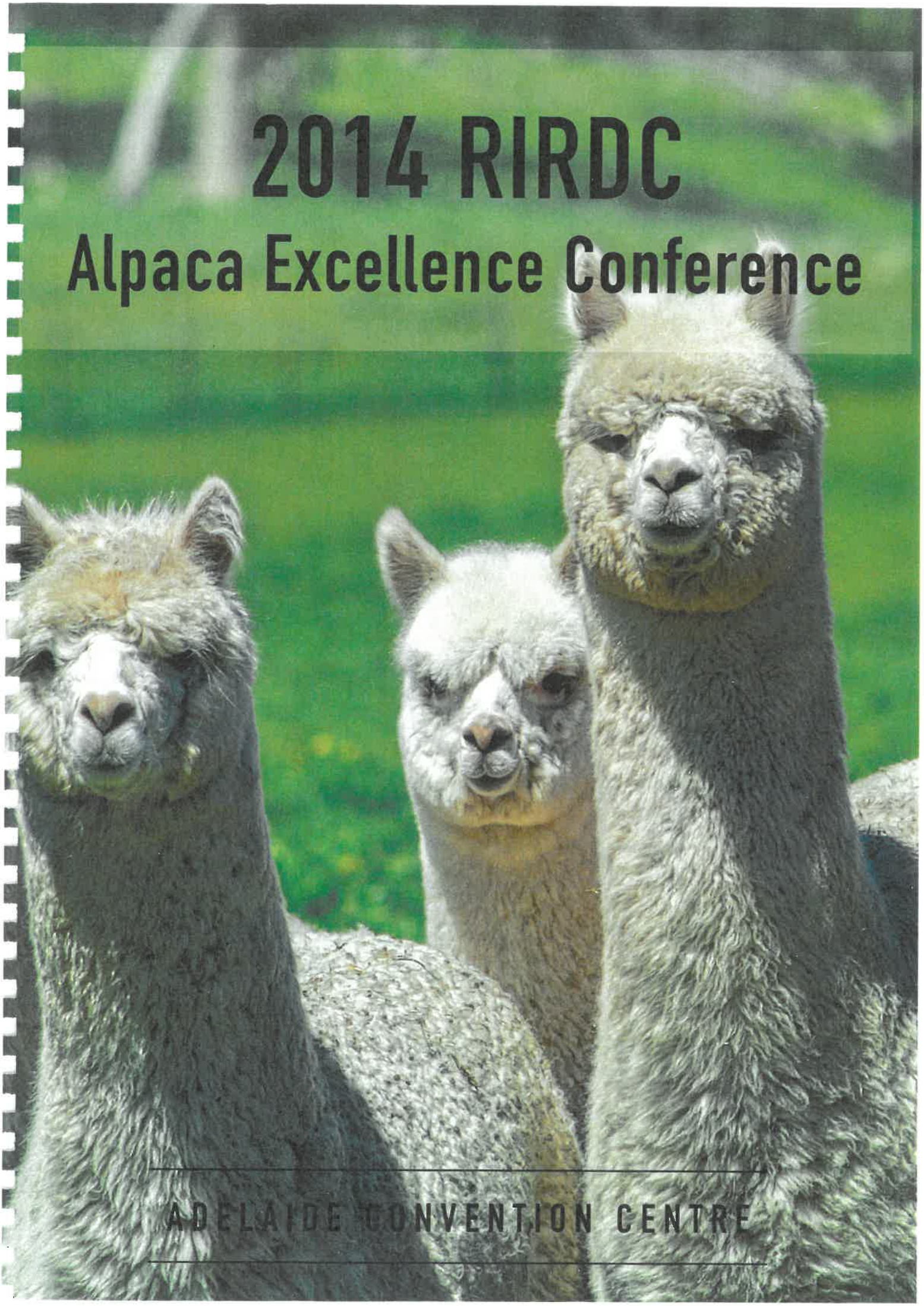
2014 RIRDC
Alpaca Excellence Conference



ADELAIDE CONVENTION CENTRE

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Welcome

Welcome to Adelaide, the South Australian capital and home of the 2014 Australian Alpaca Excellence Conference, and the SA region of the AAA. The site of the conference, the Adelaide Convention Centre, sits on the River Torrens in the heart of the city. The famous Adelaide Oval is a spectacular backdrop and tours are available, should a sporting urge strike while you are here! Perhaps some of the Adelaide cafe scene is more your pace – it's all right here.

Please take the time to enjoy some of the riches this state has to offer during your stay: our world famous regional food and wine of course, and the contrasting landscape, from coastal wonders to inland majesty, there is so much to savour and explore. Welcome and enjoy!

Greg Scarlett
AAA SA Regional President

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National Office Staff

Thanks to the National Office staff who have assisted with advice and guidance.

Craig Tabener, Kris Brown, Vicky Hawes, Janette Law

Other contributing members

The committee would like to thank Dr Jane Vaughan and Steve McNamara for his contribution.

Trade Stands

Thank you to the various studs and companies who have supported this conference by taking trade stands.

Trade Stand	**Contact person**
Alpacas Chaparral	Mark Short
Bella Textiles	Bella Head
Echo Beach Alpacas	AnneMarie Harwood
EP Cambridge	Alex Verhoef
Fibre Naturally	Gayle Herring
Millduck Alpacas	Prue Walduck
Tumi Alpacas	Rohan Dalgleish
World of Alpacas	Trevor Pavlovich
Yaringa Alpacas	Susan Haese

FRIDAY'S PROGRAM

TIME	ROOM	SESSION	SPEAKER
From 8 am		Conference Registration	
9.00 am - 9.15 am	Hall C	Housekeeping Opening Address	Michelle Malt
9.15 am - 10.00 am	Hall C	Rickets	Dr Chris Cebra
10.00 am - 10.45 am	Hall C	Neonatal Care	Dr Laura Hardefeldt
10.45 am – 11.15 am	Hall G	MORNING TEA BREAK	
11.15 am – 12.00 pm	Hall C	Development of the Alpaca Industry over the past five years	Chris Williams
	R1	Workshop: "Understanding Suri Genetics... or, How to Win at Cards"	Fiona Vanderbeek
	R3	A cross-sectional survey of South Australian alpaca serum biochemistry values and selected diseases (vet only)	Prof. Peter Cockcroft
12.00 pm – 12.45 pm	Hall C	Infant Animal Nutrition	David Reynolds
	R1	Workshop: Cria birthing	Adrienne Clarke
	R2	How do you decide when your hobby has become a business?	Greg Rundle
	R3	Glucose metabolism in alpacas (vet only)	Dr Chris Cebra
12.45 pm – 1.30 pm	Hall G	LUNCH BREAK	
1.30 pm – 2.15 pm	Hall C	Coccidiosis	Dr Chris Cebra
2.15 pm – 3.00 pm	Hall C	Parasites in Australian Alpacas	Dr Ian Carmichael
	R1	Workshop: Cria birthing	Adrienne Clarke
3.00 pm – 3.30 pm	Hall G	AFTERNOON TEA BREAK	
3.30 pm – 4.15 pm	Hall C	Alpaca Dentistry	Allison Quagliani
	R1	Workshop: Cria birthing	Adrienne Clarke
	R2	Suri Fabric project	Fiona Vanderbeek
	R3	Q-Alpaca	Dr Alison Lee
4.15 pm – 5.00 pm	Hall C	Through the Farm Gate - A Life on the Land	Angela Goode
5.00 pm – 6.00 pm	Hall C	Forum 1: Veterinary - Jane Vaughan to moderate Chris Cebra, Laura Hardefeldt, Ian Carmichael, Peter Cockcroft, Alison Lee	
6.00 pm	Hall G	***Cocktail party in trade stand area - including book signing by Angela Goode***	

SPEAKER NOTES CONTAINED IN THIS BOOK ARE IN ALPHABETICAL ORDER OF SPEAKER SURNAME.

SATURDAY'S PROGRAM

TIME	*ROOM*	*SESSION*	*SPEAKER*
9.00 am – 10.00 am	Hall C	Alpaca Nutrition	Dr Jane Vaughan
	R1	Workshop: Cria birthing	Adrienne Clarke
10.00 am – 10.45 am	Hall C	Exploding the myths about gastric ulcers: causes, diagnosis, treatment, prevention	Dr Chris Cebra
10.45 am – 11.15 am	Hall G	MORNING TEA BREAK	
11.15 am – 12.00 pm	Hall C	Delivering the Fibre of the Gods to Global Consumer	Kerryn Caulfield & Luis Chaves Bellido
	R1	Workshop: Cria birthing	Adrienne Clarke
	R2	Pasture lecture part 1	Bryan McLeod
	R3	AI in camelids	Dr Simon De Graaf
12.00 pm – 12.45 pm	Hall C	Weight loss & Ill thrift in young Camelids	Dr Laura Hardefeldt
	R1	Workshop: Fodder Selection	Dr Jane Vaughan
	R2	Opportunities for Alpaca based on the Australian Wool Industry	David Michell
12.45 pm – 1.30 pm	Hall G	LUNCH BREAK	
1.30 pm – 2.15 pm	Hall C	Alpaca reproduction 101	Dr Jane Vaughan / Chris Williams
	R1	Workshop: "Understanding Suri Genetics... or, How to Win at Cards"	Fiona Vanderbeek
	R2	Pasture lecture part 2	Bryan McLeod
	R3	Working with suri fibre	Robyn Betts
2.15 pm – 3.00 pm	Hall C	Other gastrointestinal calamities	Dr Chris Cebra
3.00 pm – 3.30 pm	Hall G	AFTERNOON TEA BREAK	
3.30 pm – 4.15 pm	Hall C	Shed sorting and Classing of Huacaya Fleece	Cameron Holt
	R1	Workshop: Cria birthing	Adrienne Clarke
	R2	Alpaca Pigmentation Genetics: Why is my alpaca that colour?	Dr Kylie Munyard
4.15 pm – 5.15 pm	Hall C	Forum 2: Alpaca Fibre - Robyn Betts to moderate Cameron Holt, David Michell, Kerryn Caulfield, Luis Chaves Bellido	
5.15 pm – 5.20 pm	Hall C	"Over the Rainbow" photographic presentation – Anne Rogers Photography – New Zealand.	
5.20 pm – 6.00 pm	Hall G	Book Signing by Cameron Holt - trade stand area	
6.00 pm	Hall B	***Dinner***	

SUNDAY'S PROGRAM

TIME	*ROOM*	*SESSION*	*SPEAKER*
9.00 am – 9.45 am	Hall C	Marketing	Kris Lloyd
9.45 am – 10.45 am	Hall C	Skin conditions	Dr Chris Cebra
10.45 am – 11.15 am	Hall G	MORNING TEA BREAK	
11.15 am – 12.00 pm	Hall C	Alpaca reproduction lesson 2 - Problem breeders	Dr Jane Vaughan
	R1	Workshop: Improving Fleece Scores - A Judge's Perspective	Chris Williams
	R2	Alpaca Meat	Ian Frith & Melanie Smith
12.00 pm – 12.45 pm	Hall C	The importance of science and statistics in the Alpaca Industry	Dr Stephen Mulholland
	R1	Workshop: Improving Fleece Scores - A Judge's Perspective	Chris Williams
	R2	Camelid immunoglobulins	Dr Andrew Padula
12.45 pm – 1.30 pm	Hall G	LUNCH BREAK	
1.30 pm – 2.15 pm	Hall C	Practical tips to keep your alpacas healthy	Dr Chris Cebra
2.15 pm – 3.00 pm	Hall C	Generating a fair assessment of alpaca fleece density	Dr Stephen Mulholland
	R1	Workshop: Using Coloured Fibre	Elizabeth Paul
	R2	Research Presentation by Adelaide University Vet School	Thomas Westermann & Ella Carapetis
3.00 pm – 3.30 pm	Hall G	AFTERNOON TEA BREAK	
3.30 pm – 4.00 pm	Hall C	Alpacas - "Where are the goalposts?"	Michelle Malt
4.00 pm – 4.30 pm	Hall C	Conference Close	

SPEAKER NOTES CONTAINED IN THIS BOOK ARE IN ALPHABETICAL ORDER OF SPEAKER SURNAME.

Australian Alpaca
ASSOCIATION

Farm Tour Number 1:

MORNING – HAYLILLA ALPACAS

Haylilla alpacas

'love the lifestyle'

59 Brunato Road, Blewitt Springs SA 5171

www.haylilla.com.au

Fifty minutes drive from the Adelaide City centre, Haylilla Alpacas is a picturesque 20 hectare property nestled between Clarendon and the McLaren Vale wine region.

Haylilla is home to a herd of approximately 100 Huacaya in a wide range of colours. Steve and Ruth Goodfellow have been developing the property for their alpacas over the last three years. The main farm tasks are carried out on weekends as they manage a business together on week days.

This visit will give an excellent opportunity to experience the lifestyle of boutique alpaca breeders, and learn their story from early mistakes to recent achievements.

A walking tour of this easy to manage farm layout can demonstrate routine tasks including toe trimming, alpaca matings and halter training. It will also allow the opportunity to examine mothers and their cria to gauge the genetic contribution of their co-owned stud sires; Leaenna Armani (solid black), Jolimont Tiberio (grey) and Ambersun Futuris (solid dark fawn). These males have had significant show ring success in recent years.

Your visit will include morning tea refreshments and tastings of local wine, while relaxing in an idyllic setting with sweeping views of the Haylilla property and distant vineyards.

Farm Tour Number 1:

AFTERNOON – AMBERSUN ALPACAS

Ambersun Alpacas
We're doing just fine

Ambersun Alpacas is located at Mount Compass, just one hour's drive south of Adelaide. The farm is on the Fleurieu Peninsula, conveniently located just 20 minutes from the quaint seaside township of Victor Harbor and close to the renowned McLaren Vale wine district, which produces some of Australia's finest.

On this picturesque property is a beautiful lake, which is home to many bird and wildlife species, and provides an idyllic background for breeding alpacas.

Chris Williams and Adrienne Clarke own and manage Ambersun Alpacas. They are among the most energetic and actively involved members of the Australian Alpaca Association. They have been breeding alpacas for over 20 years and run a herd exceeding 1,000 alpacas.

They work to a vertically integrated business model, and have embraced the opportunities offered by diversifying their breeding strategies.

The herd is mixed in colour, and Chris and Adrienne have welcomed the challenge of breeding elite quality alpacas across the range of colours. Championship results in the show ring provide testimony to the quality of their whites, fawns, browns and blacks. They have won over 120 Supreme Championships, including the coveted title of National Supreme Champion Huacaya on three occasions. The stud males contributing to these successes will be proudly on display.

Ambersun also has the largest leopard appaloosa herd in Australia and possibly in the world. Adrienne is dedicated to this breeding project and has published several articles on the inheritance of the spotted coat pattern.

Chris and Adrienne are also passionate about breeding alpacas which display advanced fleece qualities. Its best fleeces from the 2013 shearing will be displayed.

During your visit, enjoy a sumptuous lunch, with wines from the local region and tastings provided by Fleurieu Prime Alpaca.

www.ambersunalpacas.com

Farm Tour Number 2:

MORNING – SOFTFOOT ALPACAS

Softfoot ALPACA STUD

www.softfoot.com.au

8 am Depart Adelaide

10 am Morning tea & Refreshments

Softfoot's selection of gourmet Bush foods grown on farm include lemon myrtle cheesecake, aniseed myrtle Turkish delight, warrigal spinach dip, wattle seed bread, yabbies, and other delicacies.

10.30 am – 1 pm

Tour of Purpose Built Alpaca Facilities – Our farm has been developed over more than a decade to allow efficient and productive management of alpacas in a commercially relevant format.

Shearing Table demo – Softfoot's unique shearing table facilitates high value fibre production, evidenced by our numerous supreme fleece awards at major shows including 2012 National, and Jodie Christian Memorial Trophy for most successful South Australian fleece exhibitor for 5 consecutive years.

Breeding Program presentation – Find out how our "layering" breeding strategy matches fibre traits and produces elite fibre animals using advanced breeding techniques including large scale embryo transfer.

Husbandry presentation – Learn how we successfully use cell grazing on our 500 acres of high rainfall pasture to integrate parasite control and perennial pasture management.

Optional Conservation tour – Our picturesque property sustainably incorporates numerous wetlands, endangered aquatic species, a 7ha feral proof marsupial enclosure, and a state of the art endangered carnivorous marsupial facility with many other projects in early stages. A rare chance to see bettongs, wallabies, potoroos and pademelons amongst others.

12.40 pm – 1.30 pm Lunch

Fully catered Aussie style barbeque lunch in Softfoot's dedicated guest facility, Softfoot Hall

1.30 pm – Depart for Yaringa Alpacas

Farm Tour Number 2:

AFTERNOON – YARINGA ALPACAS

YARINGA ALPACAS

4A Adelaide Place, Currency Creek

Yaringa Alpacas is proud to be part of the 2014 AAA National Conference Farm Tour.

The goal at Yaringa Alpacas is to breed elite huacayas regardless of colour. Although our early emphasis was on the darker colours aiming towards black, we are now excelling with our whites and light fawns also. Yaringa Alpacas is moving from strength to strength.

Although many would consider Yaringa to be a large stud, Yaringa is managed along the lines of a smaller enterprise with each animal treated as an individual.

2.00 pm – Arrive at Yaringa Alpacas

Join the farm walk and have a chance to get "hands on" with our stud males and our donor females. This is a chance to look at our best animals and what we are achieving with them. The Yaringa herd is built on sound blood lines with generations of quality breeding behind them.

During the afternoon tea break, take the opportunity to have a look at our successful show fleeces from 2013, and our yarn project.

After the break visitors will have the opportunity to ask questions about our farm management and to discuss what has made Yaringa Alpacas the success it is today.

5 pm – Depart for Adelaide
6.30 pm – Arrive in Adelaide

www.yaringaalpacas.com.au

2013 Victorian Colourbration Show – three supreme huacayas – three supreme fleeces

Supreme White
Yaringa Hope ET

Supreme Black
Classic Darkness Before Dawn

Supreme Roan – Classic
Yaringa Lord Montgomery
ET

Robyn Betts

Professional Wool Classer

Jaegar Farming and Fibre is an integrated suri alpaca breeding and fibre production business. I have been breeding alpacas for ten years and have been commercially processing suri alpaca fibre for the past five years in Australia.

I am a professional wool classer and use these skills and my experience with suri fleece to facilitate regional alpaca fibre classing and shearing days and to do the odd bit of consultancy.

I have a belief in the qualities of suri fleece in commercial fleece production - and have been working with commercial processors to develop and improve the processing practices - It has been a real achievement to be able to continue to have my brand name products of SuriSilk, MeriSuri and Pacasnuggle developed and produced in Australia from Australian alpaca fibre.

PAPER NOT AVAILABLE

Ella Carapetis

Veterinary Student – Adelaide University Vet School

Ella Carapetis graduated with first grade honours in Bachelor of Health Science (Hon) for her thesis entitled 'Lethal consequences of ingested foreign material in seabirds', which has been accepted for publication. This inspired Ella to develop a passion in Veterinary Medicine and undertake further study. Ella is currently a 5th year University of Adelaide, Doctorate of Veterinary Medicine student.

As part of the 4th year curriculum, students are required to undertake a clinical research project for which Ella chose the topic 'Analysis of geographical and herd factors influencing dermatologic lesions in a subpopulation of South Australian alpacas'.

The aim of this cross sectional study was to give insight into the geographic and herd factors influencing skin lesions in South Australian alpacas for the first time in Australian literature. It was also intended to shed some light on factors that influence the prevalence of skin lesions.

Aims

Currently there is no literature available on geographical and herd factors influencing skin lesions in Australian alpacas. The aim of this study was to gather data on herd signalment and by means of a survey, herd and property demographics, to determine factors influencing the prevalence of skin lesions.

Materials and methods

A cross sectional pilot study was undertaken involving alpacas from 14 randomly selected herds in the Fleurieu Peninsula, Adelaide Hills and Mid-north of South Australia. Data collection comprised of an individual alpaca record sheet, skin scoring chart and survey to determine the geographical and herd factors influencing dermatological conditions.

Results

A total of 323 alpacas were included in this study. Of these, 205 (63.47%) reported skin lesions with the majority occurring in the Fleurieu Peninsula. Skin lesions were more likely to occur in those alpacas that were 3-4 year olds, white coloured, hembra and had a body condition score of 3.5. All respondents kept other animals on the property, however less uniformity was seen for herd purpose and preventative health measures. Statistical significance was found for age and gender.

Conclusions

The results of this study suggest that there is an association between the age, gender, fleece colour and the prevalence of skin lesions in alpacas. It also appears that differences in herd size and purpose reflect the husbandry and type of preventive health. This may influence the likeliness of skin lesions.

