, and several different organisms could be involved

4.2.5 Control

Generally severe–outbreaks of disease occur when the infection is introduced to a susceptible population. There are few cases of clinical disease in subsequent years, except when new animals are introduced, suggesting that the majority of deer experience subclinical infections in the endemic state. There are suggestions that control of prickly plants may help reduce the incidence of the clinical disease.
Experimental vaccination by scratching with parapoxvirus or orf virus on the inner thigh of young deer gave some protection against subsequent challenge with deer parapox, but it has not been used on commercial farms. In young animals affected prophylactic antibiotic treatment is justified to prevent secondary bacterial infection.

If velvet antlers are affected, management of an outbreak depends on the stage of antler growth. One report describes removal of antlers from those near the harvesting stage, and separation of those stags from unaffected herd-mates. Those that had affected velvet antler in the very early stage were separated and left. Their lesions resolved by the time the velvet was ready to harvest.

4.2.6 References

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5 PARASITIC DISEASES

5.1 Lungworm

5.1.1 Introduction

Lungworm infection, previously believed to be caused by *Dictyocaulus viviparus,* the cattle lungworm, has recently been proven to be caused by the deer-adapted *Dictyocaulus eckerti.* It is the most important parasitic disease of farmed deer in New Zealand, and is ubiquitous (present on every deer farm). Cattle lungworm, *D. viviparus*, has also been shown to affect deer, although it is less well adapted to red deer.

Young animals of 3-5 months of age in their first autumn are the most susceptible to infection because they have not yet developed immunity, and the environment in autumn favours survival of the parasite. Thus, explosive outbreaks can occur if this parasite is not adequately controlled. Red deer develop resistance with exposure and age and adult deer are somewhat resistant, although clinical disease can occur in situations of poor management where deer are severely stressed or nutritionally compromised.
D. eckerti has a direct life cycle. Adult worms live in the bronchial tree of the lung, where they mate and lay eggs. The eggs either hatch in the bronchi, or in the gastrointestinal tract after they have been coughed up the trachea and swallowed. Only first stage larvae are passed in the faeces. Under conditions of warmth and moisture the larvae develop on the ground to infective third stage larvae in 3-7 days. They migrate up the pasture and are eaten. They burrow through the intestinal wall and travel to the lung via the lymphatic drainage and pulmonary artery. They burrow through from the capillary bed into the airways and develop to an adult worm. The pre-patent period (time from ingestion to excretion of larvae in the faeces) is usually 23-24 days. The adults can continue to produce eggs for over 3 months. Cold weather arrests larval development on pasture, and they are susceptible to drying out in hot dry conditions. However, this is a hardy parasite and they can over-winter in cold climates, and can withstand a temperature of 4.5°C for a year.

5.1.2 Clinical signs

Clinical signs in deer include decreased appetite, gradual loss of condition and retarded growth rate, roughened coat and unexpected deaths in a herd. A soft bronchial cough may be heard, especially after exercise. Deer are usually severely affected by the time these signs are observed. Many outbreaks of lungworm are first seen by apparently sudden death.

5.1.3 Pathology

In deer that have died of acute heavy infections, the most obvious signs are “ropes” of adult worms that are usually occluding the trachea and major airways. There are usually areas of reddening of the lung (hyperaemia), emphysema and dark red areas of consolidation.

As lungworm infection is often associated poor management and loss of condition, other parasite infections, particularly of the gastrointestinal tract, may be evident, as well as other general signs of debility such as absence of fat on the carcass or the abdomen. The important difference between cattle and deer species infected with *Dictyocaulus* parasite is that deer do not develop the severe lesions of alveolar epithelialisation (thickening of the lining of the air sacs) seen in cattle. In natural infections most of the tissue reaction is seen close to the airways. In mild cases some degree of eosinophil (white blood cell) infiltration of the walls of the bronchi and bronchioles may be seen, and lymphoid follicles may develop adjacent to the bronchioles. More severe responses show irregular ulceration of bronchial and bronchiolar epithelium with eosinophils being evident in the lumen, lamina propria, the alveoli adjacent to the airways and the inter-lobular septa. Lymphoid follicles are also seen near the airways.

5.1.4 Diagnosis

Diagnosis in the live animal depends upon examination of faecal samples by the Baermann technique for the characteristic first stage larvae, which can counted. There is usually a good correlation between the faecal larval count and the size of the adult lungworm burden, although in some circumstances where susceptible animals have been recently challenged by massive numbers of infective larvae and there may be large numbers of immature lungworm present, which do not lay eggs, and therefore faecal larval counts may be low or absent.

5.1.5 Treatment, Control and Prevention

Young deer are most at risk from late summer (Feb) to early winter (June). The degree of risk depends on a number of factors including climate (temperature and moisture), stocking density, weaning practices, pasture type and length, grazing frequency, stress, and genotype. Wapiti deer appear to be more susceptible than red deer and take longer to develop a degree of resistance. Farmers should aim to prevent lungworms from limiting production by a combination appropriate pasture management, anticipating periods of risk and using preventative treatment.

Lungworm are easily controlled by treatment with appropriate anthelmintics at appropriate intervals either to reduce the risk to the animal and preferably, to break the life cycle. The latter can be achieved by treatment frequencies that prevent the immature lungworm from reaching maturity. This varies, depending -on the type of anthelmintic used and formulation. For example, pour-on formulations generally have a longer persistence than oral or injectable formulations of the same product.

Benzimidazoles (white drenches) are effective but have no persistent activity and should therefore be used at three-week intervals over periods of risk. Avermectins and moxidectin are highly effective and can be used at longer intervals according to manufacturer's recommendations, because they have varying degrees of persistent activity that prevent the establishment of infection for that period. Levamisole is not effective in deer.

Pastures heavily contaminated with larvae as a result of being grazed by untreated animals with lungworm burdens should be grazed by older deer, which are less susceptible, or spelled for a long period. Early anthelmintic treatment should prevent establishment of heavily contaminated pastures. It is important to maintain optimum nutrition and health in relation to other diseases, to maximise the young deer's immune competence, and therefore its ability to resist serious infestation. Recent research has indicated that pastures with high condensed tannin concentration, such as chicory, lotus and sulla, may have an important role to play in reducing the need for chemicals for parasite control in deer.

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5.2 Gastrointestinal parasites

5.2.1 Introduction

Most gastrointestinal (GI) nematode parasites of deer are closely related to those of other ruminants and occupy the same regions of the gastrointestinal tract as their equivalent species. The important genera of parasite affecting are *Ostertagia*-type in the family Trichostrongylidae. There are a number of species of gastrointestinal worms of sheep and cattle that infect deer, and there are a number of deer-specific worms in New Zealand.

Red and fallow deer are relatively resistant to GI parasites, but wapiti deer are quite susceptible and are prone to developing moderate to severe burdens of immature parasites in the lining of the abomasum, leading to reduced acid production, raised pH, poor protein digestion and chronic weight loss. Before the cause was clarified the clinical entity of severe chronic weight loss was referred to as the “fading elk syndrome” and is somewhat analogous to Type II Ostertagiasis of cattle. It has also been recognised in wapiti in North America.

These parasites have a direct life-cycle involving the shedding of eggs in the faeces, their hatching and development through larval stages to an infective larva and the ingestion of larvae while grazing. The larvae either continue to develop in the gut or may have a period of arrested development associated with winter. The larval stages of these parasites require high humidity and warmth in the microclimates in forage to complete their development.

5.2.2 Clinical signs

Acute heavy burdens in young deer, especially wapiti, show clinical signs similar to those of parasitic gastroenteritis in other stock. They include weight loss, or failure to thrive, staring coat, soft faeces or frank diarrhoea, and soiled tail and perineum. The syndrome akin to Type II Ostertagiasis, where immature larvae affect the abomasum, is characterised by anorexia, weight loss, weakness, dull hair coat, diarrhoea and low blood protein (hypoproteinaemia), sometimes resulting in oedema under the jaw, referred to as bottle jaw.

5.2.3 Pathology

Pathology is similar in deer to that in other ruminants. Generalised signs of debility or condition loss may be evident and the characteristic “Morocco leather” appearance of the lining of the abomasum has been seen in Ostertagia-type infections. Worm counts are usually modest, but burdens as high as 90,000 parasites have been reported in individual animals, with fourth stage larvae, which do most of the damage, being approximately 80% of the total.

5.2.4 Diagnosis

Diagnosis of GI parasitism in the live animal is difficult because the correlation between faecal egg counts and total worm number is not well researched or understood. This is further confounded because the pathogenic stage tends to be the immature stages in the abomasal lining before they produce eggs. Loss of condition and other clinical signs, combined with positive faecal samples, may be taken as positive in the absence of evidence of other specific conditions. Measurement of a stomach enzyme precursor which spills over into blood when there is damage to the stomach lining (plasma pepsinogen) has been used to confirm abomasal parasitism but its reliability in red deer or wapiti is yet to be established. The heavy winter coat of wapiti and red deer can readily mask the fact that they are in poor body condition, and suffering severe weight loss.

5.2.5 Treatment

Most modern anthelmintics appear to be relatively effective against adult Ostertagia-type nematodes in deer, although benzimidazoles appear to be less effective in wapiti than in red deer.

Moxidectin pour-on, given at the normal dose rate of 500 µg/kg, is highly effective against all stages of nematodes tested, but there is evidence that it is necessary to double the dose rate for ivermectin pour-on to 1000 µg/kg for full efficacy against the inhibited stages of Ostertagia-type parasites in the lining of the abomasum. Similarly oral ivermectin used at the standard cattle dose of 200 µg/kg was only 84% effective against inhibited abomasal larvae and it is recommended to double the dose rate for GI parasite control.
The duration of effectiveness has also been compared among products. Moxidectin pour-on has been shown to persist for 5-6 weeks, while pour-on ivermectin persisted for 3-4 weeks.

5.2.6 Control

Control measures involve good husbandry, and the principles described above for lungworm apply to gastrointestinal parasites. Indeed, in most situations, parasite control aimed at lungworm will control gastrointestinal parasites concurrently. Where these parasites have been shown to be a problem regular faecal sampling and anthelmintic treatment should be combined with pasture management to ensure that the animals have adequate nutrition. The use of pasture containing plants high in dietary tannins has recently been investigated as a means of reducing intestinal worm burdens and shows promise.

Because of their increased susceptibility to parasites, it is advisable to graze wapiti on longer pastures and to avoid grazing them with red deer. Unlike adult red deer that often need little or no anthelmintic treatment, wapiti usually require regular anthelmintic treatment, often up to four times annually depending on conditions, to maintain their productivity and avoid problems.

5.2.7 References

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5.3 Elaphostrongylosis

5.3.1 Life cycle

Elaphostrongylus cervi, also known as the “tissue worm”, occurs in deer in many parts of Europe, ranging from Britain through Fennoscandia into Russia and the Far East. It was introduced into New Zealand with deer importations around 1900. It has an indirect life-cycle that involves gastropod intermediate hosts, including a variety of native and introduced slugs and snails. The adult worms reside principally in the connective tissue sheaths surrounding skeletal muscles of the shoulders, chest and abdomen of their cervid host, as well as the arachnoid and subdural spaces of the central nervous system. Gravid female worms lay their eggs into the lymphatic or venous systems. Eggs are carried to the lungs where they are trapped in the capillary bed. The first stage larvae (L1s) hatch, invade the air spaces, ascend the trachea and are swallowed and passed out in the faeces. The L1s penetrate the foot of a gastropod and develop through to third-stage larvae (L3s) at which point they are infective if the gastropod is ingested by the deer.

In deer the L3s leave the gastropod and penetrate the gastrointestinal wall and migrate up the nerves to the level of the spinal cord. They undergo two moults and then descend down nerves to the musclular tissues or remain in the CNS.

Other than red deer and wapiti, *E. cervi* has been reported in sika deer (*Cervus nippon)*, roe deer (*Capreolus capreolus)* and experimentally in mule deer. A closely related species, *E. rangiferi,* occurs in reindeer Scandinavia.

A survey of faeces from deer farms in the early 1980's suggested infection with this parasite was present on about 35% of deer farms. There has been no recent study of this parasite, so its current prevalence on deer farms is unknown.

5.3.2 Clinical signs

In New Zealand, it is rare to find any clinical evidence of disease associated with *E. cervi* infections, which tend to be small in number. Heavy experimental infections have caused respiratory signs and occasionally CNS signs in red deer. In Europe, neurological signs are occasionally seen, and the principal clinical sign is an interstitial pneumonia. In Siberia, neurological signs are common in heavily infected maral deer, but light infections are subclinical, whereas the parasite appears to be less pathogenic for sika deer and most adult worms are found in the musculature.

5.3.3 Pathology

Heavy infections may cause lung lesions when eggs and larvae cause vascular congestion, collapsed alveoli, fibrosis and petechial haemorrhages. There are usually few gross signs of lesions associated with worms lying in the subarachnoid spaces and around the spinal cord. In the muscle tissues the worms lie like coiled strands of hair and early infections are very hard to see. Later, the immune response causes a build up of eosinophils, which cause greenish patches on the surface of the meat and is of concern to venison processors and exporters.

5.3.4 Diagnosis

Diagnosis is based on clinical signs and the finding of spiny-tailed L1 larvae in the faeces using a Baermann technique and microscopic examination. The larvae can be distinguished from lungworm larvae by morphology of the tail. However, there are promising developments in the field of serological and DNA-based detection methods.

5.3.5 Treatment

Avermectins and benzimidazoles appear to temporarily stop egg-laying, but their efficacy for killing adult worms is doubtful. Repeated doses may help to prevent the establishment of infection by killing the migrating larval stages.

5.3.6 Control

Control of the spread of infections depends on preventing movement of infected animals into areas where suitable intermediate hosts exist and on the control of gastropod intermediate hosts themselves.

5.3.7 References

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5.4 Liver flukes

5.4.1 Life cycle

Fasciola hepatica, the common liver fluke of sheep and cattle, is the only fluke causing problems in New Zealand. *F. hepatica* has an indirect life-cycle, involving Limnaead aquatic snails. Wet areas such as swamps and particularly waterways with slowly moving water and frequently irrigated paddocks, are essential for snail survival and the movement of some of the larval stages, such as cercariae and miracidia. The eggs can over-winter, but are susceptible to desiccation. The movement of infected sheep, cattle and deer can introduce infection to new areas where there are suitable intermediate hosts.

