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# Investigations into common farm management practices and diseases on alpaca farms in New Zealand

A thesis presented in partial fulfilment of the requirements for the degree of

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## Abstract

Alpacas are New World Camelid reared worldwide for fibre and meat, and since the 1980's, when they were reclassified from zoo animals to livestock, have become an increasingly popular farm animal in New Zealand. However, there is limited research performed on alpacas farmed under New Zealand conditions. Therefore the aims of this thesis were to determine common farming and health care practices among New Zealand alpaca farmers, and to determine the prevalence of certain diseases known to affect alpacas including *Candidatus Mycoplasma haemolamae*, bovine viral diarrhoea virus (BVDV) and parasitism in New Zealand.

In the first part of this study, a survey was administered to 18 alpaca owners from 9 regions of New Zealand, encompassing 1065 farmed alpacas.

Faecal egg counts were used on 75% of farms in their worming regime while on 62.5% worming was a scheduled task. Macrocylic lactones were the most popular class of anthelmintics, with Dectomax© the most commonly used of the 15 products mentioned. Ten of 18 farmers always used the same deworming product on their farm. Hand mating (64.7%) was preferred over paddock mating with females being bred around the

same time on 87.5% of farms. Repeated failure to conceive was the most common way to diagnose reproductive failure, and 12/17 farmers gave up to 3 matings before reporting reproductive failure. Spit off is the most common way to diagnose pregnancy on farms (88.2%). Vaccination for clostridial diseases and vitamin D administration occurred on 94% and 100% of farms respectively. Ten of 17 farms routinely weigh crias and 94% of farmers ensure crias suckled within 24 hours. Between 2012 and 2014 more crias died per farm (3.31) than in other management groups.

Congenital defects were the most common case of deaths in crias. The average deaths per 10 alpacas for the same period was 1.94. Voluntary tuberculosis testing was performed on 13/18 farms; facial eczema and zinc supplementation occurred only on farms in the North Island, and ryegrass staggers occurred on both islands.

The second part of the study was to determine the prevalence of *Candidatus* Mycoplasma haemolamae (CMhl), BVDV and gastrointestinal parasitism, as such 206 blood samples and 143 faecal samples were collected from 12 regions.

The prevalence of CMhl in this study as determined by PCR was 0.97%, while antibodies for BVDV were found in 2.05% of alpacas and no animals were positive for BVDV antigen. Anaemia (PCV<25%) was found in 21.76%

of animals sampled and 23.07% of alpacas had a significant gastrointestinal parasite burden (over 200epg). In New Zealand alpacas, anaemia was more likely to be associated with gastrointestinal parasites, rather than infection with CMhl.

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# CHAPTER 1

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## INTRODUCTION

Alpacas are New World camelids (from the Camelidae family) originating in South America. They are included in the suborder Tylopoda (padded foot) which includes the camelid (Camelini) and llama (Lamini) tribes [1].

The Lamini tribe includes four species: two wild, the guanaco (*Lama guanicoe*) and the vicuna (*Vicugna vicugna*), and two domestic, the llama (*Lama glama*) and the alpaca (*Vicugna pacos*). The alpaca was previously classified as (*Lama pacos*) however there is close genetic similarity between the vicuna with the alpaca which led to the reclassification of alpacas as *Vicugna pacos* [2, 3].

The New World camelid originated in North America and migrated via Central America to South America; here the South American camelids (SAC) were heavily relied upon by the Incas for transportation and fibre production [4]. In the 16<sup>th</sup> century the Inca government controlled state herds supplying pack llamas for their royal armies and alpaca wool for textile production [5].

Today alpacas are still reared worldwide for their fibre. While obtaining figures to show the growth of the fibre industry is difficult, different factors in some countries boosted the alpaca population size and the fibre industry. In the USA in 2010 the cost of buying breeding alpacas was entirely tax deductible [6]. This encouraged population growth in the USA alpaca fibre industry, and the USA now has the second largest alpaca population outside of South America, after Australia.

Recent alpaca population figures indicate that Australia has approximately 117,000 alpacas, USA approximately 53,000 alpacas, Canada and UK each with 23,500 and 20,000 alpacas respectively; these are listed as the larger alpaca populations in countries outside of South America [7, 8]. The New Zealand alpaca population size of ~16,000 which ranks it 5<sup>th</sup> among countries that farm alpacas outside of South America. Other nations with

significant alpaca populations include South Africa (~800 alpacas) and Switzerland.

Alpacas were first imported into New Zealand from Australia around 1847 but it is only after the reclassification of alpacas from zoological to farm animals and the creation of importation protocols in the late 1980's that the population of alpacas steadily increased [9, 10].

This steady increase in alpaca population is typical of reports of alpaca numbers outside of South America but even though the trend is increasing the actual population size differs depending on the source of the information [11]. Figures reported by the 2008 New Zealand National Census, reported the alpaca and llama population as 11,847, which increased to 14,168 in 2012 and 15,804 [12]. The figures reported by the Alpaca Association of New Zealand (AANZ) for alpaca number in July 2008 are higher than those of the census for the same year even though they reported only alpaca numbers[13]. This may be attributed to either the AANZ reporting registered alpacas from current and previous years or the national census recording only adult alpacas. Nevertheless the overall trend is towards increasing numbers of alpacas in New Zealand.

With alpaca populations increasing worldwide there are increased numbers of farmers transitioning from farming alpacas as part of a



lifestyle block to developing commercial opportunities; as such there is a need for increased knowledge on the management and welfare of alpacas [11]. A study done in 2006 in New Zealand found that there are many operations that have the intention of exploiting commercial opportunities in alpaca farming [14]. This may see commercial alpaca farming becoming more common, and subsequent intensive management systems may have a negative impact on alpaca health and well-being.

The aim of this study is to answer questions about diseases seen worldwide in alpacas and their prevalence in New Zealand. In addition the study aimed to collate data from alpaca owners with regards to their approaches to the health and welfare of the alpacas in their care.

## ALPACAS

Alpacas are domesticated, placental mammals from the family of even-toed feet (artiodactyls) [5, 15, 16] They are herbivores from the family of New World camelidae and were originally found in the Andes of Peru, Bolivia, Argentina and Chile, in grassy and mountainous habitats that have extremes in temperature. They have heavy wool coats to cope with the low temperatures of the Andes, as such they are commercially reared worldwide for their fibre, and in some South American countries for their meat [15]. In 1983, a report suggested that around 10,000 metric tons of alpaca meat was consumed annually in Peru [15].

The average height of an alpaca is about 99 cm at the withers; with males (machos) weighing around 60 - 80 kg, females (hembras) 55 – 60 kg, and crias weighing about 8.8 kg at birth [15, 17, 18]. There are two distinct breeds of alpaca, Huacaya and Suri, which have different fibre characteristics. The Huacaya has shorter, highly crimped fibres and Suri have a longer wavy fibre. The fibre colour varies from white to black, and all intermediate shades, with the colour tending to be uniform across the body [5, 15].

South American camelids in their natural habitat live in family groups of about 16 individuals, with a male defending his feeding territory.

Commercial farmers have found that alpacas need to be housed with other alpacas and research confirms that providing alpacas with companion alpacas decreases the stress associated with spatial and visual isolation [19].

Alpacas are forestomach fermenters with a large compartmentalised forestomach [20]. In cattle, sheep, and other ruminants the forestomach has four compartments while the alpaca forestomach is divided into three, C1, C2 and C3 (the glandular stomach).

Alpacas are reflex (copulation-induced) ovulators who are sexually receptive at 1 year of age, and ovulate 24-36 hours after coitus. The gestation period is highly variable (330 - 360 days) and is affected by body size, feeding strategy and social structure of the group [15, 21, 22].

Parturition usually occurs in daylight hours, crias are born well developed and can walk shortly after birth [15, 23].

The alpaca erythrocyte has more concentrated haemoglobin than other domestic species, is smaller in size (3.2 x 6.5  $\mu\text{m}$ ) and has an elliptical shape [24]. These features increase the surface volume ratio of the erythrocyte, and make them more resistant to osmotic lysis [25]. It also

allows for greater oxygen binding capacity, as such their red blood cells contain haemoglobin with a high oxygen saturation ( $\geq 90\%$ ) making them well adapted to live at high altitudes [15]. All these erythrocyte features contribute to their ability to live with a low percentage of circulating erythrocytes without compromising respiratory function.

## ALPACA DISEASES

### **CANDIDATUS MYCOPLASMA HAEMOLAMAE**

*Candidatus Mycoplasma haemolamae* (CMhl), order *Mycoplasmatales*, family *Mycoplasmataceae*, was first diagnosed in llamas in 1990 as an *Eperythrozoon spp* and is present in llamas (*Lama glama*) and alpacas (*Vicugna pacos*) worldwide.

The organism was reclassified as CMhl in 2001 and is the only haemoplasma specific to South American camelids (SAC). It is 0.4 – 1.0 µm in diameter and is present as a coccoid or ring shaped basophilic organism located extracellularly in an indentation on the surface of erythrocytes [26-29]. Based on sequence similarity of the 16s rRNA gene CMhl is most closely related to *Mycoplasma suis* and *Candidatus Mycoplasma haemominutum* [30].

One of the primary aims of this study was to determine the prevalence of CMhl in New Zealand; therefore haemoplasma organisms are included in detail in this review.

## HAEMOPLASMAS

Mycoplasmas are small, pleomorphic, single cell, gram negative, extracellular, obligate bacteria that affect a wide range of species, both animal and plants [31, 32]. Mycoplasmas have circular, double stranded DNA which encodes only those gene products essential for life. They lack a cell wall and therefore are resistant to agents like penicillin and other antimicrobial agents that target the cell wall, however they are generally susceptible to antibiotics such as tetracyclines and fluoroquinolones [26, 32]. In 1928 *Mycoplasma coccoides* and *Mycoplasma haemocanis* were the first mycoplasmas discovered in mice and dogs respectively [26, 33, 34]. The most common Mycoplasmas in humans and animals cause respiratory disease and are relatively species-specific; they are listed in Table 1.

Mycoplasmas may also parasitise red blood cells (haemotrophic mycoplasma or haemoplasmas), and unlike the respiratory mycoplasmas these epicellular, obligate red blood cell parasites have not been successfully grown in culture [35]. They may be rod-shaped, spherical, or ring-shaped, and may be found individually or in chains across the red blood cell surface of a wide range of vertebrate animals [26, 36].

Table 1 Common respiratory diseases caused by *Mycoplasma* organisms in different animal species

Mycoplasma species	Disease	Animals Affected
<i>M. mycoides mycoides</i> small colony	Contagious bovine pleuropneumonia	Ruminants, mainly goats and cattle
<i>M. gallisepticum</i>	Chronic respiratory disease	Turkeys, chickens, game birds, pigeons and passerine birds
<i>M. hyopneumoniae</i>	Porcine enzootic pneumonia	Pigs
<i>M. ovipneumoniae</i>	Atypical pneumonia	Sheep, goats, mountain goats and big horn sheep
<i>M. pneumoniae</i>	Atypical pneumonia	Humans

Species of haemoplasma, for example *Mycoplasma haemofelis*, have been described in all continents, except Antarctica, and it is thus believed that this family of organisms has a worldwide distribution [37].

Historically, haemoplasmas were classified as rickettsial organisms (in the order *Rickettsiales*) family *Anaplasmataceae*. However sequence analysis of 16S ribosomal RNA (rRNA) has made phylogenetic classification more precise and has led to the reclassification of *Eperythrozoon* and *Haemobartonella* as members of the genus *Mycoplasma*. The group of haemotrophic mycoplasma within the *Mycoplasmataceae* family have been given the distinct name of haemoplasmas [30, 38]. A number of haemoplasmas have the term 'Candidatus' appended to their genus and

species name in order to give them provisional status, because their taxa are new and incompletely described, for example '*Candidatus* *Mycoplasma haemolamae*' and '*Candidatus* *Mycoplasma turicensis*'. Table 2 lists the common haemoplasmas and the species affected [27, 39-45].

Table 2 Haemoplasmas and the mammalian species affected

Species affected	Haemoplasmas
California Sea lion	<i>Candidatus</i> <i>Mycoplasma haemozalophi</i>
Cat	<i>Candidatus</i> <i>Mycoplasma haemominutum</i> <i>Candidatus</i> <i>Mycoplasma turicensis</i> <i>Mycoplasma haemofelis</i>
Cattle	<i>Candidatus</i> <i>Mycoplasma haemobos</i> <i>Mycoplasma wenyonii</i>
Capybara	<i>Mycoplasma coccoides</i>
Cheetah	Haemoplasmas detected, not yet named (in the <i>haemocanis/haemofelis</i> group)
Darwin's fox	<i>Mycoplasma haemofelis</i> <i>Mycoplasma haemocanis</i> Haemoplasmas detected, not yet named
Dog	<i>Candidatus</i> <i>Mycoplasma haematoparvum</i> <i>Mycoplasma haemocanis</i>
Horses	Haemoplasmas detected (in the " <i>haemofelis</i> " cluster)
Human beings	Haemoplasmas detected, not yet named
Llamas and alpacas	<i>Candidatus</i> <i>Mycoplasma haemolamae</i>
Mice	<i>Mycoplasma coccoides</i> <i>Mycoplasma haemomuris</i>
Monkey (owl, Squirrel, Cynomolgus)	<i>Candidatus</i> <i>Mycoplasma kahaneii</i> <i>Candidatus</i> <i>Mycoplasma aoti</i> (proposed) <i>Candidatus</i> <i>Mycoplasma haemomacaque</i> (proposed)
Opposum	<i>Candidatus</i> <i>Mycoplasma haemodidelphis</i>
Pigs	<i>Mycoplasma suis</i> <i>Mycoplasma parvum</i>
Rat	<i>Mycoplasma haemomuris</i>
Sheep and goat	<i>Mycoplasma ovis</i>
White-tailed deer	Haemoplasma detected, not yet named



## CLINICAL FINDINGS OF HAEMOPLASMAS IN DIFFERENT ANIMAL SPECIES

Haemoplasmas have been identified for a number of years as disease causing organisms in a variety of animal species (See Table 2). In the 1920's, *Mycoplasma coccoides* (formerly *Eperythrozoon coccoides*) in mice, and *Haemobartonella canis* in dogs were observed to cause disease. In 1928, Schilling described small ring-shaped bodies on mice red blood cells that stained reddish brown with Giemsa or Wright's stain, and the organisms were later shown to cause anaemia in splenectomised mice [34, 46] .

As indicated in Table 2 there are a number of different haemoplasmas that affect different species causing acute haemolytic anaemia and various chronic diseases in their vertebrate hosts [42, 45, 47]. The clinical spectrum of infection ranges from asymptomatic to life-threatening, depending partially on host susceptibility and strain of bacteria contracted [39, 44, 48]. For instance, acute infection of cats with *Mycoplasma haemofelis* or *Candidatus Mycoplasma turicensis* is associated with mild to severe anaemia, but infection with *Candidatus Mycoplasma haemominutum* results in less severe anaemia or is asymptomatic [49] . Animals may be predisposed to acute infection due to age, the presence of concurrent disease, immunosuppression, or splenectomy. In chronically

infected animals, clinical disease may be occult or poorly defined. Little research has been done to determine the clinical features of disease in many of the non-domesticated animals that may be infected with haemoplasmas.

### Mice

*Eperythrozoon coccoides*, renamed *Mycoplasma coccoides*, causes mild haemolytic anaemia in laboratory and wild mice, and has been shown to cause disease in rats and rabbits experimentally. In mice, it is characterised by splenomegaly and generalised lymphadenopathy. The parasitaemia peaks between day 2 to 5 after infection, during which there is a haemolytic anaemia, however by day 8 the haematocrit is usually back within the normal range. Splenomegaly and latent infection may persist for months, and possibly for the life of the animal but without recognizable disease. Low numbers of organisms may appear sporadically for brief periods in the peripheral blood of chronic carriers. *Mycoplasma coccoides* can cause complex pathophysiological changes that affect the host response to infection, and concurrent infections of *M. coccoides* and other infectious agents can markedly alter the course of disease. For example, conversion of the benign hepatitis caused by mouse hepatitis virus (MHV1) to a fatal infection may occur when there is concurrent

infection with *Mycoplasma coccoides*, and normally harmless amounts of endotoxin produced by *Salmonella typhimurium* results in high mortality in *Mycoplasma coccoides*-infected mice. In contrast, concurrent infections with *M. coccoides* and *Plasmodium berghei* result in inhibition of growth of the *Plasmodium* organism, thus permitting longer survival of the mice [33, 50-52].

### Dogs

*Mycoplasma haemocanis* (reclassified from *Haemobartonella canis*) causes chronic, sub-clinical haemoplasma infections, and infection has been reported in both immunocompetent and immunocompromised patients [53-55]. *Mycoplasma haemocanis* is linked to clinical anaemia in splenectomised or immunocompromised infected dogs but rarely causes anaemia in dogs with normal spleens and functional immune systems. The clinical signs of acute disease include anorexia, lethargy, weight loss, fever, infertility, and haemolytic anaemia which may lead to death in severe cases [26, 32].

### Pigs

*Mycoplasma suis* can cause acute haemolytic disease and sometimes death in pregnant sows immediately prepartum, young piglets at weaning,

and feeder pigs under stress. More commonly, mild anaemia and icterus, poor growth rates and increased susceptibility to other infectious diseases is seen in infected nursery and feeder pigs [56]. *Mycoplasma suis* infection in sows may result in pyrexia, anaemia, icterus, anorexia, depression, decreased milk production, and poor maternal behaviour [57, 58]. Persistent carriers of the organism may also occur [26].

### Sheep

Infection of sheep with *Mycoplasma ovis* (Mo) may occur as a result of stress, concurrent disease or immunosuppression and result in haemolytic anaemia, jaundice and decreased exercise tolerance. The disease varies in severity but clinical signs last approximately 14 to 28 days. Disease caused by *Candidatus Mycoplasma haemovis* (CMho) is more severe in young animals and pregnant sheep on a low plane of nutrition while animals on good quality feed or pasture, with adequate trace element status develop less severe anaemia [26]. Both Mo and CMho are poorly pathogenic in healthy animals under good farming conditions but animals chronically infected with Mo, CMho or both may have a mild anaemia, decreased weight gain and wool production [59].

## Cats

*Mycoplasma haemofelis* (Mhf), *Candidatus Mycoplasma turicensis* (CMt) and *Candidatus Mycoplasma haemominutum* (CMhm) are the three most recognized haemoplasmas in cats. A fourth feline haemoplasma species *Candidatus Mycoplasma haematoparvum*-like, which has 99% morphology of *Candidatus Mycoplasma haematoparvum* has recently been identified [60]. Mhf is the most pathogenic species and is associated with a severe, regenerative anaemia [61-64]. Infections with CMt and CMhm are less severe and can be asymptomatic, resulting in carrier states [62, 64, 65] . *Mycoplasma haemofelis* can rapidly disappear from erythrocytes and then cyclically reappear, and this also is believed to allow development of a carrier state [31]. In acutely infected cats intermittent pyrexia is often seen, particularly in the acute stages of the disease [31]. Pale mucous membranes, lethargy, weakness, dyspnoea, tachypnoea, tachycardia, inappetance, dehydration, pica and weight loss are also seen in acute cases of haemoplasma infections. Splenomegaly and lymphadenopathy may occur, reflecting proliferation of macrophages and extramedullary haematopoiesis. Icterus, due to severe, acute haemolysis, is also occasionally seen [37, 62]. Chronic infection is not associated with anaemia, and carriers are common especially in CMhm infections. The

organism can be cleared with or without antibiotic treatment but factors that affect spontaneous clearance are undetermined [66].

In some studies retroviruses (feline immunodeficiency virus and feline leukemia virus) have been shown to be risk factors for haemoplasma infections and may exacerbate clinical signs of disease [26, 67-69].

### Cattle

*Mycoplasma wenyonii* and *Candidatus Mycoplasma haemobos* are two of the haemoplasmas that infect cattle. The features of *Mycoplasma wenyonii* infection include malaise, a drop in milk production and a regenerative, normochromic anaemia [70]. Hindlimb and udder oedema, pyrexia, rough coat, and prefemoral lymph node enlargement may be seen in dairy heifers, sometimes followed by loss of condition and depression, while scrotal oedema has been reported in males with *M. wenyonii* infection. Clinical signs gradually resolve and full recovery may take 10 days or longer [71]. The clinical significance of *Candidatus Mycoplasma haemobos* is unclear even though a study suggests it has a stronger effect on haematological parameters than *M. wenyonii* [70, 71].

## PREVALENCE AND TRANSMISSION OF MYCOPLASMAS

### Sheep

In sheep, haemoplasma infection is associated with any activity that transmits a very small number of infected erythrocytes from one sheep to another, for example, vaccination, ear tagging, reusing needles during herd immunization, and shearing. In sheep and goats *Mycoplasma ovis* may be transmitted by blood-feeding arthropods including ticks (*Haemaphysalis plumbeum* and *Rhipcephalus bursa*), mosquitoes (*Aedes camptorhynchus*) and *Culex annulirostris* [72]. The stable fly (*Stomoxys calcitrans*) is also a potential vector, and subcutaneous injection of sheep with a suspension of fly material has resulted in infection. Oral transmission has also been shown experimentally [26, 72]. There are no reported prevalence studies in sheep.

### Rodents

Early reports stated that the rat louse, fleas and mites were not vectors of haemoplasmas but others have since shown that *Mycoplasma haemomuris* can be experimentally transmitted by adults and nymphs of *Polypax spinulosa* (rat louse). Similarly, *Mycoplasma coccoides* has also been shown to be naturally transmitted by the rat louse [26, 73].