Dr Ian Carmichael

BVSc, DVSc

Dr Ian Carmichael graduated in Veterinary Science from Melbourne University in 1967 and was awarded a DVSc from the University of Pretoria, South Africa in 1990. He worked in Africa and Asia for two decades as a specialist veterinary parasitologist in government and corporate educational, research and developmental programs. He has been the South Australian Government Veterinary Parasitologist since 1991 and a Principal Research Scientist with the SA Research and Development Institute for 16 years.

In 1991 he confirmed the presence of *Psoroptes* mange mites in imported alpacas which had been in extended quarantine for 11 months in Chile, Cocos Island and Australia and subsequently designed a standard diagnostic protocol and a treatment schedule which enabled continuation of alpaca imports to Australia.

He was also the first to confirm the presence of the camelid biting or chewing louse, *Bovicola breviceps* in Australia in 1996. From 1994-1997 he conducted a detailed study of internal parasites (worms and coccidia) in alpacas on five farms in South eastern Australia. Dr Carmichael is currently an Associate Professor in the School of Environmental and Animal Sciences, University of New England, Armidale NSW. He has over 50 refereed publications in journals, books and professional proceedings and has provided numerous consultancy reports and project reviews to the Australian government, industry bodies and international agencies, including a comprehensive report on the 1994-1997 alpaca research.

Internal Parasitism of Australian Alpacas

Introduction

A commonality of cattle, sheep, goats, horses and alpacas is that they are not native to Australia. However, unlike the other introduced species alpacas brought very few parasites with them from their countries of origin. From the outset a system of rigorous quarantine and treatment was applied, not simply to control their parasites, but to eradicate them before arrival on the mainland. This attention to the health of imported alpacas has been highly successful in the case of gastro-intestinal worms and blood sucking external parasites and *Sarcoptes* mange mites, probably successful for *Psoroptes* mange mites and largely unsuccessful for coccidia (*Eimeria* spp) and the chewing louse of alpacas, *Bovicola brevis*.

The parasites of alpacas in Australia, therefore, represent an assemblage of gastro-intestinal worms originally brought into Australia primarily from Europe and South Africa in cattle and sheep, plus a few lamoid-specific quarantine survivors from South America. Certain dangerous South American parasites, for example *Llamanema chavezi*, a small intestinal roundworm and *Microthoracius* spp. (camelid sucking lice) appear to have succumbed to quarantine treatments and do not present a threat to Australian alpacas. Others, for example, Barbers' Pole worm (*Haemonchus* spp), Thin-necked intestinal worm (*Nematodirus*

spp) and Liver fluke (*Fasciola hepatica*), which were certainly eradicated in the import process have re-established themselves in alpacas from Australian populations maintained largely by sheep and cattle and are serious threats to the industry.

Gastro-intestinal worms of sheep and cattle, and therefore of alpacas, all have free-living developmental stages on pasture and are strongly influenced by climatic factors, mainly temperature and rainfall, leading to distinct regional and seasonal distribution patterns of worm species. It follows that each climatic region in Australia has its own dominant worm population creating regular seasonal problems which may vary greatly from year to year according to weather patterns. By contrast, coccidia are less susceptible to environmental variations, and are widespread, albeit with seasonal fluctuations in numbers.

After what seemed like good progress in worm control in early years, there is now some apprehension about the impact of parasites on the Australian alpaca industry and how to deal with the issue. Among other problems there are difficulties in interpreting laboratory results, ongoing deficiencies in management and treatment practices needed for control of worm infections, and lapses in essential quarantine. Under these circumstances confusion or expediency often drive repeated unnecessary treatments for internal parasites. In many cases the treatments are wasted because the parasites are already in an advanced stage of resistance to the drugs that are used because of similar excessive exposure in sheep, goats or cattle.

This paper describes the most important or potentially important internal parasites of alpacas in Australia, presents a profile of the dynamics of worm infections in southern Australia, and examines the factors that should be considered in deciding management and treatment interventions. The section on coccidia is limited to field findings in southern Australia, including management factors which influence the disease locally, but does not include treatment recommendations. The greater part of this information came from a significant 3-year research project funded by Rural Industries Research and Development Corporation and laboratory records from various institutions, including Primary Industries, South Australia, but there have also been valuable personal communications from two Victorian colleagues, Dr Jane Vaughan and the late Dr Paul Presidente.

The important internal parasites

Although alpacas are known to be hosts for many internal parasite species in Australia (Carmichael, 1999) there are only 5 main parasites or groups of parasites that we know have a definite impact on their general health and production or that have been confirmed to cause mortalities. These are the Scourworm complex, *Nematodirus* (Thin-necked intestinal worm), *Haemonchus* (Barbers' Pole worm), *Fasciola hepatica* (Liver fluke) and *Eimeria* species (Coccidia). More often than not one or two of these groups will be associated with a combined problem.

The Scourworm Complex

Parasites in this group include the Blackscour worms *Trichostrongylus axei* (stomach), *T. vitrinus, T.colubriformis* and *T.rugatus* (small intestine), the Brown Stomachworms ,*Teladorsagia circumcincta* and *Ostertagia ostertagi* (stomach) and *Cooperia* species (small intestine). Some species had cattle as their original primary host, and some sheep. They are widespread in eastern and southern Australia and the South-west and dominant in winter rainfall areas. Single species infections are uncommon and the adverse effects are usually due to the additive influence of several different worm species. The final outcome is modified by "host factors" such as age, nutritional status, physiological stress, acquired immunity etc., and "parasite

factors" such as the absolute numbers of worms of each species and their inherent disease producing capacity.

In on-farm research carried out by Carmichael (1999) in South Australia and Victoria and Presidente (2003) in Victoria no cases of clinical disease were found. However, Presidente (cited by Carmichael, 1999) had earlier records of field deaths of 2 alpacas. Both of these animals had been grazing paddocks either with or after ruminant livestock. Presidente (2003) also reports "mortalities in alpacas attributed to ....mixed infections with *O.ostertagi, T. axei* and *Nematodirus spathiger* in more southerly locations", presumably Victoria. The first two incriminated worm species suggest the likelihood of cattle in transfer of the parasites. There are also 2 documented cases where worm infections have caused or contributed to alpaca deaths in the Berrima-Mittagong area of NSW (Janie Hicks, personal communication, 1998). The 2 animals had a history of voluminous green diarrhoea terminating in death. In both of them the main post mortem finding was severe gastritis (C3 stomach). Large numbers of worms were seen in histological sections of the stomach; there was no damage in the small intestine. The findings are highly suggestive of infection with *Trichostrongylus axei*. Another report from southern Queensland implicates *Cooperia punctata* in deaths of young alpacas (Dr H. Straker, personal communication, 2001). No doubt the reports of clinical disease and deaths due to scourworms have accumulated since these early records.

In terms of alpaca health and management the Scourworms have been the most studied parasite complex in Australia (Carmichael, 1998, 1999, 1999a; Carmichael *et al.*, 1996, 1999; Presidente, 2003, 2007). Carmichael's (1999) research involved the collection of 1192 faecal samples between November 1994 - January 1997 from a total of 382 alpacas from sites in South Australia(2) and Victoria(3). A cross sectional sample from at least 20 alpacas representative of the whole herd was attempted on each visit (9-11 visits per farm) and, where possible, the same animals sampled each time. Worm eggs in faeces were counted at the laboratory at a high level of sensitivity (2 epg) and eggs were differentiated into various types based on size and morphology. Data from animals which had been treated for worms within 6 weeks before collection of faeces were discarded. Age classes identified were crias (<6 months old), weaners (6-12 months old), tuis (1-2 years old) and adults (>2 years). The data were derived from clinically normal healthy alpacas. Although there were variations between properties in some factors the conclusions accurately represent the general host/parasite interactions in a commercial winter rainfall alpaca venture with reasonable management standards. They could therefore be used as reference base for farms where exposure may be extremely high (e.g., close integration with other domestic livestock, overcrowding etc.) or where the susceptibility of groups or individuals to internal parasites may be increased due to stresses such as concurrent disease and inadequate nutrition and shelter, or immunological deficiencies. Presidente (2003) reported a small faecal egg count survey done in 1999 including 5 properties in South-western Victoria, in which 150 samples were tested over 1 year at a sensitivity of 1:15. The key findings of these two field surveys are again summarised because they provide a baseline resource for industry and are unlikely to be repeated.

Scourworm egg counts, their range in age classes, and natural immunity

Worm egg counts (WEC) are measured in eggs per gram of faeces (epg). All members of the Scourworm complex described above have very similar eggs, indistinguishable one from the other. The eggs of Barbers' Pole worm (*Haemonchus*) which is not a scourworm but a blood-sucking worm, are also similar but slightly smaller. Reliable differentiation of *Haemonchus* usually requires identification of hatched larvae after artificial laboratory incubation of faeces. However, *Haemonchus* was not found in either Carmichael's or

Presidente's surveys, so the reported egg counts refer in broad terms only to the important Scourworms. Other worm types (including *Nematodirus* which is technically a Scourworm) have characteristic eggs which are differentiated during counting.

Table 1. Distribution of Scourworm faecal worm egg counts(WEC) in alpacas of different age classes (Carmichael, 1999)

	Range of faecal worm egg counts							
Egg count - epg	0	1-10	11-20	21-50	51-100	101-200	201-400	>400
Adult (n = 586)	336	194	26	28	1[a]	1[a]	-	0
Tui (n = 151)	73	46	10	16	3	3	-	-
Weaner (n = 153)	60	43	17	18	10	2	1	2
Cria (n = 140)	69	36	7	9	7	6	3	3
Percentage	**52.2%**	**31.0%**	**5.8%**	**6.9%**	**2.0%**	**1.2%**	**0.4%**	**0.5%**

[a] These counts were at consecutive examinations of the same animal

The overall mean WECs for the different age classes were 3.3 epg (adults), 9.8 epg (tuis), 27.4 epg (weaners) and 33.8 epg (crias). By reference to values conventionally applied to sheep or goats the counts were low in all age classes on all properties, despite the fact that over approximately 2 years few animals were drenched for worms. It is important to emphasise that WEC would be expected to be much higher in the presence of Barbers' Pole worm (see later) and that unusually elevated WEC on any farm should raise suspicion of its presence.

Overall, 52.2% of samples had zero counts. Eggs were found in 42.7%, 51.7%, 60.8% and 50.7% of adults, tuis, weaners and crias respectively. The difference in the infection rate between crias and weaners may be due to the likelihood that many crias, especially those tested in summer, had not been exposed to infection before faeces were collected. Presidente (2003, 2007) incorrectly quotes the above results for the various age classes as 10%, 21%, 33% and 25% respectively.

Only 11.9% of egg counts from animals older than 1 year (adults and tuis) exceeded 10 epg, compared with 29.0% for animals less than 1 year old (weaners and crias). Only 2.1% of counts were greater than 100 epg. All counts greater than 200 epg were from crias or weaners. The highest individual WEC recorded for each of age class was 104 epg (adults), 152 epg (tuis), 1300 epg (weaners) and 1114 epg (crias). The highest counts were clearly in crias and weaners and in these age classes they were mostly found from June to August. Although there was no marked seasonality in the peak counts in animals older than 1 year, more than half of them occurred in late spring or summer suggesting that burdens may accumulate slowly over spring or that stress factors may influence counts in older animals at that time of the year. Only 4 animals with the highest egg counts were drenched. All others with elevated counts in which repeated observations were made returned to low or zero egg counts within 1-3 months.

In Presidente's survey more than 80% of animals had zero counts, compared with 52% in Carmichael's survey. The differences between the 2 surveys are most logically explained by the greater level of sensitivity of egg detection in Carmichael's work, but they may be associated with seasonal influences or a greater level of drenching in the flocks surveyed by Presidente.

The two authors examined the numbers of worm eggs in each age class from different perspectives. Carmichael looked at the percentage positive animals overall and in various ranges of WEC (see above), whereas Presidente calculated mean WEC from only positive results in each class. Re-examination of Carmichael's data with Presidente's method gives mean WEC values for positive animals of 5.8 epg (adults), 20.3 epg (tuis), 69.9 epg (weaners) and 68.6 (crias) which are similar to but again generally higher, than values given by Presidente.

In cases where it was possible to repeatedly sample the same animals over several months Carmichael (1999) found that the majority developed resistance during their second year, between 18-24 months of age. The clear trend of decreasing faecal egg count with age in both surveys confirms the development of resistance with increasing age, but this must not be accepted complacently as an indication that animals are "safe". Reduced WEC may simply mean that worms might continue to accumulate in the alpaca over time, either as immature stages which are not producing eggs, or as adults in which egg production is depressed, both of which are dependent on a functional immune system. Currently we do not know whether either of these dynamics happen in alpacas but it is highly likely that they do, given that both are important in sheep and cattle. The immune system can fail under nutritional and management stresses, including unusually heavy challenge from worms, leading to an immediate problem arising from worms currently present in an apparently healthy animal, exacerbated by the new worm challenge. In the long term, low worm levels will persist only in otherwise healthy alpacas.

Key messages

- *Clinical worm disease in alpacas is uncommon, but when present is serious*
- *Worm egg counts are usually low, rarely exceeding 50 epg*
- *Age immunity against worms generally develops in the second year*
- *Immunity can collapse under stresses from feeding, injuries, overriding worm challenge, illness, overcrowding, reproduction, inclement weather etc.*

Nematodirus – the Thin-necked Intestinal Worm

Nematodirus differs from the other Scourworm species in having characteristic eggs which are much more robust in the environment and require a period of drying before they can develop into an infective stage. They can therefore survive for extended periods in the environment or on fodder. If allowed to accumulate over summer on dry southern Australian pastures they can hatch *en masse* with the first autumn rains and pose a serious threat to crias born in the first half of the year. Older animals are unlikely to be affected because of pre-summer exposure and development of immunity. Anecdotally there is an association of *Nematodirus* problems in alpacas with the presence of cattle, rather than sheep.

In Carmichael's (1999) survey the overall mean *Nematodirus* WECs for the different age classes were 0.8 epg (adults), 3.2 epg (tuis), 10.8 epg (weaners) and 11.4 epg (crias). Again, the development of immunity with increase in age was confirmed. Infection intensity was also low; although 51.5% of alpacas <1-year old had positive counts, only 1.5% exceeded 100 epg. Higher levels of *Nematodirus* were found in crias born in autumn and winter. In most animals under 1 year old the infections spontaneously resolve without treatment, but in around 10% of cases they persist and the animals may be at risk. Occasional elevated egg counts (>20 epg) in adult alpacas declined within a few months to zero or very low levels and did not increase again. Elevated *Nematodirus* WECs in adult alpacas are therefore unusual and probably signify an increased general susceptibility to infection in specific individuals, which need to be observed. Apart from this *Nematodirus* species are not important in adult sheep and are unlikely to be so in adult alpacas. The importance of adults carrying *Nematodirus* infections lies mainly in their potential to contaminate the

environment with large volumes of faeces, especially over summer and thereby pose a danger to crias and weaners. However, Presidente (personal communication, 1999) brought to the author's attention his concern about the potential pathogenicity of *Nematodirus* in alpacas, especially in their first year. Apart from a clinical case where he recovered large numbers of *N. spathiger* from a cria which had been grazing a sheep paddock at Mt Moriac, Victoria, he observed that many female *Nematodirus* recovered from alpacas are not fully matured and may not be producing eggs to their full capacity. This suggests that faecal egg counts may sometimes underestimate the true level of infection, and underlines potential consequences if the natural immunity of the animal is compromised. This issue has been addressed in the previous section.

Presidente (2003, 2007) found an overall infection rate approaching 40%, strongly influenced by crias with autumn to spring infection rates of 75% to nearly 100%. In view of the differing sensitivities in detection between the 2 surveys this may indicate a true greater prevalence in his survey than that of Carmichael (1999). However, in the latter survey there was great variability between sites in prevalence and intensity of infection, so there are probably no real differences between the 2 studies. One factor however, that could have positively influenced Presidente's results, but which cannot be verified, is a greater inclusion in his samples of crias born around the time of peak pasture contamination, immediately after the first autumn rains.

Key messages

- *Nematodirus* eggs in the faeces of adult alpacas, sheep or cattle can accumulate on pastures and survive throughout summer because they are resilient to dry conditions. When moisture becomes available they mature and become infective *en masse*
- Crias born in late summer and autumn are most at risk
- Most alpacas older than one year develop a strong immunity to *Nematodirus*
- Elevated *Nematodirus* WECs in adults are unusual and probably signify increased individual susceptibility

Haemonchus spp.(Barbers' Pole worm)

This blood-sucking parasite of the C3 stomach was not found in the surveys of Carmichael (1999) or Presidente (2003). However, attention was drawn to its potential significance.15 years ago by the former author

"Until such time as more information is available concerning the interaction of the alpaca with Haemonchus, Australian alpaca owners should be mindful of the dangers it could present to their animals. This applies not only in areas where it is a perennial serious threat to the wool sheep industry and farmers are constantly reminded of its presence (eg. northern and coastal New South Wales), but also in southern Australia where it has a wide but discontinuous distribution and sudden sporadic outbreaks can cause catastrophic losses in sheep flocks on certain farms." – (Carmichael, 1999) – This has come to pass with a vengeance, because *H.placei* is also involved, which extends the danger area well into Queensland.

The earliest experience of the author with this parasite was a detailed personal communication (2001) by Dr H Straker of Cooroy, Queensland, detailing significant disease and deaths in alpacas infected with *Haemonchus placei*. Since then, especially in suitable years, it has caused serious losses in herds throughout the eastern seaboard including a wide swathe of inland Victoria in recent years (Dr. J Vaughan, personal communication, 2014). Problems occur mainly in winter or summer, suggesting transmission periods in autumn and spring when temperatures are either not too cold for worm development or too hot for survival of developmental forms on pasture. I have also heard of persistent yearly disease events in South-west Western Australia, confirmed by Dr Vaughan. There appears to be no doubt that either *H. contortus* (originally from sheep) or

H.placei (from cattle) can be involved, depending on whether cattle or sheep are the local source. Unfortunately this is only an academic consideration because both parasites comfortably maintain themselves as primary disease-causing agents in alpacas isolated from cattle or sheep, and are therefore here to stay. The main clinical sign may be sudden death of individuals but in such cases other animals would likely show ill thrift, weight loss and pale mucous membranes. Dr H Straker supplied records confirming the recovery of 3870 *H.placei* from an alpaca dying of haemonchosis.

I do not propose to deal with the issues of lifecycle and other information relating to *Haemonchus,* including diagnosis and collection of samples for laboratory analysis. All are comprehensively summarised by Dr J Vaughan in an article on www.criagenesis.cc which I strongly recommend you refer to. However, important principles relating to management in relation to general worm control in alpacas with some specifics for control of *Haemonchus*, will be addressed in later sections of this paper.

Key messages

- *Barbers' Pole worm is a serious threat to alpaca production over a wide area of Australia*
- *Individual producers should determine whether this worm is on their property and if so seek advice to design a sustainable control program*

Fasciola hepatica (Liver Fluke)

In the Peruvian Andes *Fasciola hepatica* has a low prevalence (8%) and discontinuous distribution because climatic conditions are generally unsuitable for the snail vector and the development and transmission of the parasite. However at lower altitudes, when pastures previously occupied by sheep and cattle are grazed by alpacas, acute infections with high mortality rates occur. Apparently the small liver size of alpacas, their lack of an immune response to *Fasciola* and their habit of taking food directly from the soil surface makes them particularly vulnerable to this parasite (Guerrero & Leguia,1987; Leguia, 1991).

Although liver fluke was not found in the early Australian surveys a warning, as for *Haemonchus*, was clearly sounded..."*Because of this (the susceptibility of alpacas to liver fluke), great care should be taken if alpacas are introduced into areas known or suspected to be infected with Fasciola. The greatest danger is on farms with flood irrigation or permanent streams. There are many such areas throughout south-eastern Australia, and along the coast of NSW. Liver fluke is not present in Western Australia*" (Carmichael, 1999).

Acute fasciolosis is manifested by rapid debilitation, loss of appetite, prostration and death within 2-4 days. The signs of chronic fasciolosis include anaemia, loss of appetite, progressive emaciation, diarrhoea alternating with constipation, abdominal pain and death within 3-4 months. Again, the biology of this parasite is comprehensively summarised by Dr J Vaughan in an article on www.criagenesis.cc which gives valuable information for those who find themselves in an endemic liver fluke area. By contrast with roundworm parasites there is greater general understanding and awareness of the link between liver fluke and swampy areas, so this parasite is less likely to come as a surprise to alpaca producers in a liver fluke area. Nevertheless, in an expanding industry there are likely to be instances where alpaca producers are vulnerable to losses because they are not fully aware of local conditions. Where producers are aware of the presence or likely presence of liver fluke on their properties it would be folly to rely upon drenching alone as a basis for control, no matter what the argument for this approach, for this would be expensive to maintain and would ultimately fail.