5.4.2 Clinical signs

Red deer are somewhat tolerant of *F. hepatica* and usually exhibit few signs, even when pastured on the same ground as sheep that are dying of fluke induced disease. Fallow deer are also tolerant of infection with *F. hepatica* and rarely exhibit any clinical signs. In most situations the first indication to the farmer that their deer have liver fluke is a report from the DSP, noting their presence.

There is little other information on the effects of fluke infection upon farm productivity, but reports from Scotland suggest it can cause subclinical disease and reduced productivity.

5.4.3 Pathology

In red deer infected with *F. hepatica* liver damage is rarely severe. Red deer are easily infected experimentally, but development of the adult flukes is somewhat arrested compared with the severe fibrous reaction seen in sheep. The very heavy burdens seen in sheep and cattle are not seen in red deer. The main lesion is portal cirrhosis.

5.4.4 Diagnosis

In the living animal the most reliable diagnostic tool for diagnosis of mature flukes is examination of faeces for the presence of the characteristic operculate eggs, but it cannot detect early infections. A reliable ELISA test has been developed for the diagnosis of *F. hepatica* in sheep and cattle although the rise in antibody titre is not diagnostic until 6-8 weeks post infection.

5.4.5 Treatment and Control

Triclabendazole, at 10 mg/kg has been shown to be effective against all stages of *F. hepatica* in sheep cattle and goats and appears to be very effective in red deer. It is recommended to repeat the treatment in 8 weeks.

Control depends on effective treatment of infected animals and prevention of re-infection by fencing off wetland areas harbouring the infected snail intermediate hosts.

5.4.6 References

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5.5 Ticks

5.5.1 Distribution

Haemaphysalis longicornis is the only tick of domesticated animals in New Zealand. It affects all mammals including humans, horses, pets, wildlife, as well as deer, cattle and sheep. Until recently this parasite has been restricted to warmer parts New Zealand, mainly the east coast of the North Island, and north of Waikato, with a few in Nelson. However, over the past few years, there have been heavy tick infestations in the Waikato, and south as far as the Manawatu and Wairarapa. Recently ticks have been observed on deer and cattle in Otago and Southland, causing concern that they may become an established cause of production loss in those districts. However, because this tick is temperature sensitive, it is not known whether populations will thrive and multiply to a point where they will affect production in cooler district.

Global warming may have a very significant effect in expanding the geographic range of ticks on NZ farms.

5.5.2 Life Cycle

Haemaphysalis longicornis is a three-host tick, meaning that three stages of its life cycle must engorge on a series of hosts to complete the life cycle. Each of those stages can be on a different animal species. Adult ticks lay about 1000 eggs on the soil in mid summer. They hatch to larvae late summer-early autumn. Larvae migrate up pasture and attach to a passing host upon which they engorge lymph and blood. At this stage they are about 1-2 mm. After 3-5 days of engorgement, they drop from the host and return to pasture where they moult to become a nymph, which is relatively resistant to cold, and over-winters, dormant in the base of the sward. In the early spring, the nymphs, which are about 2-3mm, become active and re-attach to a host to engorge over 3-5 days, expanding to about 5mm, and then drop off. Nymphs moult to become adults, which are about 4mm long. They attach to a host early-mid summer, and engorge to about 10mm diameter over 3-5 days and then drop off to lay their eggs. A high proportion of each stage do not survive in the environment if it is too dry, hot or cold, or do not find a host. Thus annual differences in climate and various farm management practices have a significant influence on adult tick numbers on animals in the summer.

There is evidence that in the warmer parts on NZ the life cycle may be more rapid than12 months. Thus, it is not uncommon to find more than one stage of the tick on an animal at one time.

5.5.3 Clinical signs

Signs will depend on the number of ticks that have survived to adulthood during the preceding stages of the life cycle. In cooler climates where ticks are present, insufficient numbers may survive to become a deer health concern.

Usually, ticks will be observed on the animal only when in the adult stage, which when engorged with blood, are about the size of a small grape. However, larval and nymph stages also engorge on the host, but because they are considerably smaller, they are not so commonly seen. Because deer farmers often do not examine their deer closely, particularly around fawning when adult ticks are present, infestations may go unnoticed until severe losses occur. *Haemaphysalis longicornis* infestations can cause deaths in newborn fawn if present in large numbers. Up to 160 adult ticks have been observed on ears of a newborn fawn. Each tick consumes 0.75-1 ml of blood, meaning that the calf can rapidly become anaemic, weak and die of blood loss. It is likely that growth and production are inhibited, although the only trial measuring growth showed no effect. The tick also

causes direct severe damage to hides, which is not evident until after the tanning process, thus causing considerable wastage and lost opportunity to the leather industry. Tick infestation of velvet antler can cause severe scarring, and may predispose to parapoxvirus infection. This results in downgrading of velvet, and financial losses.

5.5.4 Treatment and Control

Tick control can be very challenging. The first factor is to establish the severity of infection on a property, remembering firstly that small burdens may not affect the animal, and secondly, that it is impossible to eradicate the tick once it is established. If only small numbers are present and the environment is marginal for ticks, no action may be indicated. Control methods include animal sprays, pour-on medications, impregnated ear tags, removal of ticks from pasture, pasture renovation or spelling, and pasture treatment. A topically applied 1% flumethrin solution has been shown to both be directly effective against the tick and to have a residual action of about three weeks on deer facing natural challenge. Animal treatments must be timed to coincide with when ticks are on the deer, and can be done for any stage of the tick. However, management difficulties arise with calving hinds when the adult stage is present, because that is when they are calving, and disturbance can cause losses. Further, stags cannot easily be handled during the rut.

Removal of ticks from pasture may be done by high density grazing animals (sheep, cattle or deer) on infested pastures when conditions are right for tick attachment, such as in warm humid weather, allowing attachment, and them removal of animals for a spray or pour-on to kill them. Ploughing pastures and re-grassing, or passing through a crop will reduce tick survival. Leaving pastures fallow for 12 months will mean that there is limited access to hosts, and this will break the life cycle, although if there are a lot of wildlife species that can sustain the ticks, and this may not be totally effective. Pastures with the best environment for ticks, such as long grass, reeds, other cover and scrub, should be targeted first. It is possible to gauge which paddocks are most heavily affected by dragging a towel across parts of each paddock when they are active, and counting the attached ticks.

Pasture spraying with insecticide or acaricide has been done commonly, but only kills ticks on the grass at the time. Residual effect appears limited. The effect of these chemicals on the environment, particularly insects such as bees, and the legality of using them when bees are active should be considered.

5.5.5 References

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5.6 Lice

Both biting and sucking lice, *Damalinia spp* and *Solenopotes burmiester,* respectively, infest deer. They are not commonly observed in numbers sufficient to cause clinical problems in otherwise healthy deer, but may be present in large numbers when management is poor and animals are under significant stress. They are difficult to detect on normal deer. The main sign of lice infestation is rubbing of the neck, usually with the hind feet, causing loss of hair. This is seen commonly in adult stags, but it is difficult to find lice on those animals, suggesting that the deer does not need to have a high burden before they feel irritation.

5.7 Cryptosporidiosis

5.7.1 Introduction

Cryptosporidiosis is caused by the protozoan parasite, *Cryptosporidium parvum,* and is believed to be one of the greatest infectious causes of diarrhoea and neonatal deaths of farmed deer in New Zealand, Scotland and Canada. Hand-reared deer appear to be particularly at risk, but severe outbreaks can also occur at pasture. Mortality rates of up to 25% have been recorded. Cryptosporidiosis also occurs in wide range of animals, including lambs and calves, and is a zoonosis.

5.7.2 Transmission

C. parvum appears to have little or no host specificity and is thought to be carried in the intestinal tract by most animals, including humans, at some stage. Rodents and domestic livestock, such as sheep, cattle and deer, are likely candidates for acting as carriers in the farming environment. Infected animals can excrete millions of oocytes per gram of faeces. Neonates are probably infected by ingesting oocytes from suckling around the contaminated perineum and teats or by nibbling on contaminated pasture plants at a young age. The incubation period is 2 - 5 days in most species. The parasite multiplies rapidly in the small intestine, where the various stages lie within the lining cells of the gut, just beneath the cell membrane.

The majority of animals experience only a mild subclinical infection, and they appear to be protected by the presence of antibodies that they acquired from the colostrum in the first 24 hours of life and secretory antibody (IgA) that is present in the milk may give further local protection in the intestines during the first few weeks of suckling. However, some animals are severely affected. This may be because they had little or no colostrum, the colostrum may have lacked sufficient antibodies, or the animal may have been exposed to a massive challenge that overwhelmed the natural protective mechanisms. Hand-reared animals are often orphans that may not have had sufficient colostrum in the first 24 hours of life. They are often kept as groups in pens that can become soiled with faeces very quickly and they tend to suck each other and inanimate objects in the pen.

Outbreaks of cryptosporidiosis in young deer at pasture are unusual and the following factors may have predisposed them to the disease: (a) poorly drained, wet, muddy paddocks, (b) high stocking density in fawning paddocks, (c) poorly settled hinds or interference around the time of fawning resulting in suboptimal colostral intake, (d) a build up of faecal contamination in a fawning paddock resulting in the late calves getting heavy exposure to a contaminated environment, (f) contamination (direct or indirect via run-off) of paddocks by infected carriers such as rodents, pigs, sheep or cattle, (g) intercurrent exposure to other organisms such as pathogenic strains of E. coli.

5.7.3 Clinical signs

Affected fawns are usually 1- 3 weeks of age and may be first seen wandering behind their peers, often with their tail up, and appearing pot-bellied. There may be an explosive liquid white/yellow diarrheoa, although this may not be obvious, and they lose condition rapidly over the next 24 hours. Dehydration, recumbency and death follow quickly without treatment. Diagnosis can be made on finding vast numbers of tiny (4-6 microns) oocysts in the faeces.

5.7.4 Pathology

At necropsy there is often a full stomach of milk and sometimes there is no evidence of scour. Lesions are typically found in the mid to lower small intestine (jejunum, ileum) and caecum/colon. There may be congestion and mucosal haemorrhages. The contents may vary from pasty grey to yellow or greenish custard. The mesenteric lymph nodes may be

enlarged. Microscopic examination typically shows atrophy of the villi of the jejunum and ileum, and under high power large numbers of the organism can be seen in the epithelial cells lining the small and large intestine, just under the cell wall so they appear to be attached to the outside of the cells.

5.7.5 Treatment and Control

Treatment of clinical cases is very difficult and frequently unsuccessful. Fluid therapy is essential to control dehydration. Some apparent success has been achieved by dosing with the drug totrazuril at 40 mg/kg, given orally as a 1.25% solution. Oral colostrum and intravenous infusion of deer serum may also assist recovery. Broad spectrum antibiotics may help to control secondary infection.

Prevention is aimed at minimising predisposing factors. This is especially true of bottle fed calves where the hygiene of equipment, the environment and handlers is of utmost importance. Strong bleach solutions appear most effective. In a farming situation every effort must be made to fawn the hinds in dry, clean paddocks at low stocking density and with minimal interference prior to and around the time of fawning.

It must be remembered that this is a zoonosis and it is sensible to wear gloves and protective clothing when handling calves with scours and to disinfect clothing and equipment.

5.7.6 References

Angus, K.W. 1994. Neonatal enteritis. In: Management and Diseases of Deer. Ed T.L. Alexander and D. Buxton. A Veterinary Deer Society Publication. (2nd Ed). Pp 136-140.

Hicks, J.D. 1994. A cryptosporidiosis outbreak in fawns. Deer Branch, NZVA, Proc. Course No. 11: 246-250.

5.8 Toxoplasmosis

Toxoplasma gondii is a ubiquitous protozoal organism infecting many species. Currently there is no known disease associated with this disease in farmed deer in New Zealand, although evidence exists that infection can occur. This organism is a cause of abortion in sheep and can infect humans, causing abortion. It is unlikely that transmission would occur if normal food handling and cooking standards are applied.

6 FUNGAL DISEASES

6.1 Facial eczema

6.1.1 Introduction

Facial eczema is a descriptive name for the photosensitisation that occurs in sheep, cattle, goats and deer due to liver damage caused by the ingestion of a toxin (sporidesmin) in spores produced by the fungus *Pithomyces chartarum*. This fungus grows on dead litter in the pasture under conditions of warmth and humidity, which occur in the late summer and autumn, especially in some districts in the North Island of New Zealand. Ingestion of this material causes acute damage to and blockage of the bile ducts resulting in liver damage, jaundice and photosensitive dermatitis due to spillover of photosensitive pigments into blood and tissues.. Fallow deer are the most susceptible species, but red and wapiti deer can also be affected if conditions are dangerous enough.

6.1.2 Clinical signs

The worst affected skin is in exposed or poorly haired areas, especially the ears, conjunctiva, tip of the tongue, nose, lips, perineal area and vulva. Ulcers on the muzzle and tongue have been reported in fallow deer as a result of excessive licking due to irritation of the muzzle caused by photosensitisation. The affected animals invariably seek shade and the skin irritation can lead to excessive rubbing of the muzzle, lower jaw, eyelids and ears. Some animals may appear blind. Mucous membranes, particularly of the conjunctiva of the eye appear yellow as a result of jaundice. Acutely affected animals are distressed and have an elevated respiration rate. In animals that recover from the acute photosensitivity, there is often a prolonged period of recovery dependant on regeneration of the unaffected areas of the liver. Repeated or severe chronic damage can cause permanent liver damage and affected animals will become debilitated, lose weight and may scour. Diagnosis is from clinical signs and elevation of the enzyme gamma glutamyltransferase (GGT) in the blood.

6.1.3 Pathology

In acute cases the liver is swollen and there may be general yellowing of the carcass. The liver usually has characteristic microscopic lesions. In chronic cases the liver is shrunken and hard. The regeneration of some areas of the liver can lead to abnormal shape.