Transmission by the louse *Polypax serrata* is suggested to be mechanical, and transmission of *M. coccoides* may still occur after the louse has fasted for as long as 24 hours [74]. Transmission by mites (*Myobia musculi*, *Mycoptes musculinus* or *Radfordia affinis*) has not been documented [73, 74]. Experimental infections are produced usually by intravenous or intraperitoneal inoculation of infected blood, and by oral administration of citrated blood but attempts at transmission by smearing mucous membranes (eyes, nostrils and urogenital surfaces) with citrated blood have not been successful [75]. Haemoplasmas have also been detected in other rodent species, such as captive capybaras (*Hydrochaeris hydrochaeris*). A novel haemoplasma was detected by polymerase chain reaction (PCR) in 10 captive capybaras and 21 free-ranging animals [76].

### Cats

Despite substantial research into feline haemoplasmas around the world there is still some uncertainty around transmission of the organism. It is postulated that the routes of transmission are: social contact, arthropod vectors and vertical transmission. Fleas (*Ctenocephalides felis*) infected with *M. haemofelis* can transmit the organism and disease has been produced in a susceptible cat by this mechanism [77]. However transmission of *Candidatus M. haemominutum* by fleas was not



successful, however low numbers of fleas were suggested to be responsible for the lack of transmission in that case [78]. *M. haemofelis* and *Candidatus M. haemominutum* DNA have been isolated from fleas collected from cats and in flea faeces [79]. There are conflicting reports with regards to transmission of feline haemoplasmas by ticks [80]. The *Candidatus M. turicensis* DNA load in cat saliva and faeces was <400 copies so it is theorised that aggressive interactions are necessary for transmission of the organisms rather than oronasal exposure by mutual grooming and sharing of food dishes [80].

In New Zealand the prevalence of *Candidatus Mycoplasma haemominutum*, *Mycoplasma haemofelis* and *Candidatus Mycoplasma turicensis* was found to be 19%, 2% and 4% respectively. Combined *Candidatus Mycoplasma haemominutum* and *Mycoplasma haemofelis* infection occurred in 5.5% of cases and combined *Candidatus Mycoplasma haemominutum* and *Candidatus Mycoplasma turicensis* infection occurred in 0.5% of the cats tested [64]. The prevalences of feline haemoplasmas in some other countries are contained in the Table 3 below.

Table 3 Prevalence of feline haemoplasmas in selected countries

Country	Mhf %	CMhm %	CMt %	Reference
Australia	0.9	15.3	0.9	[81]
USA	4.8	23.2	6.5	[69]
Canada	0.7	3.3	0.1	[82, 83]
Japan	5.1	21	6.7	[84, 85]
Iran	63.6	54.5	18.2	[86]
Northern Italy	10.8	22.3	-	[87]

## Dogs

The natural means of transmission of canine haemoplasmas has not been definitively established. The brown dog tick, *Rhipicephalus sanguineus*, is likely to play a role as a vector and reservoir of canine haemoplasmas and has been shown experimentally to transmit *Mycoplasma haemocanis* [26, 88]. Cross bred dogs, and dogs kept in kennels were identified as risk factors for canine haemoplasma infection in a Portuguese study [89]. These could be related to the higher risk of exposure to fleas and ticks in kennels and that cross breeds were found more often in kennels than purebred dogs [89]. Table 4 shows the prevalence of canine haemoplasmas in selected countries.

Table 4 Prevalence of canine haemoplasmas in selected countries

Country	Mhc %	CMhp %	Mhc & CMhp %	Overall	Reference
France	3.3	9.6	2.6	15.4	[90]
Switzerland	0.9	0.3	-	1.2	[91]
Tanzania	19				[92]
Australia (Sydney)	0.8	0.8	-	1.6	[54]
Trinidad	4.9	2.7			[92]
North east Brazil	0.48				[93]
USA	0.6	0.8	-	1.3	[53]

## Pigs

Under experimental conditions arthropod vectors including lice, mosquitoes, and stable flies can transmit *Mycoplasma suis* infections in pigs [26]. Once pigs are infected with *M. suis* they remain lifelong carriers. *M. suis* has been shown to invade erythrocytes and is known to establish chronic infections. The bacteria can exist free in the cytoplasm of erythrocytes, in variable depth pits on the surface of erythrocytes, in vacuoles within the erythrocyte cytoplasm, and in deep invaginations covered by membrane material leading to the formation of invasion scars [94]. It has been postulated that the intracellular form could circumvent the killing action of antimicrobials and may be linked to the establishment of chronic infections [95]; further investigative work is needed to confirm

this [94]. A survey of 186 pigs by PCR for *Mycoplasma suis* in Southern Brazil found that 18.2% were positive for the organism [96].

## HAEMOPLASMAS IN ALPACAS

### WORLDWIDE PREVALENCE OF *CANDIDATUS* MYCOPLASMA HAEMOLAMAE

#### Peru

The prevalence of CMhl infection varies within different populations of SAC. At the La Raya Research Station in Peru, which houses approximately 3000 alpacas, 212 alpacas in a closed herd were sampled to determine the prevalence of CMhl. No animals under 9 months were positive for CMhl, while one of 40 (4.76%) 9 month old animals tested positive, and an 18 month old alpacas (4.76%) was also positive. The largest percentage of positive animals was those older than 18 months, with 25.93% of females and 55% of males positive for the organism. The overall infection rate was found to be 19.3%. Despite the disparity in prevalence, there was no statistically significant difference in infection rate between female and male alpacas [27].

#### Chile

Of the 108 alpacas that were sampled from 6 sites with closed herds in the Altiplano, 10 animals (5 male and 5 female) were positive for *Candidatus*

*Mycoplasma haemolamae* by PCR, equivalent to a 9.26% infection rate. Of the 6 groups sampled the highest percentage of animals positive in any herd was 44%, and only 2 of the 6 herds had positive alpacas. The mean packed cell volume (PCV) (30.3%) for positive animals was significantly lower than the mean PCV (33.24%) for alpacas that were negative. There was one anaemic animal, 1% of the CMhl negative group, while seven out of 10 in the CMhl positive group were anaemic animals in the CMhl positive group [27].

## Europe

Following the first reported case of CMhl in England, a further cross-sectional study was carried out to identify the prevalence of CMhl in south-east England. This study showed that the incidence of CMhl was higher in animals less than 2 years of age (40%) when compared with animals older than 2 years (22%), and overall 27.9% of females were positive, while 31% of males were positive for CMhl. Animals originating in the United Kingdom had an infection rate of 31.4% and those originating in Peru had an infection rate of 18.4%. There was no significant association between sex, country of origin, blood smear findings and molecular test results, and the overall infection rate of the sampled alpacas was 29%. Most of the positives were subclinical infections [29].

Blood samples were taken from 225 alpacas and llamas from Switzerland (194 animals) and Germany (31 animals). Of these, 169 were alpacas, 138 of which were uninfected, while 31 were positive for CMhl by real time PCR. The PCR positive animals were significantly older than the uninfected animals even though some animals less than 1 year of age were already infected. In this study it was found that the total protein content was significantly higher in animals positive for CMhl and there was a high frequency of infection in clinically healthy animals. The overall prevalence was found to be 18.7% and the likelihood infection was not affected by gender, species or country of origin [97].

### New Zealand

*Candidatus* Mycoplasma haemolamae (CMhl) was first confirmed in New Zealand in 2013. The infected alpaca had no clinical signs, but was housed on a farm with a severely anaemic animal. The anaemic alpaca died and blood samples from ten other animals in the same group were analysed for selenium, iron, copper, vitamin B12 and CMhl. The mean vitamin B12 levels in the in-contact animals was 120 pmol/L (normal range 150 to 430 pmol/L), with actual vitamin B12 values from 46 to 357 pmol/L. One of the 10 samples was positive by conventional PCR for a non-specific

haemotropic mycoplasma. This was later confirmed to be CMhl based on sequencing of the PCR product [98].

## CLINICAL SIGNS OF *CANDIDATUS MYCOPLASMA HAEMOLAMAE* INFECTION

The clinical signs of *Candidatus Mycoplasma haemolamae* in infected camelids include a mild to severe anaemia with variable regeneration, lethargy, depression, weight loss or reduced weight gain, hypoglycaemia and fever. Camelids may be asymptomatic carriers or in extreme cases present with collapse and death. The clinical signs often depend on the overall health status of affected animals [27, 97]. Young animals are suspected to be more susceptible to acute infection, with a massive parasitaemia and anaemia, especially when stressed or when there is concurrent disease [24]. Most commonly *Candidatus Mycoplasma haemolamae* causes subclinical infection, with adult alpacas potential carriers for life. Clearing of the bacteria by use of tetracycline antibiotics has not been successful [99].

## TRANSMISSION OF *CANDIDATUS MYCOPLASMA HAEMOLAMAE*

The mode of transmission of *Candidatus Mycoplasma haemolamae* is unknown, but is most likely associated with the transmission of infected blood. It has been theorised that the bacteria can be transmitted by ticks and other blood sucking parasites and/or by iatrogenic means. Further work needs to be done to confirm carrier animals as a source of the infection for transmission by blood sucking vectors.

A 2006 report suggested that transplacental infection was possible, even though transmammary spread could not be excluded [100]. In this report an uninfected dam delivered a heavily infected cria that later died. The dam was seronegative, the cria was 2 weeks premature and was delivered without complications. The cria suckled but within 48 hours of birth was observed to no longer be able to stand. The physical condition of the cria deteriorated and it became depressed, afebrile, hypoglycaemic, had a mild non regenerative anaemia, and was moderately dehydrated (so the anaemia was likely more severe than the numerical data indicated). The cria was assessed to have had adequate transfer of maternal antibodies and no external parasites were noted. A peripheral blood smear showed massive erythrocyte parasitemia and this was confirmed to be CMHl by PCR. Since the dam's milk was not tested for the presence of CMHl,



transmammary spread could not be ruled out. Transmission by blood sucking parasites in this case seemed highly unlikely and transplacental, transmammary or post natal inoculation especially during parturition was suggested [100].

However a later report suggested that *in utero* transmission of *Candidatus Mycoplasma haemolamae* is rare and colostrum from PCR positive dams is unlikely to transmit the parasite [101]. Specific CMhl PCR testing on blood and colostrum from 52 pregnant dams and crias showed that only one cria prior to colostrum ingestion was positive for CMhl, none of the 43 colostrum samples tested were positive. It should be noted that the dam who gave birth to the PCR positive cria was PCR negative and all 52 crias (including the one that tested positive) who were tested after colostrum ingestion were PCR negative for CMhl [101].

## DIAGNOSTIC METHODS OF HAEMOPLASMAS

Unlike the respiratory mycoplasmas, haemoplasma organisms are unable to be cultured *in vitro*, therefore traditionally diagnosis has relied upon cytological identification of the organisms on blood smear examination [29, 65, 80]. Smears may be stained with Romanowsky-type stains including Giemsa, Wright, Wright-Giemsa, and May-Grunwald-Giemsa

stains, and haemoplasma organisms usually appear as coccoid forms on the surface of erythrocytes, although rod and ring forms have also been observed [37, 80]. The inability to culture haemoplasmas *in vitro* has hampered the development of specific serological assays. There have been attempts using antigen derived from pigs heavily infected with *Mycoplasma suis* but the diagnostic specificity and sensitivity of the assays was poor, in addition carrier animals may be seronegative [102].

Blood smears for the diagnosis of haemoplasma infection can be insensitive and nonspecific. Stain precipitates and other erythrocyte features may be mistaken for organisms and improper drying or fixing and old or unfiltered stain can contribute to false positives (low specificity) [37, 71]. Even when blood smears are prepared shortly after blood collection in a clean environment, in order to prevent the haemoplasma from detaching from the surface of the erythrocyte, they have poor diagnostic sensitivity [71]. The number of organisms in the blood may be low in subclinical animals [99].

Acridine orange (AO) and direct fluorescent antibody staining methods were introduced as a diagnostic method and reported to be more sensitive than standard Romanowsky stains for demonstrating *M. haemofelis*. AO combines specifically with nucleic acids, and the organisms

appear bright orange with an undertone of yellow green [37]. However both these techniques are limited by the need for a fluorescent microscope.

Polymerase chain reaction (PCR) technology has permitted screening for suspected haemoplasma infections in a wide range of mammalian species. Specific amplification of the 16S rRNA gene using PCR enables specific targeting of haemoplasmas from anti-coagulated blood of infected animals. This has proven to be a more efficient means of diagnosing haemoplasma infection in cats when compared to cytology [80].

Real-time PCR assays have several advantages over conventional PCR. They are highly specific due to the use of a third oligonucleotide, a fluorogenic probe, and also allow quantification of haemoplasma DNA in the patient's blood, which helps assess the significance of the infection and the patient's response to antibiotic treatment [103]. Furthermore these assays are run as closed tube systems, reducing the risk of carry-over of PCR products as occurs in some conventional nested PCRs developed for haemoplasma diagnosis. The most noted disadvantage of PCR assays are that within a few days of antibiotic treatment the cat (proven positive for *Mycoplasma haemofelis*) may become PCR negative but become PCR positive again approximately one week after treatment is

suspended [61]. A PCR positive animal should always be interpreted together with clinical and laboratory findings as animals may be asymptomatic carriers [80].

In 2001, a polymerase chain reaction (PCR)-based assay was developed based on 16S rRNA gene of CMhl. In 2009, modifications to this method yielded a specific real-time TaqMan PCR for detection and quantification of part of the 16S rRNA gene (192 base pairs) of CMhl in llamas and alpacas [102].

## OTHER ALPACA DISEASES

### **GASTROINTESTINAL PARASITES**

Parasitic diseases (internal and external parasites) are a major health concern and the most common problem in South American camelids worldwide [104-106]. Gastrointestinal nematode infections are thought to increase in times of stress and their numbers increase in the rainy season in tropical regions and in late summer to autumn in temperate regions [107]. Nematode infestation has the largest impact on alpacas less than one year old, possibly due to immature immune system development [107]. Signs of parasitism are nonspecific and vary, but diarrhoea is the most common presenting sign and clinical disease causes severe economic losses [105, 108]. There is evidence to suggest that the productivity of alpacas is increased by regular treatment with anthelmintics, however some studies show that the reduction in egg shed on the pasture is only transient and numbers return to pre-treatment levels 4-5 weeks after treatment [104, 107]. There is increasing concern worldwide about anthelmintic resistance in alpaca herds since farms in Belgium and USA have reported resistance to macrocyclic lactone anthelmintics, and

benzimidazole in the USA [109, 110]. In New Zealand benzimidazole resistant *Haemonchus contortus* has been identified in sheep [111].

The clinical signs of gastrointestinal infestation in young alpacas include decreased weight gain, pale mucous membranes, mild to severe regenerative anaemia and sudden death with no clinical sign [108, 112]. In other farmed animals *Haemonchus contortus* is economically important and causes a severe regenerative anaemia when worm numbers are high; it is likely to be similar in alpacas especially in combination with nutritional stressors [113]. Table 5 lists the common gastrointestinal nematodes in alpacas and llamas in New Zealand and worldwide.

Table 5 List of common gastrointestinal parasites in alpacas and llamas worldwide Modified and taken from [105]

Location	Species of Nematode
C3	<i>Camelostrongylus mentulatus</i> *
	<i>Teladorsagia circumscripta</i> / <i>Ostertagia</i> *
	<i>Trichostrongylus axei</i> and <i>askivali</i> *
	<i>Marshallagia marshalli</i>
	<i>Haemonchus contortus</i> *
	<i>Graphinema auchenia</i>
	<i>Mazamastrongylus (Spiculoptera) peruviana</i>
Small Intestine	<i>Lamanema chaezi</i>
	<i>Nematodirus</i> spp. including <i>N. lamae</i> *, <i>N. filicollis</i> , <i>N. spathiger</i> , <i>N. helvetianus</i>
	<i>Cooperia</i> spp*. including <i>C. mcmasteri</i> , <i>C. oncophora</i> , <i>C. surnabada</i>
	<i>Trichostrongylus</i> spp. including <i>T. vitrines</i> , <i>T. longispicularis</i>
	<i>Capillaria</i> *
	<i>Strongyloides</i> spp
Large Intestine	<i>Trichuris</i> spp. including <i>T. tenuis</i> , <i>T. discolor</i> , <i>T. skrjabini</i>
	<i>Oesophagostomum</i> spp including <i>O. columbianum</i> , <i>O. venulosum</i>

\*nematodes reported in alpacas in New Zealand

## ANAEMIA

Moderate to severe anaemia is commonly reported in alpacas. In New Zealand in 2013 and 2014 there were reports of alpacas with severe anaemia (PCV as low as 7%) but with no clinical signs (pers comm Cristen Dywer). One report suggested llamas may be treated successfully with

parenteral iron dextran supplementation but the underlying cause of most anaemias in llamas and alpacas is often not identified [114]. The diagnosis of non-regenerative anaemia or regenerative anaemia is difficult because the criteria for defining regenerative and non-regenerative anaemia is not clear, and features of regenerative anaemia (anisocytosis, polychromasia, reticulocytosis and increased numbers of metarubricytes) may be not be consistently seen in anaemias that are regenerative [24].

Gastric ulcers occur most often in the caudal third compartment of C3 [112]. These are considered to be a common cause of anaemia by alpaca owners and have been reported to contribute to 5% or 6% of the death in some alpaca populations however other authors list it as a rare cause of anaemia [112, 115, 116]. Stress seems to be a key factor in the formation of perforating and non-perforating gastric ulcers, some reports linked stress with poor gastric emptying and increased acid content in C3 which resulted in damage to the mucosa and subsequent ulceration [112, 116]. Older reports also list high-grain diets, nonsteroidal anti-inflammatory drugs and other disease as contributory factors in alpacas and llamas with gastric ulcers [115]. The clinical signs associated with non-perforating gastric ulcers in alpacas are inappetence, scant faeces and atony of C-1, peritonitis occurs with perforating gastric ulcers [116].



## ***EIMERIA MACUSANIENSIS***

*Eimeria macusaniensis* is one of the four common coccidia that affect alpacas, guanacos, vicunas and llamas, along with *E. lamae*, *E. alpaca*, *E. punoensis*, and *E. ivitaensis* [105]. Coccidia infections can be asymptomatic to clinically significant and occur in all ages especially in young alpacas. Clinical signs of infection are frequently nonspecific but may include lethargy, anorexia, weight loss, sudden death, and mild catarrhal to haemorrhagic diarrhoea [105]. Diarrhoea is more commonly seen in young animals and symptoms of colic are more associated with *E. macusaniensis* than with other parasitic infections [117]. *E. macusaniensis* causes a protein losing enteropathy and can present with scant faeces. Anaemia is not a common finding with *E. macusaniensis* but it can be seen if the damage to the intestines is severe resulting in prolonged and excessive bleeding.

## **BOVINE VIRAL DIARRHOEA VIRUS**

Bovine viral diarrhoea (BVD) is caused by a single stranded RNA virus from the genus Pestivirus, Flaviviridae family and has a worldwide distribution [118]. It has been identified in many species other than cattle, including

pigs, sheep, goats, deer, exotic ruminants, and old and new world camelids, including alpacas [118, 119].

Bovine viral diarrhoea virus (BVDV) may cause a number of different clinical syndromes including gastrointestinal tract infection, respiratory infection, abortion, teratogenic effects, still births and weak calves. The disease syndrome depends on the age at which the animal is exposed to the virus [118]. If a fetus is infected early in gestation with noncytopathic BVDV, the fetus may develop immune tolerance and is born as a persistently infected (PI) animal that acts as the main source of infection and reservoir of infection for the whole herd [120]. Persistently infected animals may often develop mucosal disease with a mortality rate of close to 100% [121]. The main mode of transmission in cattle is nose-to-nose or sexual contact but this is unknown in alpacas and other new world camelids [122].

Pregnant llamas experimentally infected with BVDV gave birth to crias that were negative for BVDV antigen prior to colostrum ingestion but tested positive for BVDV antibody 1 month after birth. In these crias antibody titers became undetectable between 5 and 6 months of age. In this study, one pregnant female aborted but it could not be attributed to BVDV infection [118]. In another study an infected cria naturally infected a herd

of alpacas, 2 alpacas aborted (1 positive for BVDV type 1b), 17 had high BVDV antibodies for BVDV type 1, and 1 of 19 crias born to the infected herd tested positive for BVDV type 1b at birth, 3 and 26 days of age and remained positive until euthanasia at 46 days of age [119]. BVDV has also been isolated from a frozen stillborn alpaca that was from a farm of 20 alpacas with no history of reproductive problems [123].

BVDV has been reported in alpacas in Australia, Canada, USA and United Kingdom [123-125]. Recent prevalence studies in the USA reported BVD serum neutralising antibodies from 16 of 63 herds (25.4%), with 4 (6.3%) herds having recent PI crias [126]. PI crias are proving to be, like PI calves, a main source of infection. In the USA PI animals in beef herds are estimated to be 3-4% and the economic loss in the cattle industry from BVDV is substantial [127]. Similarly with a PI crias as the most important source of infection, seroprevalence of BVDV has ranged from 2.05% to 11.11% within herds, and with a mortality rate of close to 100% for PI animals it may be a cause of significant economic impact on alpaca farms [121].

## JOHNE'S DISEASE

Johne's disease (paratuberculosis) is a chronic infectious, wasting enteritis caused by *Mycobacterium avium* subspecies *paratuberculosis* (MAP). It infects many domestic and wild ruminants, including rhinoceros, South American camelids and macropods [128-131]. MAP is closely related to *M. avium* with only a 1.2% difference in DNA sequence between the two bacteria. MAP is an acid-fast, Gram positive, non-motile, non-pore forming bacillus. It is a slow growing intracellular bacteria, and has a long incubation period prior to development of clinical signs [131].