There are few records available, but it appears that alpacas have succumbed to liver fluke in endemic areas. Links et al (1992) report an alpaca death associated with chronic liver fluke infection at Tocumwal, NSW.

Eimeria spp.(Coccidia)

In Carmichael's (1999) survey in Victoria and South Australia 962 faecal samples from 374 alpacas were examined for coccidia. Some individuals were examined several times, others only once or twice. Overall 69.3% of samples were positive. Infection rates in adults, tuis, weaners and crias were 36.0%, 45.9%, 66.9% and 67.0% respectively. Animals less than 1 year old, therefore, shed oocysts in their faeces more frequently than older animals, but in practice probably contribute to the overall level of infection no more than the older animals in that they produce much smaller volumes of faeces and generally comprise only a small proportion of the herd. Some animals were confirmed to be positive only after several examinations, indicating that the true prevalence of coccidia infection in alpacas in Australia is probably higher than reported.

All 4 species of coccidia present in South American alpacas (*Eimeria lamae, E. punoensis, E. alpacae* and *E. macusaniensis*) were found, although *E. macusaniensis* only once in 2 crias from one property. The more common and least dangerous species are *E. punoensis and E. alpacae. E.macusaniensis* has also been found by Presidente (2007) in 2 out of more than 600 tests of alpacas for export and its presence confirmed in southern Queensland in laboratory records provided by Dr H Straker.

On subjective evaluation oocyst levels in crias and weaners were generally higher than those in older animals. Nevertheless, counts which were classed as moderate to high (++ and +++ classifications - several hundred up to thousands of oocysts per gram of faeces), were found in healthy alpacas in all age groups (2.7% of animals). This clearly shows that older, clinically normal animals can heavily contaminate an environment for crias and weaners. The faeces from a weaner 0.55 years old (+++ classification) had 24750 oocysts of *E. lamae* per gram. Coccidia are remarkably resistant in the environment and tend to become infective *en masse* when conditions are suitable. An apparently safe situation can become a nightmare overnight.

Leguia (1991) reports that coccidiosis is mainly a problem of young animals reared in confinement. Outbreaks of subacute and acute disease occur frequently in Peru in animals born late in the breeding season (presumably when contamination has reached high levels), or after weaning (presumably when crias are severely stressed). *E. lamae* in association with *E. macusaniensis* is a highly pathogenic partnership. The intestinal lining is completely stripped and vulnerable to secondary bacterial or viral infection, giving a strong correlation between coccidiosis and bacillary enterotoxaemia, with up to 50% mortalities in newborn animals. Rosadio & Ameghino (1994) reported a Peruvian outbreak caused by *E. macusaniensis* in crias from 25-35 days old with a history of diarrhoea and sudden death. However Lenghaus *et al.* (2004) attributed the death of an adult alpaca in South Australia to severe enteritis as a sequel to damage caused by *E. macusaniensis.*

Management to control coccidiosis in Australian alpacas is based broadly on the principles applied for control of worm parasites described under the section below, "The alpaca herd and its management to control internal parasites."

Key messages

- *All 4 coccidia species of South American alpacas are found in Australia*
- *Approximately 70% of normal health alpacas are positive for coccidia*
- *Healthy alpacas can have very high coccidia oocyst (egg) counts*
- *Clinically normal adult animals provide most of the environmental contamination*
- *Crias and weaners are most at risk*

The alpaca herd and its management to control internal parasites

Age of animal and geographical location

Under field conditions in Peru animals less than 2 years of age are usually more affected, both clinically and subclinically, than older animals. Animals over 6 years of age have also been observed to have similar worm burdens to young animals. This may be because in Chile, unlike in Australia, they are often kept under the poorest conditions as a group and rarely treated (Chavez *et al.*,1967).

In Carmichael's (1999) study in healthy alpaca herds crias and weaners had greater faecal egg counts than tuis and adults for both scourworms and *Nematodirus* , confirming a very clear age resistance to internal parasites. Counts in crias and weaners usually stabilised at low or moderate levels, and in most cases declined without treatment as the animals grew older. Egg counts in approximately 10% of animals <1 year old progressed to elevated levels. Once age resistance has become established it persists if general management and husbandry are adequate. The study suggests that crias and weaners certainly need to be monitored for internal parasites more frequently than other animals in the herd; however they do not necessarily need to be drenched frequently (see later).

Higher levels were generally found in animals born in autumn and winter because this is the time of the year most suitable for the survival and accumulation of the infective stages of worms in the southern Australian environment. This situation should generally apply to scourworms in most of southern Australia and South-western WA, but is likely to be quite different in northern NSW and coastal Queensland. Here infections with Barbers' Pole worm are likely to be dominant over summer and in the case of *Haemonchus placei* in southern coastal Queensland for an extended period from late summer through to July. Different approaches to treatment according to specific regional parasite patterns are addressed later.

Class of animal

Hembras (adult females), although not specifically identified in Carmichael's study, are probably also highly susceptible to worm infections, by virtue of the fact that they have an 11.5 month gestation and are mated again almost immediately after giving birth. This results in subjecting them to three successive stresses, namely birth, lactation and mating.

Herd concentration

In Peru, the practice of herding the animals together for giving birth increases exposure to parasites for both the cria and hembra as does concentration of animals for other routine procedures such as shearing (Leguia, 1991). In Australia, unfortunately, the practice of concentrating animals persists as a general, high-risk management procedure, but for a different set of reasons:

- Unlike sheep and goats alpacas have not yet become anonymous units of commercial livestock and many alpaca owners don't like the animals too far from sight for aesthetic and security reasons.
- Alpacas are friendly and gregarious animals and lend themselves to concentration in small areas close to the household or stockyards.
- Careful attention is essential to supervise individual matings and births.
- In many enterprises they are hand fed and regularly observed, therefore it is convenient to hold them in small enclosures near to the food source.
- Most serious of all, a general trend in the industry is that many farms are greatly overstocked.

Herd concentration presents huge threats, especially to crias and weaners. Once a holding paddock becomes heavily contaminated with internal parasites it can remain so for many months. A small change in climatic

conditions can change a contaminated paddock overnight from safe to extremely dangerous for Scourworm, *Nematodirus* or coccidia infections.

Grazing associations and herd composition

Sharing or rotating grazing with other animal species, particularly calves and lambs should be avoided. Some serious outbreaks of internal parasitism in Australia have already been associated with this practice. If difficulty presents in separating the livestock species on the farm the alternative animal species should all be cleared of worms with an effective broad spectrum drench at least 3-4 days before introduction to the paddocks. Don't use just any old drench because the odds are it will be ineffective!
Avoid grazing crias and weaners with large numbers of older animals because these comprise their major source of infection. If possible, it is preferable that they are raised entirely apart from older age classes until at least 18 months old

Grazing behaviour and feeding

In Australia the systems under which alpacas are farmed are quite different to those in their natural South American habitat. We tend to graze alpacas like set-stocked sheep, yet it is well recorded that they have distinct differences in grazing behaviour from sheep. If set stocked they tend to graze small areas to a very low pasture height and continue grazing the regrowth, leaving large areas of the paddock lank and ungrazed (San Martin, 1991, cited by Hill *et al.*, 1993). Leguia (1991) reports that they graze rather than browse and take food directly from the ground. These habits can lead to abnormal exposure to internal parasites, especially where alpacas are set stocked on small paddocks or rotated regularly in limited areas. Under such conditions the beneficial effects of the toilet habits of alpacas (see below) can not only be negated, but the latrines can provide massive concentrations of infective worm larvae which they may not be able to avoid.

To reduce exposure to internal parasites ensure that supplementary rations and feed are provided in troughs rather than on the ground.

Births

It is very important to plan a birthing area very carefully, so that hembras can deliver in a clean, uncontaminated environment. The most convenient paddock close to the sheds or the farmhouse may be the most dangerous. Do not allow birthing in a paddock or enclosure that has been housing adult alpacas. Not only are adults in the herd the greatest potential source of infection of both worms and coccidia to the cria, but the hembra is also extremely susceptible to parasite infections at this time.

Where possible, regularly change the birthing area and the paddocks where hembras and their crias spend their first few months.

Defecation behaviour (alpaca latrines)

Alpacas defecate and urinate in discrete common latrines and avoid grazing these areas (Guerrero & Alva, 1986). Given sufficient grazing area and the removal of interference through competitive grazing with other herbivores they substantially limit their own exposure to worms and coccidia. As mentioned above, enforced grazing practices which override this natural mechanism of control may have adverse outcomes.

Cleaning of pasture

Remove faeces from pasture latrines at least monthly either manually or by vacuuming, an activity successfully practiced on equine establishments in United Kingdom. Faeces should be composted for at

least 10 weeks in summer or for 20 weeks if collected from autumn to spring. Faeces from pasture latrines should never be distributed on the pasture as fertiliser without first being composted.

Rotational grazing

Rotational grazing of pastures every few weeks as practiced on many alpaca farms is unlikely to have an appreciable impact on parasite transmission, except possibly over summer. It may, under some circumstances, lead to good pasture management but compromised worm control.

Stresses

Shelter should always be provided against cold, wet weather, extreme heat and sun exposure and wind. Adequate nutrition, both in quantity and quality, is important to maintain the strong immunity that alpacas develop to internal parasites. Intercurrent diseases will modify immunity and increase susceptibility to internal parasites – retrospectively, many of the problems reported with internal parasites in alpacas both in Australia and overseas seem to be associated with a bacterial or viral condition as part of a complex aetiology. In short, stress may be a key component in disease due to internal parasites in this host.

Key messages

- *There is a very clear age resistance to internal parasites.*
- *Peak levels of infection differ regionally*
- *Common practices such as concentrating animals in small paddocks and overstocking are risky*
- *Sharing or rotating grazing with calves and lambs is dangerous*
- *Avoid grazing crias and weaners with large numbers of older animals*
- *In overcrowded conditions alpaca latrines can provide massive contamination*
- *Hembras should give birth in a clean, uncontaminated environment*
- *Supplementary feed should be provided in troughs rather than on the ground.*
- *It is advisable to remove faeces from pasture latrines at least monthly and compost it*
- *Stress is probably a key influence governing the effects of internal parasites*

Faecal worm egg counts (WEC)

Background – what do worm egg counts mean

The WEC of an animal is simply a measure in its faeces (eggs per gram of faeces) of the levels of eggs being produced by the adult worms it is carrying in its gut at the time of collection. It does not signify a degree of disease in the animal on any real or imaginary scale or serve as a portent for danger. However, experience and research have taught us what levels are to be expected in healthy animals and we use this information together with an understanding of their age, environment, clinical condition, nutritional status, stress levels, husbandry practices, and drenching history to arrive at a conclusion as to whether they are healthy and likely to remain healthy, or otherwise.

Basically, there is no magic WEC value which can have a line drawn under it as absolute. A perfectly benign WEC in one age class may signify serious disease in another age class of the same host; and what is an acceptable WEC in an age class at a certain location may need to be interpreted differently elsewhere. In addition, different worms differ in their egg producing capacity. For example, Barbers' Pole Worm, is a prolific egg layer and Scourworms are not. But both are serious problems. A life-threatening burden of Scourworms may produce only a moderate WEC whereas a low level of Barbers' Pole Worm a high WEC. Furthermore, the immature stages of all worms produce no eggs but some can cause enormous damage, and egg production capacity of mature worms (eg *Nematodirus*) can be greatly reduced by the immune response of the animal.

In short, WEC is an extremely useful tool which increases in value with experience, but it does not replace commonsense clinical judgement. Interpretation of the results of faecal egg counts of alpacas is even more subjective than for sheep, but the outcomes are, nevertheless, useful for determining drenching interventions and other management initiatives. Indeed it would be beneficial if nearly all drenching decisions were based on individual WECs of selected animals.

Collection of samples and laboratory processing

Samples should preferably be collected from the rectum and held and transported refrigerated (esky not freezing) in labelled individual containers or plastic bags with as little air as possible in them.

A standard technique is used at the laboratory to float eggs to the surface of a saturated salt solution in a chamber of standard volume where they are counted microscopically. Although counts were done to a level of sensitivity of detection of 2 eggs per gram of faeces (epg) in Carmichael's (1999), survey, most commercial laboratories do counts only at a level of 25 epg which means that some positive animals will be missed. This is not a problem because it would not be useful to drench these animals in any case. Where *Haemonchus* is present a sensitivity of 25 epg is acceptable, but in winter rainfall areas it could be useful to negotiate a more intensive microscopic search to give a detection sensitivity of 12 or 15 epg.

Eggs are differentiated into various types based on size and morphology, as follows:

- Scourworm (Trich/Ost) eggs (see earlier) - this also includes Barbers Pole worm in areas where it is expected. If there is a surprising high count in winter rainfall areas where generally Barbers' Pole Worm is not expected further specialist follow up is needed.
- *Nematodirus (Nem) (see earlier)*
- *"Chab/Oes"* These are the worms of the large intestine that have not been incriminated in disease or deaths in alpacas. Their eggs can be distinguished from Scourworm eggs. Some laboratories do not separate the counts and lump them together with the Scourworm egg count. This presents problems in southern Australia because they occur there at about the same level as Scourworms, so the count can be distorted upwards. This means that you might make a decision to drench when it is quite unnecessary. In southern Australia I suggest that you request the laboratory to separate them and if they say that they cannot, look elsewhere and find a fully trained technician that can.
- *Trichuris (whipworm), Capillaria and Moniezia* (tapeworm) are inconsequential.
- The presence of coccidia is sometimes reported on a subjective scale, but a count is not routinely done, nor is it necessay. Remember that >60% of healthy alpacas are infected. Don't panic when you see that coccidia have been reported – they are present in every flock. Further investigation including re-sampling of the individual, is justified if very large numbers of coccidia are identified (see Coccidia),

Diagnostic significance of worm egg counts – a parasite profile

- Worm egg counts of alpacas can provide a useful tool for monitoring of the herd status but are inappropriate at the levels of sensitivity routinely used for sheep, except where Barbers' Pole Worm is present. The minimum sensitivity should be 15 eggs per gram of faeces.
- Counts from only one or two animals in a herd rarely provide useful information. Selecting only those that are "doing poorly" or "looking wormy" is an unsatisfactory procedure for determining the parasite profile in a herd because many other unrelated factors may have contributed to the relatively poorer condition of the animals which are sampled. At least ten animals, but preferably 15 (or all of the herd if it is smaller than this) should be sampled if meaningful results are to be obtained. A cross section of the herd, including hembras, should be sampled. Once a simple profile of the flock is thus obtained it may be necessary to sample more widely if individuals exceeding the critical WEC levels (see below) are found.

- Bulk faecal egg counts, which are routinely used for evaluation of the internal parasite status of sheep flocks, provide no useful information in the case of alpacas and are a waste of money.
- Unless the egg types are accurately differentiated by an experienced person, the results may be confusing.

Interpretation of a worm egg count

- First, it is important to be thoroughly familiar with what a WEC is, what it can tell you, the multiple factors affecting it and to fully appreciate the principles outlined earlier under ***Background – what do worm egg counts mean.*** It would be beneficial to read this section again.
- WEC is at best a rough, but useful tool, and there are no hard and fast rules governing interpretation. It is vital that the comments below are not taken as a sliding reference scale separating healthy alpacas from unhealthy ones with a drench gun poised in between the 2 groups. Moreover, they do not replace good clinical judgement.
- *The WEC value of greatest importance on a laboratory return is that of the "Trich/Ost" component (for most southern Australian laboratories) or "Strongyle type" component (for northern laboratories).* . These are the parasites which are far more likely to exert an adverse influence on the health of the animals than any other group. Details of these are given above. For all intents and purposes the other counts except *Nematodirus* are usually of little importance.

Under southern Australian conditions, for animals in reasonable condition with adequate nutrition (and given the broad provisions mentioned earlier), I have estimated the following categories for interpretation of general herd screening of WEC. Initially my estimated values were lower (Carmichael, 1999), but were revised up (Carmichael, 1999a) after review of the survey records for repeatedly sampled animals, which confirmed that WEC of most alpacas declined over time. These values were considered by Dr P Presidente, who agreed with them and included them in his recommendations (Presidente, 2003). The only difference between the two sets of values is the base unit, in his 30 epg, in mine 25 epg.

- In crias and weaners WEC of <100 epg, 100-250 epg, 250-400 epg and >400 epg of *"Trich/Ost"/Strongyle type* eggs are defined as "low", "moderate", "elevated" and "high" respectively. A significant *Nematodirus* WEC in crias and weaners is 100 epg.
- In tuis and adults WEC of <50 epg, 50-150 epg, 150-250 epg and >250 epg of *"Trich/Ost"/Strongyle type* eggs are defined as "low", "moderate", "elevated" and "high" respectively.
  A significant *Nematodirus* WEC in tuis and adults is 50 epg. "Significant" in this context does not imply that the health of the animal is threatened, rather that the level of contamination it is delivering on to the pasture may have an adverse effect on the health of younger animals.
- As described earlier, *Trich/Ost"/Strongyle type* values will be much higher in the presence of Barbers' Pole worm.

Principles of drenching

A logical approach to drenching

- Do not seek salvation in the drench pack. Australia has an appalling history of drench abuse, which accounts for the current failure of most drenches to do what is required of them, kill worms.
- There are 2 commonsense reasons for drenching an alpaca. The first is to remove worms which threaten its own immediate health or wellbeing. The second is to reduce the level of contamination that the animal is passing to the external environment in its faeces, threatening the health of other, usually younger, members of the herd. It is illogical to drench an entire herd of alpacas as a routine for the sake of drenching them – we know that as a general rule only about half will harbour worms.

Furthermore, by selecting for drench resistance this practice can have serious long term adverse effects on the enterprise.

- Before drenching you should be convinced that the activity is absolutely essential for the wellbeing of the animals. To do this you need to know their individual worm egg counts or, in the case of larger operations, at least have an idea of the count of a representative cross section of the herd.
- In our survey work (Carmichael, 1999) working closely with alpaca farmers and veterinarians over 2 years we used a conservative approach. This involved regular monitoring of WEC to avoid a large amount of unnecessary drenching and careful documentation that the health of the animals was not compromised.
- The basic principles of monitoring of WEC are as follows. First, concentrate on the crias and weaners, especially those born in summer and autumn. Monitor their individual WEC from around 6 weeks of age regularly every 2 months in their first year. Thereafter, they can fit in to a twice-yearly monitoring cycle (in July/August and again in December) for older alpacas designed to detect those with "elevated" or "high" (*ie.* >150 epg – see above and below) counts. Alpacas with "elevated" or "high" WEC may have been more exposed than others by chance, may be more susceptible because of genetics or some other health condition, could potentially suffer ill effects or they might simply pose a threat to younger animals by contaminating the environment. Some or all of these factors might operate, but it is not imperative to immediately find the answer. What is important is that the producer now has a mechanism to implement a structured worm control program.

What animals to drench in a monitoring program

- The above process specifically identifies individuals for drenching, thereby eliminating unnecessary drenching of the whole herd. In this way selection for drench resistance is significantly reduced and maintenance of natural immunity in the herd assured.
- Animals identified as part of the regular routine monitoring with the following WEC values should be individually drenched. As mentioned earlier, for tuis and adults this may be for their benefit or to reduce paddock contamination to protect more susceptible younger alpacas.

  Crias and weaners – drenching recommended
  WEC (*"Trich/Ost"/Strongyle type)* 350 epg or greater
  WEC (*Nematodirus*) >100 epg
  WEC ((*"Trich/Ost"/Strongyle type* plus *Nematodirus*) >250 epg

  In addition any animal with a positive WEC and obvious disease condition or injury

  Tuis and adults – drenching recommended
  WEC (*"Trich/Ost"/Strongyle type)* 125 epg or greater
  WEC (*Nematodirus*) >50 epg
  WEC ((*"Trich/Ost"/Strongyle type* plus *Nematodirus*) >75 epg

  In addition any animal with a positive WEC and an obvious disease condition or injury

Other drenching strategies

Drenching based on the double summer drench concept commonly employed for worm control in sheep in winter rainfall areas in southern Australia is not recommended. There are certain rare exceptions. One for example, is the initiation of Barbers' Pole Worm control. In this instance it may be necessary to break the cycle with a herd drench at the beginning of summer to ensure no worms at all survive in the stomach of alpacas and are carried over to re-establish field infection in autumn. At most, animals should be treated once, in December, and a second drench only given to individual animals in February upon confirmation by

faecal examination that it is required. Even if regular monitoring is not practiced, drenches given at any time of the year should be in response to a requirement identified by faecal sampling, and will therefore rarely include animals older than 1 year. The necessity to give repeated drenches at short intervals is strong evidence that parasite management has failed and expert opinion should be sought.