6.1.4 Treatment and Control

Seriously affected animals should be euthanased. Mildly affected animals should be removed from dangerous pastures, kept in a dark shed or given adequate access to shade for 4-6 weeks and given supportive treatment with fluids, vitamins and good feeding to allow liver regeneration.

Preventive measures include: (a) Predicting dangerous periods and areas by doing spore counts regularly during the summer; (b) Make the pasture safe by spraying them at critical times with fungicides; (c) Administration of zinc boluses (unproven in deer).; (d) Selecting for resistant stock; (e) Pasture and stock management to minimize dead litter in pastures and adjusting grazing so the deer are not forced to graze pastures too low.

6.1.5 References

Mortimer, P.H. 1984. Facial eczema in red and fallow deer. Deer Branch, NZVA, Proc. Course No. 1: 59-63.

Smith, B.L. 1993. Health management for farmed fallow deer in New Zealand. Proc.of the First World Forum on Fallow Deer Farming. Mudgee, NSW, Australia. Ed. G.W. Asher. Pp 87-98.

6.2 Ryegrass staggers

6.2.1 Introduction

Ryegrass staggers (RGS) is a nervous disease of deer, sheep, cattle, alpaca and horses. This disease is caused by the presence of toxins in perennial ryegrass (*Lolium perens*) produced at certain times by endophytic fungi (*Acremonium loliae*) growing inside the plant. The fungal spores are present in the seed and after the seed germinates and the grass grows, the fungus grows up inside the plant and into the seed head, where it produces spores to infect the next generation of plants. The toxins, called lolitrems, are absorbed from the plant material when it is ingested and cause brain damage resulting in tremors and staggering, hence the name.

6.2.2 Clinical signs

There is a wide range of susceptibility between species and also between individuals within species. Red and fallow deer appear only moderately susceptible to RGS, while wapiti are highly susceptible. Often the signs are not very noticeable when the animals are at rest or quietly grazing at pasture. There may be a slight head tremor or trembling of the muscles of the neck, shoulder or back. However, if the animals are stressed or put under pressure, during yarding for example, the tremors and shaking become more exaggerated and severely affected animals can fall over and thrash or have convulsions. They may stand with a very stiff legged stance and shake or nod. Such severely affected individuals can die of shock or haemorrhagic stress enteropathy. Some individuals have also died of misadventure such as drowning or getting caught in electric fences.

6.2.3 Pathology

There are characteristic “torpedo-like” lesions seen microscopically in histological section in of the cerebellum.

6.2.4 Treatment and Control

There is no specific pharmacological treatment for RGS. The most effective treatment is to quietly move the animals off the dangerous pastures and either graze them on alternative safe pasture (non-endophyte ryegrass or non-ryegrass pasture such as red clover, fescue, brassicas etc) or hold them in a yard or pen and feed them on lucerne hay and concentrates. It can take 1 – 3 weeks for the signs to abate. Ensure that affected animals are kept in a safe environment where they are unlikely to fall down banks, drown in open ponds or get trapped in electric fences. Continued or repeated exposure to toxic pasture can lead to some individuals developing permanent chronic brain damage and tremors.

Prevention is dependant on developing safe pastures by using ryegrass cultivars that have been treated with fungicide or have been selected for nil endophyte or for safe endophytes, or alternatively using pastures that are free of ryegrass. Supplementary feeding with hay, silage or grain will reduce intake of the toxin. It would also be sensible to select animals for resistance to RGS.

6.2.5 References

Mackintosh, C.G., Orr, M.B., Gallagher, R.T., Harvey, I.C. 1982. Ryegrass staggers in Canadian wapiti deer. *New Zealand Veterinary Journal* 30: 106-107.

Smith, B.L. 1993. Health management for farmed fallow deer in New Zealand. Proc.of the First World Forum on Fallow Deer Farming. Mudgee, NSW, Australia. Ed. G.W. Asher. Pp 87-98.

7 TRACE ELEMENT DEFICIENCIES/TOXICITIES

This topic is the subject of a separate review for DEEResearch by Grace and Wilson. The reader is referred to that report.

8 OTHER

8.1 Post-anaesthetic deaths

8.1.1 Introduction

Post velveting deaths in stags or “stag death” is a syndrome first reported in the late 1980s, which refers to the death of stags in the 3 - 48 hour period after anaesthesia/sedation with xylazine. There is an incidence of approximately 1-2 per 1000 animals anaesthetized. Typically the stag appears to be normal immediately after the anaesthetic, but is found dead the next day.

8.1.2 Aetiology

Some deaths of stags in the post-velveting period may be due to a variety of causes, including regurgitation and inhalation pneumonia, blood loss and hypo-volaemic shock, hyperthermia, twisted bowel, injuries such as broken ribs from fighting or concurrent disease such as MCF or pneumonia. However, the “stag death” syndrome, where all these other causes have been ruled out, is thought to be due to a non-specific hypersensitivity (delayed allergic) reaction. The lungs have a very high concentration of tissue mast cells and some irritant chemicals, toxins or pharmacological compounds can cause these to spontaneously degranulate and release histamine and a range of other vasoactive amines, leukotrienes and “slow-reactive substances” that can cause lung oedema and attract massive infiltration by eosinophils. These “late-phase” reactions usually occur in 4 - 8 hours after the immediate hypersensitivity and persist for 24 – 48 hours. It is believed that a proportion of stags may be affected in this way and a number of them will succumb to severe pulmonary oedema, which fills the lungs with froth and fluid, suffocating the animal.

8.1.3 Pathology

The post mortem findings usually show froth in the trachea, bronchi and bronchioles and congestion and oedema of the lungs. Histopathological findings are mainly confined to the lungs, which show generalized oedema, thicker than usual intralobular septa, and severe infiltration of degranulated eosinophils and perivascular eosinophils.

8.1.4 Treatment and Control

There may be predisposing factors that increase the likelihood of animals reacting to xylazine in this way. Hyperthermia, excessively dusty conditions, pre-existing lung infection or infestation with lungworms and prolonged recumbency after anaesthesia may all play a role in reducing the response threshold of the animal’s mast cells in the lungs, thereby increasing the likelihood of “stag death” occurring. When velveting stags, it is recommended to: (a) keep the animals as quiet and unstressed as possible, (b) operate in cool conditions and keep animals in the shade until they are fully recovered, (c) fully reverse the sedation with yohimbine or other specific antidote as soon as possible, (d) avoid dusty conditions, (e) treat affected animals for concurrent diseases such lungworm and pneumonia. In special cases the use of anti-inflammatory drugs may be indicated.

8.1.5 References

Mackintosh, C.G., Cross, J.P 1989. Xylazine study report. Deer Branch, NZVA, Proc. Course No. 6: 136-143.

Walker, I.H., Middleberg, A. 1988. Post velveting deaths in stags. Deer Branch, NZVA, Proc. Course No. 5: 128-135.

8.2 Trauma

Injury resulting in trauma is the greatest single cause of deaths on deer farms. However, not all injuries are fatal. While some resolve, others result in permanent disability. Some injuries occur spontaneously, although most occur as a result of mustering, yarding and handling. Injuries commonly include fracture of the neck vertebrae as a result of hitting fixed objects during panic flight when an individual is isolated from the herd, or "spooking when being moved with a mob along a raceway. Limb fractures are common during yarding and mustering, and the most common course of action is for the owner to euthanase the animal immediately. It is common for stags to receive injuries during the rut as a result of fighting. These often are soft tissue injuries involving the shoulder area and hind leg muscle, tendon or ligaments causing lameness. Fractures are not uncommon in adults, especially of the ribs. Old fractures of the ribs are commonly seen at DSP's. These injuries undoubtedly reduce the animal's performance in terms of carcass weight and/or velvet antler production.

Deer are inquisitive animals and commonly suffer various misadventures such as being caught in wire or other objects. A common injury is soft tissue stripping of a lower limb, often exposing bone and tearing tendons. A common sequel is osteomyelitis, a bacterial infection of the bone, which may heal over, but later discharge pus through skin from an enlarged pus-filled bone. It is also common to find a piece of dead bone, a sequestrum, inside the bone, and this can be removed surgically with successful resolution with appropriate treatment.

It is likely that the injury rate varies considerably between farms as a result of habituation by the deer to handling and yarding, thereby minimising the risk of fright/flight responses, and reducing the flight distance of deer, reducing the predisposing factors contributing to injuries. Further study is needed to examine the farm management practices that contribute to or reduce the risk of injuries, since they are of significant animal welfare concern, in addition to the economic wastage experienced.

9 EMERGING DISEASES

9.1 Protozoal

9.1.1 Theileriosis (?)

A number of case of haemolytic anaemia, haemoglobinuria and jaundice in red deer have occurred in the Manawatu and Hawkes Bay regions since 1999. Blood smears revealed intra-erythocytic protozoal organisms thought to be a *Theileria* spp. One deer was found in an extremely weak condition with severe respiratory dyspnoea, very pale mucous membranes and elevated heart and respiration rates. A number of deer were found dead with signs of severe anaemia and haemoglobinuric nephrosis, kidney lesions. Thus, in some respects, this disease resembles leptospirosis, and mis-diagnosis is likely unless laboratory confirmation is undertaken. However, the main losses to date with this syndrome have been carcass rejection at DSP's because of jaundice. In some cases, killing of the line has stopped and the animals returned to the farm of origin where they recovered. However, animals affected by jaundice are likely to have reduced production because this condition results in loss of appetite. *Theileria orientalis* has been reported in cattle in New Zealand since 1984. These organisms are normally transmitted by ticks. Affected cattle usually show signs of illthrift, sub-optimal production and mild regenerative anaemia. The incubation period of *Theileria* is approximately 9-25 days.

This disease is currently the subject of a special investigation by MAF Biosecurity in conjunction with the National Centre for Disease Investigation, in consultation with Massey University. This investigation involves alerting veterinary practitioners to the potential for this organism to cause clinical and fatal disease similar to other causes of jaundice, as well as DSP veterinarians and inspectors. Where cases are diagnosed, trace-back to the farm will occur, and an investigation of the organism will continue until its nature has been clarified.

9.2 Viral

9.2.1 Cervine herpesvirus 1 (CerHV-1)

9.2.1.1 Introduction

The two European cervid herpesviruses have been named herpesvirus of cervidae types 1 and 2 (CerHV-1 and CerHV-2). The latter is also known as rangiferine herpesvirus 1 (RanHV-1). They are alphaherpesviruses closely related to the bovine herpesvirus BHV-1.

Recently CerHV-1 was isolated from red deer semen as part of an export testing protocol. The animals were not showing any clinical signs of disease. The prevalence of this virus infection in farmed red deer population in New Zealand is estimated to be 38% based on a serological survey of 314 deer and 161 cattle samples selected randomly from a serum bank. This is similar to the 35% prevalence found in a survey of free-living and farmed deer in the United Kingdom. In the UK it has been isolated from outbreaks of ocular disease resembling pinkeye, or contagious keratoconjunctivitis, in red deer. Outbreaks of conjunctivitis have occurred in New Zealand for some years, but the aetiology has not been determined, although CerHV-1 infection has been suspected.

The mode of transmission of CerHV-1 is unknown, but the virus replicates in both the upper respiratory tract and the eye and it appears that both contact and aerosol infection are important in disease transmission. All outbreaks of clinical disease overseas so far reported have been associated with stress such as weaning. A severe outbreak of ocular disease, with some deaths, has been reported in recently weaned farmed deer calves overseas and has been spread to several properties after a sale. CerHV-2 of reindeer spreads predominantly by the venereal route.

9.2.1.2 Clinical signs

To date, no outbreaks of clinical disease due to CerHV-1 have yet been reported in NZ. Overseas, the most obvious consequence of infection with CerHV-1 in red deer calves is a severe conjunctivitis. Excess lachrymation progresses to involve swelling and oedema of periorbital tissue, scleral injection, purulent ocular and nasal discharge, pus in the anterior eye chamber (hypopyon), corneal opacity without ulceration and photophobia. In the first outbreak reported in Scotland, several calves died, but their deaths were ascribed to trampling. In later outbreaks very severe corneal damage was seen, and corneal rupture occurred in several animals. Recovery often took as long as 2 months.

9.2.1.3 Treatment and Control

No specific treatment has been described, but supportive therapy with antibiotics may be used to control secondary bacterial infection. No control measures are described, but in common with other similar diseases an avoidance of stress should be attempted.

9.2.1.4 References

Motha, J., Jenner, J. 2001. Serological relatedness of corvine herpesvirus-1 and bovineherpesviru-1 and the prevalence of corvine herpesvirus-1 infection in farmed deer in new Zealand. New Zealand Veterinary Journal 49: 162-163.

Wilson, P.R., Cooper, B.S., Badger, S.B., Jopp, A.J., Abeynayake, P. 1981. Keratitis in Red Deer. N.Z. vet. J. 29: 92-94.

9.2.2 Rotavirus and Coronavirus

9.2.2.1 Introduction

These two viruses, which are from different viral families, have been associated with scouring in young deer in North America and may occur in New Zealand. Coronavirus has been isolated from scouring wapiti neonates and sambar deer. A coronavirus that has a close molecular relationship to bovine coronavirus has been isolated from 10-month-old clinically affected wapiti calves and grown in cell culture.

9.2.2.2 Clinical signs

The most consistent clinical sign in young deer is watery diarrhoea. As the disease progresses the calves with scours may become dehydrated, weak, and depressed. They may stagger and will often become recumbent before death. In some cases the calves show minimal clinical signs and may be identified as being sick only when the disease has progressed to the point where treatment is unrewarding, and death is impending. Bacterial agents, especially *Escherichia coli* may play an important role in exacerbating the disease. *Cryptosporidium* has also been implicated in outbreaks of neonatal diarrhoea in both wapiti and red deer. A high prevalence of antibodies to Rotavirus has been found in adult red deer.

9.2.2.3 Pathology

Affected animals develop degrees of dehydration, emaciation, and fluid faeces in the intestinal tract. Faecal samples taken from animals affected with scours for bacterial culture and virus isolation. Post-mortem tissue samples should be sent for histopathology, bacterial culture and virus isolation.