The clinical signs of Johne's disease vary depending on the species but include chronic progressive weight loss and persistent bloodless diarrhoea in a bright and alert animal with a healthy appetite. In alpacas the most consistently reported finding is progressive weight loss and emaciation but no diarrhoea. In contrast to this there are two reports in the literature of diarrhoea in alpacas with Johne's disease but also one report where no clinical signs were seen [129, 131].

The age of onset of clinical signs in cattle is usually 3 – 4 years but it can be seen in cows as young as 2 years of age. In one report, eight of ten alpacas who were naturally infected developed clinical signs of Johne's between

12 and 24 months, but it has also been reported in alpacas up to 11 years of age [129, 130].

The bacteria is shed by infected animals in the faeces, and the number of organisms shed increases as the disease progresses, and may increase when infected animals are stressed. Most animals contract the infection when young by ingesting feed or water contaminated by the organism [128]. Some animals may be infected *in utero* and small numbers of bacteria are found in infected milk potentially leading to infection of suckling animals [131]. However due to the low numbers of organisms, this route of infection is less common than the faecal-oral route.

At necropsy the gross lesions of Johne's disease include thickening of the mucosa of the small intestines particularly the ileum, lymphangitis, and enlargement of the mesenteric lymph nodes [131]. Microscopically, aggregations of epithelioid macrophages containing acid-fast bacilli are present in the intestinal lamina propria and lymph node, and occasionally the liver.

Culture of *M. paratuberculosis* from faeces (individual or pooled faecal samples) is diagnostic but relies on the animal shedding enough bacteria.

PCR on faeces can be used but is less sensitive than culture and maybe recommended when there are visible acid-fast bacilli. Interferon-gamma

assay, agar gel immunodiffusion test (AGID), complement fixation test (CFT), absorbed ELISA (Ab-ELISA) are all tests that can be used but have varying specificities and sensitivities particularly in subclinical animals [131]. In alpacas, no test is 100% reliable, sensitive or diagnostic.

## **FACIAL ECZEMA OR PITHOMYCOTOXICOSIS**

Facial eczema (FE) is primarily caused by the mycotoxin sporidesmin A produced by the fungus *Pithomyces chartarum* which grows on dead litter. The toxin is hepatotoxic and destroys medium sized bile ducts resulting in hepatocellular damage and bile duct obstruction [132]. The amount of sporidesmin produced by the fungus is directly related to temperature and UV radiation, as such FE is most common in late summer and autumn (when there is proliferation of the fungus and spores in moisture and heat) [133]. Daily spore counts as low as 50,000 spores/g could be dangerous to livestock if grazed in the sun for long periods and if forced to over graze the pasture [134]. However, spore counts of 100,000 spores/g are considered toxic to domestic ruminants [134].

The skin lesions associated with FE are caused when photodynamic pigments in the blood stream accumulate due to liver damage which prevents the excretion of chlorophyll metabolites in the bile, these reach

capillaries in the skin and react with sunlight [135, 136]. The areas affected with FE are limited to non-pigmented skin and occur more frequently on the lightly haired skin of the udder, ears, perineum, muzzle, and the underside of the tongue if exposed to the sun during licking.

Facial eczema occurs mainly in cattle and sheep but can occur in deer and alpacas [135]. Sheep, fallow deer and alpacas are highly susceptible, while cattle and red deer are moderately susceptible [135]. Goats appear to be the least susceptible ruminants, feral goats being more resistant, then Angora feral crossbred goats, which are more resistant than Saanan goats [137]. Horses, rats and mice are resistant [138].

The liver is damaged during a lag period of 7 to 20 days between ingestion of the toxin and appearance of clinical signs, inflammation and blockage of the bile ducts causes the photosensitising pigments to build up in the blood [133, 139].

Upon ingestion of the toxin there is usually diarrhoea and inappetance with an immediate drop in milk production in dairy cattle or sheep [133].

In the acute phase of the disease the liver is enlarged with rounded edges, there is mild oedema of the gallbladder wall, and thickening and occlusion of the ducts of the gallbladder. As the injury becomes more chronic the liver becomes firm and shrunken, especially the left liver lobe, this is

intermixed with hyperplasia of hepatocytes in the right lobe causing the liver to take on the “boxing glove” appearance typical of FE [140].

In most animals there are little outward signs of disease but in severe cases of acute disease there is initially erythema and oedema of the skin with pruritis due to the build-up of phylloerythrin metabolites in non-pigmented areas, which eventually progresses to necrosis, drying and sloughing of the affected skin. In these cases there is high activity of serum gamma glutamyltransferase (GGT) in the blood which indicates severe pericholangitis [141]. Serum GGT concentrations 2 - 3 weeks after sporidesmin ingestion have been shown to correlate to liver damage and decreases in body weight [141].

Zinc (Zn) as either metallic zinc, zinc oxide or zinc sulphate, if given before ingestion of the toxin, has a prophylactic effect in doses approximately 25 times the daily requirement (15 to 30 mg/kg live weight per day) [133, 142]. Intraruminal zinc decreases serum GGT and likely protects lambs against FE [143]. The protective nature of zinc is believed to be related to its inhibition of superoxide free radical generation by sporidesmin, and inhibition of absorption of copper from the intestines (an essential element of superoxide desmutase) [144]. Recent reports have indicated



that zinc supplementation as a prophylaxis against FE was inhibited when cattle were fed excess copper [145].

Facial eczema has been reported in llamas in Brazil and alpacas in New Zealand and Australia [133, 146-148].

The lesions seen in FE in llamas and alpacas are similar to those described in cattle and sheep [146, 147]. In addition, abdominal distension has been reported in a llama, and a small amount of peritoneal fluid in an alpaca in Australia who showed mild clinical signs related to FE. Serum GGT activity for the alpaca was elevated and peaked between day 6 and day 13 post ingestion [149].

## **NUTRITION**

Nutrition plays a key role in preventing disease, and nutritional diseases are the most frequently diagnosed non-parasitic problem of alpacas [106].

Although alpacas are considered to be susceptible to all nutrient deficiencies and toxicity diseases described, very few published studies are available but nutritional diseases in alpacas are believed to be similar to ruminants. A zinc-responsive dermatosis has been described but the true role of Zn deficiency is debated [114, 150-152].

## COPPER DEFICIENCY

Copper (Cu) deficiency results when less copper than is required for normal physiological functions is ingested and Cu stores are depleted.

Copper is transported in the blood by ceruloplasmin and excreted via the bile [153]. Copper is typically absorbed from the upper gastrointestinal tract, stored and regulated by the liver [153]. Physiological states determine the daily requirements for Cu, with the demand for Cu higher in pregnant and lactating females. Copper absorption rates are affected by the presence of the trace elements iron (Fe), molybdenum (Mo) and sulphur (S) as these antagonise copper absorption and utilisation [154].

In cattle serum levels of copper do not predict the liver copper levels [155]. Serum copper levels only accurately indicate low copper levels (0.5µg/ml) in cattle when liver copper values were also low and therefore are of limited diagnostic value.

Copper deficiency may cause sway back (a nervous disorder), osteoporosis, fragile bones, changes in coat colour, poor weight gain and impaired reproductive performance [154]. Routine application, in New Zealand and Australia, of Mo and S on pastures used for cattle and sheep effectively decreases the availability of copper and thus copper deficiency

is the most common trace element deficiency in cattle and sheep in these countries [154].

In SAC, two llamas with anaemia and poor condition were reported to have low serum copper concentration and responded to copper supplementation [114]. As with cattle, it is postulated that alpaca serum copper levels alone are of limited value in diagnosing copper deficiency. However the normal plasma copper concentration in alpacas (4.20 -5.9  $\mu\text{mol/L}$ ) is low when compared to mean copper concentrations for healthy sheep (11.6  $\mu\text{mol/L}$ ) [114, 154, 156].

## VITAMIN D DEFICIENCY

Alpacas and llamas seem to be highly sensitive to vitamin D deficiency, especially in the winter [150]. Vitamin D is naturally synthesized in the skin upon exposure to ultraviolet light. Dark coloured and heavily fleeced alpacas have lower serum vitamin D<sub>3</sub> concentration, and shearing increases skin exposure and subsequently increases serum vitamin D<sub>3</sub> concentrations [157]. In winter crias born to hembras with lower serum vitamin D concentrations (due to less solar radiation) may develop rickets since they receive less vitamin D through the placenta or colostrum [158]. The intensity and angle of the sun at the most distant latitudes within the

northern and southern hemispheres may be insufficient during the winter months to maintain adequate vitamin D status without supplementation [114].

The clinical signs of vitamin D deficiency include skeletal deformities, delayed eruption of teeth, enamel hypoplasia, and enlarged costochondral joints. Vitamin D is required to actively absorb dietary calcium and phosphorus so when serum concentration is low, calcium is poorly absorbed resulting in hypocalcaemia and hypophosphataemia.

Vitamin D is toxic in excess and therefore care must be taken in not exceeding recommended doses. Alpacas in Southern Australia have been successfully treated with a subcutaneous dose of 1000 IU D<sub>3</sub>/kg body weight [114, 150]. A study using treatment with intramuscular vitamin D found that between 1500–2000 IU/kg of vitamin D could increase serum vitamin D concentrations for 90 days [158].

## CHAPTER 2

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### INTRODUCTION

Alpacas are domesticated, herbivorous new world camelids originating in South America, and are divided into two breeds, Huacaya and Suri. They are reared commercially worldwide for their fibre but in some countries for their meat. In their natural habitat alpacas live in large family groups of about 16 individuals.

Alpacas have been present in New Zealand since 1847, when they were imported from Australia. It is suggested that some of the herd owned by the then Governor of New South Wales, Charles Ledger, was exported to Canterbury, New Zealand [9]. Alpacas successfully adapted to the New

Zealand climate but the population remained small until the 1980s. In 1985, attention was generated around rearing alpacas when Ian Nelson's work helped to reclassify alpacas from zoological animals to farm animals [10]. This, along with the protocol for the importation of alpacas from Chile created by Murray Bruce, allowed for the importation of several alpacas from South America in 1987 [9, 159].

By the late 1980's alpaca farming in New Zealand had commenced and research began on alpacas in New Zealand in the 1990's [160]. Alpacas are farmed in New Zealand mainly for their fibre, which is produced in 22 natural colours, and is used to produce high quality soft garments (Appendix: Table 1)

In 2001, the Alpaca Association of New Zealand (AANZ) was formed out of the Alpaca and Llama Association of New Zealand (ALANZ) when alpaca farmers recognised the uniqueness of the alpaca as an animal to be used for lifestyle blocks, commercial farms or as pets.

The alpaca industry in New Zealand continues to grow, and the population of alpacas has increased from 1607 alpacas in 1990, to approximately 15,000 in 2008 [161]. AANZ census results from 2000 to 2005 estimated that the population of alpacas in New Zealand had consistently increased annually, and by 2005 there were 8500 alpacas.

From 2007 the national numbers for llamas and alpacas have been recorded as a combined number in the national census [161]. It is believed that most of the recorded population increase in New World camelids is from the more rapidly growing alpaca population rather than llamas, however this is difficult to substantiate. From 2007 to 2013, with the exception of 2010, the national census figures indicated a continual increase in combined alpaca and llama numbers.

The alpaca population and fibre industry are well established with specialised groups, events, and organisations. The AANZ has increased membership from less than 50 in 1996, to over 700 members and 14,000 registered alpacas in 2014 (SFF AANZ census results) [162]. The AANZ is also joined by other organisations and events that support alpaca farmers, notably the Alpaca Breeders Association New Zealand (ABANZ), Alpaca Sellers NZ, and the national alpaca show.

Information on current treatment practices by farmers is lacking and if inadequate, may impact on the health and well-being of animals and economics of farming. Therefore in exploring alpaca health in New Zealand obtaining baseline information on current practices is important.

Internal parasites are a common problem in alpacas worldwide and are listed on the Alpaca Association of New Zealand (AANZ) website as one of

the alpaca diseases for farmers to keep in mind when doing regular husbandry checks [105].

A number of different infectious diseases may also affect South American camelids [112, 163-165]. Black leg (caused by *Clostridium chauvoei*), bovine viral diarrhoea virus, equine herpes virus 2, leptospirosis, tetanus, tuberculosis, toxoplasmosis, and other clostridial diseases all have all been reported in New Zealand and can affect alpacas, but there are few studies that identify the incidence of these diseases. Other common non-infectious conditions seen in alpacas include hypovitaminosis D (rickets), facial eczema (FE), and ryegrass staggers [166].

Reproduction of alpacas for producing replacements, increasing herd size, and improving fibre characteristics is an important part of farming alpacas. Female alpacas (hembras) are reflex ovulators with a gestation period of 330- 360 days. In their native habitat, hembras can delay reproduction until conditions are favourable for the survival of crias. As a result, birth usually occurs in day light hours and is generally unassisted.

Research on alpacas in New Zealand has examined base line blood parameters and the general concerns of alpaca farmers. In 2004 a comprehensive survey of 11 farms was conducted. It focussed on collecting data on general health of the alpacas, common problems



encountered, common problems associated with reproduction and crias, and reasons for deaths. There is no data on the health and reproductive practices of alpaca owners in New Zealand, and so the aims of this study were to examine the practices of alpaca owners that may impact the health of alpacas in their care. It focuses on worming procedures, and herd management practices, in order to obtain information on how owners deal with death and reproduction, and if they test or vaccinate the herd for diseases that are commonly found in New Zealand.

## METHOD AND MATERIALS

### **SAMPLE COLLECTION**

The survey was created in collaboration between alpaca clients of a local veterinary clinic, and a specialised alpaca veterinarian. It was targeted at alpaca owners, in the 16 regions of the North and South Islands of New Zealand, in order to record the common health practices on their properties. It was collected from alpaca owners who volunteered to be interviewed by phone, email, or in person, in order to complete the survey, and all surveys were done between October 2014 and March 2015. There was no selection based on herd size or any other criteria.

The questionnaire was emailed out to farmers, and for most farmers this was followed up with a call or subsequent emails to ensure that each question was answered by all farmers. The questionnaire was made available to any farmer of the Alpaca Association of New Zealand, any owner who subscribed to a free lifestyle block website, and to farmers who had heard by word of mouth about the questionnaire. The questionnaire is attached in Appendix 2.

## RESULTS

### FARM AND FARMER CHARACTERISTICS

The number of alpacas farmed ranged from 8 to 228 alpacas, with mean of 59 alpacas. Two farms had over 200 alpacas, and 3 farms had 10 or less alpacas. Most owners farmed predominantly Huacaya (9 farms), 2 farms had Suri alone, and 7 farmed both Suri and Huacaya alpacas.

The largest farm in this survey was 48 ha and the smallest was 1.7 ha. The mean was 10.5 ha and the median was 7.0 ha. One farmer in the Otago region leased an additional 30 ha thus increasing his usable farm land from 6.4 ha to 36.4 ha. Other farms in the Wellington, Canterbury and Auckland regions farmed only 8.9 ha out of 10.1 ha, 3 ha out of 4 ha and 1.3 ha out of 7 ha respectively, with not all the land having been developed for farming. The smallest area used to rear alpacas in this survey was 1.3 ha.

The location (by region), number of farms surveyed and land area of the 18 farms surveyed from 9 regions are recorded in Figure 1.

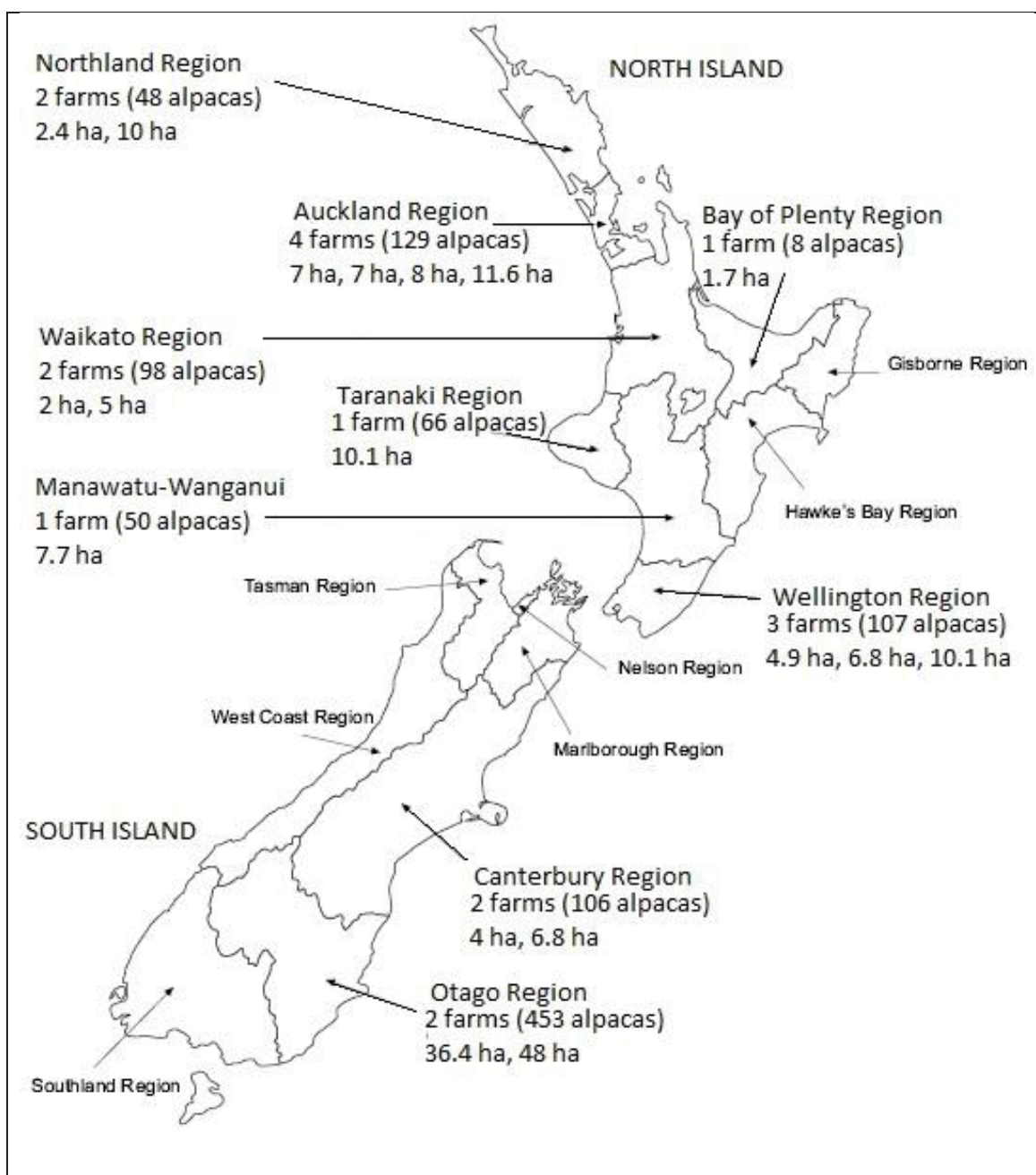


Figure 1 The number of farms, location by region and total number of alpacas on the farms surveyed, and farm sizes

Table 6 shows the sizes of the farms surveyed, and the number of alpacas per farm, by breed and management group. The stocking density is also

tabulated. Stocking density in this survey was taken as the number of alpacas divided by the area of the farm in hectares (ha). The stocking densities ranged from 1.0 to 15.8 with the average stocking density being 6.6 alpacas / ha and the median 6.1 alpacas / ha.

Table 6 Number of alpacas by breed, management group and the stocking density in hectares of the 18 farms surveyed

Farm ID	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	TOTAL
Alpaca numbers	8	9	10	14	18	19	27	35	37	38	50	61	62	66	79	79	225	228	1065
Size (ha)	1.7	4.9	10.0	11.6	7.0	2.0	4.0	7.0	6.8	2.4	7.7	10.1	8.0	10.1	5.0	6.8	36.4	48.0	
Suri	8	1	0	0	0	0	0	0	33	0	50	0	4	0	74	9	4	35	218
Huacaya	0	8	10	14	18	19	27	35	4	38	0	61	58	66	2	70	221	193	844
Crias	3	0	3	0	2	5	8	8	9	13	15	9	5	15	23	17	40	43	218
Adults	5	9	7	12	15	12	18	23	24	23	35	51	47	51	56	61	175	153	777
Geriatrics	0	0	0	2	1	2	1	4	4	2	0	1	10	0	0	1	10	32	70
Stocking density	4.7	1.8	1.0	1.2	2.6	9.5	6.8	5.0	5.4	15.8	6.5	6.0	7.8	6.5	15.8	11.6	6.2	4.8	
Deaths per 10 alpacas	5.00	-	3.00	0.71	3.89	2.78	1.48	0.43	4.30	0.46	2.40	1.15	0.81	0.76	0.65	-	-	1.23	
Status	hob	hob	hob	hob	both	both	bus	both	both	hob	both	both	both	both	bus	bus	bus	both	

bus refers to a business farmer

hob refers to a hobbyist farmer

both refers to farmers that consider themselves both a hobbyist and business farmer

The most experienced alpaca farmer had been farming for 19 years, and the least experienced alpaca farmer had farmed for 2 years. Most farmers had 6 to 10 years' experience rearing alpacas. Figure 2 shows the variety in the years of experience of the alpaca farmers who participated in the survey.