The drenches

- There are no drenches specifically registered for alpacas in Australia, hence comments on this matter are general and not given as personal professional advice.
- There is widespread resistance of worms to most of the chemical groups.
- A full list of all drenches available on the market in Australia and comprehensive information about them and their application can be found on an excellent website www.wormboss.com.au It is important to refer to this.
- It is suggested that the two drenches most likely to be effective [(moxidectin (Cydectin) and monepantel (Zolvix)] are used at double the dose recommended for sheep.

Drench resistance and quarantine of purchased animals

- Australia and New Zealand have among the highest levels of drench resistance in the world. Although originally limited to the white (benzimidazole) and clear (levamisole) groups of drenches and their combination, strains of sheep, and more recently, cattle parasites resistant to the macrocyclic lactones (ML's) ivermectin and moxidectin are now widespread. The ML's are extremely suitable for administration to alpacas because they are safe, highly effective and easily administered in low volume injectable formulations. They have justifiably become the treatment of choice for internal parasites in alpacas. However, the end is nigh. Ivermectin has largely run its course and abamectin a related compound in an oral formulation plus injectable moxidectin remain. We are aware that in some areas these drenches are also failing against certain important parasites. There are now many drench products available containing multiple actives to offset drench resistance, but in reality, they too have a limited lifespan.
- This grim situation is complicated by the fact that there is no information whatsoever whether the drenches in current widespread use by alpaca farmers are in fact effective at all and, if effective, at what level. It is a sobering thought that some farmers may be wasting their time and money drenching their animals. There is absolutely no point in guessing what might be happening or hoping the situation will miraculously improve. The first personal responsibility of every alpaca producer who is serious about their business is to check WEC values before drenching, drench only according to a known WEC for each of several individuals and then have another WEC done on the same individuals 7-10 days later. This will give an answer and perhaps some peace of mind.
- Another serious issue relating to drench resistance is its potential to move great distances around the country in transferred animals. It is courting disaster to bring animals on to your property without ensuring that they have been effectively treated beforehand. A quarantine drench incorporating both Zolvix and Cydectin, each at double the recommended dose for sheep, both given at the same time, is the minimum that should be administered. The addition of a combination white/clear drench as well is suggested. The animals should be treated 2-3 days before despatch and held preferably indoors or on concrete to allow worm eggs present in the gut at the time of treatment to pass.

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Kerryn Caulfield

Kerryn Caulfield, COO Apical International Pty Ltd and Non Executive Director, Australian Alpaca Fleece Ltd.

DELIVERING THE FIBRE OF THE GODS TO GLOBAL CONSUMERS

Kerryn will outline the journey of our Australian alpaca fleece to the consumer; through the complex global production supply chain across oceans, against challenging global forces, against uncertain retail conditions and yet fortified by strategic partnerships.

With over 30 years experience in the fibre, textile, advanced manufacturing and related industries, Kerryn was appointed to the AAFL Board in 2011.

Through her management company, Apical International, Kerryn contracts its services to a number of collectives and associations that represent the interests of Australian manufacturers and recyclers.

Kerryn participates in government and industry-driven committees and forums specifically to design and formulate policy at state and federal levels and manages many transformative industry programs.

Prior to forming her company, Kerryn held senior international trade positions with the Australian Chamber of Manufacturers and large manufacturing firms. Her contribution towards Australian exports was recognized by the Australian Trade Commission and the College of International Business and provided her with a legacy of global contacts.

Co-presenter Luis Chaves Bellido will outline the alpaca breeding and production capabilities in Peru and the purchasing system of the large fibre collectives, including their role in supplying alpaca products to European and Japanese fashion houses. Peru is the largest producer of alpaca fibre in the world today.

PAPER NOT AVAILABLE

Luis Chaves Bellido

Luis Chaves Bellido, Director Inca Group Companies and Non Executive Director, Australian Alpaca Fleece Ltd

Luis Chaves Bellido, General Manager Incalpaca TPX SA and Non Executive Director, Australian Alpaca Fleece Ltd.

Luis has worked for more than 30 years, in a career dedicated to the Alpaca and Wool industries of Peru. His numerous years of work are indicative of his absolute passion for The Fibre of the Gods.

Luis has extensive knowledge and experience in all related areas, inclusive of fibre, textiles, manufacturing, production and retail. Luis was, and is revered by his peers in these fields.

Recruited by Inca Tops S.A.A, Luis worked as general manager for 9 years (1992 – 2011), before moving on to work – once again as general manager, for Incalpaca TPX S.A – one of the largest manufacturers of alpaca products in Peru, in 2010. He currently retains that role.

In addition to this, Luis is also an esteemed and valued member of the Board of Inca Group Companies. He was also President of IPAC (Peruvian Institute of Alpaca & Camelids) [2006 – 2007], and of the Arequipa Chamber of Commerce [2008 – 2010]. Luis has been a keynote speaker in numerous events locally and internationally.

A STRATEGIC APPROACH TO THE ALPACA FIBRE

Textile industry is one of the most ancient industries and certainly one of the most competitive and fast changing industries of present times. Alpaca Fibre industry around the world is not isolated from this reality. If we want to succeed in this highly competitive industry its necessary to design and strategy.

Price is variable that moves the markets, industries and key factor in any strategy; therefore is necessary to review its evolution, the common fields and differences of the Luxury and Noble fibres prices considering changes across time and its relation with the fineness of the fibre.

In transforming the raw Alpaca Fibre into Clothing is necessary to considerer the different implications of using Alpaca, ranging from Markets, Manufacture requirements and weather to Comfort Factor. A panoramic view of all this factors and its possibilities in the highly competitive

Textile / Fashion world will help the Alpaca Industry to find the right paths to approach consumers markets.

It is true that most of the Alpaca fibre of the world is produced in the Southern Highlands of Peru, but in order to have a clearer idea of the who is who in the Alpaca world market its necessary to review and know what is the real influence of the biggest producer and will be known who the biggest players are.

The textile tradition of Peru goes back in history to 1,650 BC; Incalpaca TPX SA in its quest to renew this ancient glory its constantly questioning and perfecting its strategic approach which already has some success stories to share and new challenges to overcome.

Dr Christopher Cebra

VMD, MA, MS, DACVIM

The Glen Pfefferkorn and Morris Wendorf Professor of Camelid Medicine
Oregon State University College of Veterinary Medicine

Dr. Christopher Cebra has worked with llamas and alpacas for over 20 years as a clinical veterinarian and researcher. He has authored or co-authored over 50 peer-reviewed scientific reports on camelids and numerous lay articles, reviews, and book chapters. Most recently he finished his chapters for a new comprehensive textbook on llama and alpaca health care.

As a specialist in internal medicine, Dr. Cebra has concentrated most on the diagnosis and treatment of abnormal conditions, but has also worked in general physiology, parasitology, and overall health care to prevent disease. He is currently a professor of large animal medicine at the Oregon State University College of Veterinary Medicine where he runs an annual course in camelid medicine and teaches about and treats all farm animal species

RICKETS OR HYPOPHOSPHATEMIA SYNDROME IN ALPACAS

The doses of medications described in this report have not been extensively scientifically tested and should be used with caution.

Rickets is a reasonably well-known disease: most alpaca owners have heard of the human form of it, even if they are not as aware of their alpacas' susceptibility. They know it is a bone disease and related to a deficiency, but do not appreciate the variety of disease signs it can cause. Additionally, although this disorder was well publicized in the mid- to late-90s, when reports and research came out of Oregon and New Zealand, and a few years later with a new wave of research, it seems to drift out of view on occasion, then strike unexpectedly.

Rickets in alpacas usually relates to Vitamin D deficiency. Although some of this may be dietary deficiency, especially in hay-fed alpacas, most relates to a seasonal lack of production due to lack of adequate exposure to ultraviolet (sun)light. In winter, especially in areas with cloudy weather and at higher latitudes, or places where the weather is bad enough that alpacas are kept largely indoors, blood vitamin D concentrations drop to as little as one-sixth their summer levels. The decline appears to be especially bad in alpacas with fuller or darker fiber coats. In rare cases, rickets may also result from a phosphorus-deficient diet.

Lack of exposure to ultraviolet light leads to a drop in vitamin D. Vitamin D helps in the uptake of dietary calcium and phosphorus, the re-uptake of urinary calcium, and the normal mineralization of bone. When vitamin D drops and blood calcium drops, parathyroid hormone increases and starts degrading bone. In mature alpacas, this can lead to some thinning of bone, but it is rarely clinically significant. The greater problems occur in developing animals, either during the fetal period or growing alpacas in the first 2 years of life. In the fetus, bone disease may develop immediately. In more mature animals, changes is bone seem to lag a few months after the drops in blood vitamin D.

The fetus is dependent on its mother's circulating vitamin D. Fairly little crosses the placenta, leaving the nursing cria dependent on getting enough through colostrum and milk. If the period of skeletal growth (third trimester) coincides with maternal deficiency (winter), crias may be born affected. More commonly, fall-born crias develop abnormalities during their early skeletal growth, which often coincides with a period of low sunlight. Similar seasonal maldevelopment may occur any year until the alpaca achieves skeletal maturity.

There are a variety of different clinical presentations, ranging from covert to overt. In newborns, the most common sign is abnormal angulation of the carpus or tarsus. This may affect the ability to stand or the cria's gait. The gait may be inconsistently abnormal, because pain may shift from leg to leg. Severely affected crias may be reluctant to move or stand. In older animals or crias with more chronic problems, angulations are less notable. It becomes more common for them to stand abnormally, usually with their weight rocked back onto their hind legs. The carpus remains the most commonly affected joint, and it may appear slightly swollen or painful to palpation. In severe cases, the leg may break from a weakened cortex or ligaments may pull loose from their attachments. There is also growing evidence for vertebral column involvement, resulting in pain, reluctance to stand or move, or possible neurologic deficits. Poor weight gain and a generally unthrifty appearance are other findings. These may relate to pain and reluctance to move to eat, or possible to some other effect of vitamin D deficiency. Some affected alpacas appear to have an immune deficiency, which can also contribute to ill thrift.

Screening for at-risk animals may be accomplished by measuring dietary vitamin D, calcium, and phosphorus, or more directly, by measuring these same parameters in blood. Young, growing animals usually have blood phosphorus concentrations at the high end of or even above adult reference ranges, so finding lower values in younger alpacas is suggestive. Ideally, serum phosphorus is above 7 mg/dl and the Calcium:Phosphorus ratio is greater than 1.5 in growing camelids. Vitamin D concentrations should be above 50 nmol/L. Blood values usually become abnormal weeks to months before clinical disease develops.

Radiographs or more advanced imaging studies may give further evidence. Angular limb deformities, fractures, thin bone cortices, and widening and irregularity of the growth plates are all characteristic. The distal radius and tibia are the best places to find lesion, but any bone may be affected.

If the disorder is caught early, prompt treatment may lead to complete resolution. This may involve vitamin D supplementation, mineral supplementation, supportive care, and possibly surgical or splint management of the legs. Supportive care may involve pain medications, convenient provision of feed, water, and shelter, sometimes slinging and physical therapy. Improvement may be noted within 1 to 2 weeks, but it may take several months for all signs to disappear.

Prevention strategies have been developed to avoid this disorder. These include keeping the blood vitamin D and phosphorus of most alpacas above the values stated above, especially pregnant females or growing crias and juveniles. Managing animals to optimize UV light exposure is recommended, but not always practical. If UV light exposure is inadequate, vitamin D should be fed or injected. The oral requirement in alpacas appears to be around 30 to 40 U/kg/day. Gel supplements, milled pellets, or mineral mixes may be used. Injecting vitamin D is the other choice. The recommended dose is 1000 to 2000 U/kg of vitamin D under the skin. This should be repeated every 2 to 3 months during the low-light season. Giving too much vitamin D may damage the kidneys.

If alpacas do not respond to the standard treatment of vitamin D supplementation, an imbalance of dietary calcium and phosphorus may be the culprit. These may respond to rebalancing these minerals in the diet.

WHAT WE CURRENTLY KNOW ABOUT GLUCOSE CONTROL IN CAMELIDS

Background

Fats, sugars, or proteins all may be broken down to provide energy for cellular function. Fats and sugars are by far the most common sources in domestic animals, with the proportion of fats to sugars being heavily dependent on the type of diet, or in the case of herbivores, the products of microbial fermentation the diet.

When dietary energy is insufficient to meet body needs, body stores of energy are mobilized. Fat contains the greatest energy per unit mass and is malleable, and thus is the preferred storage form of energy for mobile creatures. Sugar stores are smaller and rapidly expended.

Camelids are pseudoruminants. Thus, after maturation and population of their gastrointestinal tract, they use microbial fermentation to digest plant products such as cellulose and hemicellulose. The presence of this fermentation chamber in the forestomach also promotes fermentative digestion of most dietary protein and carbohydrate. The end products are the volatile fatty acids (VFA) acetate, propionate, and butyrate, which appear to be produced in similar proportions to those produced by ruminants on similar diets. These VFA supply most of the energy used by camelids either by direct oxidation in tissues, conversion in the liver to other lipids for use by other body tissues, or providing precursors for hepatic gluconeogenesis (propionate). Unlike ruminants, the gastric wall of camelids does not appear to convert butyrate to beta-hydroxybutyrate.

Carbohydrate Metabolism

Cattle and many other ruminants have adapted to lack of dietary carbohydrate absorption by limiting somatic utilization. They have mild insulin resistance and slower glucose clearance than carnivores or omnivores. Thus glucose, which must be produced by the energy-losing process of gluconeogenesis, is relatively spared for tissues with an obligate glucose requirement, such as the

fetus, mammary gland, brain, and erythrocytes. These tissues take up glucose in an insulin-independent fashion. Other somatic cells are still able to take up glucose, particularly when it is abundant and stimulates insulin production and release. Because of their requirement for gluconeogenesis, most ruminants have blood glucose concentration lower than nonruminant mammals.

Although camelids presumably have a similar requirement for gluconeogenesis, glucose utilization and regulation is physiologically very different in camelids. Camelids have higher resting blood glucose concentrations, clear exogenous glucose slowly, have lower blood concentrations of fasting and stimulated insulin, and have considerably greater insulin resistance than cattle. These appear to be the result of greater glucose economy, that most somatic cells are unable to upregulate glucose uptake and usage much above basal needs, even in times of glucose and insulin excess, as opposed to exuberant gluconeogenesis or greater gastrointestinal absorption of carbohydrate.

Glycogen is the major storage form of glucose. Epinephrine and cortisol both appear to mobilize glycogen stores in camelids, similar to in other species. Epinephrine leads to near-instantaneous increases in blood glucose. Cortisol takes longer (90 to 120 minutes), but leads to longer, higher peaks. Epinephrine, cortisol, and other glucogenic factors are likely to increase blood glucose to a greater degree in camelids than in other species because of the lack of effective countering by insulin.

Glucose Transport

Blood glucose comes primarily from one of three sources: dietary absorption of glucose, glycogen breakdown, and gluconeogenesis. It leaves the blood by two main routes: urinary excretion and transport into cells. Dietary absorption has not been tested in camelids, but we can assume it is minimal on most roughage-based diets, because the diet itself contains little simple carbohydrate, and the gastric microbes likely rapidly ferment what is there. Glycogenolysis likely also occurs under hormonal conditions that are present under similar conditions in other species, though epinephrine challenge suggests that camelids have inferior stores to some other species. That leaves gluconeogenesis, which provides the bulk of circulating glucose in most ruminants.

Urinary excretion occurs when blood glucose concentrations exceed a certain threshold, which has not been established for camelids, but appears to be in the range of 200 mg/dl. Cellular uptake occurs either through the sodium-linked transporter (SGLT) or a family of glucose transporters referred to as GLUTs. The SGLT has not been investigated in camelids. In other species, it is responsible mainly for absorption of glucose across epithelial surfaces from the ingesta or the urine, and thus has the role of introducing glucose into the blood from one of these sources.

The more interesting transporters affecting blood glucose in camelids are the GLUTs. Of this family, there are 4 major representatives that principally transport glucose between the intracellular and extracellular spaces. GLUT-1 is widespread and probably serves to ensure each cell its basal glucose supply. GLUT-2 is very bidirectional, and regulates glucose movement in and out of hepatocytes depending on the relative supply on each side, and regulates glucose uptake and thereby insulin production by the rodent pancreas. GLUT-3 is mainly found on the neuronal plasma membrane, and thus has a role in maintaining glucose supply for the central nervous system. GLUT-4 is found mainly in adipose cells and skeletal muscle, and is responsible for insulin-mediated glucose uptake.

Immunohistochemical examination has revealed what appear to be substantial populations of GLUT-1, GLUT-2, and GLUT-3 in the appropriate places in camelids. GLUT-4 was not found, but whether that was due to absence of the transporter or lack of cross-reactivity with the antibody was not determined. GLUT-4 was recently found in abundance in camels, but the authors suggested some sort of problem with its moving to the membrane.

Adult camelids can be characterized as slow-transporters of glucose out of their blood, even when stimulated by short-acting exogenous insulin. Some of this action relates to a generally sluggish and inadequate pancreatic insulin response to hyperglycemia, but the slow clearance in insulin-stimulated camelids also suggests a sluggish or inadequate GLUT-4 response. The higher efficacy of long-acting insulin or insulin CRI in decreasing plasma glucose suggests the former: that adult camelids have the mechanism for a GLUT-4 response, but that it is somewhat dormant due to chronic understimulation.

Crias are notably different in their insulin response, and their glucose clearance with or without exogenous glucose. This makes sense, as they are on a high carbohydrate diet and have a gastrointestinal tract that presumably allows for greater carbohydrate absorption than an adult. They appear to have similar pancreatic anatomy and GLUT populations, so something appears to make the pancreas more reactive to hyperglycemia and the peripheral tissue more response to insulin.

Disorders of Carbohydrate Metabolism

Stress hyperglycemia is a well-known phenomenon in camelids. Whereas ruminants and horses rarely develop hyperglycemia >300 mg/dl, camelids occasionally have glucose concentrations up to about 500 mg/dl. The frequency and severity of stress hyperglycemia most likely relates to the poor mechanisms for glucose clearance. No adverse signs are associated with this condition, and no treatment is necessary.

Diabetes mellitus has been described empirically, but not reported scientifically in camelids. The usual basis for diagnosis is persistent hyperglycemia with or without confirmation of slow glucose clearance or glycosuria. In rare cases, diabetic complications develop, such as cataracts.

I have examined a small number of camelids with persistent hyperglycemia and found normal insulin concentrations, normal glucose clearance, and normal pancreatic architecture. They also show no evidence of decreased function of tissues that take up glucose in an insulin-independent fashion. Thus, overproduction of glucogenic factors (cortisol, epinephrine, others) appears more likely than pancreatic insufficiency, pancreatic exhaustion, or somatic insulin resistance.

Hyperosmolar disorder is one consequence of persistent and severe hyperglycemia. Glucose is an osmotic agent that does not move readily between body compartments, especially in camelids. Factors which increase exogenous glucose, such as endogenous or exogenous catecholamines or glucocorticoids, pancreatic insufficiency, or exogenous glucose, are poorly countered by insulin. Because of slow cellular glucose uptake, renal clearance becomes the primary method of combating hyperglycemia. It has not been determined scientifically, but our clinical experiences suggest that camelids have a similar threshold to renal glucose excretion as other domestic species, around 180 mg/dl.

Under normal conditions, glucose is a minor component of blood osmolality, 5 to 10 mOsm/l out of a total of around 300. Its contribution can be estimated by the following equation:

Blood osmolality (mOsm) = 2 x (Na + K) + Glucose/20

Even with increases of several hundred, the direct effects of glucose on blood osmolality are minor. However, as glucose climbs to 600 mg/dl or above, fluid shifts from the intracellular to the extracellular space may decrease cellular function. Additionally, glycuresis leads to fluid loss. If this fluid loss exceeds intake, blood sodium concentration increases, and hyperosmolality worsens. Mineralocorticoid release to combat hypovolemia may exacerbate hypernatremia.

The clinical signs of hyperosmolar disorder relate to the stage of the disease. Early signs include frequent urination, fine muscle tremors, and a base-wide stance. Later signs include recumbency, seizures, and coma. The camelids most susceptible to hyperosmolar disorder are those with limited fluid intake and exposed to exogenous glucose and/or glucogenic agents, i.e. neonates.

Hyperadrenocortism or Glucocorticoid administration: Exogenous glucocorticoids have an exaggerated effect on blood glucose in camelids because of camelid's inherent slow glucose clearance. Thus, glycuresis and hyperosmolar syndrome may ensue in dehydrated camelids treated with glucocorticoids. Mineralocorticoid activity of glucocorticoids may worsen hypernatremia by promoting renal reabsorption.