9.2.2.4 Treatment and Control

Treatment of scouring deer is especially challenging, as the decision to treat may often mean a concomitant decision to proceed to hand rearing. Dams may refuse to accept a

deer calf that has been taken away for periods longer than a few hours. If treatment is attempted, fluid replacement is of paramount importance, but the use of appropriate fluids is essential. Hypernatremia (low sodium) has been identified as an important mortality risk factor in wapiti calves treated with oral replacement fluids designed for bovines. Scouring wapiti calves rapidly become severely hypoglycaemic and immediate attention must be given to this situation. Fresh drinking water should also be supplied to calves, even if they are receiving intravenous therapy.

Many deer North American farmers use combined cattle vaccines that incorporate one or both viruses and sometimes *E. coli* antigen as well, despite the lack of any objective data on efficacy. Better results may be achieved by altering environmental conditions and management programmes, than by vaccinating the stock.

The occurrence of neonatal diarrhoea may be associated with many determinants other than bacterial, parasitic or viral agents, such as wet environmental conditions, overcrowding and poor nutrition of the dam. If a herd problem arises it would be prudent to try to identify not only a causative agent specific to the herd, but also other environmental factors that may be significant contributors to the development of the disease.

9.2.2.5 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10 EXOTIC DISEASES OF IMPORTANCE

10.1 Viral

10.1.1 Foot and mouth disease

10.1.1.1 Introduction

Foot-and-mouth disease (FMD) is caused by an apthovirus in the family Picornaviridae and there are seven immunologically distinct strains. With the exception of Australasia, the disease is known world-wide.

Many animal species, including humans, and all artiodactyls, are susceptible. Among cervids, infection has been reported in reindeer, moose, muntjac, white-tailed deer, Eld's deer, sika deer, fallow deer and red deer In a series of trials with British deer, type O and type C virus were used to infect animals. Although all species can be infected, there appears to be a range of susceptibility. Red deer and fallow deer develop only mild lesions and are much less susceptible than muntjac and roe deer, which develop severe potentially fatal disease.

FMD was diagnosed in feral deer in an outbreak in California, USA, in the 1920s and was eradicated through a strenuous deer control operation. Spill-over of FMD into the feral deer population in NZ in the event of an outbreak in farm animals could jeopardise control procedures, but experience from the 2001 outbreak in the UK suggests that feral deer may not play an important role in maintaining the infection in the wild. In the 2001 outbreak in the United Kingdom, the disease was diagnosed on a deer farm, which had four species of deer. A number of deer developed suspicious clinical signs. The first case was a sika deer that stopped eating, was salivating, drinking, trembling and had a vesicle in its mouth. Samples were taken but they were negative. There were four similar cases, all of which had mild mouth lesions but all were negative to tests. Among red deer, fallow deer, Père David's deer and sika deer in the herd, clinical signs were seen only in sika deer, in the period between initial diagnosis and herd depopulation

10.1.1.2 Transmission

FMD is extremely contagious, and is readily spread via respiratory secretions, saliva urine and faeces. Mechanical spread via contaminated animal products occurs and a variety of fomites can carry the virus, which, within a pH range of 6 and 9 is relatively resistant to environmental conditions. Red deer can shed similar amounts of virus to cattle and sheep, and could so infect in-contact animals, while fallow and sika deer can become carriers. During the 1967-8 epidemic some deer, particularly fallow, shared grazing with infected cattle and sheep but none were reported as showing clinical signs.

10.1.1.3 Clinical signs

Experimentally infected red deer, whether they were infected by exposure to infected cattle, or by inoculation of virus, developed either inapparent infection or only mild clinical signs. Vesicles developed in the mouths of only a small proportion of the infected animals. Those that did develop vesicles remained alert, did not show excess salivation, and were not lame. By contrast sika deer developed severe signs, with copious salivation, depression, lameness, and ulceration of the oral mucous membranes.

10.1.1.4 Pathology

In experimentally infected red deer the main pathological signs are restricted to the epithelium of the mouth and hard palate, where the small vesicles mentioned above were seen. No lesions developed on the coronets, the bands above the horn of the hoof, in deer.

In other species that die of acute infection, degeneration of the heart muscles is a feature of the disease.

10.1.1.5 Diagnosis

Diagnosis is usually based on clinical signs, and confirmed by virus isolation and serological testing. Specific strain identification is achieved either with ELISA or PCR testing.

10.1.1.6 Control

The old adage 'prevention is better than cure' applies particularly to FMD as control in free-ranging populations of deer is likely to be impractical or impossible. In most countries FMD is a reportable disease. Therefore control measures are mandated under animal health regulations. In the face of an outbreak one or more of the three basic control measures of eradication of infected and nearby stock, quarantine and vaccination may be used.

10.1.1.7 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.1.2 Bluetongue and epizootic hemorrhagic disease

10.1.2.1 Introduction

Both bluetongue (BT) and epizootic haemorrhagic disease (EHD) are insect vector borne virus infections that affect deer and other ruminants in North America. The viruses are closely related members of the Orbivirus genus. They both produce a similar haemorrhagic syndrome in susceptible white-tailed deer, but they are distinct diseases and can occur simultaneously in a single animal, or be detected during the same outbreak.

10.1.2.2 Bluetongue (BT)

The natural vertebrate host of BT is the bovine, but non-clinical infections can become established in wapiti. Cattle, wapiti and goats can have high concentrations of circulating virus for prolonged periods and can probably act as long term carriers. Vertical transmission from a female wapiti to her calf has been demonstrated. Bluetongue virus was isolated between 5 and 9 days post-infection from five experimentally infected wapiti, three of which developed mild clinical signs, and also from two of them that were given steroids 105 days later.

10.1.2.3 Epizootic haemorrhagic disease (EHD)

White-tailed deer are the most susceptible species to EHD, and outbreaks that have occurred are usually characterised by local die-offs of a high percentage of animals in a small area. In spontaneous outbreaks mule deer and pronghorn antelope have also been affected, but not to the same extent as white-tailed deer. Serological evidence of infection has been found in black-tailed deer, red deer, wapiti, fallow deer and roe deer, as well as domestic cattle. Experimentally, the EHD virus has been inoculated into wapiti, red deer, fallow deer, roe deer, muntjac, cattle, sheep, goats and domestic pigs. All but the goats and pigs developed viraemias, but none showed clinical signs.

10.1.2.4 Transmission

Transmission of both diseases is primarily via biting gnats or sand-flies of the genus *Culicoides*. In North America the main vector of BT is *C. sonorensis,* which has a limited range and this may explain the distribution pattern of the disease and its virtual absence

from Canada and its low to nil seroprevalence in the upper mid-west and northeastern USA. However, many other members of this insect genus have been incriminated as potential vectors. Bluetongue infection is most commonly seen in late summer and early autumn, especially during wet seasons. Windborne movement of vectors over long distances has been incriminated in new outbreaks of BT in some parts of the world.

EHD has been reported in most of the states of the USA as well as in western Canada. The disease occurs less frequently in northern areas, which is no doubt related to the lack of vectors at these latitudes for much of the year. *Culicoides variipennis* is considered to be the principal biological vector of EHD. The incubation period is usually from 10-20 days, and outbreaks are seen in late summer and early autumn, often associated with wet weather. Cattle develop chronic viraemias and may be reservoirs of infection.

It is believed that the potential insect vectors of BT and EHD would survive only in the north of New Zealand if these diseases were introduced to this country.

10.1.2.5 Clinical signs

The clinical signs of Bluetongue vary widely according to the species affected, as well as the strain of the virus and the immune state of the animals. White-tailed deer are the most susceptible cervid to BT. They can die in less than 24 hours, after suddenly developing a high fever, and showing signs of respiratory distress. There may be a bloody nasal discharge and marked reddening of the buccal and nasal mucosa. Oedema, especially of the head and neck, is often evident. Diarrhoea and dysentery may occur, and inflammation of the coronary band may cause severe lameness. Mortality in white-tailed deer herds may reach 50%, but in enzootic foci outbreaks of clinical disease are rare. Following recovery from BT animals are generally unthrifty, have poor weight gain, develop chronic foot lesions and may have impaired reproduction.

In white-tailed deer EHD manifests as an acute or peracute haemorrhagic disease. Mortality rates may be high, and up to 90% of infected deer usually die within 8-36 hours of the onset of signs after an incubation period of 5 to10 days.

10.1.2.6 Pathology

The gross pathological picture varies according to the severity and the two diseases cannot be distinguished from one another by either gross or histopathological examination. In peracute forms severe oedema of the head neck, tongue and lungs may be evident. In the acute form the oedema may be accompanied by haemorrhages in many parts of the body including the heart, and the entire gastrointestinal tract. There may be areas of necrosis or ulceration on the dental pads, tongue, hard palate, rumen and abomasum.

Histologically, a disseminated vasculitis and thrombosis, associated with haemorrhages, degenerative changes and necrosis is seen in many organs, but the lesions can be very subtle if the animals die early in the course of the disease.

10.1.2.7 Diagnosis

Haemorrhagic diseases present with such a diverse range of signs that field diagnosis is often very difficult, except in the more severe forms. The occurrence of a haemorrhagic disease during vector seasons should increase the index of suspicion. For confirmation, virus isolation and serological tests must be carried out. Presence of antibody alone is not enough to confirm diagnosis, and may only indicate that exposure to the agent has occurred.

There are a number of serological tests available to assist in BT and EHD diagnosis. Agar gel immunodiffusion (AGID) tests, serum neutralisation (SN) and the competitive ELISA (cELISA).

The only means of confirming the causative agent of haemorrhagic disease is virus isolation from blood (collected in anticoagulant), lung, liver, lymph node or spleen or molecular techniques including dot blot, *in situ* hybridisation and PCR.

10.1.2.8 Control

Infected animals are only viraemic for a short time, so importation of animals outside vector seasons is the most effective means of avoiding these diseases. Stringent testing for antibodies to BT or EHD should preclude movement. It appears that New Zealand may not have suitable insect vectors for these diseases. It is important that such vectors are not introduced in this country.

10.1.2.9 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. (2002). Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.2 Prion

10.2.1 Chronic Wasting Disease

10.2.1.1 Introduction

Chronic wasting disease (CWD) is one a group of fatal mammalian neurodegenerative diseases known as transmissible spongiform encephalopathies (TSEs) that is characterised by accumulations of abnormal proteinaceous material that is a protease resistant (PrP^{res}) form of cellular protein (PrP^{C}) normally produced in the central nervous system. PrP^{res} is a transmissible agent and catalyzes the conversion of PrP^{C} to PrP^{res} in susceptible hosts. CWD has been found only in elk, mule deer, white-tailed deer, and black-tailed deer in North America. First recognized as a clinical "wasting" syndrome in 1967 in mule deer in a wildlife research facility in northern Colorado, it was identified as a TSE in 1978. CWD is typified by chronic weight loss leading to death.

Cases from wild cervids of these species were first reported in 1981 but it is likely that the disease was present before then. In the mid-1980s, CWD was detected in free-ranging deer and elk in contiguous portions of northeastern Colorado and southeastern Wyoming. It has subsequently also been documented in free-ranging deer herds in Nebraska, Wisconsin and the Canadian province of Saskatchewan. It also has been documented in captive herds in Colorado, Montana, South Dakota, Oklahoma, Kansas, and Nebraska. It has more recently been diagnosed in the Canadian provinces of Saskatchewan and Ontario. It is very likely to be diagnosed in other states as surveillance involving surveys of hunter-shot animals and material submitted from farmed animals is undertaken.

Transmission of CWD is thought to be primarily horizontal, that is, from animal to animal within a herd. The youngest animal diagnosed with CWD to date has been 17 months of age, but the date of infection was not known, so that a true incubation period in natural infections remains undetermined. There is ongoing research to further explore the possibility of transmission of CWD to other species. To date there is no evidence of transmission to humans, which make it similar to scrapie and unlike BSE.

10.2.1.2 Clinical Signs

Most cases of CWD occur in adult animals. The disease is progressive and always fatal. The most obvious and consistent clinical sign of CWD is weight loss over time. Behavioural changes also occur in the majority of cases, including decreased interactions with other animals, listlessness, lowering of the head, blank facial expression, and repetitive walking in set patterns. In elk, behavioral changes may also include hyperexcitability and nervousness. Affected animals continue to eat grain but may show decreased interest in hay. Excessive salivation and grinding of the teeth also are observed. Most deer show increased drinking and urination. Some deer may gradually lose the ability to swallow, so pneumonia associated with inhalation of ingesta is one of the terminal signs in some animals.

10.2.1.3 Diagnosis

Currently, definitive diagnosis is based on postmortem examination (necropsy), typical histopathological changes in the brain, and immunohistochemical testing for the abnormal prion. Gross lesions seen at necropsy reflect the clinical signs of CWD, primarily emaciation. Aspiration pneumonia, which may be the actual cause of death, also is a common finding in animals affected with CWD. Thus, in New Zealand, veterinarians are advised to submit brains from deer that die of respiratory disease, as part of this country's surveillance for CWD.

10.2.1.4 Control

Control of CWD in wild populations is not possible currently. Control and hoped-for elimination in farmed and other captive cervids in Canada regulated by the Canadian Food Inspection Agency (CFIA), and is based on slaughter of all deer in any herd containing infected animals and measures to clean the environment.

10.2.1.5 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.3 Bacterial

10.3.1 *Brucella abortus* and *Brucella suis* type 4

10.3.1.1 Introduction

In North America, brucellosis due to *B. abortus* biotype 1 occurs among wapiti that congregate on winter feeding grounds in north-western Wyoming and in wapiti from Yellowstone National Park. To date, brucellosis has not been reported in farmed wapiti.

Reindeer (*Rangifer tarandus*) in Alaska present with a range of disease syndromes caused by *B. suis* type 4. *B. abortus,* a previous disease of cattle in New Zealand has been eradicated from this country and *B. suis* has never been reported. *Brucella* organisms are highly infectious and transmission may occur via the oral, nasal, conjunctival, vaginal or rectal mucosa.