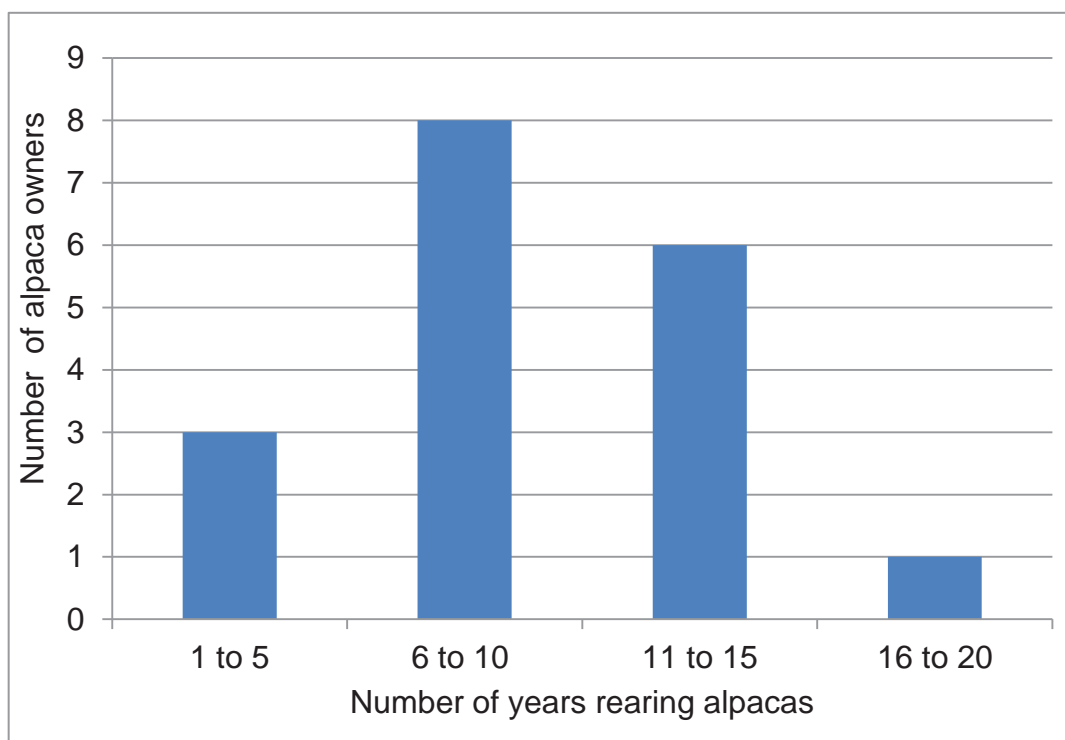


Figure 2 Number of years alpaca owners surveyed have reared alpacas

All alpaca farms in the survey had shelter for the alpacas. Shelter was provided in some of the paddocks on 4 farms, most of the paddocks had shelter in 1 farm, with the remaining farms indicating that the alpacas had

access to shelter. The shelters consisted of both natural vegetation (trees and shelter belts) and erected covered paddocks or sheds.

Alpaca farmers commonly also farmed other animals (11 farms), with only 7 properties farming only alpacas. The most common species on the farm with alpacas was cattle, (7 of the 11 farms), followed by horses or ponies on 5 of the 11 farms, and sheep on 3 farms. One farm had goats, and one farm kept pigs, chickens and pigeons as well as alpacas.

Of the 11 farms with other species present, 8 of those cross grazed with the other species, but 2 did not cross graze. Of the 7 farmers with only alpacas on their farm, 2 farmers borrowed cattle from neighbours to occasionally cross graze with their alpacas. Most farmers cross grazed with cattle or sheep but one farmer used pigs to graze after the alpacas.

Twelve of the seventeen farms had the soil tested, and pasture treatments had been applied on 14 of the farms. Lime application was the most common treatment performed on 42.9% of the farms, while AgriSea® was used on 21.4% of farms. Applications of Selenium, Magnesium, dicalcium phosphate, SuperTen® and Mycotac® were used by at least one farmer each in the study.



Computer software was used by 44.4% owners to track the alpacas while the remaining 55.6% used no computerized tracking system. Alpaca Manager<sup>®</sup> was the most popular tool, and was used by 5 of the 8 owners. Alpaca Plan<sup>®</sup>, Microsoft Excel<sup>®</sup>, and a self-made computerized tracking system was used by 1 farmer each in the survey. One farm had Alpaca Manager<sup>®</sup> but was yet to use it in its management of the alpaca farm. All persons interviewed were members of the AANZ, and 1 person was also a member of the New Zealand Llama Association (NZLA).

Forty four percent of alpaca owners (8/18) surveyed reared alpacas as both a hobby and a business, while 29.4% (5/18) each produced alpacas either solely as a business, or as a hobby.

Farmers in the study reared alpacas for many reasons with the most common reasons including pleasure 88.8% (16 owners), fibre production 83.3% (15 owners), and to sell as breeding stock or pets 77.7% (14 owners). The least common reason was meat, with only 2 farms rearing alpacas for this reason. Seven farmers showed their alpacas regularly, while one owner only showed his alpacas occasionally. One farmer recorded that his alpacas provided a service as stud animals for other farms. Figure 3 summarises the data on reasons why alpacas were farmed.

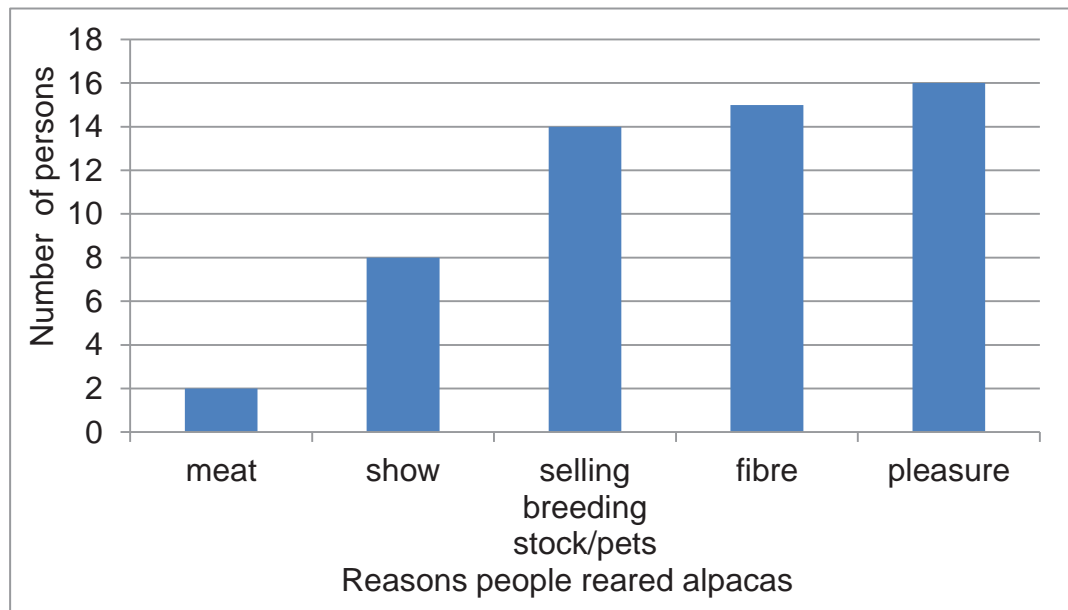


Figure 3 Reasons given by farmers as to why they farmed alpacas

All the farmers who kept alpacas for a hobby listed pleasure as one of or the only reason for farming alpacas. Other reasons hobbyist farmers had alpacas were for fibre (4/5), selling breeding stock/pets (2/5), for meat (1/5), and showing (1/5).

All business farmers listed fibre, selling breeding stock/pets, and pleasure as reasons for ownership. No business owners kept alpacas for meat, and 4 of 5 business owners showed their alpacas.

## SELECTION CHARACTERISTICS

Of the owners interviewed 12 considered fibre quality when choosing a breed or when buying an alpaca. The most common fibre characteristic listed was small fibre diameter, with an additional emphasis on low standard deviation of fibre micron diameter. Other characteristics mentioned were stable length of the fibre, structure and fleece density as measures of fleece quality, fineness, and fleece weight. All farmers who put fibre as a breeding criteria owned alpacas for fibre production or for showing. Physical characteristics, including conformation (6), comparison to breed standard (5), and size (1) also featured highly in the selection criteria. Fibre colour and temperament were considered by 7 farmers each when breeding or buying alpacas. The criteria considered by owners when breeding or buying alpacas is summarised in Table 7.

Table 7 Characteristics considered by farmer when breeding or buying alpacas

Characteristic considered by farmer	Tally
Physical characteristics	12
Fibre quality	12
Fibre colour	7
Temperament	7
Health	5
Cost	3

Two farmers who were Suri breeders, mentioned genetics as important, and they bred for breed standards, while the other two farmers who mentioned genetics as important selection criteria mentioned fibre characteristics as the most significant in that regard.

## ANIMAL HEALTH CARE SPEND

In this survey animal health care included anthelmintic costs, other animal treatments, and all fees associated with veterinary care. The average money spent on health care on the farms surveyed was \$1516.47 per year, with a minimum of \$200 spent, maximum of \$3500 and a median amount of \$1500. The average annual animal health care spent per alpaca was \$48.20 and the median was \$29.40. The range of the annual animal health care spent per animal for business farmers ranged from \$6.67 to

\$74.07, the range for the hobbyists was \$20 to \$37.50. The widest range was in the category of farmers that counted themselves as both hobbyist and business farmers, and ranged from \$6.58 to \$194.44. Veterinary care accounted for most of the money spent on animal health care in 10 of 11 farms while on 1 farm the veterinary care budget equalled the money spent on anthelmintics (Figure 4).

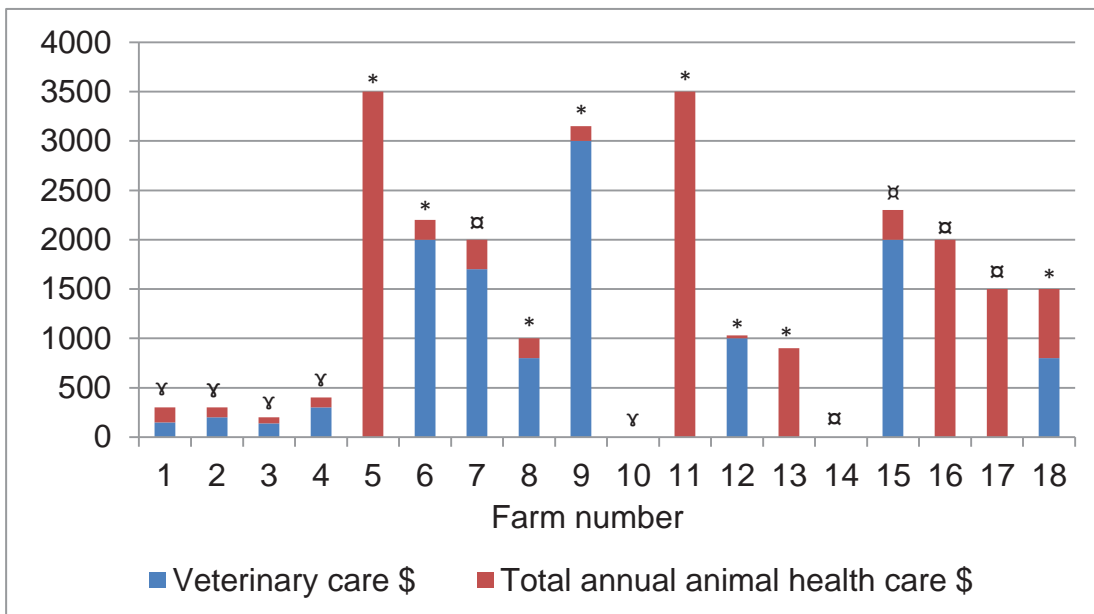
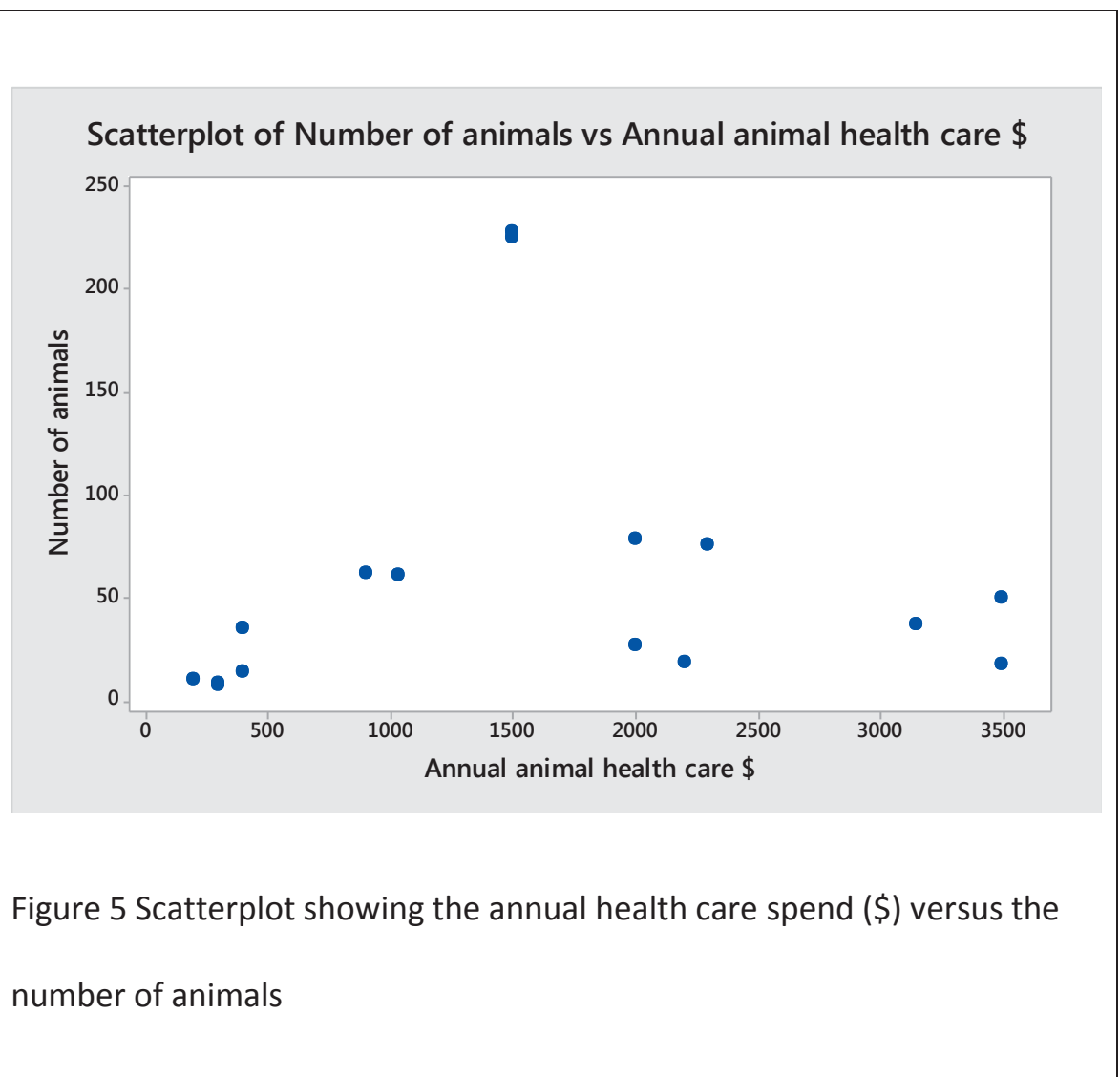


Figure 4 Total money spent on animal health care annual showing veterinary care

\* both business and hobby    x business    γ hobby

The average spent on veterinary care was \$1099 annually, with a median

of \$800 while the average spent on anthelmintics annually was \$208.20 and the median was \$150. Figure 5 shows the money spent on alpaca health care annually and the numbers of animals farmed of the farmers surveyed and figure 6 shows the range in the annual health care per alpaca for all farms surveyed.



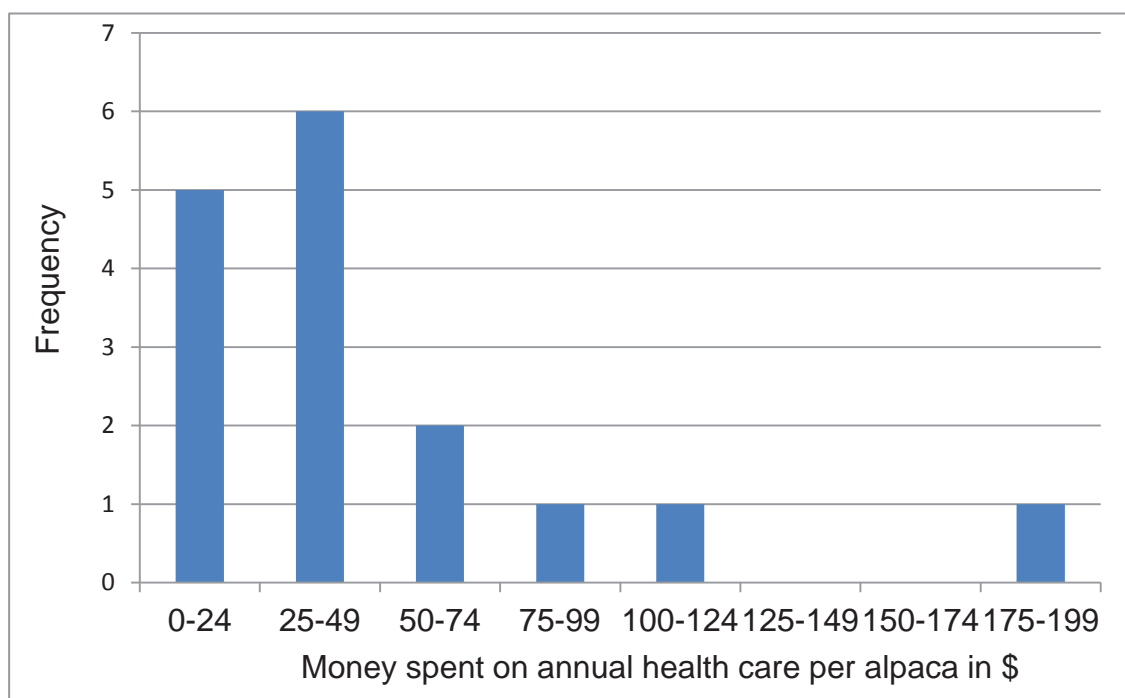


Figure 6 Frequency graph of annual health care per alpaca in New Zealand

## PARASITE CONTROL

All alpaca owners dewormed their animals, with 44.4% using only subcutaneous injections, 16.7% using only oral drenches, and 38.9 % using a combination of both parenteral and oral routes. No farmer mentioned using pour-on formulations. On 66.7% farms all crias were dewormed annually, and half the farmers scheduled the deworming of all their alpacas. Of the 9 farms that dewormed all animals, 6 are dewormed twice yearly (most commonly in autumn and spring) and one each dewormed

once, thrice and four times a year. Deworming in spring and autumn was done on 4 of 6 farms, and only on 1 farm were the animals dewormed in summer and winter. When deworming only occurred once a year (2 farms) the treatment was performed in autumn.

On the 9 farms where deworming was performed as needed, 87.5% (7/8) used faecal egg counts (FEC) to determine when to deworm, however 62.5% of farms with a scheduled worming routine also used FEC on their farms (Table 8). Overall, on all farms 75% performed FEC at least as needed.

Table 8 Faecal egg count (FEC) performed on farms that schedule deworming and farms that deworm as needed

FEC	Scheduled Deworming	Deworming as needed	TOTAL
Annually	2	3	5
As needed	3	4	7
Don't do	3	1	4
TOTAL	8	8	16

Dectomax® (doramectin) was the most commonly used of the 15 products mentioned by the alpaca farmers surveyed. The classes of anthelmintics represented in the survey included the benzimidazoles,



macrocyclic lactones, combination drenches, natural wormers and coccidiostats. Table 9 lists the products, active ingredient, company, classes of anthelmintics used, and the number of farmers surveyed that currently used or previously used the products.

Macrocyclic lactones were the most popular class in use, with Dectomax®, Cydectin®, Vetdectin® and Noromectin® replacing the older members of this class (Ivomec® and Baymec®). Some farmers mentioned the use of herbal wormers but only one farmer identified willow among the anthelmintics used as part of a natural worming regime.

Seven of the 18 farmers surveyed have discontinued use of certain anthelmintics, while 10 farmers were still using the same anthelmintics they first used. Of the 7 who discontinued drugs, reasons for this included having a drug rotation policy (3 farms), discontinued use because of the cost (2 farms), and 1 farm reported drug resistance as the reason.