Endogenous glucocorticoids are harder to assess. They have long been blamed for stress hyperglycemia, but because of their delayed onset of efficacy, they are likely only contributing to hyperglycemia in camelids stressed for 2 hours or more. Some abnormality of the pituitary-adrenal axis may be responsible for persistent hyperglycemia and diabetes-like signs in certain camelids, though this has not been investigated sufficiently.

Pancreatitis with damage to islet cells would be another possible cause of hyperglycemia. We have seen both acute and chronic forms. Acute pancreatitis appears to cause colic signs. Its effects on glucose homeostasis have not been described, but it appears to affect peri- and intrapancreatic fat more than the pancreatic parenchymal cells.

Chronic pancreatitis with islet atrophy has also been described and possibly associated with a picorna virus similar to Equine Rhinovirus. Diabetes-like clinicopathologic changes and glucose response were described. However, recent work suggests that these assessments of pancreatic form and function were not that abnormal for camelids.

Hypoglycemia is an uncommon finding in camelids, even sick neonates and older camelids with longstanding anorexia. Poor glucose clearance limits its rate of use, even if camelids lacking other forms of energy. Lactating or pregnant camelids may be at higher risk because of greater insulin-independent glucose uptake. Septic camelids or those with Mycoplasma hemolama infection may also develop hypoglycemia from bacterial usage. Hypoglycemia is best treated by intravenous glucose administration. Because of the infrequency of this disorder and the potential for aggravating hyperglycemia, confirmation of hypoglycemia before treatment is advisable.

Monitoring Disorders of Carbohydrate Metabolism

Measurement of blood glucose is relatively straightforward and informative. Blood insulin may be measured by radioimmunoassay, but the standard curve must be adjusted for the very low insulin concentration found in camelids. Glucose tolerance tests and insulin response curves may provide evidence about glucose clearance and insulin resistance, but again, the slower clearance and lower insulin sensitivity of camelids must be kept in mind.

General assessment of renal function, protein and electrolyte concentrations, and so forth are recommended, especially in camelids at risk for developing hyperosmolar syndrome.

Treatment of Disorders of Carbohydrate Metabolism

As glucose itself appears to be marginally useful to camelids and the chance of deficiency in low, most efforts are directed at avoiding the complications of hyperosmolality. Transient, severe hyperglycemia can be left to resolve on its own, as long as the camelid remains adequately hydrated. If glucose remains high, or the rise in blood sodium concentration is becoming a concern, insulin may be administered to reduce blood glucose. Regular insulin given intravenously has a near-instant effect that lasts about 1 hour. Long-acting preparations given subcutaneously have effects lasting up to 18 hours. Both porcine and human recombinant forms appear to be effective. Glucose should be monitored over the period of insulin efficacy to prevent hypoglycemia (rare), keeping in mind that hyperglycemia in camelids does not usually reflect an ongoing overproduction of glucose, but rather an acute overmobilization or peripheral resistance.

Treatment of hyperosmolar disorder is more complicated. The need to reduce blood glucose becomes more urgent, as well as the need to reduce blood sodium and total osmolality. Hypernatremic hyperosmolar conditions in calves and pigs require slow correction, likely because of the amount of time they take to develop and the production of idiogenic osmoles in the brain. Our understanding of hyperosmolar disorder in camelids is that it develops rapidly, that water is drawn out of the intracellular and intraneuronal space by the sudden spike in blood glucose, and that the brain has not had time to compensate. Thus, affected camelids appear to respond to rapid, intravenous and oral fluids to correct hypernatremia. If there is reason to believe that the condition developed over several days, a more measured treatment effort is recommended.

Hypoglycemia may be treated with exogenous intravenous glucose. Other glucogenic agents are not recommended due to or potential complications.

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COCCIDIOSIS IN NEW WORLD CAMELIDS

A variety of parasites affect the gastrointestinal tract of New World camelids. Some of these are unique to camelids, but many also infest or infect ruminants, other domestic animals, cervids, or other wildlife as well. As a rule, parasitic infections are more associated with ill thrift than more specific and overt signs of GI disease, such as diarrhea or colic, but as such, they are among the most common causes of poor-doing in domestic camelids. Awareness of the importance of protozoal enteritis has been growing steadily. This is reflected both in the number of scientific publications, and the overall recognition that parasite control strategies must extend beyond anthelmintics. Also, once considered diseases of crias, certain protozoal enteritides are now widely recognized as important disorders of all ages of camelids.

New World camelids appear to be susceptible to at least 5 species of *Eimeria*. These appear to be camelid-specific, unlike many worms, *Giardia*, and *Cryptosporidium*. Thus, transmission from ruminants or wildlife is not thought to be important. Camelid coccidia do, however, affect all species of New World camelid, and whether they also affect Old World camels is an open question. Whereas coccidiosis is primarily thought to be a disease of the young in many species, and is well publicized as a cause of illness and death in crias in South America, it is becoming more commonly recognized worldwide as a cause of adult morbidity and mortality as well.

Camelid coccidia can be lumped into categories of small and large. The small coccidia are relatively conventional in appearance and life cycle, and will be familiar to anyone acquainted with coccidial disease in domestic poultry or ruminants. The large coccidia of camelids are relatively unique. The small coccidia include, by increasing size of oocysts, *Eimeria punoensis* (17 to 22μ in length), *E. alpacae* (22 to 26μ), and *E. lamae* (30 to 40μ). Oocysts become more ovoid as they get larger, so that *E. punoensis* is about 16% longer than it is wide without a clearly visible polar cap (by standard light microscopy), whereas *E. lamae* is about 60% longer than it is wide and has an obvious cap. These follow the same general lifecycle as *Cryptosporidium* with a few notable exceptions. *Eimeria* oocysts are not thin-walled and are not capable of autoinfection. They do not sporulate and become infective until they have spent 4 to 12 days (for *E. lamae*) or more outside of the host. Sporulation rate is highly dependent on environmental conditions and currently under investigation. After ingestion, sporulated oocysts usually release 4 sporocysts each containing 2 sporozoites, which penetrate epithelial cells. The host cell nucleus and organelles are marginalized by the parasitophorous vacuole. When this has matured, the cell ruptures and hundreds to thousands of merozooites are released. The level of multiplication for camelids is unknown; for *E. macusaniensis*, roughly 300 oocysts were produced for each infectious oocysts ingested by healthy adults. New generations of merozoites repeat the cycle a number of times, creating more mucosal damage. Eventually, some merozoites develop into sexual forms and oocysts subsequently appear in the feces. The prepatent periods are approximately 10 days for *E. punoensis*, 15-16 days for *E. lamae*, and 16-18 days for E. alpacae.

The small coccidia are best associated with hemorrhagic, watery diarrhea progressing to weakness, lethargy, weight loss or poor weight gain, feed refusal, dehydration, and eventually shock, coma, and death. Colic, respiratory distress, and cerebral signs are uncommon or late findings. The gut, particularly the terminal jejunum and ileum, is occasional hemorrhagic or markedly edematous, and may have areas of mucosal hemorrhage, fibrinonecrotic pseudomembranes, or punctate white lesions, but is more commonly grossly non-remarkable. Histologically, lesions are most pronounced in the villi. There is mucosal loss and villus shortening. Immature and mature forms of the coccidia may be present. The submucosa is often filled with hemorrhagic or eosinophilic infiltrates. In severe cases, the mucosa is lost to the basement membrane. Protein loss is considerable, and hypoproteinemia is the most consistent blood abnormality. Anemia, hyponatremia, and hypochloremia are other common abnormalities.

The small coccidia primarily cause clinical disease in crias up to around 8 months of age. South American crias are usually shedding by around 23 days of age, with earliest shedding by 15 days, meaning they become infected shortly after birth. Shedding increases until 40 to 50 days of age, then gradually tapers off. Illness usually occurs in those first 2 months. Under rare circumstances, clinical disease is seen in older crias or adults. This usually reflects overwhelming exposure or a poor immune response.

The large *Eimeria* of New World camelids are *E. macusanensis* and *E. ivitaensis*. These are 3 to 4 times larger than small coccidia, and are approximately 80 to 100µ in length. As such, they resemble *E. leuckarti* of horses, *E. camelli* of camels, and other large *Eimeria. E. ivitanensis* oocysts are elongated ellipses, whereas *E. macusaniensis* is ovoid and pyriform, resembling a cut avocado or watermelon seed in shape. Both have an obvious polar cap. There is some heterogeneity in size and shape of *E. macusanensis*, and it is possible that future research will reveal that distinct species exist. *E. macusanensis* also has a thick wall (approximately 8.5 to 11µ), which makes the cyst extremely durable; identifiable cysts have survived approximately 10,000 years in mummies.

The life cycles of the large coccidia resemble those of small coccidia, except that everything generally takes longer. The prepatent period for *E. macusaniensis* is from 31-43 days; that from *E. ivitaensis* has not been reported. Sporulation times for *E. macusaniensis* range from 2 to 3 weeks, with faster times under warmer conditions. Sporulation appears to arrest at 7ºC or below. Sporulation times for *E. ivitaensis* has not been reported, but appears to be in the 7 to 10 day range in our laboratory. The longer lifecycle means that patent infections appear later than with small coccidia, but not necessarily that disease occurs later. Severe disease and death appear to be able to occur within 3 weeks of initial exposure and 2 weeks before establishment of patency. There is growing evidence that crias shedding small *Eimeria* oocysts or showing signs of enterotoxemia, may actually be dying of prepatent *E. macusaniensis* infection. Additionally, there are increasing reports of prepatent or patent disease in adult camelids. Some of these are long-time herd residents, but most have a history of transportation and mixing with new groups of animals. Whereas shows, sales, and movement for breeding may cause stress and inhibit the immune response, the simplest explanation may lie in eating habits: new entrants in a herd are more likely to eat off the ground than out of feeders, or more likely to eat in the less desirable areas of pasture. Thus, they may ingest larger doses of the parasite and be more likely to show disease signs.

The characteristics of clinical disease associated with *E. macusaniensis* have been studied and reported more extensively than any other parasitic gastroenteritis in camelids. It is likely that much of this information pertains to other coccidial infections and to parasitic gastroenteritis in general. In younger camelids, gastroenteritis is more likely to result in clinical diarrhea, whereas in older camelids, diarrhea is often absent or easy to miss. In fact, in adults, it is frequently counterintuitive to directly link the presenting signs to GI illness. In addition to or instead of diarrhea, general signs include weight loss or poor weight gain, ill-thrift, and increasing lethargy, weakness, and loss of appetite. It is one of the most common causes for weakness, weight loss, hypoproteinemia, or ill thrift in our area. Some animals show colic signs, probably more with *E. macusaniensis* than with any other parasitic gastroenteritis. As the disease progresses, hypoproteinemia worsens without commensurate anemia; mild anemia is common, but hypoproteinemia and hypoalbuminemia often become severe (such as 3.8 to 2.2 mg/dl for serum total protein and 1.8 to 0.6 mg/dl for serum albumin concentrations). There is also often some reduction in serum sodium and chloride concentrations, and hypokalemia become more marked with anorexia. With worsening disease, the animal becomes susceptible to translocation of bacteria or toxins through the damaged mucosa, the effects of hypoproteinemia, and circulatory shock. Signs of other organ systems are seen: camelids may develop ascites, hydrothorax, hydropericardium, pharyngeal edema, and cerebral edema, with ensuing abdominal distention, increased respiratory effort, lethargy, tachycardia, hypothermia, dysphagia, salivary loss, and cerebral signs. Eventually, there is also clinicopathologic evidence of the systemic disease including azotemia, metabolic (usually lactic) acidosis, high liver enzymes, hyperbilirubinemia, and increases in fat fractions. Abdominocentesis usually yields a transudate. Abdominal imaging is also inconclusive: colicky camelids may have ileus and fluid-distended intestine, though usually to a lesser degree than camelids with GI obstruction. Thickened bowel walls are rare (<10% of cases). In some cases, signs are insidious, and the animal is simply found dead.

There is also a growing body of evidence, most of it from South America, that *E. macusaniensis* is an important facilitator for subsequent enterotoxemia. Many crias diagnosed as dying from enterotoxemia have been found to have extensive coccidial damage to their bowels, and herds that adopt aggressive anticoccidial strategies have experienced significant reductions in losses due to enterotoxemia. South American crias have been found to have fecal *E. macusaniensis* oocysts as young as 5 days of age. While these particularly oocysts are just passing through, their presence indicates that crias of this age are likely also eating infective oocysts and suffering gut damage. Considering that most enterotoxemia deaths occur between this early ingestion period and the onset of patent infections (around 32 to 40 days later), the role of *E. macusaniensis* in these infections cannot be appreciated by fecal flotation alone.

Light microscopic examination of fecal samples is also the standard method of detecting coccidia. There are a number of pitfalls, namely prepatent or peripatent disease, intermittent shedding, and oocysts with a range of densities. Prepatent disease is more common in the coccidia with longer life cycles, namely *E. macusaniensis*. For *E. macusaniensis*, clinical disease and death can occur up to 2 weeks before the appearance of oocysts in the feces. For small coccidia, clinical disease usually appears around the same time as fecal cysts passage, but may precede it by a day or two. If prepatent disease is suspected, serial fecal analyses at two to four day intervals may be used to confirm the organism; presumptive treatments may be instituted in the interim. When looking for prepatent or peripatent shedding of protozoan parasites, it is important to keep the shedding curve

in mind; *Eimeria* oocyst passage resembles a bell-shaped curve, with few at the earliest stages, peaking in 1 to 2 weeks before tapering off.

Small *Eimeria* and *E. ivitaensis* oocyst appear to float readily in flotation solutions with a specific gravity of 1.18 (saturated saline, 33% $ZnSO^4$). *E. macusaniensis* requires denser solution (S.G. >1.025?).

We have recently developed a fecal PCR to detect *E. macusaniensis* and another to detect *E. lamae.* Whereas similar tests have been used for a variety of protozoa, previous use has focused on finding DNA in shed oocysts, or similar fecal forms. We are reasonable certain with *E. macusaniensis*, partially because the cysts are so hard to break, that we are finding DNA that is not associated with oocysts, and may be useful in identifying prepatent infections. Validation of this test is ongoing.

The most commonly used anticoccidial medications for camelids in North America are amprolium (10 mg/kg at the label dilution, PO, q24h for 5 days) and sulfa antibiotics. Both are more effective against the immature forms of the parasite in ruminants and should not be expected to immediately reduce fecal shedding. Efficacy in camelids remains empirical, and pharmacokinetic trials on sulfa antibiotics suggests that uptake (and therefore efficacy) is negligible once the forestomach develops. Likewise, amprolium put in the water supply is often not drunk in sufficient quantities to be effective; individual animal dosing is preferred. Treatment with amprolium during the prepatent period does appear to reduce subsequent shedding, and also decrease the viability of oocysts. There has been some concern that protracted treatment with amprolium may cause polioencephalomalacia, but I have not seen that with the 5-day course. Simultaneous treatment with Thiamine should be reserved for animals in which polio is a legitimate concern, as it decreases the efficacy of the amprolium. Milling decoquinate (0.5 mg/kg for 28 days) into pelleted feeds may also be helpful, if the crias are old enough to eat the pellets. Ionophore antibiotics have been associated with toxicosis, albeit often when given at high concentrations. In the absence of safety date, their use in camelids is generally avoided.

Benzeacetonitrile compounds have been gaining popularity as a coccidia treatment in the U.S. and are in heavy use in other countries. Ponazuril and toltrazuril (5 to 20 mg/kg, PO, q24h for up to 3 days) are effective against multiple stages of the parasite and rapidly stop shedding. The higher doses and longer courses are for treatment of individuals; the lower doses may be used for control.

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GASTRIC ULCERATION IN NEW WORLD CAMELIDS

Gastric ulceration is one of the best publicized health problems to affect New World camelids outside of their natural range. It was one of the first disorders recognized in these transplanted camelids and often the only one recognized postmortem. A fair amount of research was conducted, particularly on the use of medications to treat ulcers, but the results of this work were often unsatisfactory. First off, the medications often did little to change stomach acidity, one of the putative causes of ulcers, and second, it was often difficult to assess who to treat.

Ulcers are loss of the surface lining of the stomach or intestine. Traditionally, since the part of the stomach where ulcers most commonly occur is also the area with the greatest acidity, we have thought of ulcers as being a chemical burning away of this lining. Other factors, often extrapolated from people, were also thought to contribute. Among these factors were stress, the use of aspirin-like medications, dehydration, and high dietary carbohydrate. How much each of these factors contributed could vary from case to case, and in many cases, none of them were present.

Camelids secrete stomach acid into their third compartment at a fairly constant rate. This is typical of herbivores and particularly ruminants, because they are constantly eating or chewing their cud, and thereby fairly consistently releasing feed from their first stomach into subsequent compartments. With fairly constant secretion, the acid needs to be diluted or neutralized to bring stomach contents to a higher pH to go down the intestine. Dilution happens through the passage of first compartment contents into the third compartment. These contents may be more or less acidic, based on how much soluble sugar the animal eats. Neutralization happens further along in the stomach and intestine, where more basic fluids are added.

The roles of stress, aspirin-like medications, and dehydration are harder to define. Again extrapolating from humans, their roles were often described as either increasing gastric acid secretion or compromising the stomach lining's defenses by decreasing the protective mucus coat or by decreasing blood flow. The theory of increasing acid secretion may not have much validity, because camelids likely secrete acid constantly anyway. The other way these factors worked their way into the popular psyche was how sick camelids often spent their last days under veterinary care. Aspirin-like drugs are important contributors in many situations due to their beneficial effects,

dehydration is common in sick animals, and the whole situation of being sick and being hospitalized is often considered stressful. When the animals finally succumbed, and an ulcer was identified postmortem, rather than considering it the cause of the primary disease, it was often considered a fatal complication of the treatment period. Various recommendations were made, including limiting the use of aspirin-like drugs and providing companion animals for the treatment period. Antiulcer medications, although they were repeatedly shown in scientific studies not to be absorbed or effective in reducing gastric acidity in camelids were routinely prescribed and used in large quantities. No appreciable success was noted through this approach, but in the presence of failure, continued failure did not seem noteworthy.

In the following years, we learned a lot about normal camelids and the diseases that affect them. Our differential diagnosis lists grew longer, and we identified more diseases that had specific treatments, including even some that responded positively to treatment. We also learned that there are different types of ulcers and that their development occurred for different reasons.

The first compartment is essentially a fermentation chamber, where increasing acid production is the result of increase fermentability of the ration. First compartment ulcers are a poorly recognized entity. They can be focal, multifocal, or diffuse and usually lead to vague signs of poor growth, weight loss, or intermittent diarrhea. Complications are usually due to protein loss or invasion of microorganisms, particularly fungi. Causes of these ulcers include previous or current forestomach acidosis, tumors, foreign bodies, and fibrous balls within the gastric lumen. There is generally no suitable treatment, as the lesions are not identified premortem.

The third gastric compartment is long and tubular, running craniocaudal in the right abdomen. Within the first two-thirds of its length, fermentative digestion is still taking place. At its caudal extreme where the organ abruptly turns dorsal and cranial, there is a small region of hydrochloric acid secretion. Ulcers here cause about 6% of all camelid deaths in North America. Interestingly, their occurrence appears to be diminishing, potentially due to a "maturation" of the industry and a decrease in the number of neophyte owners.

Ulcers typical occur at the elbow of the third gastric compartment or up toward the pylorus - the places where the luminal contents are most acidic. Given the location, it is likely that gastric filling and emptying functions are very important in ulcer development. In a normal camelid, less acidic fermented feed would push into this regions from the first and second compartments, and the newly secreted acid would be rapidly propelled out of the tubular stomach into the intestine for neutralization. Under conditions of poor gastric filling and emptying, such as anorexia or intermittent feeding, dehydration, electrolyte or acid-base abnormalities, and gastric hyperacidity (grain feeding), the acid contents would remain unneutralized and possibly damage the mucosa. Reflux of bile from the duodenum would exacerbate acid injury.

Gastric ulcers are most commonly recognized on perforation. Affected animals frequently are depressed, weak, and anorexic, and may appear colicky or shocky. Fecal production and gastric motility are greatly reduced. Blood and peritoneal fluid provide evidence of septic inflammation. Some camelids lose most of their acid-secreting mucosa and are chronic poor-doers for months before perforation, but most are outwardly completely healthy until perforation. Thus, it is difficult for us to recognize non-perforating ulcers, and their frequency is unknown. Fecal occult blood tests

are not a reliable method of diagnosing non-perforating gastric ulcers, and melena is more common with gastric masses or massive ulceration due to copper toxicosis.

Treatment after perforation is usually heroic. Camelids do not always die acutely: many are able to surround the lesion with omentum, fibrin, and adhered loops of bowel, but these camelids do not do well long-term. Pharmaceutical treatment of non-perforating ulcers has also been fairly unrewarding: it is difficult to identify which camelids to treat and most anti-ulcer medications are poorly efficacious in camelids. Parenteral administration of proton pump inhibitors appear to offer the most promise.