10.3.1.2 Clinical signs

The most common signs of brucellosis due to *B. abortus* in wapiti are abortion, stillbirth and infertility in females, and orchitis (inflammation of the testicles) and epididymitis in males. Severe lameness and swollen joints may also be observed. Similarly, *B. suis* type 4 causes abortions, infertility and severe septic arthritis in reindeer.

10.3.1.3 Pathology

The most significant lesion is a necrotising placentitis, characterised by a thickened placenta covered with purulent exudate. Lesions in male wapiti are usually milder than those seen in cattle or bison, with some testicular swelling and oedema, in addition to variable necrosis in the testicular parenchyma.

10.3.1.4 Diagnosis

Bacterial isolation of *B. abortus* or *B. suis* from aborted foetuses and/or placenta is the most reliable method of confirming infection. Gloves should be worn when handling aborted material because of the zoonotic risk. Serological tests, including the complement fixation test, tube agglutination and the ELISA, can be used for presumptive diagnosis and export tests.

10.3.1.5 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.3.2 Anthrax

10.3.2.1 Introduction

Anthrax, caused by the sporulating aerobic bacterium, *Bacillus anthracis*, presents as an acute or peracute disease affecting multiple species. The disease has a world-wide distribution and over the centuries, severe outbreaks have occurred in red deer (*Cervus elaphus*), wapiti (*Cervus elaphus* spp.), moose (*Alces alces*) and fallow deer (*Dama dama*) in Europe and the former Union of Socialist Soviet Republics (USSR). Anthrax must always be considered as a potentially serious disease risk to humans. Anthrax has occurred sporadically in cattle on a small number of gazetted farms in New Zealand. There have been no cases reported in NZ deer.

10.3.2.2 Transmission

In herbivores, transmission is essentially by ingestion of contaminated soil or water, and in some cases, chewing infected bones. Aerosol infection at dusting sites has been postulated as a source of infection.

10.3.2.3 Clinical signs

In the acute form, the incubation period is less than two days and the animal dies rapidly from septicaemia (blood poisoning). Affected animals may appear dull, and show evidence of abdominal pain by grinding their teeth. Animals may stagger or have a stiff-legged gait, and have bloody discharges from the nostrils and anus.

10.3.2.4 Pathology

If anthrax is suspected, no autopsy should be conducted until blood films have ruled out the possibility of this disease. The reason for this is that *B. anthracis* will readily sporulate once exposed to oxygen, and these spores are exceedingly resistant to environmental decontamination, remaining viable in the soil for decades. If an autopsy is mistakenly performed, copious sero-sanguinous fluid will be observed in the body cavities, the spleen will be distended and dark, and unclotted blood will be found in the heart and major vessels. These fluids contain vast numbers of the organism, which are highly infectious for humans handling the carcass. A cutaneous form of anthrax results in humans, involving marked local swelling and redness.

10.3.2.5 Diagnosis

Diagnosis is confirmed by detection of characteristic spores in stained blood films. Other clinical presentations which must be distinguished from anthrax include lightning strike, trauma, poisoning, yersiniosis, malignant catarrhal fever and any peracute infectious disease.

10.3.3 Lyme disease

Lyme disease is a zoonosis caused by the bacterium *Borrelia burgdorferi.* It is transmitted to humans by the bite of an infected tick. Various species of cervid deer are the preferred hosts for adult, black-legged ticks (*Ixodes scapularis* and *Ixodes pacificus*) in the United States. Although frequently exposed to the agent of Lyme disease (*Borrelia burgdorferi*), these animals, for the most part, are incompetent as transmission reservoirs

10.4 Parasitic

10.4.1 Giant Liver Fluke

The giant liver fluke, *Fascioloides magna,* occurs in North America and parts of Europe and can cause serious disease in a range of cervids. It has an indirect life-cycle which involves transmission through aquatic snails, usually of the genus *Lymnaea,* as does *F. hepatica,* described above. The definitive hosts, which are all cervids, are white-tailed deer, wapiti, caribou, black-tailed deer, mule deer, red deer and fallow deer. The dead-end hosts include moose and sika deer. *F. magna* causes serious disease, which is often fatal, in aberrant hosts including domestic sheep, goats and cattle.

Diagnosis is made on finding the characteristic operculate eggs in faeces. The prepatent period of *F. magna* may be as long as 30-32 weeks, and eggs would not be detected in faeces of infected animals before this time. Special flotation or sedimentation techniques are required to detect the eggs, which will not usually be found upon routine faecal flotation.

10.4.1.1 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.4.2 Parelaphostrongylosis

Parelaphostrongylus tenuis, also known as meningeal or brain worm is a parasite of white-tailed deer and other species that reside in the eastern half of North America. It has an indirect life-cycle with a snail intermediate host. *P. tenuis* adults are found in the meninges, tissues surrounding the brain and spinal cord, of the white-tailed deer and generally do not cause any clinical disease. Abnormal hosts, including moose, wapiti, red deer, woodland caribou, mule deer, black-tailed deer, fallow deer, sheep and goats, llamoids, domestic cattle and several exotic bovids can become infected and some may show clinical signs relating to CNS damage. These may include ataxia, twisting of the neck (torticollis), knuckling, circling, fearlessness, depression, nystagmus (sideways twitching of the eyeballs), apparent blindness, paresis, inability to stand, and weight loss. Pneumonia can also occur with heavy infections of *P. tenuis.*

Diagnosis is based upon clinical signs and the finding of spine-tailed larvae in the faeces.

Control of the spread of infections of this parasite depends on prevention of movement of infected animals into areas where suitable intermediate hosts exist and control of gastropod intermediate hosts.

10.4.2.1 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.4.3 Throat bot

Several throat bots of the genera *Pharyngomyia* and *Cephenemyia* are seen in the upper airways of cervids in the northern hemisphere. Their life-cycles involve the deposition of first stage larvae in the nasal cavity. The commonest site where large numbers of third stage larvae may be found is the retro-pharyngeal pouch where they may grow to about 20 mm in length. The hatched larvae migrate to the pouches where they may either complete their development in 30-35 days or remain until spring. They then leave the host through the nostrils, fall to the ground, pupate and go on to complete the life-cycle. No treatment has been described.

Cephememyia trompe, one of the bot species, was imported into New Zealand from Canada in a Wapiti in the mid 1980's. It was an incidental finding at post mortem. Herd mates were treated with ivermectin. Surveillance suggests that it did not become established in this country.

10.4.3.1 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.4.4 Warble flies

Warble flies of the genus *Hypoderma* do not appear to be important parasites in wapiti or red deer in North America, but heavy infestations have been observed in deer in the UK. *H. tarandi* occurs as an important parasite of reindeer and has a Holartctic distribution. It causes severe irritation and damage to hides, but there is a degree of age-related increased resistance. *H. diana* and *H. actaeon* are specific warble flies of deer in Europe, but not in North America.. Serological tests have been developed for cattle that can be applied to deer. Pour-on organophosphates or ivermectin are effective, but reinfection of farmed red deer from wild red deer may occur.

10.4.4.1 References

Haigh, J.C., Mackintosh, C.G., Griffin, F. 2002. Viral, parasitic and prion diseases of farmed deer and bison. Rev. sci. tech. Off. Int. Epiz. **21** (2): 219-248.

10.4.5 Ticks

The North American winter tick *Dermacenter albipictus* is common on wapiti in Canada. It was found on a Wapiti imported to NZ from Canada in the mid-1980's, but it appears not to have become established in this country. A number of other tick species affect deer in various parts of the world, and some are vectors of protozoal blood-borne diseases such as *Anaplasma* and *Babesiosis.*

10.4.6 Other exotic parasites include *Eleaophora*, *Filaria*

11 COST &/OR OTHER IMPLICATIONS OF HEALTH/DISEASE PROBLEMS

The true costs of health/disease problems can only be crudely estimated because for virtually all these problems the prevalence of infection, the incidence of clinical disease, and the production losses due subclinical disease are not known.

11.1 Bovine Tb

There are direct and indirect costs to farmers and to the deer industry. The National Tb Control Scheme requires farmers to test their animals at their own expense and there is no compensation for reactors, so accurate calculation of the cost is difficult. There is also the potential for Tb to be used as non-tariff trade barrier. New Zealand is the only significant international exporter of venison and could be singled out by competitors in the marketplace. At worst we may not be able to export venison in the future until we are officially "Tb free" as a country. At a lower level, importers may insist that venison is certified to have come from "Tb-free" farms, districts or regions.

The direct costs of the Tb scheme are:
Tb testing: MCT $3-5 per animal plus travel
Ancillary tests: CCT $15 per animal plus visit fee and travel.
BTB ~$100 per animal plus cost of blood sample and travel.
ELISA ~$10 per animal plus cost of blood samples plus travel
AHB levies
Wildlife control on farm

Indirect costs include:
Down-grading of Tb reactor animals to "local consumption"
Down-grading to "local consumption" of suspect lesion animals in DSPs

Additional costs for Movement Control farms:
Movement control restrictions on trade: unknown cost, but high for stud breeders
Losses in production from diseased animals: unknown cost.

Statistics:
In the year ending Mar 2002 there were 582,000 deer MCT tests and 982 "reactors".
Over this period there were 6587 CCTs, 2255 BTBs and 2299 ELISA tests. We estimate that 1000 reactor deer were killed for local consumption.
Costs:

MCT 580,000 @ $4	$2.3 mill
CCT 6587 @ $15	$100,000
BTB 2255 @ $110	$250,000
ELISA 2299 @ $12	$28,000
Reactors killed for "local": est. 1000 @ a loss of $5/kg per 50kg carcass	$250,000
Suspect lesion for "local": est. 1000 @ a loss of $5/kg per 50kg carcass	$250,000

Total estimated direct and indirect costs of testing for 2001/02 $3.2 million
Plus hidden costs of labour, movement control restrictions and loss of production.

11.2 Johne's disease

An Agriculture-NZ economic evaluation (Brett, 1998) estimated that JD costs the NZ deer industry $200-340K per annum based on estimated 1998 figures. However, this is likely to be a gross underestimate of the future annual costs, because the disease is spreading and there have been an increasing number of serious outbreaks in deer aged 8 to 15 months old. These outbreaks have generally involved approximately 10% of yearlings in a group and have caused serious economic losses on affected properties, which can amount to

$20 – 30, 000 per season. This type of serious outbreak in yearlings is a significant difference between JD in deer and JD in cattle and sheep, where losses are usually confined to 2 to 4 year old animals and annual mortalities rarely rise above 3% under NZ conditions. Sporadic losses of adult deer have also occurred on farms throughout NZ and it is acknowledged that there is under-reporting of infection. The effects of finding JD on stud farms can also be financially crippling. Thus the annual costs of JD are projected to increase significantly. Currently JD has been positively diagnosed on over 300 deer farms, which is around 5% of commercial deer farms in New Zealand.

Of even greater concern than the direct cost of JD to individual farmers is the potential for JD to be used against New Zealand venison exporters as a non-tariff-trade barrier in the same way that bovine tuberculosis might be. If *M. paratuberculosis* is shown to cause disease in humans then consumers of venison may demand product free of this organism. Such a scenario has major implications for the deer industry in New Zealand. It should be noted that the Food Standards Agency in the United Kingdom has devised a strategy for the control of *M. paratuberculosis* in cows' milk

11.3 Yersiniosis

The true incidence of yersiniosis is not known, but the animal health surveillance data from veterinary diagnostic laboratories for 1997-2001 shows that yersiniosis was identified in 14% of ill-thrift cases in deer in 1997, 16% in 1998, 9% in 1999 and 6% in 2000. For the last few years nearly 500,000 doses of Yersiniavax were sold annually for the estimated 700,000 weaners. With two doses needed per deer, this equates to approximately 35% of weaners being vaccinated overall. However, 40-56% of weaners are vaccinated in the major deer producing areas. The vaccine costs approximately $1.50 per dose to the farmer ($3/deer) and thus the total direct cost to the industry is $750,000 plus administration and labour costs.

Experimental evidence and anecdotal reports suggest that vaccination has significantly reduced the number of serious outbreaks of yersiniosis in farmed deer. The annual reduction in calf losses throughout the industry is likely to be in excess of 1%. Small losses from yersiniosis do occur in vaccinated weaners due to a variety of reasons, including incorrect vaccination, use of only one dose, concurrent stress or disease, extremely heavy challenge and genetic susceptibility. AgVax NZ Ltd, the suppliers of the vaccine, have data to show that in most outbreaks in vaccinated herds the losses are 3% or less. By comparison outbreaks in unvaccinated herds usually involve losses of 10 – 30%. However, on a national basis a 1% reduction in weaner losses is equivalent to a saving of 7,000 calves @ $250 or $1.75 million, which makes vaccination cost-effective on an industry-wide basis. On an individual basis a farmer has to save only one $250 weaner to pay for enough vaccine for 83 animals.

11.4 Leptospirosis

Surveys of deer suggest that infection is widespread throughout deer herds in New Zealand, but the most common serovar is *L. hardjo*, which appears to be relatively non-pathogenic in the endemic situation. Infections with *L. pomona* and *L. copenhageni* cause sporadic outbreaks of haemoglobinurea, jaundice and sudden death in weaners, with high apparent morbidity but usually with low mortality. An analysis of Animal health records from 1987 – 1992 showed that leptospirosis was a significant cause of death in deer, especially those less than 12 months of age and particularly in the autumn. Over this period 345 deer were reported dead in association with evidence of leptospirosis, at an estimated loss of ~$100,000. The deaths of 50 two-year-old stags on one property were attributed to leptospirosis.

Leptospirosis causes abortions and perinatal mortality in cattle and, although there is little direct evidence, it is possible, although yet to be proven, that leptospirosis contributes to

the losses of 0.6 - 0.8% that occur in pregnant hinds between pregnancy scanning and calving, as well as perinatal losses of 9 – 17%.

Two leptospiral vaccines are licensed for use in deer, but the number of animals vaccinated is not known. If the vaccines are effective, then the high number of lesions seen in deer at DSPs suggests that only a small proportion of deer are vaccinated, and it probably occurs mostly on individual properties that have experienced clinical outbreaks in the past. Vaccination is becoming more common as farmers attempt to reduce their exposure to OSH risk. The zoonotic aspects of leptospirosis should not be overlooked. The main driving force for vaccination of dairy cattle and pigs has been to prevent the number of cases of leptospirosis occurring in farmers and other people at risk in the livestock industry. This is also a valid reason for vaccinating deer. While there is little risk to handlers or consumers of venison, food safety concerns are often based on perceptions rather than the reality of risk, so the potential cost of the zoonotic nature of this organism if it became an issue in the marketplace should not be overlooked. Thus the cost of this disease is difficult to assess.