Table 9 Anthelmintics in use or used by alpaca farmers in the survey

Class of Anthelmintics	Product Name	Active Ingredient	Company	Number of farmers	
				Currently use	Previously used
Benzimidazole	Bomatak©	Oxfendazole	Bayer Animal Health New Zealand	1	0
	Panacur©	Fenbendazole	MSD Animal Health	1	1
	Rycoban©	Ricobendazole	Novartis Animal Health	0	1
Macrocyclic Lactone	Ivomec©	Ivermectin	Merial New Zealand	0	2
	Baymec©	Aabamectin	Bayer Animal Health New Zealand	0	1
	Dectomax©	Doramectin	Pfizer New Zealand Ltd.	13	3
	Cydectin©	Moxidectin	Zoetis Inc.	2	0
	Vetdectin©	Moxidectin	Zoetis Inc.	1	0
	Noromectin©	Ivermectin	Norbrook Laboratories	1	0
	Genesis©	Abamectin Praziquantel	Merial New Zealand	3	0
Combination	Matrix© (Hi Mineral)	Ivermectin Levamisole Oxyfendazole Praziquantel	Merial New Zealand	6	1
	Unspecified drench			1	1
	Untrademarked Off patent product			1	0
Natural Wormer	Willow trees			1	0
Coccidiostat	Baycox C©	Toltrazuril	Bayer Animal Health New Zealand	1	0





## REPRODUCTIVE PRACTICES

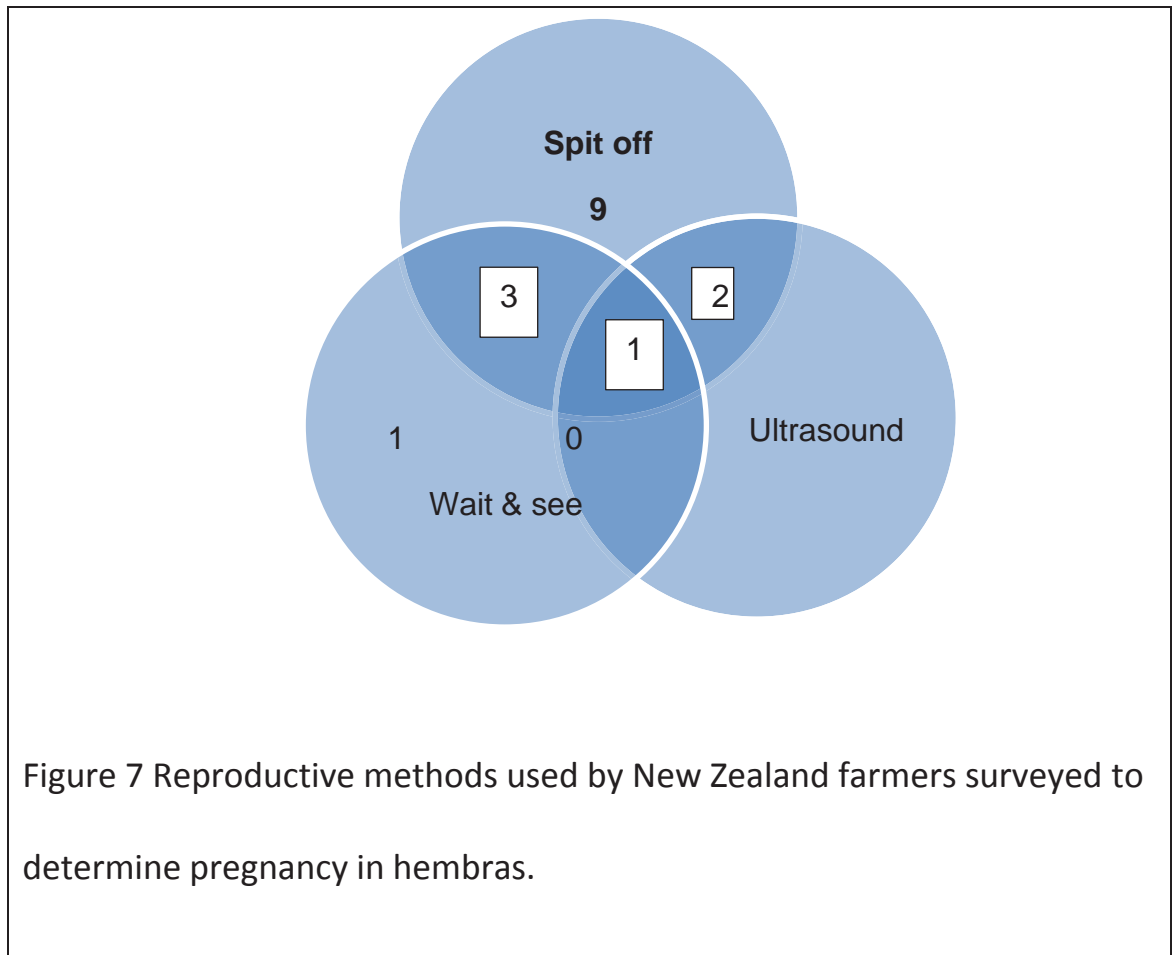
Male reproductive problems did not occur on 5 of the 15 (33.3%) farms that have breeding males. Inability of the male to impregnate the female was the preferred way to diagnose a reproductive problem in males on 53.3% (8/15) of farms. Half (4/8) of these farms allowed the male more than 2 matings to achieve pregnancy while one farmer supervised the mating before concluding infertility in the macho. On 2 farms offspring defects or poor quality crias was used as an indicator of reproductive problems in the male.

Two farms indicated that males may have difficulty impregnating females with changes in environmental conditions associated with heat stress and physical defects in the male genitalia (problems with penile extension or undescended testes).

Hand mating (64.7%) was preferred over paddock mating (17.6%), and 17.6% of farmers used a combination of both hand and paddock mating. Females were bred around the same time annually on 87.5% of farms.

Spit off was the most common method used to diagnose pregnancy on farms (88.2%), while 29.4% of farms used a wait & see approach.

Ultrasound and observed mating were the two least common methods used on 17.6% and 5.8% of the farms respectively (Figure 7).



One farm did not breed its own animals therefore of the 17 remaining farms 88.2% specifically recorded reproductive failure. On 40% of farms reproductive failure was considered when a hembra failed to conceive after being bred three times. On other farms 1, 4, or 5 breedings without conceiving were considered evidence of reproductive failure (Figure 8).

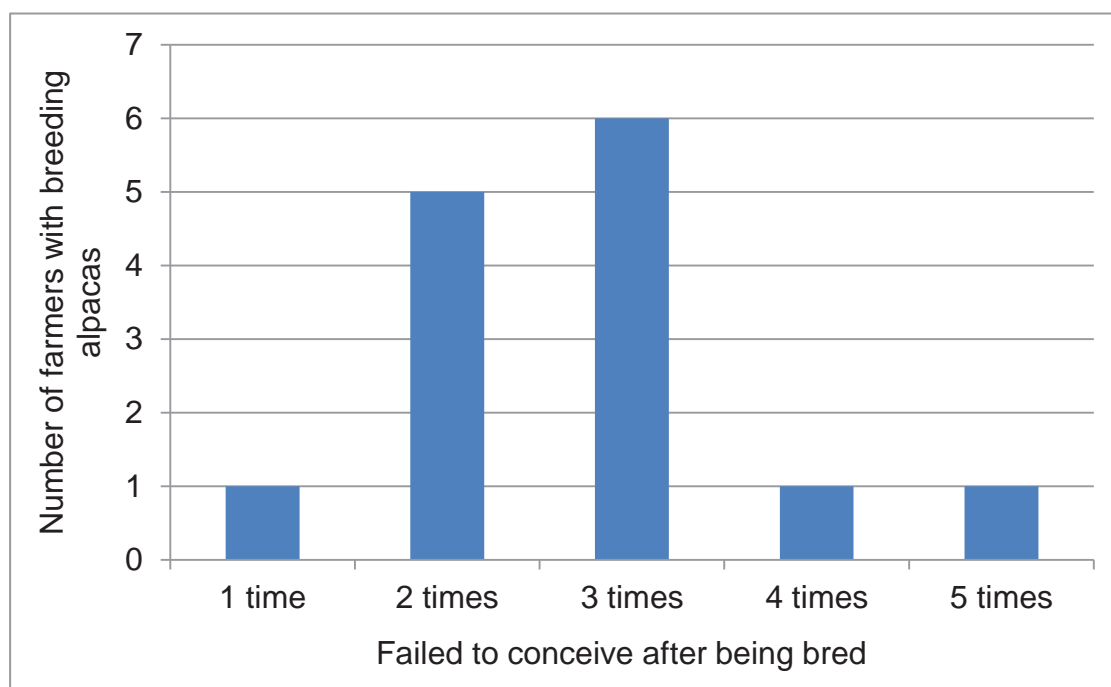


Figure 8 Number of times a female is unsuccessfully bred before reproductive failure is considered

Repeated failure to become pregnant after mating was the most common way owners diagnosed reproductive problems in their hembras, as reported by 60 % of farms. On 13.3 % of farms both a vet diagnosis and failure to become pregnant were used to diagnose reproductive failure. However, 46.7 % used only failure to become pregnant, and 26.7 % only a vet diagnosis.

Barren animals were sold on 6 farms, used to produce fibre on 3 farms, culled on 2 farms, and on 2 farms not mated or allowed to stay unbred for the year.

The age at which tuis were introduced to machos varied on New Zealand farms. On 10 farms female receptivity to the male was a factor, 12 farms had a minimum age for breeding (Figure 9), 7 farms a minimum size, and 5 farms a minimum weight for breeding tuis. No farmer in the study bred females less than 1 year old.

Fifteen of the 17 breeding farms indicated that BCS or weight factored into determining if adult females were healthy enough to be bred. The majority of farms (54%) indicated that a BCS of 2.5 to 3 was acceptable for mating, while 45.4% preferred a BCS of above 3. Thirteen farms used a fixed time after unpacking for subsequent matings, most commonly 2-4 weeks on 7 of 13 farms. However on one farm females were mated when crias were weaned.



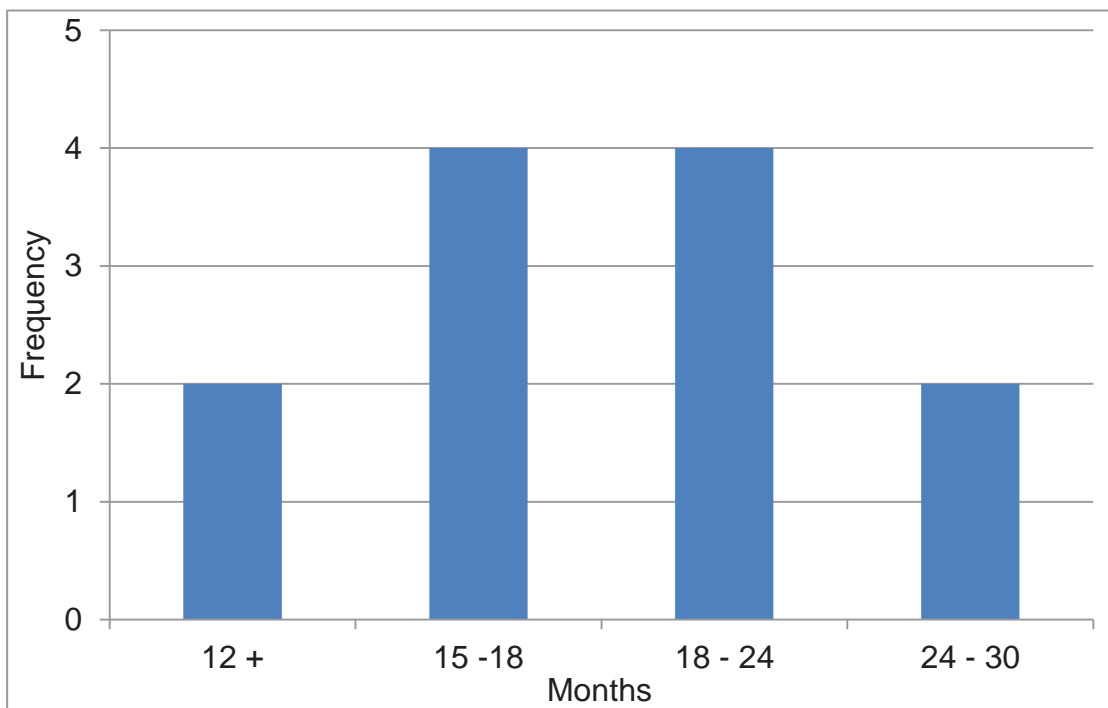


Figure 9 Age ranges used by alpaca farmers to determine if tuis are old enough to be bred

Common health practices associated with pregnancy included vaccinations, giving vitamins and minerals, and feed changes (Figure 10). Nearly all (94%) farmers vaccinate against clostridial diseases. The clostridial vaccines commonly used by the farmers include the 5 in 1 or Covexion® 10 (10 in 1) sold by MSD Animal Health for sheep and cattle. On most farms the clostridial vaccines were given annually, however on 3 farms the vaccine was given twice a year.

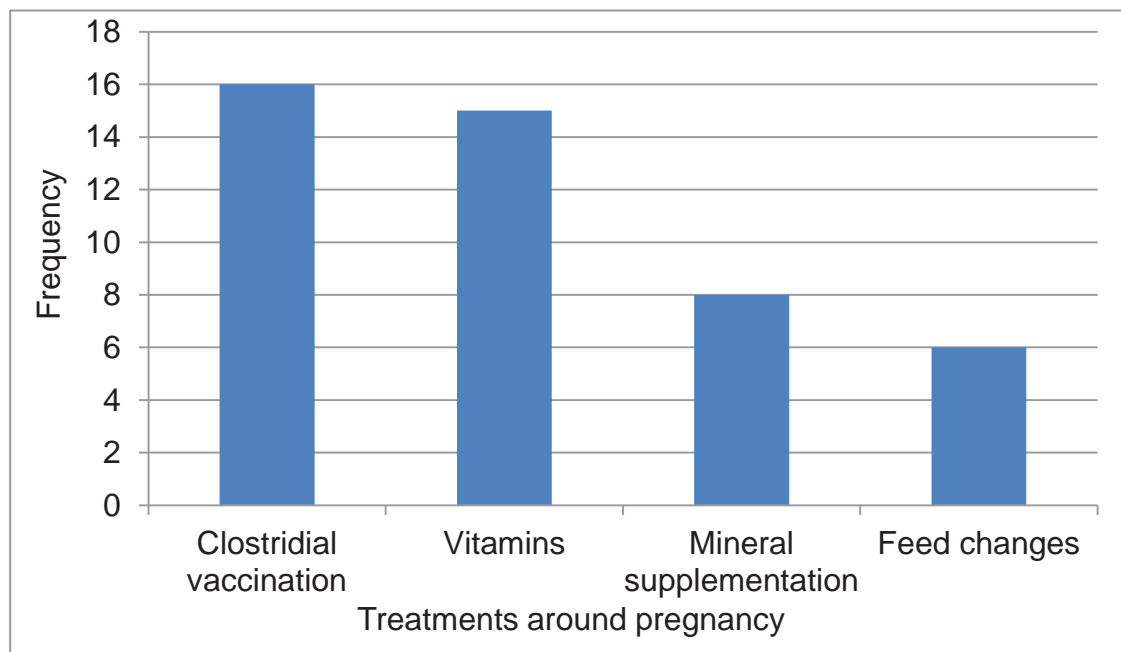


Figure 10 Treatments and their frequency on the 17 alpaca farms with breeding females

Vitamins, most commonly, vitamin D, were given to pregnant hembras on all farms. Eight farmers indicated that they routinely supplement pregnant females with minerals. Mineral supplements included salt lick/blocks, Agrisea® seaweed tonic, and Ringrose alpaca supplement.

Of the 6 farmers that change the feed of the pregnant herd, the diet was modified by adding protein (2 farmers), and adding zinc for FE prevention (2 farmers). One farmer each increased the feed intake during the last trimester, and in the last 6 weeks of pregnancy, respectively.

Ninety four percent of the farmers noted when crias suck within 24 hours, with 82.3% of them ensuring that the crias suckled within 12 hours after birth. Eleven of the 18 farmers regularly check the paddock when birthing was expected. One farmer surveyed sent the pregnant hembras to a maternity facility so that perinatal events could be adequately monitored, while on one farm closed circuit television was used to ensure that the alpacas were monitored pre- and post- natally.

Ten of the 17 breeding farms routinely weighed crias. On these farms the timing of weighing was concentrated around birth, and the first weeks after unpacking, with 7 farms weighing at birth, and 7 weighing crias 1 or 2 times weekly after birth. In 4 herds the weighing patterns eventually amalgamated with routine herd weighing, which was weekly on 2 farms, monthly or with each paddock rotation on one farm each. One farmer each reported that in addition to scheduled weighing, weights were assessed if there was a concern or if alpacas were not seen feeding. Weight measurements ceased when the cria reached 20 kg on one farm.

## **DEATHS AND ALPACA DISEASES**

Of the 16 farms surveyed the maximum number of deaths on any farm in 2014 was 9, 2013 was 12, and 2012 was 7. The average number of alpacas

that died in 2014, 2013 and, 2012 were 2.19, 2.81 and 2.0 alpacas respectively. The total deaths on farms over the 3 year period of 2012 - 2014 averaged 6.88 alpacas with a median of 5 deaths but 28 was the highest recorded number of deaths on one farm.

Over the period of 2012 and 2014 greater numbers of crias died on farms (maximum 14), when compared to other age groups (adults maximum 8 and geriatrics maximum 9). The average number of crias that died between 2012 – 2014 on farms surveyed was 3.31 which was higher than adult (2.31) and geriatric (1.2) alpacas. The average deaths per 10 alpacas (Figure 11) from the farms surveyed was 1.94 with the highest (5.00) recorded on the smallest farm, and 0.43 alpacas per 10 the lowest recorded (Table 6). The deaths per 10 alpacas that died in 2014, 2013 and 2012 were 0.43, 0.61, and 0.40 alpacas respectively.

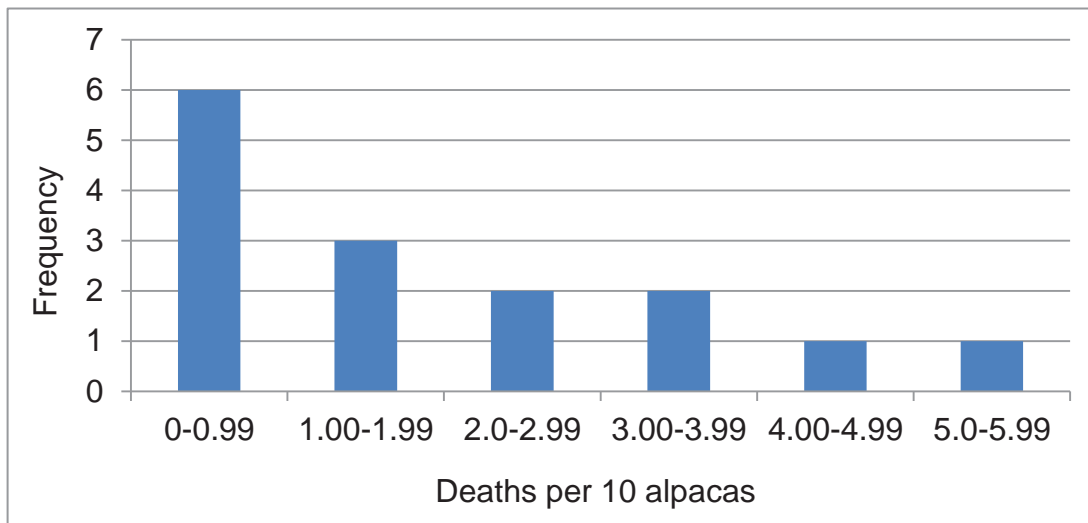


Figure 11 Frequency for deaths per 10 alpacas among surveyed farms

Three farmers (2 businesses and 1 hobbyist) did not record deaths while 15 farmers recorded deaths annually. Weak crias that failed to thrive or still born crias were reported in 5 of 15 farms and congenital defects were reported on 5 farms. In older crias *Haemonchus contortus* caused death on 2 farms and Canna lily toxicity occurred on 1 farm. In adult and geriatric alpacas, accidents (3) were the most common cause of death. Table 10 lists the causes of death recorded in the survey.

Table 10 Causes of death recorded in the survey

Management group	Causes of death and tally		
Crias	Weak/still born crias		5
	Congenital defects	Defects in cardiovascular system	3
		Choanal atresia	2
	<i>Haemonchus contortus</i>		2
	Toxicity	Canna lily	1
Adults and Geriatrics	Accidents	e.g. broken neck, broken leg	3
	Infection	Uterine and pneumonia	2
	Facial eczema		2
	Cancer	e.g. Lymphoma	2
	Old age	Blindness/ arthritis	2
	Toxicity	e.g. Bracken fern	2
	Rickets		1
	Ulcers		1

The method of diagnosis mostly commonly used by farmers surveyed was a veterinarian diagnosis (5/12), while 3 farmers used their own experience and a veterinary diagnosis. One farmer only used their own experience to diagnose deaths on the farm.

Thirteen of the farms surveyed tested for tuberculosis (TB). All farmers who showed their animals had them TB tested. Four farms indicated that TB testing on their farms was associated with it being a requirement for showing the alpacas, and therefore was usually done in September/October. On 1 farm TB testing was done every 3 years.

Bovine viral diarrhoea virus (BVDV) and Johne's disease were tested for on 1 farm each. No farmers surveyed vaccinated for equine herpes virus 2 (EHV2). Nine of seventeen farms quarantine animals when new animals came onto the farm.

Ryegrass staggers was not recognised on 58.8% of farms, with no ryegrass staggers reported in Northland, Taranaki, and the Bay of Plenty. Waikato, Otago, Wellington and Auckland reported farms with and without ryegrass staggers. Both farms from Canterbury reported ryegrass staggers.

Two farms indicated that they supplement hembars with Zn when pregnant while 10 indicated that the entire herd is supplemented with zinc (Zn) for facial eczema (FE) prevention. Farmers supplemented with Zn usually between Dec – April, when spore counts were highest (6 of 10 farmers). One farmer supplemented with Zn from December to August.

## DISCUSSION

The results from the 18 farms in the survey give a snapshot of current practices on alpaca farms of varying sizes throughout the country.

Fourteen farms were surveyed in the North Island and four in the South Island, yielding 506 and 559 adult alpacas respectively.