Because of our failure in recognizing ulcers before they perforate and of treating them after the due, the emphasis should be on recognizing at-risk camelids and changing their management to decrease the likelihood of ulcer formation or progression. Clinical evidence also suggests that ulcers can form and perforate in under 24 hours, so following this guidelines may help save animals. To preserve good filling and emptying, camelids should be provided with, and eat, diets mainly consisting of roughage. Too much grain, fruit, or other fermentable feed increases forestomach acid production and inhibits motility. Roughage must be eaten to be effective. Animals that are bullied or are bullying or other wise preoccupied do not eat. They should be identified and separated from their distractions. This, of course, does not mean isolation, but rather maintenance in a smaller group of friends. Breeding males are especially prone to stress, particularly what facing competition, and show or shipped animals are also at high risk due to the changing environments and intermittent feeding associated with travel. Sick animals also tend not to eat well. Correction of their underlying diseases should be the focus of treatment, and with success in that department, ulcers rarely become a problem. In summary, maintenance of good gastric emptying, and avoiding causes of stress or anorexia are probably the best way to avoid gastric ulcers.

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OTHER GASTROINTESTINAL CALAMITIES

Grain Overload

Camelids are adapted to survive in a harsh, nutrient-poor environment. Their gastric fermentation of fiber is the most efficient of hoof stock species. It is likely that the non-keratinized epithelium of their gastric saccules contributes to this efficiency by providing surface area for rapid absorption of volatile fatty acids from the gut lumen. Bicarbonate is generated when fatty acids are absorbed under low energy conditions, leading to speculation that camelids have greater gastric buffering capacity than cattle, and thus are protected against grain overload. It is unlikely that this bicarbonate would be generated under high energy conditions and it is unwise to think that camelids are anything but highly susceptible to grain overload.

The perception that camelids are less susceptible to forestomach acidosis than conventional ruminants is flawed. Camelids are purported to be discriminate eaters, and thus not prone to engorgement, and they rarely are fed large quantities of highly fermentable feeds. They also are reported to actively secrete bicarbonate into their forestomach, which has led to the belief that they have exceptional gastric buffering capacity. It is unlikely that saccular bicarbonate functions as physiologically-important buffer. It also is probable that high density confinement and the availability of highly-fermentable feed increase competition between animals, alter feeding behavior, and increase the risk of forestomach acidosis. As with ruminants, dividing grain feedings over the day, making feed changes slowly, providing adequate roughage, and preventing individual animal from engorging on grains are useful methods to prevent forestomach acidosis. Deleting or decreasing the barley from grain mixes has been helpful in preventing future cases..

Feeding accidents are a common cause of forestomach acidosis. We also have noted two other syndromes of susceptiblity. The first of these occurs when trying to increase body condition in a debilitated, elderly camelid through increased grain consumption. The abrupt addition of a small amount of grain often leads to acidosis, and frequently is fatal. Ulceration of the forestomach mucosa may be extreme in such cases. The other syndrome involves group-housed camelids, which are fed a combined ration for all animals. Though the overall amount of grain would appear reasonable if equally divided, certain aggressive eaters may get more than their share. Pathogenesis of the disease is very similar to cattle. Clinical signs include acute depression, gastric atony and fluid distention, a stumbling gait, and arching of the head and neck over the back. Diarrhea is a convalescent finding, and polio-like signs have been reported. Diagnosis is made by determination of gastric fluid pH. Metabolic acidosis is the most common blood abnormality.

Normal forestomach pH in camelids is between 6.4 and 6.8. These values tend to climb with anorexia or salivary contamination. Camelids with acute acidosis often have values of 4.0 to 4.5; these may climb to 5.0 or more with time. In general, anything under 6.0 should be considered suspicious. Some camelids on grain diets have gastric pH around 5.5 for long periods; they typically have intermittent bouts of diarrhea and poor doing, unlike ruminants which can adjust to these lower gastric pH.

Treatment follows the guidelines established for ruminants. Correction of dehydration and systemic acidosis is critical as is prevention of continued forestomach acid production and systemic

absorption. Treatment with intravenous fluids containing bicarbonate and cold oral fluids containing magnesium hydroxide usually is sufficient, although gastric lavage or gastrotomy may be necessary with severe disease or if intravenous fluids are impractical. Treatment with antibiotics and thiamine HCl also are recommended to prevent secondary complications. Most affected animals survive with treatment. A small percentage become chronic poor doers with hypoproteinemia and weight loss, most likely due to deep, non-healing ulceration of the gastric mucosa.

Gastroliths

The non-keratinized epithelium of the gastric saccules is protected from the abrasive action of ingested fiber by muscular sphincters coated with keratinized squamous epithelium. If large particles pass this sphincter, they are frequently trapped in the saccular lumen. Wedge-shaped grains, particularly sunflower seeds, appear more prone to do this. Once in the lumen, mineral is laid around the objects, forming stones. In general, these stones do not result in overt clinical disease and they are a frequent finding in aged camelids. However, because of the important role of the saccular lining in nutrient absorption, it is likely that camelids with most saccules blocked are poor doers. Also, on rare occasions, these liths are large and jagged, and may cause fatal peritonitis by rupturing through the serosal surface of the gastric compartment. Gastroliths are easily recognizable on abdominal radiographs, but there is currently no treatment for them.

Neoplasia

Squamous cell carcinoma is the most commonly reported primary neoplasm affecting the gastrointestinal tract of camelids, while multicentric lymphoma is the most commonly reported neoplasm in camelids overall, and commonly invades the terminal third compartment or saccular regions of the first compartment. Other neoplasms, particularly those of glandular tissue or smooth muscle origin occur sporadically. In general, neoplastic disorders cause gradual weight loss with progressive lethargy and anorexia. Diarrhea might be noted early in the course of the disease or terminally. Lymphoma may cause palpable masses on other body parts. Often, the early clinical signs are missed and the llama is either found dead or extremely debilitated. Antemortem diagnosis and successful treatment of malignant internal neoplasms are rare, but could be increased by earlier identification of the sick animal, and greater awareness of the tumors, and a diagnostic work-up directed towards neoplastic disorders.

Twists, blockages, and related disorders

Most veterinarians are more familiar with horses than they are with camelids, and that may be useful of detrimental when dealing with camelids with various intestinal accidents. These occur fairly rarely, but even so, are more common in camelids than in ruminants. The major issues relate to blockage of the gut with undigested plant material or twists of the most mobile part. Specific plants, ectoparasites causing the alpacas to chew themselves, and dental issues may promote blockage formation, but that is speculative. Coccidia may promote twists; that is slightly more scientifically supported.

For the owner, the most important thing is to recognize what is happening and how severe it easy. Alpacas with abdominal pain usually lie down and get up frequently, lie restlessly, and kick some of

their legs out to the side. In other cases, they are more mopey and develop a pot-bellied look. In all cases, their appetite and fecal production diminish, but it may take days for complete cessation of these functions.

For the veterinarian, recognizing severity is also crucial. Blood tests may reveal evidence of obstruction similar to what is seen in ruminants with abomasal outflow compromise, but these changes are only seen with certain types of blockage. The most important, initial, on-site evaluation is confirmation that bowel is becoming distended and losing its function. This is best determined by ultrasonographic examination, mainly concentrating on the right flank. Fluid-filled loops which do not contract and propel well is typical. Unfortunately, most of the problems respond best to surgical repair, but if that is done early, prognosis can be quite good.

Non-parasitic diarrhea

Most of the disorders causing diarrhea in camelids are the result of parasitic infections, but a few notable ones are not. The most important of these is viral diarrhea, usually associated with Coronavirus. This is most likely a bovine virus that has crossed over into alpacas at least once. It tends to affect animals without immunity to it. When a crossover event first occurs, all camelids are susceptible and herd outbreaks of diarrhea affecting all ages are seen. These usually begin with animals that were off property, for a breeding or a show usually, who brought the virus back with them. Once these outbreaks have run their course for a while, only crias are susceptible. Rotavirus has also been described, but appears to be less common.

Diarrhea associated with viruses is usually explosive and watery. Affected alpacas can be reasonably bright, but also can become lethargic with dehydration and electrolyte loss. Water and electrolyte replacement are the cornerstones of treatment, and in adult animals, provision of shelter, adequate water, and a salt block may be all that is necessary, and the disease runs its course in 4 to 9 days. Neonates are much more vulnerable, and may require supplemental oral or even intravenous fluids. Oral calf electrolyte products often contain too much salt and sugar for camelids, and should therefore be diluted or used in combination with other water sources.

Diagnosis can be tricky. Bovine ELISA or PCR assays may work, or the virus may be found on electron microscopic examination of fresh feces.

CAMELID DERMATOLOGY

Camelid skin conditions have been some of the most enigmatic and frustrating disorders that we deal with. Owners are often very concerned, diagnostic tests are not enlightening, and treatment response is mediocre or temporary at best. References are fairly sparse and most reports are anecdotal.

Normal skin

Camelid skin may be divided into zones. It is thickest in areas of the densest fiber, including the trunk and the back of the neck. It is thinnest in areas of light fleece and heat exchange, including the inguinal and axillary regions, the escutcheon and the head. To facilitate heat exchange, thin skinned

areas frequently have extensive dermal plexi. Skin also contains a number of eosinophils, which can confound diagnosticians.

Alpaca wool is fine and has few primary guard hairs. Llama wool tends to be less dense with a greater percentage of long, coarse guard hairs. The follicles exit the skin obliquely.

The muzzle, pinnae, legs and front of the neck are covered with short, thin hair. These regions are more extensive in llamas, whereas in alpacas, the heavy fleece covers larger portions of the legs and head. The axilla and inguinal region are relatively hairless.

Skin disorders

Camelid skin disorders can be roughly divided into disorders affecting one of four areas: 1) fleeced areas 2) haired and hairless areas, and 4) mucocutaneous junctions. Disorders affecting haired areas and the mucocutaneous junctions are the most common. In all cases, the most common findings are alopecia and dry hyperkeratosis.

Fleeced areas

The heavily fleeced areas of camelids are infrequently affected. The most common diagnosis is pediculosis. Camelids are affected by both sucking and chewing lice, with sucking lice more common in my practice area, but less common in other areas. Infestation is most common during the colder months, partially due to the long fleeces during that period, as well as the possibility that confinement housing or huddling eases transmission. Lice are usually easy to find by parting the fleece near the affected area, and pediculosis is one of the relatively few pruritic dermatoses that we see. Patchy fiber loss is also common. The type of louse may be identified by microscopic examination of the mouthparts.

Shearing and exposure to sunlight and fluctuating temperatures are very effective at reducing lice numbers, but may be impractical certain times of the year. Parenteral avermectins may be effective against sucking lice. Topical livestock anti-louse treatments are effective against both types.

Dermatophilosis (Rain Rot) may be seen, particularly in areas with wet, mild areas. Lesion are most common on the back. Affected areas have wet, clumped wool, which comes out in clumps. The underlying skin is erythematous, with erosions and exudate. Impression smears of the exudates may reveal the classic "railroad track double chains of *Dermatophilus congolensis*, gram-positive cocci. Culture or biopsy may be used to confirm the diagnosis. Treatment is by use of parenteral antibiotics (usually penicillin or ceftiofur) and possibly clipping and topical disinfectants.

The other major consideration regarding the fleeced area is its possible contribution to heat stress. In hot, moist areas, especially during exercise, the inability to dissipate heat can lead to a pathologic rise in body temperature. Although the fleeced area is important in the pathogenesis of this disorder, the lesions are most common in the axilla, inguinals regions and perineal region, where erythema and edema may be seen.

Haired and hairless areas

The lightly haired areas are not as protected as the fleeced areas, and hence appear to be much more vulnerable to trauma, infection, and ectoparasitism. Bacterial folliculitis can lead to patchy, crusty, exudative hair loss, usually with minimal to mild pruritus. Staphylococcus intermedius is the most common isolate. Diagnosis is by culture and biopsy. Systemic antibiotics and topical antiseptics usually lead to resolution of signs within about 2 weeks.

Dermatophytosis tends to affect the legs, face, or peritoneum. Winter confinement appears to increase risk. A variety of *Trichophyton* and *Microsporum* have been reported. Lesions tend to be non-pruritic and alopecic, with thick crusts. Crusts tend not to be as thick as the "asbestos-type" lesions seen in cattle, and the diagnosis may be missed without further testing. Diagnosis is achieved by identifying the organism by microscopic inspection of affected hairshafts or growth in dermatophyte test medium. Culturing *T. verrucosum* may require special techniques. Treatment of dermatophytosis involves clipping and cleaning affected areas, followed by topical treatment with iodine, chlorhexidine, lime sulfur, Captan, Miconazole, or bleach. Using landscape-grade lime sulfur to make a 2% solution appears to be effective.

A variety of mites affect the predominantly haired regions. These include the Sarcoptic, Chorioptic, and Psoroptic mange mites, *Demodex*, possibly sheep keds, the Northern Fowl mite, and harvest mites. Of the mange mites, *Sarcoptes* is the most common in South America. It is a deep burrower, affecting primarily the legs, neck, face, axilla, ventrum, and perineum. In severe cases, the infestation becomes generalized. Pruritus is usually intense. *Psoroptes* is less common, and affects primarily the ears. Both these mites may be found by deep skin scrape, or potentially microscopic examination of exudates, particularly from the ears. Parenteral avermectins are very efficacious, and have severely limited the prevalence of these mites outside of South America. Treatment should be repeated weekly to biweekly for 2 to 4 treatments to eliminate mites newly hatched from eggs.

Chorioptes is the most common mite in camelids in North America and Europe. It resides near the skin surface, and primarily affects the legs, feet, tail base, and ears. Typical lesions include non-pruritic or mildly pruritic alopecic areas of thickened, crusty skin. These spread and worsen slowly, eventually leading to large patches of dry, elephantine skin. Certain individuals appear to be affected much worse than others. A hypersensitivity reaction is suspected. Detection of the mite is through skin scrape or occasionally biopsy. Because *Chorioptes* dwells near the surface, the scrape need not be deep. Although the edges of the lesions occasionally yield mites, scraping the skin between the toes, even if unaffected, appears to offer the best success. The axilla also frequently yield mites.

Chorioptic mange mites are extremely difficult to eradicate. Because we think the lesions are due to a hypersensitivity reaction in individuals, even a few surviving mites can elicit disease signs. The superficial habitat of the mite potentially decreases the efficacy of parenteral antiparasitical agent. Non-clinic herdmates or the environment can serve as sources for reinfection. Unlike lice or the other mange mites, *Chorioptes* appears to survive up to about 2 months off the host. The best treatment options augment individual treatment with either removal from the herd to a clean environment (semipermanently), or concurrent herd and environmental treatment. Individual treatments that appear to have some efficacy include parenteral avermectins, topical permethrins, topical lime sulfur, or topical fipronil. All are likely to fail unless the reservoir populations are also

eradicated. Continuously treating over the entire 60 day off-host survival period has not successfully eliminated infestation.

Demodex infestation is rare. When it occurs, it results in papular or nodular alopecic lesions on the face, neck, and brisket. Mites may be expressed from the nodules and identified microscopically. The Northern Fowl mite and harvest mites have also been described as causes of dermatitis of the distal extremities. Fowl mites usually come from chickens, so that reservoir population must be addressed. Harvest mites are difficult to diagnose because of their short time on the host. They tend to be seasonal, and may be treated with topical repellents or permethrins. Removal of the camelid from the affected pasture may be necessary.

Non-mite related hyperkeratotic lesions were once thought to be very common. A sizeable percentage of those are now being blamed on the Chorioptic mange mite, but some remain unexplained. Among the syndromes are Dorsal nasal alopecia, Zinc deficiency/responsive dermatosis, Superficial hyperkeratotic dermatitis (Munge), Fly bite allergies, and Solar dermatitis. Most of these appear similar clinically and on biopsy, with parakeratosis with variable amounts of various cellular infiltrates and necrosis. Solar dermatitis tends to affect exposed areas with short white hair, such as the ears. Diagnosis is made easier by the presence of unaffected pigmented spots. The most effective treatments are increasing time indoors or covering affected areas with an effective zinc oxide or titanium dioxide based sun block.

Fly bite allergies tend to be seasonal and may respond to housing or insect repellants. Dorsal nasal alopecia may relate to halters or feeders. Otherwise, these disorders tend to get lumped together in the idiopathic category. Zinc responsive dermatosis gets its name from the fact that some lesions appear to respond to zinc supplementation, even though the camelids do not appear to have zinc deficiency. Organic forms such as zinc methionine are much more efficiently absorbed than inorganic forms, such as zinc oxide or sulfate. Given that many camelid mineral supplements now contain abundant zinc, the percentage of camelids with idiopathic hyperkeratotic dermatosis that respond to zinc appears to be declining. Other treatments that have been tried include many of the standard treatments for skin disorders, including steroids, vitamin E and selenium, antibiotics, antiparasiticals, and antiseptic watches.

An idiopathic congenital form of diffuse hyperkeratotic dermatosis (Ichthyosis) has been described. A fungal diorder (Entomophthoramycosis conidobolae) has also been described.

Mucocutaneous junctions

The lips, nares, periorbital areas, and mucocutaneous junctions of the urogenital system are affected by some of the same hyperkeratotic disorders that affect haired areas, particularly Munge and zinc-responsive dermatosis. Additionally, a number of discrete, proliferative lesions may be seen. Contagious ecthyma or Orf may occasionally be seen, particularly in camelids exposed to infected small ruminants, or also potentially in camelids inhabiting a facility infected with the virus. Similar lesions may be seen in camelids exposed to camels with Camelpox. These diseases may be diagnosed by history and appearance, with electronmicroscopy of crusts or histopathology used to confirm the diagnosis. Lesions should regress over about 2 months.

Recently, a specific camelid Fibropapilloma virus has been identified. Associated lesions are usually multifocal and proliferative without crusting. They do not typical regress. Surgical removal can be curative, but also frequently lead to regrowth. In some cases, the regrowth is actually granulation tissue, not the original fibropapilloma. Cryosurgery and injection with cisplatin have been used on persistent, recurrent lesions.

Miscellaneous

Myiasis can occur in a variety of areas. The most common sites are around injuries or other lesions, such as dermatophilosis, or around the perineum and tail base.

Squamous cell carcinoma of the skin has been described. It appears to be an unusual tumor, affected either areas of previous injury or near the mucocutaneous junction. Oral and preputial tumors have been seen. Diagnosis is by biopsy. Excision and local chemotherapy may be curative.

TIPS

In principle, if we would like to discuss how best to keep alpacas healthy, it is best to start with a discussion of what makes alpacas unhealthy. If you look at everything possible, this becomes a lengthy list, but if you narrow the focus to things likely to affect groups of animals, it becomes much shorter. In my estimation, the most common challenges to group health in alpacas include:

1. Parasitic diseases
2. Nutritional deficits
3. Nutritional excesses
4. Herd stresses
5. Weather stresses
6. Infectious diseases earn an honorable mention; they are not as common in herd challenges as the previous five, but can be devastating.

Parasitic Disorders

There are a number of parasitic disorders that can have severe impact on individual alpacas or herds. These include diseases caused by the Barber Pole worm [Haemonchus], liver flukes, whipworms [Trichuris], Lamanema chavezi, Eimeria macusaniensis, Cryptosporidium, Giardia, Parelaphostrongylus tenuis, the sarcoptic mange mite, and the blood parasite Mycoplasma haemolama. The gastrointestinal worm (Haemonchus, Trichuris, and Lamanema) all infect animals through ingestion of contaminated fecal material or feeds contaminated by fecal material. Their life cycles are slightly different. The Haemonchus egg hatches fairly quickly and the larva matures in the environment. It is generally believed that the larvae climb stalks of grass, and thus a higher rate of successful new infections on pasture. Lamanema is fairly rare outside of South America. Its larva

matures within the egg, then hatches and also requires ingestion, probably associated with pasture. The Trichuris larva matures within the egg, which hatches on ingestion. The keys here are that all of these initially are shed by infected alpacas as eggs, and then require a variable period of maturation outside, followed by ingestion by the new host. Distribution of eggs out of feces occurs most readily when they become wet. Removal of feces while it is still dry is the most effective way of controlling any of these parasites, and may be the only effective way of controlling whipworm. Haemonchus and Lamanema require pasture, and thus mowing may have an effect as well.

Crias are an exception to this pattern: they may have looser feces that allows easier distribution throughout the environment and they also tend to eat feces, dirt, and whatever else they find. Thus, infection of crias with any of these parasites, is near impossible to prevent without timely fecal removal.