If a bold assumption was made that 0.1% of weaners were affected annually, this disease could cost $300,000, plus the cost of currently used vaccine.

11.5 Pasteurellosis

Sporadic losses occur due to infections with Pasteurella spp. But it does not appear to be a major cause of wastage on deer farms.

11.6 Colibacillosis

Some neonatal losses have been attributed to colibacillosis, especially in wapiti, but it is not considered to be a major cause of wastage in the deer industry.

11.7 Clostridial infections

Tetanus, pulpy kidney, blackleg, malignant oedema and wound infections occur sporadically at a low level. A small proportion of deer farms routinely vaccinate with multi-strain clostridial vaccines as a form of cheap insurance against losses, although the efficacy of these vaccines has not been demonstrated. Of greater concern is the effect that images of post-velveting clostridial infections could have on the future of velvet antler removal as a farming practice or profit, if they became available to animal welfare activists. Thus the cost of this disease is difficult to assess.

11.8 Fusobacteriosis

This is primarily a problem on fallow deer farms due to the high susceptibility of young fallow fawns to necrotic mouth and foot infections. Outbreaks of foot lesions occur in red deer sporadically and are usually associated with wet muddy underfoot conditions. Various killed bacterin vaccines appear to have given good protection, but none are currently available in New Zealand because the cost of licensing is too great for the number of doses likely to be sold. Thus the cost of this disease is difficult to assess.

11.9 Brucellosis

To date there have been only a small number of properties where *B. ovis* infections in stags have been reported. Potentially this disease could cause widespread problems if it was allowed to spread uncontrolled. However, evidence that there have been no natural occurrences of this disease in the past 4 years suggests this is unlikely. Further, sheep are the primary source of infection, and the incidence of this disease in that species is reducing.

11.10 Malignant catarrhal fever

When deer farming first started 30 years ago this was one of the major causes of mortality in adult deer. Subsequently the incidence rate has dropped, but it still causes an estimated 0.25 – 1.0% mortality rate in adult deer throughout the country. The incidence is low in dairying areas but is higher in sheep farming areas, especially in the South Island. Surveys in the Manawatu/Hawkes Bay showed the loss rate for hinds was 0.17% and stags 0.52%, but the incidence is believed to be higher in Canterbury.

Assuming that there are 1.8 million mixed age hinds and stags, this equates to losses of 5 - 20,000 animals per year @ $350 or $1.75 – 7 million pa.

11.11 Parapox

This causes sporadic outbreaks on farms, but these appear to occur when it is introduced onto a farm where it is not endemic, or when a large group of susceptible animals are moved onto a farm where it is endemic. In the endemic state it does not appear to cause significant problems because the animals get exposed gradually at an early stage of life. The outbreaks can be severe and cause considerable lost revenue if they affect stags in velvet. Some farms have lost virtually their entire crop of velvet for a season, amounting to thousands of dollars. The velvet cannot be sold because of the zoonotic risk and the fact that processing may not kill the virus. There have been some instances of weaners dying, but this is often associated with secondary infection. Overall deer parapox causes some low level losses each year to the industry but it is not considered a major threat.

If 0.1% of velvet was rejected, this would cost $34,000 annually

11.12 Lungworm

This is the most significant parasite for deer and potentially the biggest threat to weaners in the autumn. Currently *D. eckerti* can be effectively controlled by good grazing management and regular use of anthelmintics. However, every year there are cases of clinical disease due to lungworm infection reported by diagnostic laboratories and vets. These may be due to inexperience, poor management, seasonal differences or misunderstanding that “white” drenches do not have any persistent activity compared with avermectin/moxidectin anthelmintics that continue to be effective for up to 6 weeks after dosing, and that levamisole is not effective at all. There is also a move towards “organic” farming and the avoidance of anthelmintics, and this can lead to problems with lungworms. It has been estimated that deer farmers spend up to $10 million annually on anthelmintics, primarily against lungworm. Although lungworm are still sensitive to the current anthelmintics, research should continue into alternative control measures that are more ecologically acceptable and sustainable, including vaccines and “natural” parasiticides.

Cost of anthelmintic at $10million, and production losses of 1% through growth retardation due to lungworm and gut worms combined would result in reduction of value of venison of about $2.8 million on 2001 export figures.

11.13 GI nematodes

These are becoming an increasing problem as deer farming is becoming more intensive and stocking rates are increasing. There is also an increasing frequency of wapiti genes into the breeding stock and wapiti are used as terminal sires. Wapiti are more susceptible to GI parasites and this may result in increasing susceptibility of breeding hinds and weaners to parasitism. Pure wapiti herds, which are generally stud animals or heavy velvet producers, are very valuable and are particularly at risk from the “fading elk syndrome”. This increasing susceptibility is likely to result in clinical and subclinical losses and increased cost of anthelmintics used for treatment and prevention.

11.14 Elaphostrongylus cervi

There do not appear to be any significant losses associated with clinical disease due to elaphostrongylosis, but there is occasionally some minor loss of product from trimming affected carcasses in Deer Slaughter Plants.

11.15 Flukes

There do not appear to be any significant losses associated with clinical disease due to liver flukes.

11.16 Ticks

Although ticks have been recognised as a problem for over 20 years, losses are patchy and they have tended to be more prevalent in the upper and eastern North Island. However, they appear to have been spreading further south recently, especially in association with the movement of deer and dairy cattle to the South Island and have been reported in Southland. There have been increasing reports of tick problems on deer farms, especially in young fawns, which can get very heavy burdens and die of extreme anaemia. All classes of animal can carry ticks and, as well as causing loss of production associated with anaemia, they cause damage to hides and velvet antler. With "global warming" developing it is likely that ticks will become an increasingly serious problem on deer farms. Current technology can effectively control ticks, but a longer-acting animal treatment would greatly enhance the simplicity of tick control.

If 0.05% of calves born died of ticks, this would represent a lost opportunity for 600 venison carcasses at $300: $180,000, plus treatment est. 200000 doses at $1 = $200,000. Total $380,000

11.17 Cryptosporidiosis

Sporadic neonatal deaths attributable to cryptosporidiosis have been reported on a few deer farms, especially in the South Island.

11.18 Facial eczema

This is a very seasonal problem and is largely confined to well known dangerous areas in the North Island where it also affects sheep, cattle and horses. Fallow deer are particularly sensitive to facial eczema. However, when conditions get extreme, clinical disease can occur in red and wapiti deer. However, subclinical liver damage can also reduce productivity even when clinical signs are not apparent. Control measures that are effective for sheep and cattle also appear to be effective in deer. It is not possible with current information to assess the cost to the industry, but it is likely to be substantial in some years, due mainly to lowered growth rates, and interference with reproductive performance of hinds.

11.19 Ryegrass staggers

Red deer appear to be relatively resistant to ryegrass staggers. However, pure wapiti, and to a lesser extent wapiti hydrids, are very susceptible to this disease. With the increasing use of wapiti and wapiti terminal sires, the national herd may become more susceptible to this disease. Global warming may exacerbate the problem by causing more drought-stress to ryegrass and increase the amount of toxins. Conversely, the availability of new specialist non-ryegrass pastures and non-endophyte ryegrass cultivars may reduce the amount of toxic pastures. Although not usually directly fatal, ryegrass staggers can result in accidental deaths through misadventure and make management difficult. There is also evidence from sheep and cattle that subclinical levels of toxins in the pastures can reduce palatability and productivity. The cost of this disease is difficult to assess.

11.20 Cu deficiency/toxicity

Many areas of New Zealand are deficient or low in available copper. Osteochondrosis in young deer and enzootic ataxia in yearlings and older deer are the main manifestations of copper deficiency. However, the incidence of clinical disease is relatively low, even in herds where serum samples indicate the majority of animals have very low copper status. Few trials have shown a growth response to copper supplementation in their first year of life in groups under these conditions. However, supplementation may be economic if it prevents clinical disease occurring, even in a small number of animals. Wapiti are more susceptible to deficiency than red deer and have a copper higher requirement. Supplementation of hinds in mid to late pregnancy may the most cost-effective means of preventing osteochrondrosis in young fawns. The long-term effects of copper deficiency on hind productivity is not well understood.

There are a number of methods of copper supplementation including dosing with copper oxide wire particles (copper bullets), injections and pasture topdressing. The relative merits of these methods needs to be investigated, to determine which are the most cost-effective under a range of conditions. It is estimated crudely that in excess of $1.5million is spent annually on copper supplementation of deer, and a substantial amount would be spent on diagnostic testing and monitoring. If average losses, clinical and subclinical combined were 0.1%, this cost would be about $300,000. Total $1.8 million.

Copper toxicity is diagnosed occasionally and is usually due to overenthusiastic or poorly controlled copper supplementation.

11.21 Se deficiency/toxicity

Most of New Zealand soils and pastures are deficient or marginally deficient in selenium. Clinical disease is rarely diagnosed in deer and there is little information on what liver or circulating blood levels indicate “adequate”, “marginal’ and ‘deficient” states in deer. As insurance against deficiency it is likely that the majority of farmers supplement their deer with selenium by topdressing pastures with selenium prills, adding selenium to drenches, giving selenium boluses or selenium injections or using pour-on selenium. These are all relatively cheap forms of supplementation compared to the value of the animal.

11.22 Incidental/misadventure/”stag death”

The incidence of “stag death” has been estimated at 1 per 1000 anaesthetics. It is likely that less than one third of stags receive xylazine for velvet antler removal. Thus, approximately 100 stags could be lost annually, as a meat value cost of $50,000. However, a number of high value stud stags die of this condition, so the real cost would be higher. It is possible that the administration of anti-inflammatory drugs may help to prevent the non-specific hypersensitivity that is believed to be the cause of the problem, but there have been not trials conducted. The increasing use of physical restraint and local anaesthetics may reduce the number of cases occurring. Similarly, the development of a drug-free means of analgesia will also eliminate the risk of these deaths.

Other causes of misadventure include stags fighting, especially during the rut, water deprivation, which usually occurs in summer due to natural water sources drying up or an interruption to the piped water supply, and broken legs or necks as a consequence of panic or some stressful procedure. Improvements in management can reduce the likelihood of these occurrences. However, an estimate of the cost of deaths due to injuries can be made, based on figures in Table 2. Deaths due to injury accounted for 10% of hind deaths, 16% of stag deaths, 12.5% of weaner deaths, and 3% of calf deaths. At carcass values of $300 and $500 ascribed for hinds and stags, respectively, (ie. lost opportunity to achieve optimum production) the total cost approximates $765,000 for 2250 hinds,

$600,000 for 1200 stags, $2.5million for 8400 weaners, and $108,000 for 3600 calves. The total cost of injuries is therefore estimated at **$3,973,000** annually.

11.23 Verification of zoonotic organism status of venison

Food safety is increasingly becoming a serious international marketing issue. There are many organisms that have the potential for affecting food safety. These may be involved in a disease process in the animal at the time of slaughter, or they may simply be present in the tissues or blood stream or present in the gut contents or on the skin surface. The most common organisms include: *Yersinia pseudotuberculosis*, *Yersinia enterocolitica*, *Listeria* spp, *Salmonella* spp, *Escherichia coli*, *Campylobacter* spp, *Clostridium* spp, *Mycobacterium bovis*, *M. avium*, *M. paratuberculosis*, Staphylococci, Streptococci, *Leptospira* spp, etc.

A survey of carcass contamination with *Y. pseudotuberculosis* yielded only one organism from more than 300 carcasses, indicating the risk to humans from handling venison is very low. However, food safety concerns are often based on perceptions rather than the reality of risk, so the potential cost of the zoonotic nature of this organism if it became an issue in the marketplace should not be overlooked.

There are also issues of food spoilage, which will not be discussed in this review.

12 SUMMARY OF FINANCIAL LOSSES

12.1 Financial cost of Mortalities

The following is a summary of the major financial losses that can be estimated from current knowledge, with a number of assumptions, to provide a ready relative importance of each:

Disease	Possible cost ($Million Annually)
Tuberculosis	3.2
Johne's Disease	0.4 – 0.6
Yersiniosis	2.5
Leptospirosis	0.3
MCF	1.54
Parapoxvirus	0.034
Internal Parasites (lung and gut worms)	12.8
Ticks	0.38
Facial Eczema	?
Copper	1.8
Injuries	3.97
Other	?
TOTAL	**>$27 million**

12.2 Financial Cost of Subclinical disease

Because the real production losses caused by subclinical disease are unknown, only estimates can be made. Subclinical disease causes production losses such as failure of conception, failure to rear progeny, reduced growth rate and its effect on limiting the lifetime production of the individual animal or herd affected, reduced venison production, and failure to achieve potential velvet antler production. It is generally assumed that the total cost of subclinical losses is greater than the cost of mortalities *per se*.

Thus, the total annual cost of subclinical disease may exceed $27 million currently.

Combined, clinical and subclinical disease may be contributing to annual losses within the deer industry of around $54 million in 2001. This figure will increase as the number of farmed deer increases, unless there is better application of existing technologies to reduce wastage. In the future, new technologies will be devised that have the potential to further reduce losses caused by current diseases. However, the industry must be realistic in its expectations in this regard, because experience indicates that there are always new disease threats arising, and these will inevitably continue to represent a cost to the deer industry into the future.

13 PREVENTIVE MEDICINE PROGRAMMES

Most veterinary practitioners find that deer farmers are generally reluctant to seek veterinary attention to individual sick or injured deer. Thus the opportunity for farmers to have accurate diagnoses, and improve their understanding of means of prevention of disease is largely lost. Formal and informal surveys by one of the authors (PRW) suggest that more than half the mortalities on deer farms are preventable. Further, there is significant wastage due to lack of implementation of current technology for disease prevention resulting in higher mortality and subclinical disease rates than necessary, and often misdirected or unnecessary spending of money on animal health.