The average size for business farms was 12.46 ha, hobbyist farms was 6.12 ha and farmers who consider themselves both hobbyist and businessmen had farms with an average size of 12.08 ha. The median farm sizes of 10.1 ha (business), 4.9 ha (hobbyists), and 7.35 ha (both) was also noted. This suggested that those farming as business have greater land area and alpaca numbers, likely associated with maintaining a profitable operation, while hobbyists were more likely to be on lifestyle blocks.

The average size of farms in the North Island was smaller (6.7 ha) compared to the average size of the farms in the South Island (23.8 ha). Similarly, the average herd size in North Island was 36 alpacas while South Island average herd size was 140 alpacas. The alpaca herd sizes and farm areas were bigger in the South Island but this is also true of dairy herds and dairy farm sizes when compared with the North Island and maybe associated with greater availability of good quality farming land in the



South Island [167]. The stocking density of the alpacas in this study ranged from 1.0 to 15.8 alpaca/ ha, with an average of 6.6 alpacas/ha. The recommended stocking density for camelids grazing full-time in a paddock is 12 – 17 camelid per ha [168, 169]. With the existing farm sizes it appeared that most farmers could increase alpaca stocking densities without compromising animal health. In 2012/2013 the stocking density of sheep in New Zealand was approximately 6.5 su/ha, which is comparable to the average stocking density of alpacas in this study [170]. Alpacas can be stocked at a higher density because the slow passage of forage through the digestive tract allows better feed conversion (i.e. use less forage to satisfy nutritional requirements) [158]. They also have lower nutrient requirements so more alpacas can be maintained per paddock when compared to other ruminant species [150].

Pleasure featured highly (16/18 farmers) in the reasons alpaca farmers reared alpacas, even though the main reason alpacas are reared in New Zealand is for their fleece (15/18 farmers). As a result fibre characteristics were the main selection criterion when breeding or buying alpacas. Even farmers who rear alpacas as a business also considered pleasure as one of their main drivers for farming these animals.

More than 80% (9/11) farms spent more on veterinary care than anthelmintics. The average annual cost was \$1045NZD, with business farmers (\$34.08 per alpaca) spending more than hobbyist farmers (\$29.85 per alpaca). In this study it was observed that most of the deaths on the farm were seen in crias (<1year) and therefore increased veterinary intervention may decrease the mortality in this management group. Quick summoning of the farm veterinarian may decrease mortality among crias and should be considered more often by farmers. Similarly, post-mortem examinations on crias that die may also assist in preventing future deaths.

No farmer in this study used pour-on anthelmintics and this is most likely associated with ensuring the quality fleece is not stained [169]. In addition the use of pour-on anthelmintic applications is discouraged because they are not considered effective against common gastrointestinal parasites [171]. However, a recent report suggested that in llamas plasma levels following oral Ivermectin® (0.2 mg/kg) failed to reach quantifiable levels when compared with plasma levels reached by pour-on (0.5 mg/kg) and subcutaneous injectable treatments (0.2 mg/kg) [169].

Cross grazing alpacas with other animals species to assist with parasite control, was seen on 10 of the 18 farms surveyed. Three of the 18 farms have a drug rotation policy in place while 55.6% (10/18) of the farmers are

still using the first anthelmintic introduced on their farm. These practices increase the risk of developing resistant nematodes. One farm in Northland that cross grazed with cattle reported drug resistance to Dectomax© (macrocyclic lactone) and therefore switched to a combination wormer. There are reports of resistant gastrointestinal nematodes in many species [113, 172, 173]. Resistance of *Haemonchus contortus* to doramectin (the active ingredient in Dectomax©) has been shown to occur in alpacas [109]. In this global climate of emerging resistant strains of nematodes, alpaca farmers in New Zealand need to ensure that current practices do not produce resistant nematodes in the alpaca population. Targeted strategies to manage anthelmintics across the industry are required as well as evaluation of management practices already implemented on some farms in New Zealand.

Drug rotation policies, cograzing with horses or ponies, the use of natural wormers and pasture vacuuming are all utilised by the alpaca farmers surveyed. The effectiveness of these practices has not been evaluated in alpacas in New Zealand and further work should be done to gauge their efficacy against *Haemonchus contortus* (the most common nematode in alpacas) and other gastrointestinal nematodes.

Willow is a natural wormer used by some farmers. Willow reduces *Haemonchus contortus*, females of *Teladorsagia circumcincta* and *Cooperia*, and therefore determining the dose and cost effectiveness when compared to commercially produced wormers may be helpful to farmers who opt for natural wormers [174]. Pasture vacuuming is a technique employed by some farmers to remove faeces presumably before infective larvae hatch from eggs. Removal of faeces is encouraged as an excellent strategy but the efficacy of pasture vacuuming is unknown in New Zealand alpacas [169].

Reproductive practices are relatively consistent in diagnostic techniques, and post natal practices but very varied in number of times females are rebred and determining reproductive problems. Reports in the literature suggest that alpacas have low fertility and most problems in reproduction may be associated with a low sperm count in males [22]. In this study most farmers who had hembras that failed to conceive after the first mating got success with repeat breedings. This need for repeat breedings may be associated with low sperm count in males but more research into reproductive failure is needed.

In females the most common causes of infertility in the USA were found to be uterine infections and uterine fibrosis [175, 176]. Farmers (26.7%) in

this study indicated that veterinarians are used to investigate reproductive failure in hembras but no specific information was gathered to identify the causes of reproductive failure on these farms. Fertility was also reported as a problem in New Zealand alpacas in the Sustainable farming fund report of 2004. Further work needs to be done to determine the most common causes of reproductive failure in New Zealand alpaca males and females.

There are no mandatory disease surveillance programmes in New Zealand alpacas but this survey indicated that 72.2% (13 of 18) farms volunteered for TB testing. This figure is most likely related to a negative TB result being a requirement for showing alpacas. As a result all 7 farms that show alpacas participated in the TB program.

FE was reported as a common cause of death on one North Island farm who reported all of the 5 deaths in the 2012 to 2014 to be in adult (2-11 year old) alpacas. There are no reports of FE on South Island farms and therefore no Zn supplementation was reported. FE is seen usually in late summer and autumn in the North Island. In alpacas initially there are no outward clinical signs, eventually chronic weight loss occurs due to cumulative destruction of the medium sized bile duct, hepatocellular damage, and hepatic fibrosis [177]. The administration of Zn inhibits free

radicals production from the causative mycotoxin sporidesmin which when untreated causes pericholangitis and subsequent liver damage [144]. The sporidesmin is produced by the fungus *Pithomyces chartarum* which is only present in the North Island. As a result preventive Zn treatments are given on 71% (10/14) of North Island farms.

Congenital defects are well documented worldwide in crias. The most frequently congenital defects in alpacas are cardiac (3) and facial defects (2), as was also seen in this survey. The most common cardiovascular defects are ventricular septal defects; while choanal atresia (the most widespread congenital defect) and wry face are also common in New Zealand crias [178]. It is believed that a narrow gene pool accounts for the congenital defects that are relatively common in alpacas. The Sustainable Farm Fund also reported the causes of deaths in 2004 and the findings were similar, with FE, stomach ulcers and euthanasia related to old age notable similarities.

## CONCLUSION

This survey gives a starting point for further investigation into alpaca farming practice and herd health. In particular it has highlighted the need for further research into the causes of reproductive failure, and of death on farms and to determine the best practice for anthelmintic usage.

## CHAPTER 3

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### INTRODUCTION

Alpacas are even-toed, herbivorous mammals originating in the Andes of South America [4, 5, 16]. The two distinct breeds, Huacaya and Suri, are commercially reared worldwide for their fibre, and have been farmed in New Zealand since the 19<sup>th</sup> century [179]. In 2012, the total alpaca population in New Zealand was estimated to be 23,000 animals [4].

Haemotrophic mycoplasma, bovine viral diarrhoea virus (BVDV) and gastrointestinal parasites affect alpaca populations worldwide [26, 105].

*Candidatus* Mycoplasma haemolamae (CMHl) is a haemotrophic mycoplasma that infects alpacas and llamas. It is a small (0.4 – 1.0  $\mu\text{m}$ ), pleomorphic, wall-less, gram negative, extracellular bacteria, and is present as a coccoid or ring shaped basophilic organism on the surface of



erythrocytes [26-29]. Young animals are more susceptible to acute infection but infection is most common in adult animals [24]. The clinical signs of infection in alpacas include mild to severe anaemia, lethargy, depression, weight loss or reduced weight gain, hypoglycaemia, and fever [27, 97]. The mode of transmission is unknown but is associated with transmission of infected blood.

Traditionally diagnosis has been by blood smear examination, but this technique has poor sensitivity and specificity [71]. The introduction of polymerase chain reaction (PCR)-based assays for the organism has improved our ability to diagnose infection with CMhl.

In 2013, CMhl was first confirmed in New Zealand in a clinically normal alpaca that was housed on a farm with a severely anaemic alpaca. On that farm, ten animals were tested for non-specific haemotropic mycoplasma, only one tested positive and it was later confirmed by sequencing to be CMhl [98].

Bovine viral diarrhoea (BVD) is caused by a single stranded RNA virus from the Pestivirus genus, Flaviviridae family [122]. BVDV causes many clinical syndromes depending on the age at which the animal is exposed to the virus. BVDV may cause gastrointestinal tract infections, respiratory infections, abortion, act as a teratogen, and cause still births or the birth

of weak calves [118]. In addition the fetus may develop immune tolerance, resulting in persistent infection (PI), and thus act as a reservoir of infection for the whole herd [120, 121]. In cattle the infection of a pregnant dam after day 30 of gestation (usually between day 25 and day 90 of gestation, during the self-recognition phase of the immune system ontogeny) can lead to a persistently infected calf. The timing of infection required for the development of persistently infected crias has not been identified but is believed to be in the first trimester. The gestation length of alpacas (345 days) is longer than cattle (284 days) and therefore the self-recognition phase may be longer, meaning persistent infection could develop up to day 145 of gestation [121]. The main mode of transmission in cattle is nose-to-nose or sexual contact, whether this is also the case in alpacas is unknown [122]. Diagnosis of BVDV in alpacas is by serological testing for BVDV antigen and antibody or PCR for antigen.

Parasites are the most common disease problem in alpacas worldwide [104]. Gastrointestinal nematodes are known to have the largest impact in young alpacas, causing non-specific clinical signs such as diarrhoea (most common presenting sign), reduced weight gain, pale mucous membranes, mild to severe regenerative anaemia, and sudden death [105, 108].

Alpacas that are stressed are more likely to be anaemic and have

gastrointestinal nematode infestations [24, 107]. Gastrointestinal nematode numbers increase from January to April in New Zealand i.e. late summer to autumn.

With the recent confirmation of CMhl in New Zealand, the primary aim of this study was to determine the prevalence of Mhl in New Zealand alpacas. In addition we also aimed to determine the prevalence of anaemia, exposure to BVDV, and the common gastrointestinal parasites in alpacas, as potential risk factors for disease due to CMhl.

## METHODS AND MATERIALS

### **SAMPLE COLLECTION**

The alpacas sampled were more than one year of age, and were selected from alpaca farms around New Zealand with herd sizes of more than forty animals. In addition, alpaca blood samples sent for routine haematology to New Zealand Veterinary Pathology Ltd (NZVP Ltd) diagnostic laboratories around New Zealand were also collected. Approximately 10 - 20% of the adult alpacas in each herd were sampled, based on estimated prevalence of CMHl in other studies [27, 29, 97]. In total, samples from 206 alpacas from 12 regions around New Zealand were collected (Figure 12). There was a positive selection bias towards those with reported risk factors for CMHl, including imported animals, and animals with a history of anaemia and/or lethargy. All samples were collected between June 2013 and September 2014.

Blood samples were collected by jugular venipuncture into plain evacuated tubes, and tubes containing EDTA anticoagulant. Blood smears were made within 4 h of the blood being taken. The plain evacuated tubes were centrifuged at 1800 x g for 15 min, the serum removed and stored in

microcentrifuge tubes at -20°C until required. EDTA blood was also stored at -20°C until needed.

Faecal samples were collected either from the rectum of each animal or when the animals were observed defecating and the sample collected immediately from the ground. These were chilled and stored in airtight plastic containers until processing.

Each of the 12 farms completed a one-page questionnaire around the time of sampling to assess the risk factors associated with CMHl infection. The questionnaire included eight yes or no questions on the health status of the herd and the management practices of the farm sampled, and gathered general information on the number of animals, breed and sex distribution of animals on the farm. The risk factor questionnaire is attached in Appendix 1.



Figure 12 Map of New Zealand showing the number of alpacas 1 year of age or older sampled in each region between June 2013 and September 2014

## **FAECAL EGG COUNTS AND FAECAL CULTURE**

Faecal egg counts (FEC) were performed within 48 h of sample collection on 145 individual faecal samples. The faecal samples were then pooled by farm, and faecal cultures were performed on each pool. The faecal analyses were performed by the Parasitology laboratory, Massey University. Briefly, FEC were performed using a NaCl solution with a specific gravity of 1.2, and faecal cultures were performed on a vermiculite, water and faeces mix, and incubated at 23 – 25°C for 10 d followed by larval identification.

## **BLOOD SMEARS**

A total number of 205 smears were examined, 35 were peripheral blood smears and 170 were central vein blood smears, representing 170 animals. The smears were stained with Siemens Diff-Quik® solutions as per the standard protocol. Each slide was evaluated for haemoplasma infection and erythrocyte morphology using 1,000 x magnification.

## **PACKED CELL VOLUME AND TOTAL SOLIDS**

The packed cell volume (PCV) and total solids (TS) were determined for each animal where possible. EDTA blood was centrifuged at 900 x g in a microcapillary tube for 10 min. The PCV was determined using a PCV

reader card, and TS were measured using a temperature compensated refractometer (Reichert Vet 360).

## **BVD SEROLOGY AND PCR**

As per the manufacturer's instructions the BVD p80 antibody ELISA test (ELISA BVD/MD/BD p80 kit, Institut Pourquier IDEXX, France) can be used on bovine milk, plasma, and serum, and sheep serum. It has also been used on alpaca serum in New Zealand and in the literature to diagnose BVD positive animals (Table 11) [180]. BVD antibody ELISA screening test was done on pooled serum samples from each farm with a maximum of 10 samples per pool. Multiple pools were done from farms where more than 10 animals were sampled. Positive pool samples were then individually tested. The BVD antigen PCR test was done on pools of 10 samples. In this way 195 serum samples were tested for BVD antibody and BVD antigen.



**Table 11 Conclusions on status of animals tested for Bovine Viral Diarrhoea (BVD) Virus antibody and antigen based on the immunological responses**

BVD p80 antibody	BVD antigen	Conclusion
negative	positive	Exposed to virus but no immune response (retest to confirm persistently infected)
negative	negative	Not exposed to virus
positive	positive	Active infection or transiently infected with BVDV, mounted an immune response
positive	negative	Was exposed to the virus and mounted an immune response or passive transfer from colostral antibodies

## **DNA EXTRACTION FOR REAL-TIME PCR FOR CMhI**

DNA was extracted using a Qiagen DNeasy® Blood and Tissue kit

(Germany) as per the manufacturer's instructions. The DNA was stored at -20°C until real-time PCR was performed.

## **REAL-TIME PCR**

Real-time PCR assays were performed using an Applied Biosystems

StepOne Plus® Real-time PCR machine and Applied Biosystem TaqMan®

Gene Expression Master Mix (Lifetech Technologies, USA). Each reaction

consisted of 5 µL of TaqMan® mastermix, 18 µM of probe 18S or CMhI,

and 1 µL of DNA, made up to a final volume of 10 µL with DNA-free water.

In each PCR run, a negative control (water) was included to check for

contaminants, and a positive control (known positive Mhl) to confirm the presence of amplifiable DNA and absence of PCR inhibitors [98]. The real time PCR conditions consisted of an initial incubation at 50°C for 2 min and 95°C for 10 min. This was followed by 40 cycles of 95°C for 15 s and 60°C for 1 min. Fluorescence data was collected at the end of each combined elongation annealing stage.

Primer-probe combinations were ready made mixes from Applied Biosystems. The 18S primer-probe mix was a universal primer-probe (HS99999901\_S1). The Mhl primer-probe sequences had previously been published but were 5'd AAAAGCAGGATAGGAAATGATTCTG 3' for forward and 5'd TGCTGGCACATAGTTAGCTGTCA 3' for reverse and 5' CCATTGGAGGGCAAGTCTGGTGCCA 3' for probe [102].

Five-point standard curves were generated for CMhl (Figure 13) and 18S DNA (Figure 14) in order to determine the efficiency and  $R^2$  of each reaction. For the CMhl the  $R^2$  and efficiency were 0.99 and 93.849% respectively, and for 18S the  $R^2$  and efficiency of 18S probe were 0.996 and 94.387% respectively.

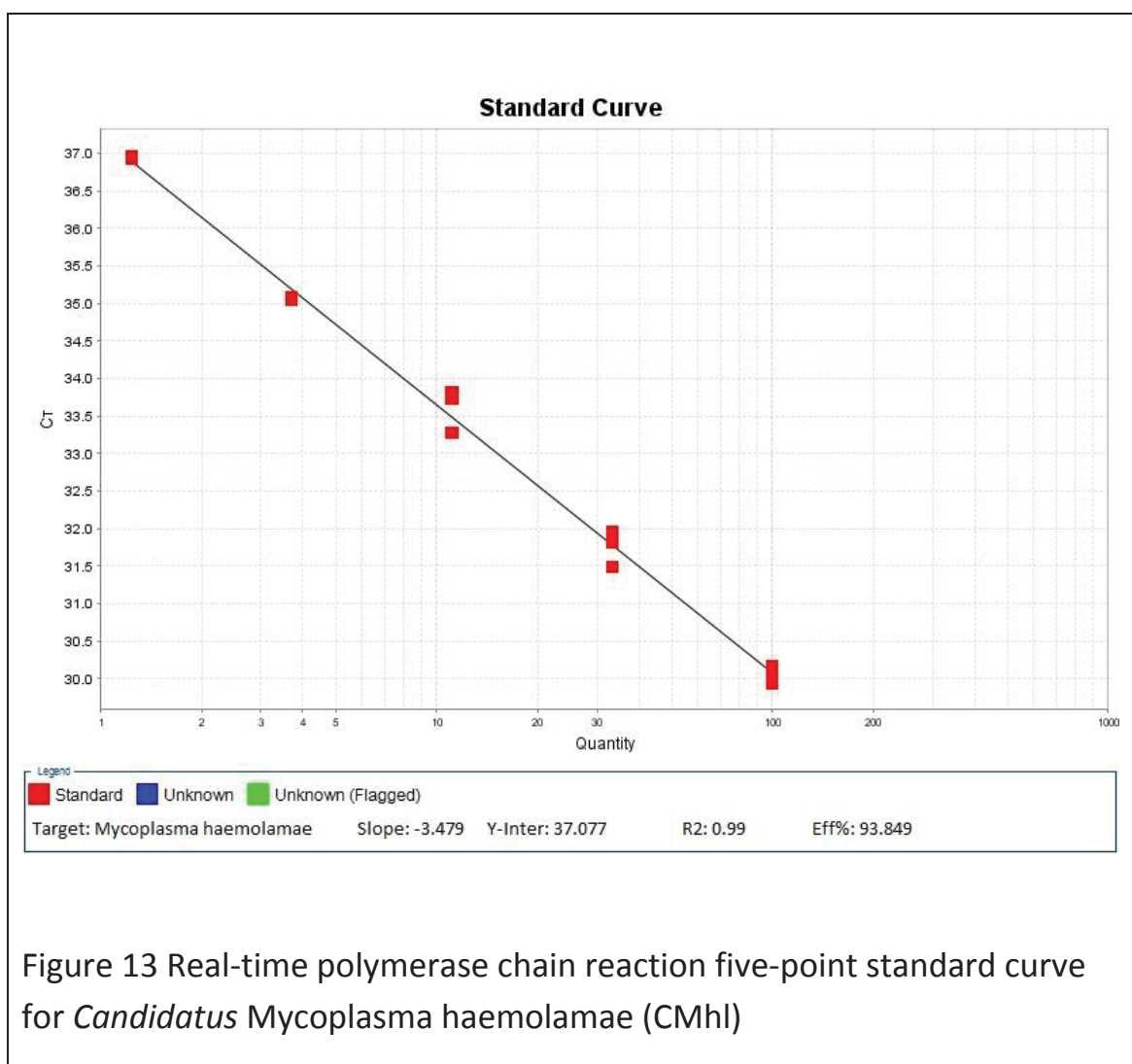


Figure 13 Real-time polymerase chain reaction five-point standard curve for *Candidatus* Mycoplasma haemolamae (CMH)

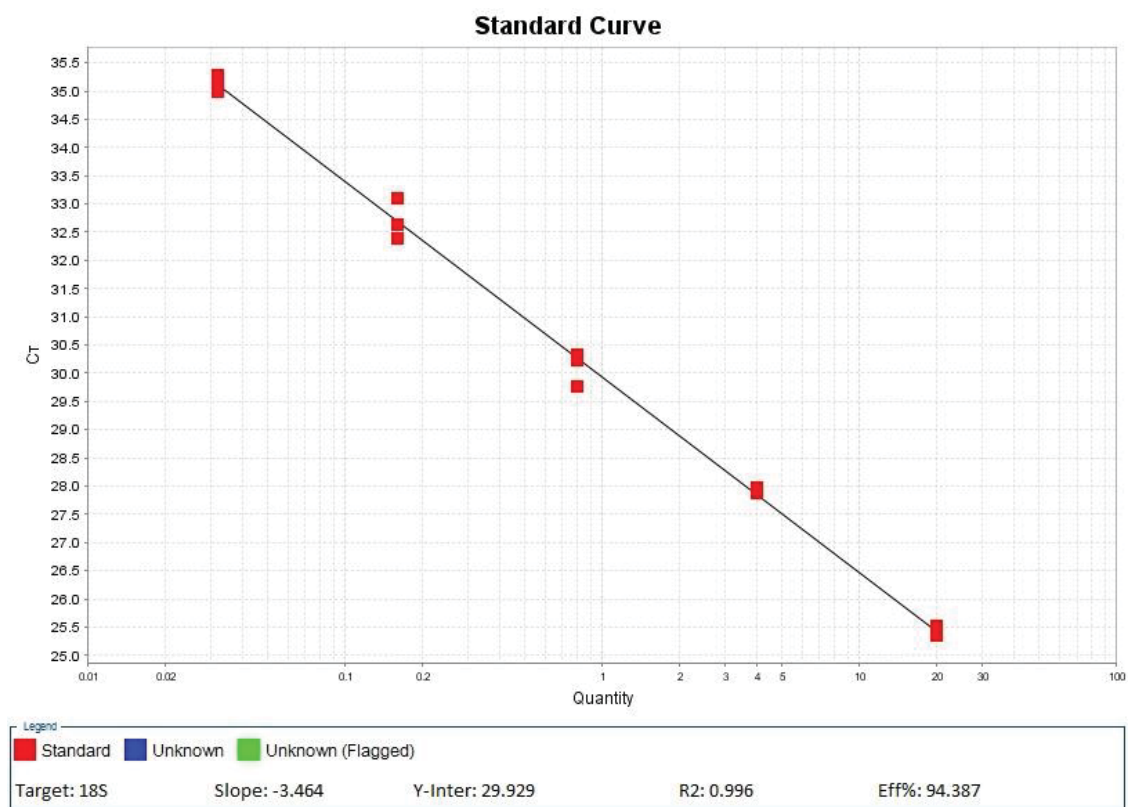


Figure 14 Real-time polymerase chain reaction five-point standard curve for 18S

## RESULTS

### ANIMAL STATISTICS DERIVED FROM THE RISK FACTOR QUESTIONNAIRE

Of the 206 animals sampled, 104 were Huacaya, 32 were Suri, however for 70 animals the breed was not reported. The distribution of breed and sex is shown in Table 12. The adults sampled ranged in age from 2 – 23 years, with 3 year olds being the most commonly sampled animal (Figure 15).