Eimeria has a similar life cycle to Trichuris: it matures in the egg and must be eaten from the surface of the ground. Thus animals eating off the ground, especially in dirt lots are most vulnerable. Crias, being the poop eaters that they are, are also vulnerable. Besides fecal removal, which should be done every 2-3 weeks to control Eimeria macusaniensis, feeding susceptible animals from above ground feeders with limited spillage may provide effective control.

Liver flukes are usually residents of cattle, sheep, or in the US, deer. The larger versions have an aquatic snail as the intermediate host, and thus are most common in moist areas or around water sources. Fowl and anti-snail agents and restricting access to damper areas may be more useful than medications in controlling them.

Cryptosporidium and Giardia are most commonly found in sick crias. They are especially important parasites because they affect that age group and because man can get them too. You are unlikely to get Giardia from your alpacas but transmission of Cryptosporidium from alpacas to man is fairly well documented. These were rare when farms were all small, such as less than 10 animals with only a couple of crias a year. As farms became bigger, these became greater problems. Both may come from other domestic animals or wildlife. Giardia is associated with contaminated water sources and damp pasture, but Cryptosporidium is not. Giardia may thus be controlled by restricting crias access to the source of infection. Hygiene is currently the only way to combat the spread of cryptosporidiosis.

Several of the other listed parasites are not common here.

Nutritional Deficits

Both nutritional deficits and excesses are becoming more common in the US for the same reason: with the pullback in alpaca prices, many owners are looking for ways to economize. This may result in not feeding enough of something or discontinuing an essential supplement, or in replacing a quality product designed for alpacas with a less suitable but cheaper substitute.

The most common nutritional deficits are everything at once (starvation), in some cases protein deficiency, and rarely single vitamins or minerals. Starvation is a sad event and the keys to recognition and prevention are clear. If it is partial, herd stressors may be more evident than starvation signs. Using lower quality feeds may also lead to deficiencies in individual nutrients, that

lead to more subtle signs. Protein deficiency is a controversial example. Alpacas appear to have a fairly high requirement for protein compared to cattle or sheep. Feeding too little results in caloric sufficiency, and hence good body condition, but may compromise liver function and energy metabolism, leading to poor milk production and/or fatty liver. The flip is production of ultrafine fiber: too much dietary protein is associated with an increasing fiber diameter. The trick is balancing, and to optimize that, using the same animals as breeders and fiber producers may be problematic in some cases.

Regarding individual nutrient deficiencies, the most-widespread and best known is the vitamin D deficiency associated with dark coat colors, long fleeces, and short days. This is covered in another talk.

Nutritional Excess

Although too much of anything is likely to be bad, only a few nutritional "poisonings" have been identified in alpacas. The most important of these are carbohydrate overload, vitamin D overdosing, and copper poisoning. Regarding vitamin D overdosing, there appear to be different philosophies in different parts of the world, and I would refer you to your veterinarians for their specific recommendations. Vitamin D supplementation is common around to world to combat the deficiency associated with rickets. This supplementation may be in feeds, oral gels, or injections. Most of the acute overdoses have been associated with injections. This usually occurs in crias; they quickly lose their appetite and show blood or radiographic evidence of renal disease, including mineralization. Chronic poisonings are more common in adults and related to an excess in feed. Signs are the same, but are more subtle and develop more slowly. Treatment is less rewarding than prevention in these cases, so ensuring proper amounts of supplements are given is key.

Carbohydrate overload is fairly rare. It usually occurs with inexperienced owners or holiday feeders who simply give too much grain supplement. Most mineral mixes for camelids have a forage base and are fairly safe, so this tends to relate to pure grain mixes. These are seldom necessary in alpacas, so avoidance is not too hard.

Copper poisoning usually occurs when pellets or mineral mixes designed for cattle, horses, poultry, or swine are fed to alpacas. Copper slowly damages the liver, so signs are also fairly subtle until the animal starts to lose weight and stop eating. Blood or tissue evidence combined with knowledge of what was fed are the keys to recognizing this problem, and avoiding the wrong feeds is the key to prevention.

Herd Stresses

Interactions between individuals often appear to work themselves out fairly quickly, but may have longer effects than is commonly appreciated. The key is usually space. If alpacas can get away from each other, they tend to do well. When they are confined or have to come into smaller spaces for feeding, weather protection, or other issues, the old rivalries emerge, and the stress on the animals increases. This stress is enhanced by feeder space or feed inadequacy, as there is greater competition for what is available. Various reports out there list recommendations for feed trough space or square footage per animal, but your observation is important here too. If specific animals are shy to come in, bullied when they do arrive, or end up picking up the leftovers, their

management needs to change. Groups can be separated, feeding space improved, or anything that decreases these negative interactions. The effects of stress are hard to describe. Gastric ulcers, one of the best known maladies of alpacas, are frequently blamed on stress, and are covered in a separate paper. Other effects may include decrease immunity to infectious and parasitic disorders, increased exposure to these disorders, reproductive failure, and nutritional deficits. All of these increase unthriftiness.

Weather Stresses

Heat, cold, wet, and dry each pose a different challenge, and may create still other problems when in combination. Overheating is commonly described, and appears to be worst when combined with humidity. The native alpaca environment tends to be moist in the shoulder seasons and dry in the summer and winter. They appear to tolerate arid conditions very well, as long as they have adequate potable water. They also tolerate dry cold, but can become quite hypothermic in cold, snowy climates. Shelter is really the only solution; occasional recommendation to feed more calories are problematic due to carbohydrate overload. Heat alone is also fairly well tolerated as long as they are shorn and have shade: there may be decreased reproductive efficiency, but that is not well documented. In hot, moist areas, shearing and shelter are also essential, but the ability of the heat to permeate even sheltered areas becomes problematic. This is especially true of alpacas engaged in any sort of physical activity. This includes packing, trekking, herd infighting, males excited by nearby breeding females, etc. In addition to the general recommendations, soaks appear to be helpful, but they must be kept stocked with clean water and used by the animals. Kiddie pools and mist sprayers both seem to be effective in delivering enough water close to the skin for cooling to occur.

Examining healthy, sick, or suspect alpacas

Several routine surveillance tools may help you keep ahead of several of these problems. These include routine weighing or body condition score assessments, routine fecal examination and/or membrane color checks, and routine behavioral observations. Eating, moving around, lying down, cud-chewing, defecating, and urinating are all part of the daily routine, and the more you see your animals doing normal things (lying down is the one to be careful of here), the greater confidence you can have that they are healthy. If you fail to see the desired activity, or its frequency appears decreased (or increased, in the case of lying down), single that animal out for closer observation, or do a physical assessment. A variety of problems ranging from emaciation to a rounded abdomen are better assessed by getting your hands on the animal than from afar. If there is still a question, that's where the veterinarian comes in. Blood tests are best when done early in the process when it is still correctable, and may reveal much that is invisible to the external examiner. Imaging studies are another step, but they are best when you know what you are looking for. All in all, your success as an alpaca owner hinges on your ability to minimize the effects of most of the things in this report; individual animals with uncommon disease are another matter, but your ability to keep your herd healthy is the basis on which you build a happy, successful operation.

Adrienne Clarke

Adrienne Clarke began breeding alpacas in 1992. She is tertiary educated, but attributes her alpaca knowledge and expertise to more than 20 years of hands-on involvement with the alpacas. With her husband Chris Williams, she runs *Ambersun Alpacas*, one of Australia's largest alpaca herds of approximately 1,100 animals.

Adrienne has assisted with many birthings in her own herd, and is often called in for help by other breeders. In 2011 she began running informative courses for alpaca breeders to increase their confidence to assist with an alpaca birthing, especially when veterinary assistance was delayed or not available. The courses were well attended and enthusiastically received.

Adrienne is a dedicated member of the SA region, and currently holds the position of Education and Training Officer. Adrienne was a recipient of the Australian Alpaca Association's Certificate of Appreciation for service to the industry. Adrienne is also developing her qualifications for appointment as a judge for the AAA.

Adrienne also has a passion for the breeding of leopard appaloosa alpacas and has been researching the genetics behind the inheritance of this coat colour pattern.

CRIA BIRTHING WORKSHOPS

The birth of a cria is usually a very exciting and pleasurable event. Most alpaca breeders tend to feel most fortunate to be able to witness the birthing process, from the first presentation through to the delivery of a healthy and responsive cria. This pleasurable experience may however, quickly change to apprehension or anxiety if the delivery process fails to advance and the cria is not delivered within the expected time frame.

A small percentage of deliveries do not advance smoothly. Sometimes, with minimal manipulation of the cria, it is possible for the breeder to facilitate the birthing process and as a result, improve the likelihood of survival of both the cria and its dam. Preparedness to offer assistance may be most critical if veterinary assistance is not immediately available.

The workshop addresses the following areas

- The normal presentation of the cria at birth
- The identification of signs of a problem delivery
- A description of the most common cria mispresentations
- The process of assessment

- Strategies for manipulation of the cria to assist with the delivery
- A description of more complicated mispresentations where immediate veterinary assistance is most critical
- Post delivery care of the cria and its dam
- Useful equipment and resources to have on hand.

Using a simulated birthing box, workshop participants will be given the hands-on opportunity to develop and practice their skills in assisting with the delivery of a cria under simulated conditions. Participants are advised that during the workshops, they will be exposed to cria that have died, in order to simulate birthing conditions as close to reality as possible.

The workshop has a proven history of providing alpaca breeders with the confidence of assisting with the birthing of a cria when its presentation at birth is not normal and veterinary assistance may not be immediately accessible.

Professor Peter Cockcroft

School of Animal and Veterinary Science
University of Adelaide

Peter Cockcroft is Professor in Ruminant Health at the School of Animal and Veterinary Sciences, University of Adelaide. His current research interests include the transitional ruminant and targeted disease monitoring and surveillance.

Teaching interests include: clinical diagnostics, evidence-based veterinary medicine and population medicine. Peter has published 4 veterinary books. He is a Cambridge veterinary graduate and previously taught at the Cambridge Veterinary School for 11 years. He participated in the veterinary services which provided primary and referral alpaca consultations.

A CROSS-SECTIONAL SURVEY OF SOUTH AUSTRALIAN ALPACAS: SERUM BIOCHEMISTRY AND HAEMATOLOGY REFERENCE VALUES AND SELECTED DISEASES

This paper presents the outcomes from a cross-sectional survey of 13 alpaca herds resident in the Adelaide Hills in South Australia performed in 2011. This survey was conducted by the academic staff and 10 Veterinary students in partial fulfilment of their Clinical Research Course within Doctor of Veterinary Medicine programme at the School of Animal and Veterinary Science, University of Adelaide.

The scope of the survey included:

- Haematological references ranges
- Serum biochemistry reference ranges
- Prevalence of nematodes
- Prevalence of Faciola hepatica
- Prevalence of Eimeria macusaniesis
- Serological prevalence of Neosporum caninum antibodies
- Serological prevalence of Bovine Viral Diarrhoea antibodies

The information is summarised in 7 abstracts presented below:

HAEMATOLOGY REFERENCE RANGES FOR ALPACAS IN SOUTH AUSTRALIA

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Introduction

The alpaca (*Lama pacos*) is increasingly popular as an alternative production animal in Australia. Although published reference ranges for haematological parameters are available for alpacas, the results from these studies are varied and sample sizes are often too small to reflect true population values. The objective of this study was to determine normal haematology reference ranges for alpacas in South Australia and to investigate differences relating to age and gender.

Methods

Ninety blood samples were collected by jugular venepuncture into EDTA from 8 alpaca properties within the Adelaide Hills region of South Australia. Haematological analyses were performed (Cell-Dyn 3700, Abbott Diagnostics, Illinois, USA) and white blood cell differential counts were achieved using standard methods. Statistical analysis in Microsoft Excel was performed, reference ranges calculated, and paired t-tests were performed to compare data on the basis of age, gender and method of analysis.

Results

Reference ranges for haematological parameters are consistent with findings in similar studies; however, MCHC (396.3 - 518.5 g/L) was considerably lower than previous findings. Monocyte, eosinophil and basophil reference ranges were not in accordance with other studies. Female alpacas had significantly higher MCV and MCH values compared to males ($P<0.05$), whilst adults had significantly higher MCH values ($P=0.03$). Females had significantly higher monocyte and basophil counts ($P=0.03$) and lymphocytes were considerably higher in juvenile alpacas ($P=0.001$). Manual and automatic counts were significantly different for all white blood cells except for lymphocytes, with P values considerably less than 0.05. Table 1, Table 2 and Table 3 provide further details.

Discussion

The haematological reference ranges reported in this study show variations in age and gender for several parameters. Differences reported between similar studies may be due to environment, laboratory techniques, equipment and experience in performing data analysis. Automatic and manual counts are significantly different and so further investigations are needed to determine the cause of this. The results of the present study may be used as a basis for evaluating health in alpacas.

Table 1. Sample population structure according to gender and age

Category		N	Total number of Alpacas
Gender	Males	19	
	Females	71	
			90
Age	Adults	67	
	Juveniles	23	

N= sample size

Adults are animals ≥ 2 years of age, juveniles (including crias) are < 2 years of age

Table 2. Haematological reference ranges determined using an automatic analyser (Cell-Dyn 3700) which includes the mean, standard deviation lower and upper limits (90% confidence intervals) and reported values from two similar studies.

Parameter	Units	N	mean	±SD	Lower limit (90% CI)	Upper limit (90% CI)	Haematological parameters (Hajduk, 1992)[2]	Haematological parameters (Hengrave et al, 2005)[§ 12]
RBC	10^{12}/L	89	11.8	±1.5	**8.1** (7.8-9.4)	**14.5** (14.3-14.6)	11.2-14.4	10.5-15.6
HGB	g/L	89	132.1	±17.1	**100.2** (93.4-108.5)	**167** (164-174)	232-303*	109.6-166*
HCT	L/L	90	0.3	±0.04	**0.18** (0.12-0.24)	**0.35** (0.34-0.36)	ND	0.26-0.37
MCV	fL	89	24.7	±1.9	**21.1** (20.6-21.7)	**28.8** (27.8-29)	26-31	21.5-26.3
MCH	pg	89	11.2	±0.7	**10** (9.9-10.2)	**12.6** (12.2-13)	ND	9.6-10.95*
MCHC	g/L	89	454.6	±27	**396.3** (384-422.3)	**518.5**(509-522)	ND	410.9-464.07*
RDW	%CV	90	26.9	±3.8	**21.5** (21.2-22.1)	**35.2** (33.9-42.5)	ND	ND
PLT	10^9/L	81	1571	±1296.3	**321.3** (245-421)	**5204.6** (4581.6-5760)	ND	ND
PCV	%	90	0.29	±0.04	**0.23** (0.1-0.24)	**0.38** (0.36-0.38)	0.30-0.42	ND
WBC	10^9/L	89	11.9	±3.5	**6.6** (6.3-7.2)	**19.6** (18.6-20.3)	6.0-20.90	7.3-16
Neutrophils	10^9/L	90	8.1	±3.25	**3.2** (2.1-4.3)	**16.4** (14.6-16.9)	2.0-13.30	2.9-9.3
Lymphocytes	10^9/L	90	2.3	±2.04	**0.6** (0.3-0.7)	**6.89** (4.47-7.04)	2.1-6.80	1.1-5.9
Monocytes	10^9/L	88	1.1	±0.9	**0.1** (0.07-0.2)	**4.1** (3.02-4.3)	0-0.50	0.1-0.9
Eosinophils	10^9/L	84	0.3	±0.3	**0**	**0.35** (0.1-0.4)	0-3.0	0.1-3.6
Basophils	10^9/L	88	4.9	±4.9	**0**	**1.8** (1.6-2.0)	ND	0-0.3

* Values have been converted the same units as shown in this study to enable comparison
ND- Haematological parameter not determined in this study
SD- Standard deviation
N=sample size (taking into account removal of significant outliers determined using Reference Value Advisor)
§ Data transformed to reference ranges for all individuals (excluding gender and age differences)

Table 3. Mean haematological parameters in alpacas according to gender and age

	WBC(x 10^9/L)**	RBC(x 10^{12}/L)**	HGB(g/L)**	HCT(L/L)**	MCV(fL)	MCH(pg)**	MCHC(g/L)	RDW (%CV)	PLT(x 10^9/L)	PCV(%)
All individuals	11.96	11.78	132.14	0.29	24.68	11.22	454.62	26.91	1571	29.29
Male	11.57	12.12	133.21	0.28	23.36[a]	10.81[b]	463.22	27.41	2018.10	29.58
Female	10.78	11.68	130.72	0.28	24.70[a]	11.19[b]	452.56	26.70	1475	28.75
Juvenile	12.07	11.75	130.30	0.28	24.69	10.96[c]	456.18	27.64	1442.60	28.74
Adult	10.63	11.66	130.57	0.29	24.20	11.20[c]	452.92	26.73	1426.20	28.67

[a,b,c]: significantly different (t-test) at the 5% level if sharing the same letter

** Significant outliers removed

BIOCHEMISTRY REFERENCE INTERVALS IN ALPACAS (*Lama pacos*) AND COMPARISONS OF OTHER SOUTH AMERICAN CAMELID VALUES

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Abstract

A robust definition of the "normal range" for molecular components of alpaca serum is a useful tool when examining health status in a clinical setting. Although the four South American camelids species are closely related, reference values cannot always be used interchangeably between these species. For this study animals were restrained by an experienced handler and blood was collected by jugular venepuncture from 83 alpacas from 13 semi-rural properties in South Australia and the serum samples were performed by Beckman Coulter AU480 chemistry analyser and Rx Monza analyser for GLDH. Reference values were determined using non-parametric intervals generated by the freeware Reference Value Advisor V 2.0 Add-In for Excel, with confidence intervals for lower and upper limits given at 90%. Data for eight other South American camelid reference intervals were also compared with these results. These data were generally clustered when compared to one another and even though some values out of the 19 analytes investigated were very different from the other values, the remaining intervals closely matched one another. These juxtapositions gave a better indication of healthy South American camelid biochemical values. The aim of this research was to establish a reference interval for clinically healthy alpacas in South Australia to be used in veterinary clinical practice. This interval, along with other published values, will be useful, instructive and meaningful in a clinical setting. Tale 1 and table 2 provide further details.

Table 1. South American camelid serum biochemistry reference intervals for 19 analytes, a comparison between South Australian alpacas (*Vicugna pacos*) (A; n=83, ≥1 year old) from 13 semi-rural properties, and other published values (B–I), including those of other alpacas, as well as llamas (*Lama glama*) and guanacos (*Lama guanicoe*). Data were converted to SI units where necessary. (ALP=Alkaline phosphatase, AST=Aspartate aminotransferase, GGT= Gamma-glutamyltransferase, GLDH= Glutamate dehydrogenase, Mg= Magnesium, K= Potassium, Na= Sodium).

Analyte	Maguran-Pivas *et al.* 2012	Dawson *et al.* 2011	Foster *et al.* 2009	Hengrave *et al.*2005	Simons *et al.* 1993	Lassen *et al.* 1986	Foster *et al.* 2009	Hengrave *et al.*2005	Zapata *et al.* 2003
	A	B	C	D	E	F	G	H	I
	Alpaca	Alpaca	Alpaca	Alpaca	Alpaca	Llama	Llama	Llama	Guanaco
Albumin (g/L)	27.2–45.4	29–43	25–42	28.4–38.6	25–45	23–45	29–50	30.1–40.5	26–50
ALP (U/L)	20.6–196.4	18–113	–	–	35–198	15–121	0–610	–	–
AST (U/L)	143.3–489.8	128–308	137–391	–	65–202	66–235	128–450	–	68–227
Bicarbonate (mmol/L)	17.2–33.7	22–34	–	–	–	–	–	–	–
Bilirubin-total (µmol/L)	0.3–2.5	0–1.7	1.5–7.5	0.2–1.1	0.1–3.2	0–17.1	0–13.4	0.2–1.4	–
Ca (mmol/L)	1.9–2.5	2.1–2.5	2–2.7	2.1–2.5	2.2–2.9	1.95–2.45	1.9–2.7	2.3–2.6	1.2–2.9
Chloride (mmol/L)	105.2–120.6	106–116	97–111	108–146	101–117	105–127	98–120	104–134	–
Cholesterol (mmol/L)	0.5–2.4	0.4–2.3	0.24–2.15	–	–	–	0.34–2.3	–	–
Creatinine (µmol/L)	80–160	88.4–212.2	54–177	97–168	128–288	97.2–300.6	79.5–247.5	121–203	–
Creatine kinase (U/L)	38.2–437.4	29–120	56–662	38–276	9–451	–	0–137	49–663	12.0–540
GGT (U/L)	4.4–42.7	8–35	13–50	–	11–38	12–37	3–28	–	–
Glucose (mmol/L)	3.5–8.6	0.3–8.3	5.1–9.1	5.4–7.3	–	5.38–12.4	4.3–9.9	5.4–7.5	2.7–10.6
GLDH (U/L)	3–25.5	3–19	–	3.4–29.8	–	–	0–25	6–50.3	–
Mg (mmol/L)	0.7–1.1	0.7–1	0.6–1.1	0.8–1.2	0.9–1.5	0.75–1.6	0.8–1.06	0.7–1.2	–
Phosphate (mmol/L)	0.5–3.6	1.1–2.5	0.51–3.12	–	–	–	–	–	–
K (mmol/L)	4.6–9.2	4–5.7	3.9–6.2	4–5.3	4.4–6.5	2.7–7.9	3.6–6.2	4–5.5	–
Protein-total (g/L)	46.3–67.1	5.7–7.2	51–79	56.2–70.4	54–75	50–78	47–73	57.3–72.2	44.0–71.0
Na (mmol/L)	139.1–163	146–155	142–154	148–155.6	141–165	144–168	148–158	147–158	–
Urea Nitrogen (mmol/L)	3–12.3	7.14–21.4	3.9–10.2	4.5–9.7	5.0–12.4	9.28–30.7	3.2–12.8	5.8–11.5	1.5–7.9

Table 2. Comparisons of published sample sizes, ages, statistical analyses and laboratory methods used to determine alpaca (*Vicugna pacos*), llama (*Lama glama*) and guanaco (*Lama guanicoe*) reference intervals as a means of comparing published values against reference intervals generated in this study. RI= Reference Interval; IQR= Interquartile Range.