Preventive medicine programmes, also known as whole herd health and production programmes, have been shown by numerous formal studies to be highly cost-effective in other farmed animal species. No such formal economic analyses have been done for deer herd health and production programmes, but experience of individual professionals has confirmed their effectiveness on deer farms. In New Zealand, group health and production projects include the now concluded Canterbury DeerMaster and Hawkes Bay Richmond-Wrightson Deer Performance Projects, and the current DeerSouth Project. A characteristic of almost all group schemes is that they have a relatively short life-span. On the other hand, individual farm programmes based on commercial relationships with professional advisors generally have a longer persistence, and are likely to be of greater individual farmer benefit in the long term. The optimum is a combination of both group and individual relationships. The former provides benchmarking, social contact and motivation, whereas the latter provides individualised targeted programmes.

These programmes work by improving financial returns to farmers by a number of outcomes that are inextricably linked. That is, many of the factors that contribute to improving one outcome, for example feeding and nutrition for health, will also have a positive influence on other outcomes, such as growth of venison and improved reproductive performance, that also yield profit. Such programmes have a dual aim of improving health and production at the same time as reducing wastage in terms of unnecessary animal losses and animal health and management expenditure.

The key element of health and production programmes is farmer education. They need to be farmer focussed, because it is the farmer that implements management practices. The animal is a “reflex responder”, meaning that if the optimum is provided for the deer, the optimum will be produced by the deer: the deer does not choose.

The general methodology for health and production programmes for individual farms is generic, and well established. The biological potential for production and health is well understood within current limits, from extensive benchmarking data collected over the last 10 years. There are many well understood management practices that contribute to achievement of biological potential. There are a number of existing technologies that can significantly reduce the current cost of clinical and subcinical disease. There are new technologies available to farmers to improve production and health, which do not have a high adoption level within the deer industry. Thus, there is enormous opportunity for the deer industry to improve productivity and individual farm profitability.

It is the authors’ opinion that one of the most cost-effective projects the industry could embark upon is development of industry initiatives that encourage farmers to harness existing technology, through whatever is the most appropriate means for the individual farmer. This would have the following impacts:

Improve health, thus reducing wastage through mortalities and subclinical disease.

1. Concurrently improve productivity through improved growth and therefore average carcass weights, and reproductive performance, and hence greater numbers of animals for slaughter.
2. Improve cost-effectiveness of farm expenditure by targeted expenditure based on animal health and production surveillance.
3. Enhance the image of the NZ deer farming by improving the health and wellbeing of deer and minimising chemical usage.

This review has suggested that reducing animal health wastage, in terms of mortality and subclinical disease and expenditure on control by only 20%, could yield an industry return in excess of $10 million based on current national deer herd numbers.

13.1 Tools for Preventive Medicine Programmes

The most efficient means of maximising the health of farmed deer is to have a Preventive Medicine Programme (PMP) fully integrated with the management programme for the farming enterprise. This PMP will have elements of disease prevention, control, management and treatment. It will require the relative risk of all the relevant diseases to be assessed and subjected to simple cost/benefit analyses. The appropriate measures should be integrated into a package and a calendar of action points drawn up. Animals brought onto the property should also be subjected to a range of tests. The available tools for disease prevention, control, management and treatment (and the diseases that they are most relevant for) include:

- Vaccinations (yersiniosis, leptospirosis, clostridial infections, fusobacteriosis, salmonellosis, possibly JD)
- Anthelmintics (lungworm and GI parasites)
- External parasiticides (ticks, lice)
- Skin testing (Tb)
- Blood testing (Tb, *B. ovis*, JD, Leptospirosis etc)
- Faecal examination for parasites (lungworm, GI parasites, liver fluke, E. cervi, Cryptosporidiosis, coccidiosis)
- Faecal culture (JD)
- Physical examination (*B. ovis*, parapox, lice, congenital defects, injuries, semen quality, penile injuries, teeth, feet, heart and lung sounds)
- Blood and liver sample analysis to assess the need for treatment with trace elements
- Cu, Se, I for deficiencies
- Zinc boluses for facial eczema
- Individual and mass treatment with antibiotics or coccidiostats for treatment/ control of cases/outbreaks (yersiniosis, leptospirosis, cryptosporidiosis, pasteurellosis etc)
- Employing pasture species that reduce the need for anthelmintic, trace element supplementation and avoid the risk of RGS
- Management to reduce disease transmission between livestock (MCF, JD, Tb, parasites)
- Fertilizers containing trace elements (Cu, Se, Co)
- Management and QA systems to optimise nutrition, minimise stress and provide adequate shelter (yersiniosis, MCF, trauma, cryptosporidiosis, pasteurellosis
- Selection for increased resistance to disease (yersiniosis, MCF, Tb, JD, RGS, parasites)
- Selection and management of pastures to avoid animal health risk, such as nitrate poisoning, fungal toxicities, and goitrogenic effects

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14 POTENTIAL FOR APPLICATION OF TECHNOLOGY FROM OTHER SPECIES TO DEER HEALTH ISSUES

Most of the diseases that affect deer have a similar or equivalent clinical condition in other domestic livestock, especially cattle and sheep. For example there are many similarities between Tb, JD, leptospirosis, MCF, lungworm, GI parasites, facial eczema, and ryegrass staggers in deer and cattle and between, pasteurellosis, parapox, *B. ovis* and clostridial diseases of deer and sheep. Thus many inferences can be drawn in terms of epidemiology, clinical diagnosis, diagnostic tests, vaccines and treatments, although it is dangerous to assume that all features will be identical, as has been shown with *B. ovis*. Most diseases affecting deer have unique characteristics and differences from similar conditions in other animals. Some examples are:

Metabolism, requirements and some clinical manifestations of copper deficiency in deer are different from those of sheep and cattle. There are also big differences between deer species and subspecies, with wapiti being much more susceptible to deficiency than red deer.

Lungworms appear to be much less pathogenic in terms of damage to lung tissue in deer than in cattle. The species affecting deer is different to that affecting cattle and although morphologically, cattle and deer lungworm are very similar, the vaccine against the cattle lungworm offers less protection against lungworm in deer.

Deer can develop a much more acute form of Johne's disease, with outbreaks occurring in 8 – 15 month old animals, compared with the more chronic sporadic form that usually affects sheep and cattle at 2 – 4 years of age.

Drugs and anthelmintics do not necessarily have the same efficacy or length of activity in deer as they do in sheep and cattle. Levamisole, which is an effective anthelmintic in sheep and cattle, is ineffective in deer. Benzimidazoles also appear to have a shorter half-life in deer. Leptospiral and clostridial vaccines appear to promote lower antibody responses in deer than in other animals, although the protective nature of the titres has not been evaluated. Some antibiotics such as "Terramycin LA" have a shorter half life in deer.

Early cases of Tb in deer involve the head lymph nodes in over 80% of cases with relatively little lung involvement, whereas 50% of early cattle Tb cases involve the lungs.

MCF has occurred as outbreaks with multiple cases in deer. This is uncommon in cattle.

The behaviour of deer means that detection of disease in its early stage is usually more difficult that it is in sheep and cattle.

Wapiti appear to be especially susceptible to ryegrass staggers.

Young deer appear more susceptible to outbreaks of yersiniosis than other farmed species, but less susceptible to salmonellosis.

The suspect disease, theileriosis, described above appears to be more pathogenic in deer than in other species in New Zealand.

Thus it is extremely important that we understand the key differences and unique feature of the major diseases affecting deer. It is important to acknowledge that recognising these differences relies on the presence of veterinary practitioners on farms to investigate disease occurrence and manifestation.

15 CURRENT RESEARCH ON DEER HEALTH PROBLEMS

The following are main current research areas relating to deer health problems in New Zealand and overseas (as far as the authors are aware):

15.1 New Zealand

- Tb
- JD
- Lungworm
- GI parasites
- Leptospirosis
- Copper deficiency and supplementation
- Velvet antler removal
- Iodine supplementation
- Role of forages for parasite control
- Role of forages for trace elements
- Suspect *Theileria* (MAF Surveillance project)
- Cer HV-1 (Preliminary investigations)
- *B. ovis*
- Evaluation of vaccines and therapeutic agents (Pharmaceutical industry)

15.2 Overseas

- Tb
- JD
- Brucellosis
- CWD
- Parelaphostrongylosis
- Flukes
- MCF
- Protozoal infections
- Lyme disease
- Cryptosporidiosis

15.3 Related programmes

- Tb
- JD
- Parasites
- Leptospirosis

15.4 Linkages to FoRST and other funding (existing and potential)

15.4.1 Current funders

- FoRST currently funding deer JD and lungworm research at Invermay
- DEEResearch agreed in principle to fund JD research based primarily at Invermay
- DEEResearch agreed in principle to fund Tb research based primarily at DRL
- DFA/GIB funding deer leptospirosis epidemiology project at Massey
- Animal Health Board funded work on Tb vaccines in deer based at DRL/IDF/Invermay
- Animal Health Board funded work on diagnostic tests for Tb in deer based at DRL and Invermay/IDF

- DRL/ Deer farmer/Animal Health Board funded work on Tb resistance at DRL and Invermay/IDF
- VARNZ/AgResearch/Massey University/Western College of Veterinary Medicine, Canada, jointly funding evaluation of antler analgesia.
- Private company funding of products and forages at Massey University. Linkages exist between Massey University and research groups at AgResearch Invermay, AgResearch Grasslands, and Otago University.
- Massey University contributes financially through staff time and overheads, and a number of internal research funding agencies within the Veterinary and Agricultural disciplines.
- Fertiliser industry and DEEResearch funded research into copper requirements (AgResearch/Massey collaborative study)
- Recent trial work on copper requirements funded by the Canterbury Branch, DFA plus NZDFA plus Technology NZ

15.4.2 Potential funders

- Livestock industry consortium (Dairy/Meat/Wool/Deer) and FoRST may fund JD research
- AgVax has previously funded Yersiniavax trials and may continue to do so
- AgVax may contribute to other deer disease projects such as combined vaccines
- Commercial companies may fund trials, especially of vaccines, anthelmintics, anaesthetics and antibiotics from time to time

15.4.3 Linkages with allied work

- FoRST currently funding JD cattle vaccine research at Wallaceville
- FoRST currently funding JD sheep infection model research at DRL
- MeatNZ currently funding JD sheep vaccine and epidemiology work at Invermay
- Commercial companies fund anthelmintic and vaccine trials in other species

15.5 Deer health and disease research capability

15.5.1 AgResearch Invermay

- Dr Colin Mackintosh (BVSc, PhD) – Veterinary Scientist – general deer research, especially Tb, JD and parasites
- Marion Johnson (BAgSci, BSc Hons, MSc Vet.Paras) – Agricultural scientist and parasitologist – PhD study on lungworm

- Invermay Deer farm – 122 hectare deer farm, wintering 1100 deer (600 hinds, 300 sire and velveting stags, 200 weaners) plus indoor feeding pens for 80-100 deer, plus an Isolation Building for 8-12 deer.
- Infected Deer Farm (leased) – 20 hectares
- Woodlands Deer Farm (Southland) – 7 hectares
- Flockhouse Deer Farm (Manawatu) – 40 hectares
- Winchmore Deer Farm (Canterbury) – 62 hectares
- Deer Science Group – range of scientists and technicians undertaking deer research
- Otago Venison DSP on Invermay site and cooperative owners
- Genomnz Laboratory – national deer blood-typing and parentage testing laboratory
- Genomics Lab/Molecular Biology Lab (Invermay and Otago University) – ruminant genomics laboratory, markers for disease resistance, etc

15.5.2 AgResearch Wallaceville

- Dr G.W. de Lisle (BVSc, Dip. Micro. Biol., PhD) Microbiology, Dr. B.M. Buddle (BVSc, Dip. Micro. Biol., PhD) – Immunology, Dr D.M. Collins (PhD) –molecular biology of mycobacteria. Tb and Johne's group and culture laboratory –expertise in Tb diagnosis, Johne's disease diagnosis, vaccine development etc
- Parasite group – Led by Dr C. Shoemaker (PhD) Largest parasitological research group in NZ.
- Ruminant Immunology – Dr. W. Hein (BVSc, PhD)

15.5.3 AgResearch Grasslands

- Dr Neville Grace (BAgSci, MAgSci, PhD)- Scientist – trace elements, especially copper
- Biochemistry Group collaborating with Massey University researchers on plant compounds with anti-parasitic activity
- Aorangi deer farm - 16 hectares

15.5.4 University of Otago, Disease Research Laboratory (DRL)

- Professor Frank Griffin (PhD) – Immunologist
- Scientists, technicians, post-graduate students, honours students – undertaking research projects especially relating to Tb and JD in deer and sheep

15.5.5 Massey University

- Professor Peter Wilson (BVSc, PhD, MACVSc)–Veterinarian, deer health and diseases, production and welfare.Dr Dave West (BVSc, PhD, FACVSc) and AnnRidler (BVSc)– *B. ovis* research
- Dr Pomroy (BVSC, PhD)– parasite research
- Dr Allan Murray (BSc PhD) – JD research
- Professor Tom Barry (BAgSci, PhD, DSc) and Dr Simone Hoskin (BAgSci Hons, PhD), – grazing/food/parasite interactions
- EpiCentre – Associate Professor Peter Davies (BVSc, PhD) and Dr Cord Heuer (DVetMed) expertise in veterinary epidemiology and food safety
- Drs Julie Collins-Emmerson (PhD) and Ann Midwinter (PhD) leptospirosis laboratory science
- Dr Allison Quinn (BVSc) Trace elements
- Other staff with expertise in pathology, microbiology, agronomy and fundamental animal sciences are employed as needed.
- Post-graduate students

The Massey University Deer Research Unit is 24 Ha, with ~100 breeding hinds and replacements, ~70 venison-producing weaners, and ~12 hand-reared rumen, abomasal and oesophageal fistulated castrated stags for digestion and metabolism studies. It has a large purpose-built facility housing 15 individual metabolism cages. This building can be used for other deer research requiring housing. The veterinary clinic has a full surgical facility and surgical expertise.