Table 12 Breed, sex distribution and numbers of neutered male alpacas sampled in New Zealand *Candidatus* Mycoplasma haemolamae study, surveyed between June 2013 and September 2014

Breed	Total Males (Neutered males)	Total Females	Sex Not reported	Total
Huacaya	20 (1)	74	10	<b>104</b>
Suri	1	31	0	<b>32</b>
Not reported	11 (1)	29	30	<b>70</b>
<b>Totals</b>	<b>32</b>	<b>134</b>	<b>40</b>	<b>206</b>

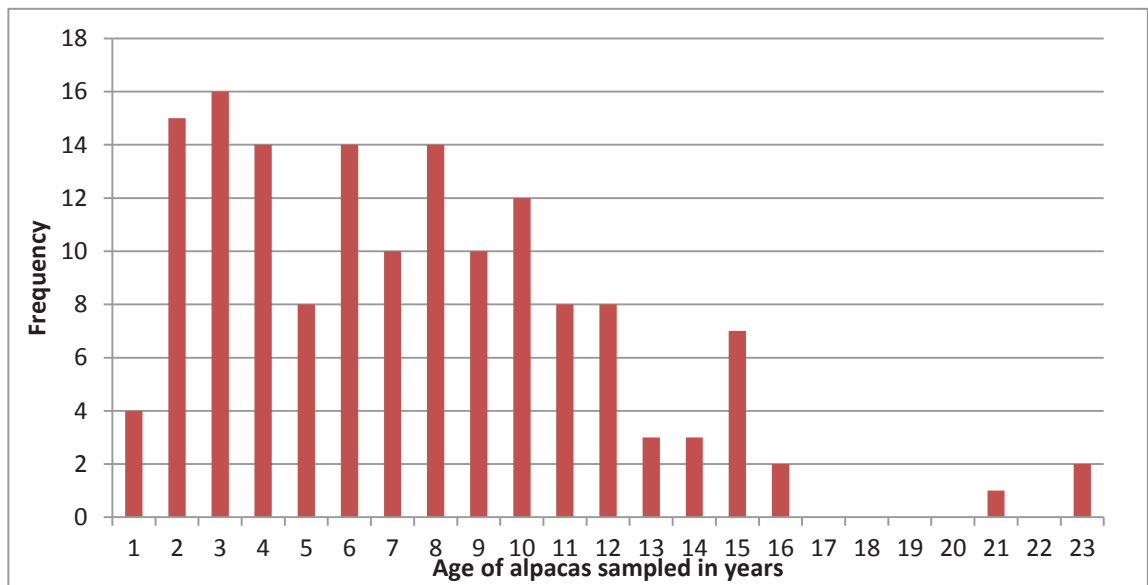


Figure 15 Age frequencies and distribution of adult alpacas with known ages between June 2013 and September 2014

All farmers who completed the questionnaire had 5 plus years of farming experience. Imported alpacas were present on 10 out of 12 farms sampled. Of these 10 farms, all had imported alpacas from Australia, one farm from the USA, one from Peru, and one from Chile. Other animal species were present on 42% (5 out of 12) of farms, with all farmers reporting that the alpacas grazed alone, and that the other species were low in number and used primarily to graze around faecal piles. Cattle and sheep were the most common species on the farm with alpacas (4 farms), while horses were on three farms, and goats on one farm.

Dectomax<sup>®</sup> (Doramectin, Pfizer New Zealand Ltd), a macrocyclic lactone, was used for gastrointestinal parasite control on 50% of farms, and Matrix<sup>®</sup> (Ivermectin, Levamisole, Oxyfendazole and Praziquantel, Merial New Zealand) a combination worming product was used on 42% of farms. Genesis<sup>®</sup>, Genesis Ultra<sup>®</sup>, Genesis Horse<sup>®</sup> (Abamectin and Praziquantel, Merial New Zealand), Zolvix<sup>®</sup> (Monepantel, Novartis Animal Health) and Bomatak<sup>®</sup> (Oxyfendazole, Bayer Animal Health New Zealand) were each used on one farm in the survey, with one farmer using an unspecified combination of wormers routinely. Alpacas were treated for gastrointestinal parasites as required based on FEC on 42% of farms. Scheduled treatment was used on 3 farms.

All of the 12 farms supplemented with vitamin D. Supplementation with copper, vitamin B12 and zinc was routine on 5 farms, and selenium was supplemented on 4 farms.

One farmer reported BVD had previously been diagnosed on the farm, in a PI alpaca and one farm reported having had PI cattle previously on the farm. Facial eczema was reported on only one farm. No farms reported previous diagnosis of CMHl infection.

## FAECAL ANALYSIS

### FAECAL EGG COUNT (FEC)

Of the 143 FEC performed, 55 were free of eggs (38.46%), while the remaining 88 had eggs from gastrointestinal parasites. Of these, 55 contained 200 eggs per gram (epg) or less (38.46%) and 33 had clinically significant numbers of eggs (greater than 200 epg (23.07%) [181]. Figure 16 records the FEC and PCV of the 143 alpacas tested.

### FAECAL CULTURE

There were 13 faecal cultures performed, 3 on individual faecal samples and 10 on faeces pooled by farm. *Haemonchus contortus* was the most common species of nematode cultured in adult alpacas in this study.

*Trichostrongylus spp* and *Cooperia spp* were also common in the alpacas surveyed. Table 13 contains the frequencies of the gastrointestinal nematodes seen in the study. Of the 143 FEC examined *Capillaria spp* was identified in 4 samples, indicating a prevalence of 2.8% in the alpacas tested.



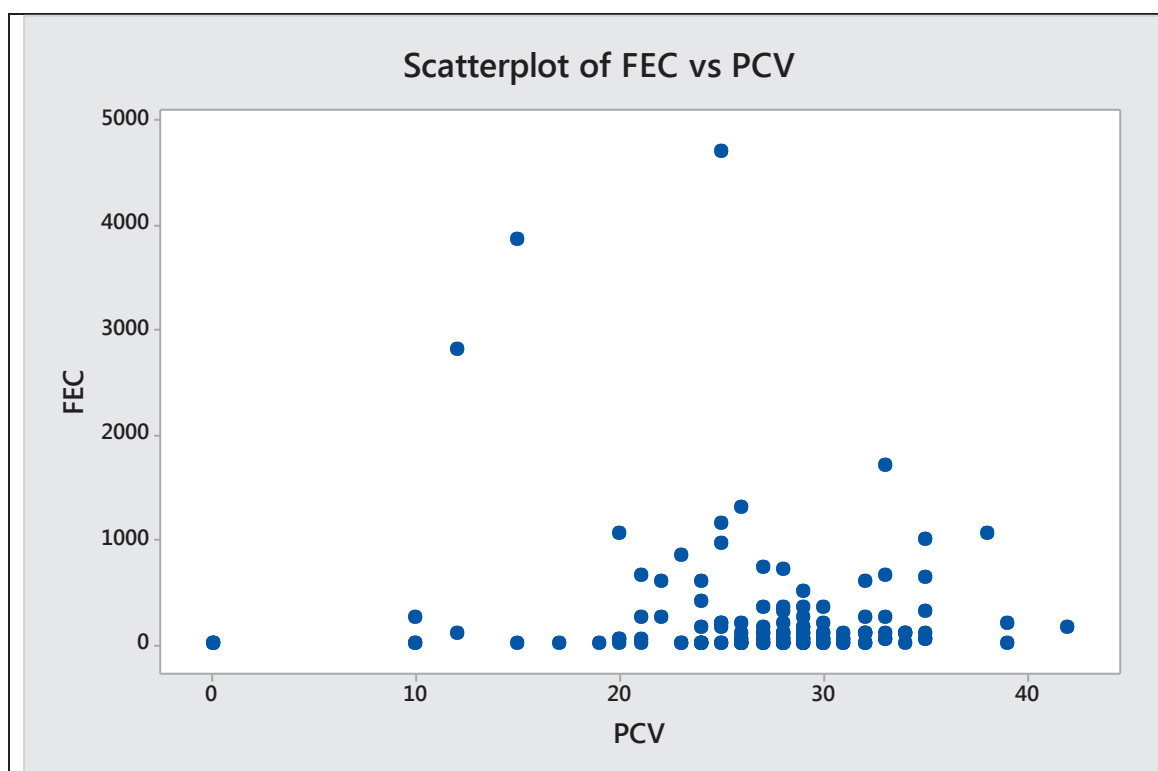


Figure 16 Scatterplot of the Packed Cell Volume (PCV) of adult alpacas versus Faecal Egg Count (FEC) values recorded in the New Zealand *Candidatus* *Mycoplasma haemolamae* survey

Table 13 Frequency of occurrence of gastrointestinal nematodes cultured from the 13 faeces of adult alpacas in New Zealand between June 2013 to September 2014

Nematode larvae reported in alpacas in New Zealand	Frequency
<i>Haemonchus contortus</i>	11
<i>Trichostrongylus axei</i> and <i>askivali</i>	9
<i>Cooperia</i> spp	9
<i>Teladorsagia circumscripta</i> / <i>Ostertagia</i>	5
<i>Capillaria</i> spp	4
<i>Nematodirus lamae</i>	2
<i>Camelostrongylus mentulatus</i>	1

## **BLOOD ANALYSIS**

### **PACKED CELL VOLUME (PCV) AND TOTAL SOLIDS (TS)**

Of the 193 alpacas tested, 42 (21.76%) were outside the normal range (25% – 45%) and were therefore considered anaemic. The PCV, TS and FEC for the 42 alpacas outside the normal range are seen in Table 14. This showed that 11 of the anaemic animals had a significantly high faecal egg count (epg of over 200), with 12 untested for gastrointestinal parasite eggs in the faeces. Therefore 37% of animals with a low PCV had high faecal egg counts (11 of 30). Of the remaining alpacas tested 23.81% (30 of 126) had a normal PCV and a high FEC while 76.19% (96 of 126) had a normal PCV and a low FEC. Thirty-six of the 193 alpacas tested had a normal PCV but no FEC was performed.

Total solids were tested in 177 of the 193 blood samples analysed and the values ranged from 17 to 79 g/L, with any value below 51g/L reported as low. Six of the 177 samples (3.38%) were low. Figure 17 shows the variation in TS and the relationship between the TS and PCV.

Table 14 The packed cell volume (PCV), total solids (TS) and faecal egg count (FEC) of the 42 anaemic alpacas of the 193 adults tested for the *Candidatus* Mycoplasma haemolamae study between June 2013 and September 2014 in New Zealand

PCV	TS	FEC		PCV	TS	FEC		PCV	TS	FEC
7	-	-		17	58	0		23	53	-
<10	44	-		19	59	0		23	61	0
<10	56	0		20	52	0		23	58	850
<10	56	250		20	53	50		23	-	-
10	58	0		20	57	1050		23	58	0
12	53	2800		20	63	50		24	47	-
12	66	100		21	57	250		24	57	600
13	-	-		21	61	0		24	60	0
14	-	-		21	62	-		24	73	0
15	62	0		21	62	50		24	61	400
15	67	3850		21	57	250		24	66	0
16	-	-		22	-	-		24	63	0
17	-	-		22	52	250		24	62	0
17	-	-		22	61	600		24	63	150

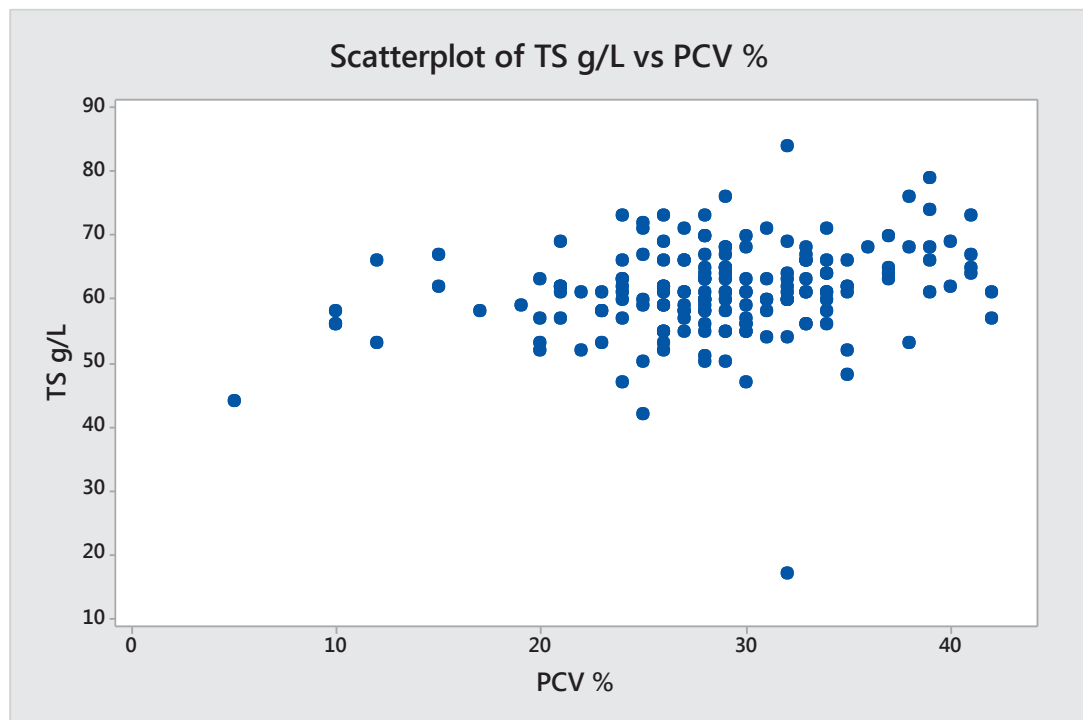


Figure 17 Scatterplot of total solid (TS) and packed cell volume (PCV) of adult alpacas sampled in New Zealand between June 2013 and September 2014

## BLOOD SMEARS

Of the 170 central vein blood smears and the 35 peripheral blood smears examined, 8 contained suspected CMhl organisms (based on blind assessment), while 18 had dacrocytes, spindle shaped cells and hypochromic erythrocytes (Figure 7) likely associated with iron deficiency anaemia [24]. The examination of the peripheral and central blood smears revealed identical findings. Marked numbers of polychromatophils

(seen as cells with uneven distribution of haemoglobin) were seen in 11 of the 18 slides with tear-drop-shaped cells, this is consistent with a regenerative response, even though the haematology parameters often stay within normal ranges [24]. The prevalence of CMhl infection from blood smear examination was 4.7% (8/170).

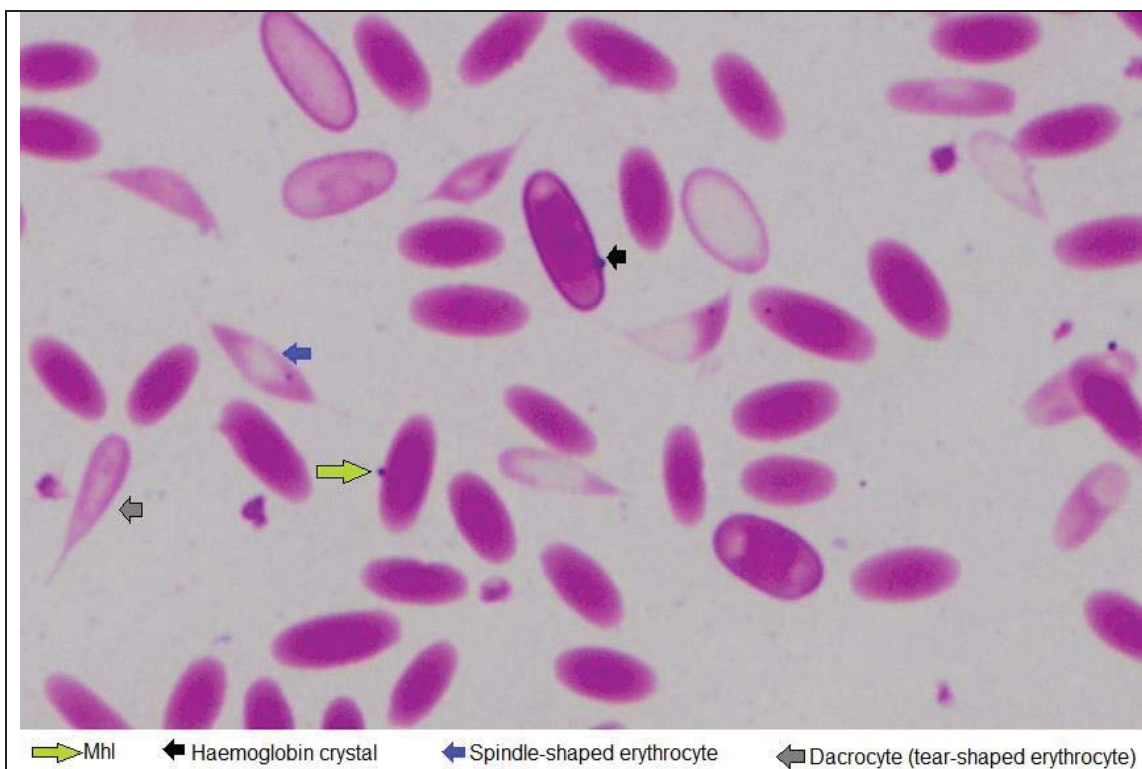


Figure 18 Photograph of blood smear used for cytological examination of an adult alpaca with hypochromic anaemia

## **BOVINE VIRAL DIARRHOEA VIRUS (BVDV)**

None of the 195 serum samples were positive by PCR test for BVDV antigen and therefore there were no animals persistently infected with BVDV or animals with active or transient infections detected in this study (see Table 11). Of the 195 serum samples tested by ELISA for BVDV antibody 4 animals were positive, from 3 of 16 pools. The prevalence of BVDV serum antibodies was therefore 2.05% (4 out of 195), with 18.75% of farms having animals that were exposed to BVDV. The alpacas with antibodies to the BVDV virus were negative for the BVDV antigen, indicating they had been infected with BVDV in the past and mounted an immune response. Most animals in the study were not exposed to the BVDV.

## ***CANDIDATUS MYCOPLASMA HAEMOLAMAE (CMhl)***

Two samples were positive for CMhl by real-time PCR. The prevalence of CMhl in the adult New Zealand alpacas sampled was 0.97% (2 of 206). The complete blood analyses for the two animals' positive by PCR are contained in Table 15.

Table 15 Complete blood and faeces test results, age and place of birth of the *Candidatus Mycoplasma haemolamae* PCR positive alpacas tested between June 2013 and September 2014 in New Zealand

Patient ID	Positive 1	Positive 2
Age /years	5	2
PCV %	32	12
TS g/L	60	53
FEC epg	250	2800 - 50 Capillaria
Place of birth	New Zealand	New Zealand
Blood smear	Positive	Positive

## DISCUSSION

This study showed that the prevalence of CMhl by PCR in New Zealand adult alpacas is 0.97%. Anaemia was common with 21.76% of animals sampled having a PCV <25, and 37% of alpacas with anaemia had significant gastrointestinal parasite burdens (over 200epg). The prevalence of BVDV antibody was 2.05%, while none of the animals surveyed were persistently infected or positive to BVDV antigen.

The prevalence of CMhl in New Zealand alpacas by PCR was 0.97% (2/206) but by blood smear examination the prevalence was 4.7% (8/170). Blood smear exam is not as specific as PCR. Haemoglobin and stain residues may be mistaken for positive CMhl diagnosis on blood smear (see Figure 18) and older stain residues can form clumps that are difficult to distinguish from CMhl organisms attached to the blood cell membrane [37, 80]. CMhl is the only haemotrophic mycoplasma identified in alpacas so it is assumed that all organisms identified by cytology are CMhl but a generic Mycoplasma PCR was not performed, so the presence of unidentified Mycoplasmas cannot be definitively ruled out. In other species where there are more than one haemoplasma, for example in cats, identifying a haemoplasma by blood smear is not a definitive diagnosis and further



work is necessary to identify the specific mycoplasma species [64, 71]. In time, as diagnostic methods improve and subspecies or new species of haemoplasmas are discovered, more specific testing, like PCR, is required to further classify mycoplasmas. Therefore the overall prevalence of CMhl in this study is 0.97% and this study highlights the unreliability of blood smear examination. After re-examination of the blood smears it is most likely that in our study the higher prevalence on blood smear examination was due to false positive identification of Mycoplasma organisms.

The prevalence of CMhl in New Zealand is lower than that reported in other countries. In Peru at the La Raya Research Station the prevalence is 19.3%, and in Chile the prevalence is 9.26% [27]. In Europe the prevalences for South-east England, and Switzerland and Germany are 29% and 18.7% respectively [29, 97].

The differences in the prevalence of CMhl in New Zealand compared to those reported worldwide could be due to a variety of factors. Iatrogenic route of transmission was described as a potential route especially in sheep. Ear-tagging and reusing needles during herd vaccination are suggested as opportunities for transmission of infected erythrocytes. Even though the practice of reusing needles between alpacas cannot be ruled

out on on all farms it is believed to be less prevalent on alpaca farms than on sheep farms since the financial investment per alpaca is higher than per sheep. Subsequently because alpacas are more valuable, owners take less risks with their alpaca health resulting in less needle sharing and other practices that would allow iatrogenic transmission of CMHl.

Another important reason might be the difference in distribution of the cattle tick (*Haemaphysalis longicornis*) (a likely mode of transmission) and the location of the alpacas within New Zealand. The cattle tick (the only tick in New Zealand) is more prevalent in the North Island, while there are more alpacas in the South Island, concentrated particularly in the Canterbury region, where 36% (see Figure 12) of the samples were collected. However, this region has a high number of imported alpacas and this may be associated with the fact that both positive animals were from this region. Despite the positive animals being New Zealand born they were from a farm that imported a large number of animals.

In England there is some association between younger animals and higher infection rates [29]. Most farmers in New Zealand limit the interaction between humans and crias when the animals are young. This 'hands off' approach with minimal intervention and limited use of tools like

hypodermic needles may have worked well in preventing transmission of CMhl from infected to uninfected animals. In addition, good management practices by farmers may also have limited iatrogenic transmission of infection, thus contributing to a lower prevalence.

Nationally New Zealand does not allow importation of potentially sick alpacas. Even though imported alpacas are not tested specifically for CMhl, a complete blood count and PCV are done to ensure that imported animals are not anaemic. This may have inadvertently selected animals that are CMhl free since in one herd in Chile, a country with prevalence of 9% and from where alpacas are imported, CMhl was most often seen in anaemic animals [27].

Anaemia was present in 21.76% of alpacas sampled. Animals with a low PCV are difficult to determine clinically because alpacas are stoic, rarely show signs of poor health, and are known to mask illness well. In addition their thick fibre coat makes it difficult to determine declining physical condition. Only when alpacas become severely anaemic are obvious behavioural changes such as recumbency and reluctance to move seen. Farmers should be encouraged to check mucous membranes of the gums or inner eyelids to check for signs of anaemia in their alpacas. It is believed

that the increased oxygen carrying capacity of the elliptical erythrocyte allows them to maintain function with a PCV as low as 7%, as seen in an alpaca in this study. There are cases in New Zealand of death in alpacas associated with severe *Haemonchus* worm burdens resulting a severe blood loss anaemia (pers comm Cristin Dwyer).

Only 1 of the 2 animals PCR positive for CMhl was anaemic, and this animal had a very high FEC of 2800. This along with the fact that 37% (11 of 30) of the anaemic animals had a significantly increased FEC suggests that anaemia in alpacas is more likely associated with significant parasite burdens than CMhl infection.

In this study we used a normal reference range for PCV of 25% to 45%, as used by NZVP Ltd, the diagnostic laboratory. A study published on 50 alpacas in 2004 suggested that a PCV of 21% to 41% could be considered normal for alpacas [182]. If we used this reference range rather than the laboratory reference range, 20 (10.36%) of alpacas were anaemic.

Regardless of the reference range used, high FEC were associated with anaemia.

The most common parasites in this study were trichostrongyle-type nematodes including *Haemonchus contortus* (11), *Trichostrongylus axei*

and *T. askivali* (9), *Cooperia* (9), *Teladorsagia circumscripta* (5), and *Camelostrongylus mentulatus* (1), which made up 85.36% of the worms cultured. Most of the nematodes recorded in the survey were the typical nematodes seen in alpacas worldwide, although this is the first report of the gastrointestinal parasite *Capillaria spp.* in alpacas in New Zealand [108] [104]. The high incidence of *Haemonchus*, a blood sucking parasite, is likely related to the anaemia seen in alpacas with a high FEC.

Controlling these parasites will impact the number of anaemic animals in the herds in New Zealand. Anthelmintics were used by all farmers in the study however none of the products used by farmers are licensed for use in alpacas. Anecdotally common practice appears to be that most anthelmintics are used at one and half times the recommended dosage for sheep but no studies have been published that indicate if this dose is effective in alpacas. In this study no data on the dose rates and frequency of deworming were collected but the common occurrence of significant FEC suggests that parasite control practices in alpacas need examining and improvement.

Methods that decrease the amount of worm eggs ingested by alpacas will decrease the number of anaemic animals in the herd. Mixed grazing is one

method that can be used by alpaca farmers to decrease worm burdens.

Cattle, sheep and horses are commonly used especially to graze around communal faecal piles where the contamination is highest. However farms in this study with other species to graze paddocks did not have lower FEC than farms where only alpacas were grazed, possibly because the parasite species seen in alpacas are also common to sheep and cattle.

In this study only 2% of alpacas had serum neutralising antibodies to BVDV, despite BVDV being common in cattle in New Zealand, with a prevalence as high as 88% in beef herds in one study but believed to be  $\geq$  60% in cattle in New Zealand [183, 184]. In a recent study of 63 herds in the USA, 25.4% of herds had alpacas with antibodies to BVDV [126]. In our study with 16 pools of samples, 3 pools tested positive for BVDV serum antibodies indicating a herd prevalence of 18.75%. This is comparable to the USA study. In farms surveyed in our study 1 farmer (6.25%) reported a persistently infected alpaca although there were no laboratory reports to confirm this. However, this reflects similar data from the USA where 4 of the 63 (6.3%) farms had reported PI crias [126].

The animals positive for BVDV antibody came from 3 farms, 2 completed the questionnaire and both had other animals imported from Australia.

Both of the farms reared only alpacas on their farm and have been farming on that property for at least 6 years. It is therefore unlikely that the alpacas contracted the BVD virus on the property from contact with infected cattle.

## CONCLUSION

The prevalence of *Candidatus Mycoplasma haemolamae* in adult New Zealand alpacas is much lower than in other countries where studies have been performed.

Regenerative anaemia in young alpacas has been attributed to CMHl infection but in New Zealand is more likely associated with gastrointestinal parasite infection.

Abbreviation	
AANZ	Alpaca Association of New Zealand
Ab-ELISA	Absorbed Enzyme-linked immunosorbent assay
AGID	Agar Gel Immunodiffusion test
AO	Acridine orange
BD	Border Disease
BVD	Bovine Viral Diarrhoea
BVDV	Bovine Viral Diarrhoea Virus
BVD/MD	Bovine Viral Diarrhoea/ Mucosal Disease
C1, C2, C3	Compartment 1, compartment 2, compartment 3
CFT	Complement fixation test
CMhl	<i>Candidatus Mycoplasma haemolamae</i>
CMhm	<i>Candidatus Mycoplasma haemo</i>
CMho	<i>Candidatus Mycoplasma haemovis</i>
CMt	<i>Candidatus Mycoplasma</i>
DNA	Deoxyribonucleic acid
EDTA	Ethylenedinitrilotetraacetic acid
EHV2	Equine herpes virus 2
ELISA	Enzyme-linked immunosorbent assay
epg	eggs per gram
FE	Facial eczema
FEC	Faecal egg count
GGT	Gamma glutamyltransferase
MAP	<i>Mycobacterium avium subspecies paratuberculosis</i>
Mhf	Mycoplasma haemofelis
MHV1	Mouse hepatitis virus 1
Mo	<i>Mycoplasma ovis</i>
PCR	Polymerase chain reaction
PCV	Packed cell volume
PI	Persistently infected
RNA	Ribonucleic acid
rRNA	ribosomal Ribonucleic acid
SAC	South American camelids
TB	tuberculosis
US / USA	United States of America
UV	Ultraviolet
Zn	Zinc



# Appendix 1

Some of the natural colours of alpaca fibre



White

Beige

Light fawn

Medium fawn

Dark fawn

Light brown

Medium brown

Dark brown

Bay black

True black

Light s grey

Medium s grey

Dark s grey

Light r grey

Medium r grey

Dark r grey

## APPENDIX 2

## RISK FACTOR QUESTIONNAIRE

Name: \_\_\_\_\_

Address of farm: \_\_\_\_\_

Number of alpacas older than 1 year? Male \_\_\_\_ Female \_\_\_\_  
Breed Huacaya \_\_\_\_\_ Suri \_\_\_\_\_

### Health Status

1. Have you imported alpacas from overseas onto your farm? No  
Yes ☐ Number and years: \_\_\_\_\_  
Country/s of origin: \_\_\_\_\_ ☐
2. Are there other species of livestock on your farm?  
cattle ☐ sheep ☐ Other ☐ Please specify \_\_\_\_\_
3. Have any alpacas tested positive for Bovine Viral Diarrhoea antibodies?  
Yes ☐ No ☐  
  
Any alpacas confirmed with persistent infection? Yes ☐ No ☐  
  
Any persistently infected cattle? Yes ☐ No ☐
4. Have you previously had *Mycoplasma haemolamae* diagnosed on your farm?  
Yes ☐ No ☐
5. Have you had cases of facial eczema in the last 3 years?  
Yes ☐ No ☐ Approximate number? \_\_\_\_\_
6. What supplements do you currently give?  
  
Copper ☐ Selenium ☐ Vitamin B12 ☐ Vitamin D ☐  
  
Zinc during facial eczema season ☐ Other ☐ Please specify \_\_\_\_\_  
\_\_\_\_\_
7. Do you deworm (drench/pour-on) regularly? Yes ☐ No ☐  
With what product? \_\_\_\_\_ How often? \_\_\_\_\_

### Management Practices

8. Do you mix graze with cattle or sheep? ☐ Yes ☐ No

How long have you been farming alpacas on this property? \_\_\_\_\_ years

### APPENDIX 3 ALPACA FARM HISTORY

NAME OF FARM: \_\_\_\_\_ ADDRESS: \_\_\_\_\_

EMAIL: \_\_\_\_\_ PHONE NUMBERS: \_\_\_\_\_

WHAT SIZE IS YOUR FARM? \_\_\_\_\_

HOW MANY PADDOCKS ARE ON YOUR FARM? \_\_\_\_\_ SIZES: \_\_\_\_\_

DO YOUR ALPACAS HAVE ACCESS TO SHELTER? \_\_\_\_\_

HOW LONG HAVE YOU FARMED ALPACAS ON PROPERTY? \_\_\_\_\_

HOW MANY ALPACAS DO YOU HAVE ON YOUR FARM? \_\_\_\_ Suri \_\_\_\_ Huacaya \_\_\_\_

Crias \_\_\_\_ Last year crias \_\_\_\_ Adults (2-11years) \_\_\_\_ Geriatrics (12+) \_\_\_\_

DO YOU CLASSIFY YOUR OWNING ALPACAS AS A BUSINESS OR A HOBBY OF BOTH?

DO YOU HAVE SELECTION CRITERIA WHEN YOU BREED OR BUY ALPACAS? Y / N  
EXPLAIN \_\_\_\_\_

WHY DO YOU OWN ALPACAS?

Pleasure Show Fibre Selling breeding stock / pets Meat

ARE THERE OTHER SPECIES OF LIVESTOCK ON YOUR FARM? Y / N

Cattle Sheep other \_\_\_\_\_

DO YOU CROSS GRAZE? Y / N EXPLAIN \_\_\_\_\_

ESTIMATE HOW MUCH IS SPENT ANNUALLY ON ANIMAL HEALTH? \_\_\_\_\_

OR

ESTIMATE HOW MUCH IS SPENT ANNUALLY ON WORMING? \_\_\_\_\_

ESTIMATE HOW MUCH IS SPENT ANNUALLY ON ADDITIONAL VET CARE? \_\_\_\_\_

DO YOU USE A SOFTWARE PACKAGE TO KEEP TRACK OF YOUR ALPACAS?

Y / N Which one? \_\_\_\_\_

WHAT ALPACA ASSOCIATIONS OR CLUBS ARE YOU A MEMBER? \_\_\_\_\_

COMMENTS ON FARM HISTORY SECTION:

## WORMING PROCEDURES

DO YOU DEWORM? Y / N (drench / injectable)

HOW OFTEN DO YOU DENCH?

HOW MANY ANIMALS GET DRENCHED IN 1 YEAR?

\_\_\_\_\_

	Frequency	Totals in 1 year
crias		
Last year's crias		
Adults (2-11)		
Geriatrics *		

\* 12 years old or older

WHAT INDICATORS DO YOU USE TO DECIDE WHEN TO DRENCH?

Consistency of stool    Body condition    Pregnancy status / expected due date    Finances  
 Age of animal    Weather    Faecal Egg Count (FEC) test results  
 Season    Vet recommendation    Quarantine    Other.

Explain \_\_\_\_\_

WHAT INDICATORS DO YOU USE TO DECIDE DOSAGE OF DEWORMER?

Manufacturer recommendation for sheep    Experience  
 Vet recommendation    Body weight estimates    Weight on scale    Other.

Explain \_\_\_\_\_

LIST PRODUCTS CURRENTLY IN USE ON YOUR FARM TO DEWORM ANIMALS

	Product Name	Route of Administration	Dose rate used mls per weight	Years product used on farm

LIST PRODUCTS PREVIOUSLY USED ON YOUR FARM TO DEWORM ANIMALS

	Product Name	Route of Administration	Dose rate used mls per weight	Years product used on farm

1-Dectomax 2-Ivomec 3-Matrix 4-Zoltil 5-Cydetin 6-panacur 7-Scanda 8-

WHAT IS YOUR REASON(S) FOR DISCONTINUING PRODUCT(S)? Put the number of the product next to the reason for discontinuing products. NB Product numbers may be used more than once.

Cost                      Unavailability      Drug resistance                      Rotation policy.

Explain \_\_\_\_\_

HOW OFTEN ARE FEC DONE ON YOU FARM?  
DONE IN 1 YEAR?

HOW MANY ANIMALS HAVE FEC

	Frequency	Totals in 1 year
Crias		
Last year's crias		
Adults (2-11)		
Geriatrics *		

HAVE ANY FAECAL EGG COUNT REDUCTION TEST (FECRT) BEEN DONE ON YOUR FARM?              Y / N

WHEN DO YOU DEWORM?

Crias	
Last year's crias	
Adults (2-11)	
Geriatrics	

DO YOU DO ALTERNATIVE REMEDIES OR TECHNIQUES OF PARASITE CONTROL?

Pasture vacuuming Y / N              EXPLAIN

\_\_\_\_\_

Natural wormers Y / N              EXPLAIN

\_\_\_\_\_

Other

\_\_\_\_\_

\_\_\_\_\_

COMMENTS ON WORMING PROCEDURES

## DEATHS AND REPRODUCTIVE PRACTICES

DO YOU RECORD DEATHS ON YOUR FARMS? Y / N If no skip to questions on Reproductive Practices (next page)

HOW MANY DEATHS HAVE YOU HAD ON YOUR FARM IN 2014?

Total number of deaths: \_\_\_\_\_

	Summer	Autumn	Winter	Spring	Total
Crias <1 week					
Crias					
Last year's crias					
Adults					
Geriatrics					

HOW MANY DEATHS HAVE YOU HAD ON YOUR FARM IN 2013?

Total number of deaths: \_\_\_\_\_

	Summer	Autumn	Winter	Spring	Total
Crias <1 week					
Crias					
Last year's crias					
Adults					
Geriatrics					

HOW MANY DEATHS HAVE YOU HAD ON YOUR FARM IN 2012?

Total number

of deaths: \_\_\_\_\_

	Summer	Autumn	Winter	Spring	Total
Crias <1 week					
Crias					
Last year's crias					
Adults					
Geriatrics					

COMMON CAUSES OF DEATHS

\_\_\_\_\_

METHOD(S) OF DIAGNOSIS?

\_\_\_\_\_

## REPRODUCTIVE PRACTICES

WHAT ARE YOUR MATING PRACTICES?      Hand mate / Pasture or paddock mate      If  
pasture mate skip to last 2 questions on page?

DO YOU BREED FEMALES AROUND THE SAME TIME ON YOUR FARM?      Y / N  
EXPLAIN \_\_\_\_\_

HOW DO YOU DIAGNOSE PREGNANCY?      Spit off      Ultrasound      Wait & see  
Explain \_\_\_\_\_

DO YOU RECORD REPRODUCTIVE FAILURES ON YOUR FARM?      Y / N      EXPLAIN  
\_\_\_\_\_

HOW MANY TIMES IS A FEMALE BRED BEFORE THERE IS A CONCERN FOR HER  
REPRODUCTIVE STATUS? \_\_\_\_\_

HOW DO YOU DETERMINE IF A MALE HAS REPRODUCTIVE PROBLEMS? \_\_\_\_\_  
\_\_\_\_\_

WHAT IS THE PRACTICE ON YOUR FARM TO DEFINITELY DIAGNOSE REPRODUCTIVE  
PROBLEMS? Vet diagnosis      Repeated failure in reproduction      Other.  
Explain \_\_\_\_\_

WHEN HE/SHE IS PROVEN BARREN, WHAT IS STANDARD PROCEDURE ON YOUR  
FARM? \_\_\_\_\_

WHAT INFORMATION IS USED TO HELP YOU DECIDE WHEN A FEMALE IS HEALTHY  
ENOUGH TO BE BRED? Fixed time after unpacking      Y / N      EXPLAIN \_\_\_\_\_

Body condition score      Y / N      WHAT BCS DO YOU USE? \_\_\_\_\_

Accepting male      Y / N

Weight      Y / N      WHAT WEIGHT? \_\_\_\_\_

Size      Y / N      WHAT SIZE IS ACCEPTABLE? \_\_\_\_\_

Age      Y / N      WHAT AGE DO YOU USE? \_\_\_\_\_

WHAT TREATMENTS ARE ROUTINELY GIVEN WHEN FEMALES ARE PREGNANT?  
Vaccinations      Y / N      EXPLAIN \_\_\_\_\_

Vitamin supplementation (A, D & E)      Y / N      EXPLAIN \_\_\_\_\_

Mineral supplementation      Y / N      EXPLAIN \_\_\_\_\_

Feed changes      Y / N      EXPLAIN \_\_\_\_\_

WHAT EVENTS AROUND BIRTH ROUTINELY OCCUR ON YOUR FARM?

Cria suckles within first 12 hours      Cria suckles within first 24 hours  
Regular paddock checks      Other. Explain \_\_\_\_\_

DO YOU ROUTINELY WEIGHT YOUR CRIAS? Y / N      WHEN? \_\_\_\_\_

COMMENTS ON DEATHS AND REPRODUCTIVE PRACTICES

## TESTING, VACCINATION AND HERD MANAGEMENT

WHAT DISEASE PREVENTION OR SURVEILLANCE PROGRAMMES ARE ROUTINELY DONE ON YOUR FARM?

TB testing      Y / N      HOW OFTEN? WHEN? \_\_\_\_\_

BVD testing      Y / N      HOW OFTEN? WHEN? \_\_\_\_\_

Johne's Disease testing Y / N      HOW OFTEN? WHEN? \_\_\_\_\_

Zinc supplementation      Y / N      HOW OFTEN? WHEN? \_\_\_\_\_

Vaccination for EHV2      Y / N      HOW OFTEN? WHEN? \_\_\_\_\_

Vaccination for clostridial diseases Y / N      HOW OFTEN? WHEN? FOR WHAT? \_\_\_\_\_

DO YOU HAVE QUARANTINE PROCEDURES FOR ALPACAS COMING ONTO YOUR FARM?      Y / N      EXPLAIN \_\_\_\_\_

HAVE YOU SOIL TESTED ON THIS PROPERTY?      Y / N      EXPLAIN \_\_\_\_\_

DO YOU APPLY ANY PASTURE TREATMENTS?      Y / N      HOW OFTEN? \_\_\_\_\_

HOW DO YOU MANAGE RYE GRASS STAGGERS? \_\_\_\_\_

DO YOU CULL FOR REPEATED RYE GRASS STAGGERS?      Y / N      EXPLAIN \_\_\_\_\_

COMMENTS ON TESTING, VACCINATION AND HERD MANAGEMENT SECTION





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