Massey University has a suite of research and diagnostic laboratories.

15.5.6 Lincoln University

- Dr Alastair Nicol (PhD) and Professor Andrew Sykes (PhD): Trace elements and internal parasitism
- Deer farm - 20 hectares
- Indoor feeding facilites for 28 individual deer and pen facilities for 80 deer.

15.5.7 Diagnostic Laboratories

- Veterinary expertise and capacity for servicing research projects.
- Alpha Scientific Ltd
- Ruakura AgriQuality
- Batchelor AgriQuality
- LabWorks Animal Health Ltd
- LABNET Invermay

15.5.8 National Centre for Disease Investigation (NCDI) and MAF Biosecurity

- Surveys, disease surveillance, investigation of new or emerging diseases, exotic agent investigations

15.5.9 Landcare

- Dr Graham Nugent (PhD)– wild deer studies including Tb

15.5.10 Commercial companies

- R & D on commercial products related to deer and carry out trials for registration.

16 RESEARCH TOPICS AND PRIORITIES

In determining research priorities, consideration must be given at two levels: industry, and individual farm. In turn, industry level prioritisation is driven by multiple market imperatives of access in accordance with international disease status according to OIE standards, product (food) safety, and farming systems that provide for freedom from disease, among the other four freedoms defining animal welfare. For example, some diseases such as salmonellosis and parapoxvirus may have a very small importance to the individual farm, but could have a major impact on the market if contamination of venison or velvet, respectively, were to occur, and cause a human disease epizootic. The deer industry already has experience with *Toxoplasma* in this regard. Furthermore, other diseases, such as clostridial diseases, may not be common but cause severe suffering when they do occur. There are easy, apparently efficacious and low cost preventive measures existing, which should be considered by the industry to protect the marketing image of the production system.

At the farm level, disease priorities should be determined by two main considerations. First is the effect of diseases on the financial viability or profitability of the farm. Second is the disease impact on human health and Occupational Health and Safety imperatives for farm staff and workers in the production chain.

With respect to the first consideration, there are a range of direct and indirect effects of clinical and subclinical diseases, as well as time spent involved in preventative measures such as Tb testing and lost opportunities (for example JD may prevent live sales).

With respect to the second consideration, leptospirosis is currently the most important disease in this regard, although other diseases may also be important, including Tb, yersiniosis, parapoxvirus, salmonella and possibly Johne's disease because of its suspected link with Crohn's disease. There may also be psychological effects on the mental health of the farmer and his family (eg of serious outbreaks of Tb, JD or yersiniosis affecting the viability of the enterprise).

The following is a priority list of disease/health research topics. This list is based on risk at the market only for deer products, and not live deer or deer genetic material, since the latter is unlikely to be a significant financial component of the industry. The rankings are from highest to lowest, in the consensus opinion of the authors, proposing market and on-farm scores out of 5:

	Market	**On-farm**	**Combined**
Tuberculosis	5	5	10
Johne's disease	5	5	10
Injury and trauma prevention	3	5	8
Leptospirosis	3	3	6
Verify venison zoonotic risks	5	0	5
Velvet antler removal	3	2	5
Lungworm	1*	5	5
Effects of subclinical disease	1*	5	5
GI parasites	1*	4	4
Trace elements, particularly copper	0	4	4
Perinatal deaths	0	4	4
Mortality survey	0	3	3
Ticks	0	3	3
MCF	0	3	3
Suspect Theileria	0	?	?
Cer HV-1	0	?	?

* conservative estimate due to potential residues

Of the diseases present in this country, Tb and JD continue to pose the biggest threat to the New Zealand deer industry in terms of market access, and ultimately in financial impact.

(a) Tb is relatively well understood and, although not perfect, the diagnostic tests allow significant control of the disease in farmed deer at the herd level. However, at the individual animal level there are a few situations where the infection may not be eradicated from a herd by a test-and-slaughter programme, because some infected deer remain test negative. Nevertheless, as the incidence declines it will become exponentially more difficult to reduce the prevalence further with existing technology since deer will always be at risk as long as there are other deer and wildlife reservoirs infected. In the absence of infected feral vectors, the disease can be eradicated from problem herds by depopulation, as is successfully applied in other countries such as Canada. The eradication of bovine tuberculosis from deer will require the elimination of spread of infection from wildlife to farmed deer. The majority of the generic research on bovine tuberculosis will be focused on stopping the spread from wildlife into farmed deer herds. However, there will continue to be a need for some research that focuses directly on tuberculosis in deer. This should focus primarily on improving diagnostic techniques to increase the efficiency and cost of Tb diagnosis and surveillance, and secondarily on exploring genetic resistance as a means of reducing the number and severity of herd breakdowns. The future of vaccination for farmed deer should be evaluated concurrently with testing strategies that can differentiate vaccination from infection responses. The investigation of oral vaccines for at-risk wild deer populations is a relatively high priority.

(b) JD is a serious problem because of growing on-farm production and potential market-related issues. Currently we lack the fundamental tools to deal with JD in deer. The available immunological diagnostic tests have poor sensitivity and poor specificity due; a) to the similarity between *M. ptb* the rest of the *M. avium* group, resulting in cross-reactivity, b) to the intra-cellular nature of the disease, which allows the organism to hide away from the immune system, c) the initial cell-mediated host reaction, which requires expensive and technically difficult tests to detect, and d) the fact that an antibody response is a late event and signals serious clinical disease. Culture is relatively sensitive but difficult and expensive. This lack of tests makes thorough epidemiological studies difficult and expensive. There is an urgent need to better understand the risk factors that cause herd serious outbreaks in weaners. There is currently no vaccine licensed for use in deer, and any live or killed bacterin is likely to cause interference with the Tb control scheme. However, it is likely that vaccination may be the most cost-effective strategy to prevent clinical losses in young deer in venison production operations if the disease continues to spread and cause severe losses in individual herds. In that situation, vaccine may be available in identified animals that must be slaughtered before the age of 2-years, and not enter the breeding population in the case of females. Ultimately it may be possible to select for increased genetic resistance to help combat this disease, because the severe outbreaks in yearling animals suggests that deer are more susceptible to JD than sheep and cattle in New Zealand.

(c) Injuries are clearly an important component of clinical losses on deer farms, and potentially, in the perception of the consumer, farming systems that result in high injury rates could not be considered welfare friendly. Factors that contribute to injuries need to be studied, so farming practices can be modified to better provide for the safety of the deer. Indeed, the importance of this issue goes beyond simply the occurrence of injury. It needs to address the question about stress in deer farming *per se* since this will also impact on the susceptibility to a range of diseases and parasites, by reducing the capacity of the immune system. Thus, this aspect of

health needs to be built into a broad study of deer welfare, including methods of measuring wellbeing, interrelationships with the physical facilities, environment, and human interaction.

(d) Leptospirosis poses a human health risk and the planned epidemiological study and vaccine trials should enable better prevention and control of this disease. The most significant component of this research should be to reduce the risk of transmission to humans involved in the production chain.

(e) Lungworms are the major parasitic problem for deer farmers and the heavy reliance on anthelmintics is expensive and may not the ecologically sensible or sustainable in the long term. Lungworms are also the major impediment to totally organic deer farming and a vaccine or plants or “natural” compounds with anthelmintic properties need to be developed. Improved methods of predicting pasture challenge will allow more strategic use of anthelmintics. Improved methods for accurate and quantitative diagnosis of lungworm infection are needed to compliment reduction of chemicals for prevention, ie. Reduced use of chemicals will require increased surveillance of infection.

(f) GI parasites are likely to become increasing important in the future as deer farming increases and intensifies and as global warming changes the farming environment in New Zealand. Comments above in relation to lungworm apply to GI parasites.

(g) Verification of zoonotic organism status of venison
Food safety is increasingly becoming a serious international marketing issue. There are many organisms that have the potential for affecting food safety and this aspect of preventive health needs to be addressed. The preventive medicine programmes operating on the farm, the management and handling of animals on the farm immediately prior to transport, during transport and in lairage is likely to have a bearing on the potential for pathogenic organisms to multiply in the gastrointestinal tract enter the blood stream or soil the skin of the animal, thus increasing the risks of contamination of the carcass

(h) MCF remains a significant cause of wastage but research into it is hampered by the inability to isolate the causal virus. Dr Hugh Reid at The Moredun in Scotland, who has made most progress in recent times into this disease, is still researching the problem and it may be possible to develop a collaborative project in the future.

Of the exotic diseases CWD and FMD pose the biggest threats. As well as supporting all efforts to prevent the introduction of these diseases in this country, we must continue to monitor the international literature and maintain close links with research groups in North America. We must stay up-to-date with diagnosis, epidemiology and control of both these diseases, especially CWD, which could be introduced, it could arise spontaneously in New Zealand, or it could already be here in deer imported from North America before the borders were closed. This is achieved by the presence of a vigilant and knowledgeable practising veterinary workforce, combined with MAF, who has a role in maintaining a watching brief on international disease trends.

17 A TWO PAGE POPULAR SUMMARY ARTICLE

Review of Diseases of Farmed Deer

Dr Colin Mackintosh, AgResearch Invermay
Professor Peter Wilson, Massey University.

This review summarises the main diseases that currently affect farmed deer in New Zealand, together with a few diseases that have recently appeared and are starting to cause problems. It also summarises a number of diseases that occur in deer overseas and would cause problems here if they were introduced. It provides a summary of the economic impacts of deer diseases, the role of preventive medicine programmes and proposed priorities for research in the future. This research will provide information to enhance deer health, welfare and production, and protect market access into the future.

Over the last 30 years the capturing, domesticating and farming of deer has resulted in a steep learning curve to deal with health and diseases issues. There was no precedent elsewhere in the world, so much of the world's understanding of deer diseases has evolved from New Zealand. Wild deer appeared to be very healthy, but their capture and adaptation to a farming environment resulted in wastage due to misadventure, trauma and stress related diseases such as malignant catarrhal fever and yersiniosis. Initially the deer tended to be kept in large paddocks on poorer land, but increasingly farmers found that they could be successfully farmed on more productive pasture land. This led to increased numbers of animals on smaller areas and this soon led to parasite problems, especially lungworm. In the late 1970s tuberculosis (Tb) became a major problem. Trace element deficiencies, especially copper, became apparent in the early 1980s and then Johne's disease first arose in the late 1980s. Concurrently, a wide range of other herd or individual animal diseases of a more sporadic nature, similar to those seen in other domesticated livestock, have been encountered.

Research and development over the last 25 years has led to major advances in understanding of deer health, and technology for controlling and preventing most diseases. This has been achieved by the combined efforts of researchers at Invermay, Wallaceville, Massey University, Otago University, diagnostic laboratories and Ministry of Agriculture and Fisheries (MAF), as well as veterinary practitioners.

- The development of Tb tests has led to the ability to control and eradicate Tb on deer farms, in the absence of reintroduction from feral vectors. The national deer Tb control scheme was developed as a partnership between the deer industry, veterinary practitioners and MAF.
- Improvements in management, plus the development of Yersiniavax, have provided technology that can significantly reduce the risks of serious outbreaks of yersiniosis.
- Anthelmintics have been developed, trialled for efficacy and registered for deer and these, plus diagnostics tests and good management, are able to effectively control lungworm and gastro-intestinal parasites. However, continued reliance on anthelmintics may not be sustainable in the long term. Anthelmintics are not acceptable for organic farming other than for treatment of clinical illness.
- Improved management systems and the introduction of the Deer Farm Quality Assurance scheme have reduced stress and trauma of deer on farms. Improvements in pastures and provision of shelter have helped to reduce stress-related disease such as malignant catarrhal fever, yersiniosis, parasites and ryegrass staggers.

However, despite the availability of technology for the control and prevention of many diseases and subclinical losses on deer farms, there still appears to be significant preventable wastage occurring. Thus, wider application of existing technology would significantly improve deer health, welfare and profitability. Planned animal health

programmes integrated with good management have the potential to maximise productivity and minimise losses.

Despite progress in many areas, there are still a number of disease and health issues that are a serious threat to the deer industry.

- Bovine tuberculosis, which is a potentially serious zoonosis, continues to be a threat in vector risk areas. The development of new diagnostic tests, vaccines and selection for resistance will provide additional tools to limit this risk.
- Johne's disease is potentially the most serious threat to the New Zealand deer industry and is the disease for which there are the fewest tools to control it. New diagnostic tests, an effective vaccine and epidemiological information on risk factors are the highest research priorities. There is also great potential for selection for increased resistance to Johne's disease, which may be related to Tb resistance.
- New developments in the field of genomics may also lead to the ability to select for resistance to a raft of other disease including yersiniosis, ryegrass staggers and parasites.
- Leptospirosis does not appear to cause major production losses in farmed deer on a national basis, but serious outbreaks have occurred on individual farms. It is a serious zoonosis and the risk for workers in the deer industry may be reduced by vaccinating deer, as it has in the dairy and pig industries.
- Research has led to improved understanding of trace element requirements of deer, and data for interpreting, diagnosing, treating and preventing trace element problems should reduce losses and result in more cost-effective production.
- This review has highlighted that trauma is a significant but largely overlooked cause of loss on deer farms, and research into predisposing factors and preventive measures is warranted.
- Malignant catarrhal fever remains an enigma, but the incidence appears to be declining.

The introduction of exotic diseases remains a major threat to deer farming in New Zealand. While foot-and-mouth disease is a threat to all livestock enterprises, chronic wasting disease (CWD) is probably the biggest threat to deer farming. It has severely damaged the deer industry in North America and all efforts must be taken to prevent its introduction to this country. The deer industry must limit the risk of introduction of exotic disease by maintaining conservative criteria for importation of live deer and genetic material.

The financial impact of a range of clinical and subclinical diseases, and mortalities on deer farms is difficult to assess because there are insufficient survey data of their prevalence, causes or production losses. However, estimates suggest that clinical disease and current disease control measures may cost the deer industry $27 million per annum currently, and it may be speculated that at least that amount may be lost due to loss of production from subclinical diseases, resulting in costs of over $50 million. Thus, investment to improve technology for disease diagnosis, control and eradication should yield significant dividends for the deer industry.

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