Reference	Location	Species	Sample sizes	Age of animals	Statistics	Biochemistry analysis
Maguran-Pivas *et al.* 2012	South Australia	Alpaca	72–83	≥ 1 year	RI: Reference Value Advisor v2.0 for Excel (National Veterinary School, Toulouse, France) Non-parametric: Obviously aberrant outliers excluded only	Beckman Coulter AU480 chemistry analyser (2010 Beckman Coulter Inc, Brea, CA, USA)
Dawson *et al.* 2011	Central New York, USA	Alpaca	71	>2 years (Females >7months pregnant or ≤2 months postparturition were excluded)	Analyse-it for Excel 2010 v14.0 (Analyse-it Ltd., Leeds, UK) Shapiro-Wilk test Non-parametric: outliers excluded if obvious or outside x3 IQR RI: from lower 2.5th and upper 97.5th percentiles	Hitachi P modular automated chemistry analyser and an ABL-800 Flex blood gas analyser
Foster *et al.* 2009	UK	Alpaca Llama	60–149 54 females	≥ 6 months	Non-parametric: Outliers, those more than x3 the IQR and above the 75th or below the 25th percentiles were excluded RI: the estimated 2.5th percentile and the 97.5% percentile after outlier exclusion	Various biochemistry analyses, did not identify a particular analyser
Hengrave *et al.*2005	Switzerland	Alpaca Llama	105 112	≥ 6 months		
Simons *et al.* 1993	Victoria	Alpaca	39–326 females (22 for K)	Adults (age not specified)	Microstat (Ecosoft Inc.) used for means and SDs RI: Percentile method (Sunderman 1972)	Cobas Mira UV/visible spectrophotometer automatic analyser (Roche Diagnostics, Indianapolis, IN); Titan III cellulose acetate plates (at Helena Laboratories, Texas)
Lassen *et al.* 1986	Western Oregon, USA	Llama	37–64	1–7 years	Descriptive statistics: mean, SD and range RI: Lowest and Highest values (Min–Max)	Multistat III microcentrifugal analyser (Instrumentation Laboratory Inc, Lexington, MA); Gliford system 103 clinical chemistry analyser (Gliford Instrument Laboratories Inc, Oberlin, Ohio); Model 920M chloride meter (Corning Medical, Medfield, MA)

Zapata *et al.* 2003	Central Chile	Guanaco	40	Not specified, used animals 2 and 3 yrs old	Tested data normality by Kolmogorov-Smirnov test: CK was log transformed Descriptive statistics: mean, SD and range RI: Lowest and Highest values (Min–Max)	Various biochemistry analyses, did not identify a particular analyser

FAECAL SURVEY OF GASTRO-INTESTINAL NEMATODES OF ALPACAS (LAMA PACOS) IN SOUTH AUSTRALIAN HERDS IN THE ADELAIDE HILLS

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Introduction

Gastrointestinal nematodes, which includes worms such as *Haemonchus contortus* , *Teladorsagia circumcinta*, Trichostrongylus species, Nematodirus species, Trichuris species and Oesophagostumum species, may cause parasitic gastroenteritis, and contribute to production loss among many species, including alpacas (*Lama pacos*). Outside of their native South America, the parasites found are usually shared among ruminant species. There has been little study of these parasites in alpacas. While there is currently limited no information on gastrointestinal nematodes of alpacas in Australia, it is thought that the species found will be similar to that of sheep in the same region. The aim of this project was to assess the presence and species of gastrointestinal nematodes in South Australian alpacas.

Methods

Thirteen farms were approached and faecal samples obtained from approximately 15 animals per farm. Faecal egg counts (FEC) were performed by a modified McMaster's method. Samples were then cultured to hatch infective larvae for a week on a per farm basis, to assess the species of nematodes by means of the morphology of the L3 infective larvae. Surveys were also given to the farm owners to assess parasite management practices on farm.

Results

The preliminary results of the faecal studies show that the farms assessed had low average egg counts. The speciation of larvae showed Teladorsagia was the most common enteric parasite found, but Trichostryongylus, Oesophagostomum and Strongyloides were also found.

Discussion

The low egg counts this should be considered in context with the results of the survey undertaken, as drenching regimens may have affected the results. Speciation data for some farms was incomplete because some faeces samples did not have enough samples left after FEC to be cultured, including some of the higher egg counts. Overall, the gastrointestinal nematode species found in alpacas in the Adelaide Hills appear to be similar to those found in sheep.

SEROLOGICAL PREVALENCE OF LIVER FLUKE (*FASCIOLA HEPATICA*) WITHIN THE SOUTH AUSTRALIAN ALPACA (*LAMA PACOS*) POPULATION

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Introduction

Liver fluke infections have not been well documented in alpacas. However it is known that alpacas are susceptible to infection with *Fasciola hepatica*. Transmission occurs through the accidental ingestion of the infected definitive host, a lymnaeid snail. Infections may be acute, presenting with lethargy, paresis and recumbancy, or more commonly as a chronic condition presenting with ill thrift, poor fibre quality and a higher than normal mortality rate. Infected animals will also have extensive fibrosis of the liver leading to further clinical conditions. The objective of this study was to determine the prevalence of F.hepatica infection within a sample of the alpaca population of South Australia.

Methods

Blood and faecal samples were collected from 195 alpacas on 13 farms in the region of the Adelaide Hills area of South Australia. Both faecal and blood samples were tested using separate Bio-X ELISA kits to detect the presence of *F. hepatica* antigens and antibodies. A questionnaire was also used to determine the management practices and health issues currently present on each of the sampled farms.

Results

The results of the ELISA kits showed only one positive coproantigen sample, this sample coincided with the farm which had an identified *F. heptica* egg on faecal egg floatation. There were no positive results from the serum antibody analysis.

Discussion

The results suggest that within the alpaca population of the Adelaide Hills region of South Australia there is little evidence of current or recent liver fluke infections. This is a positive result for the farmers. Further study is required to examine the infection rates at other times of year, and in other more at risk areas of the state.

PREVALENCE OF *EIMERIA MACUSANIESIS* IN ALPACAS (*LAMA PACOS*) IN SEMI-RURAL SOUTH AUSTRALIA

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Introduction

Eimeria macusaniesis is an important pathogenic coccidian parasite that has been shown to cause malabsorptive diarrhoea in neonates and possible secondary bacterial infection in adult alpacas leading to death. This project aimed to determine the prevalence of *E. macusaniesis* in alpacas in semi-rural South Australia. This is the first Australian prevalence study.

Methods

A total of 181 faecal samples were obtained from alpacas from 13 farms. Faecal samples were analysed using a modified faecal float using 1.35 specific gravity saturated sugar solution. They were centrifuged at 2500 rpm for 5 minutes and examined on a slide under a light microscope on the 10x lens. The slide was completely systematically scanned for Eimeria macusaniesis using a scale bar. The parasite was identified by its pyriform shape, dark brown colour, having a prominent micropile a thick wall and measuring 81-107μm long.

Results

Analysis of the slides for *E. macsaniesis* found a prevalence of 3/181 or 1.66% (95%CI 0-3.5%). Other parasites found included other coccidia, Trichuris, Nematodirus, Strongyle eggs, Giardia, a tapeworm egg and Capillaria. One farm found positive for *E. macseniesis* had a sample prevalence of 23% for Trichuris. The positive samples were recovered from three separate farms situated within 26 km of each other.

Discussion

Although the prevalence of this study was 1.66% the true prevalence for E. macusaniesis may be differ. It is possible that some alpacas were not shedding the parasite at the time of collection or that the parasite was not in the faecal pellet used in the faecal floatation method. However, the original aim to determine a presence of the parasite was achieved. The 23% Trichuris prevalence finding for one farm is significant and warrants further investigation.

SEROLOGICAL PREVALENCE OF *NEOSPORA CANINUM* IN ALPACAS (*LAMA PACOS*) IN REGIONAL SOUTH AUSTRALIA

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Introduction

The commercial production of alpacas (Lama pacos) in South Australia has increased over the past 15 years as a result of demand for high quality fleece fibre. However, the high incidence of reproductive losses and infertility reported in alpaca herds is of significant economic concern to the industry. Neospora caninum, a cyst-forming protozoan observed worldwide, has been implicated as an infectious cause of abortion outbreaks in seropositive alpacas in South America, with characteristic parasitic lesions observed in aborted foetal tissues similar to those found in infected cattle. There is currently no data quantifying the prevalence or implications of *Neospora caninum*

infection in South Australian alpaca herds, despite the presence of the organism and severity of clinical signs observed in Australian cattle. The objective of this study was to confirm the presence and establish the serological prevalence of Neospora caninum in commercial alpaca herds in South Australia.

Materials and Methods

A sample of 183 alpacas was randomly selected from 13 herds located in the Adelaide Hills and 20 mL of blood collected from each animal via jugular venepuncture. Serological analysis was performed via IDEXX anti-Neospora enzyme-linked immunosorbent assays (ELISA) in order to detect the presence of antibodies against the protozoan indicative of an immunological response to infection. An initial test was carried out using the supplied cattle specific conjugate and a subsequent modified assay performed using Protein G, a non-species-specific conjugate.

Results

All samples tested were negative for *Neospora caninum* antibodies using both conjugates.

Discussion

These results suggest that there is currently no significant prevalence of *Neospora caninum* in alpaca herds located in the Adelaide Hills. Analysis of herds over a wider geographical range across South Australia and in conjunction with the incidence of infections recorded in cattle may provide a greater understanding of disease prevalence and the implications for reproduction in

A SEROLOGICAL SURVEY OF BOVINE VIRAL DIARRHOEA VIRUS EXPOSURE IN SOUTH AUSTRALIAN ALPACAS

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Introduction

Bovine virus diarrhoea virus (BVD) has recently been identified as an infectious disease that affects alpacas. It is known that alpacas may develop antibodies to the virus after exposure, as well as becoming persistently infected if exposed during the correct times during foetal development. However, while the prevalence of this disease has been found in a number of other countries, the prevalence of BVD has not been determined in Australian alpacas.

Methods

A pool of 182 alpacas were sampled from 13 alpaca farms. All of the properties used were located in the Adelaide Hills in South Australia and ranged from having few alpacas to a fully functioning stud. The serum samples collected were tested using a PrioCheck BVDV antibody ELISA kit.

Results

Of the 182 animals sampled, 5 animals were positive. Of the five animals testing positive, four were from two farms (two from each) and another positive was from a separate third farm. No antigen

test was run to detect persistently infected animals as it was considered to be of little value given the limited positive serological results.

Discussion

The results show that exposure to BVD is present in South Australian alpacas. However, more animals need to be sampled in order to gain a more accurate picture of the disease prevalence in South Australia. A questionnaire could be used to determine the history of the alpacas that have tested positive to BVD virus antibodies. This may indicate any patterns linking the positives which may help to provide the basis for a larger, more comprehensive survey.

Acknowledgement

The authors would like to acknowledge the owners of the alpaca herds who participated in the survey for their patience and assistance during the survey sampling protocols.

Dr Simon De Graaf

Dr Simon de Graaf specialises in the applied reproduction of domestic and wildlife species and is recognised as a world expert in sheep reproduction, sperm sexing and artificial insemination.

He graduated from the Faculty of Agriculture, University of Sydney in 2003 with a Bachelor of Science in Agriculture, receiving first class honours and the University Medal before going on to complete a PhD in 2006 in the Faculty of Veterinary Science, on 'The development of sperm sorting for the sex-preselection of sheep'.

After a brief period as a lecturer in Veterinary Basic Sciences at The Royal Veterinary College, London, Dr de Graaf joined the Faculty of Veterinary Science, University of Sydney as a lecturer in 2009 and was promoted to Senior Lecturer in 2013.

Dr de Graaf has a strong international research profile in the field of applied reproduction of farm and wildlife animals and manages an active research program in subjects as diverse as controlled breeding, sperm sexing, semen cryopreservation and artificial insemination, as well as proteomic and functional investigations of the interaction between sperm, seminal plasma and the female reproductive tract.

His published works include a book chapter on reproduction in sheep, over 20 refereed articles in international journals and presentation of research findings at several national and international conferences. He consults to the Australian artificial breeding industry, including the provision of instructional courses for standardisation of semen assessment, processing and freezing for semen processing centres.

Dr de Graaf is a member of the International Embryo Transfer Society, Society for Reproduction and Fertility, Society for Reproductive Biology, British Andrology Society, Australian Reproduction Trade Advisory Group and the Association of Applied Animal Andrology and currently acts as a reviewer for eight international scientific journals, all ranked as category 'A' by the ERA scheme. He is currently on the editorial board of Animal Reproduction Science (ERA: category A) and the executive board of the Association of Applied Animal Andrology.

He has a passion for developing future research talent to benefit the Australian sheep industry and has attracted outstanding honours, masters and PhD students to his group.

THE DEVELOPMENT OF ASSISTED REPRODUCTIVE TECHNOLOGIES IN CAMELIDS – ESPECIALLY THE ALPACA

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Assisted reproductive technologies have not been well established in camelids due to their unique reproductive physiology. Camelids, including the alpaca, are induced ovulators, meaning they require an external stimulus to ovulate, unlike spontaneous ovulators such as sheep and cattle, which ovulate as part of their oestrous cycle. In camelids, a potent factor termed ovulation-inducing factor (OIF) present in the seminal plasma of the male is deposited into the female reproductive tract during mating (Chen *et al.*, 1985, Adams *et al.*, 2005) inducing ovulation. Additionally, the high viscosity of their seminal plasma and low sperm concentration and motility makes handling and dilution of spermatozoa difficult. Development of cryopreservation of spermatozoa has been hindered by these semen characteristics, as the viscosity of the seminal plasma prevents cryoprotectant agents from reaching the plasma membrane. Recently, the proteolytic enzyme papain has been shown to reduce viscosity without damaging spermatozoa (Kershaw-Young *et al.*, 2013), facilitating renewed attempts to develop cryodiluents for this species.

Five experiments have been conducted to optimise an alpaca sperm cryopreservation protocol, examine the induction of ovulation with β-NGF and validate a novel method for sperm quality assessment.

Experiments 1 and 2 aimed to determine the optimal egg yolk and glycerol concentrations for use in an alpaca cryodiluent. In experiment 3, SHOTOR, INRA-96®, skim milk- and lactose-based diluents were used to dilute alpaca semen prior to freezing and their effects on post-thaw sperm survival were compared. Experiment four aimed to validate a heterologous oocyte-binding assay for the assessment of alpaca sperm fertility and experiment five was a field trial in which the effect of varying doses of β-NGF on the incidence and timing of ovulation, corpus luteum (CL) size and plasma progesterone concentration were compared.

In experiments 1 and 2 semen was collected from four males, diluted with a lactose-based diluent and treated with papain (0.1 mg/mL final concentration) to reduce viscosity. In experiment 1: semen was further extended with lactose diluent supplemented with 5, 10 or 20 % egg yolk and 5 % glycerol, chilled to 5˚C over 2 h and frozen in 0.25 mL straws. The same procedure was followed in experiment 2, except the samples were chilled in the diluent with 5% egg yolk and 2, 5 or 10 % glycerol. After thawing, samples were incubated for 2 h and assessed for motility, plasma membrane and acrosome integrity pre-freeze and 0, 30, 60 and 120 minutes post-thaw.

In experiment 1, spermatozoa were found to have higher post-thaw motility, acrosome integrity and plasma membrane integrity when frozen with 5 % egg yolk compared to 10 or 20 % ($P < 0.001$). The mean decline in motility from pre-freeze to 0 h post-thaw was only 18.0 ± 2.8 % for the 5 % egg yolk treatment. In experiment 2, glycerol concentration did not affect the motility ($P = 0.12$) or acrosome integrity ($P = 0.21$) of spermatozoa post-thaw. However, 10 % glycerol had lower plasma membrane integrity than the 2 or 5 % treatments ($P = 0.002$). These results suggest that papain-reduced alpaca spermatozoa should be frozen in a diluent containing 5 % egg yolk and 2 or 5 % glycerol.

In experiment 3, semen was collected from four males, diluted with each of the four diluents and treated with papain (0.1 mg/mL final concentration) to reduce viscosity. Diluted semen was further extended with each diluent supplemented with 5 % egg yolk and 5 % glycerol, chilled to 5˚C over 2 h and frozen in 0.25 mL straws. After thawing, samples were incubated for 2 h and assessed for motility, plasma membrane and acrosome integrity pre-freeze and 0, 30, 60 and 120 mins post-thaw. DNA integrity was assessed pre-freeze and 0 and 60 mins post-thaw. Spermatozoa diluted and frozen in the lactose-based diluent had higher motility at 0 and 30 mins post-thaw ($P < 0.001$) and higher plasma membrane and acrosome integrity across all time points, than those frozen in the three other diluents ($P < 0.001$). DNA integrity did not vary between SHOTOR, skim milk- and lactose-based diluents, but was significantly lower for INRA-96® treated spermatozoa ($P = 0.001$). These results suggest that a lactose-based diluent is preferable for dilution and cryopreservation of papain-reduced alpaca spermatozoa.

Experiment 4 was the heterologous oocyte-binding assay. Alpaca, sheep and cattle ovaries were collected from the abattoir and transported in Dulbecco's phosphate buffered saline (DPBS; 30°C) to the laboratory. Follicles were aspirated using a 23G needle and 2 mL syringe. Oocytes were placed in DPBS with bovine serum albumin (BSA; 4 mg/mL) and hyaluronidase (1 mg/mL) to facilitate removal of cumulus cells. The oocytes were washed three times in DPBS before being stored in 500 µL of oocyte salt storage solution per 20 oocytes at 5˚C for up to 2 months. Prior to use, they were washed five times in DPBS and placed in 50 µL drops of sperm capacitating medium (CM; sperm TALP plus 10 µg/mL heparin) under embryo culture oil and equilibrated for one hour at 38˚C under 5 % CO_2.

Semen was collected from one adult male alpaca, one bull and one ram using species appropriate techniques. The ejaculates were diluted in sperm TALP and centrifuged at 300 × g for 10 minutes. The supernatant was discarded and the pellets resuspended in CM to a final concentration of 150×10^6 spermatozoa per mL. Spermatozoa were stained with Hoechst 33342 fluorescent stain and incubated for 15 minutes at 38˚C under 5 % CO_2. Two million motile spermatozoa were added to each oocyte-containing drop for coincubation over 1 hour. The 9 treatments included each species' oocytes coincubated with spermatozoa from all three species' spermatozoa in separate drops. After coincubation, the oocytes and their attached spermatozoa were washed twice in sperm TALP before being fixed in 1% formalin and stored in Quench solution. The number of attached spermatozoa was determined by mounting the fixed oocytes onto slides and visualising with fluorescent microscopy.

The results found that spermatozoa from all three species were able to bind to the oocytes of every species. Alpaca oocytes had the largest number of attached alpaca spermatozoa. There was a significant difference between the number of alpaca and ram spermatozoa bound to sheep oocytes, suggesting that sheep oocytes would not be suitable for a heterolologous oocyte-binding assay in alpacas. In contrast, there was no difference between the binding of alpaca and bull spermatozoa to cattle oocytes, suggesting cattle oocytes would be suitable for use in an alpaca zona-binding assay.

This is the first report of an attempt to validate a heterologous zona-binding assay for the in vitro assessment of alpaca spermatozoa. The results suggest that preserved cattle oocytes are a suitable substitute for alpaca oocytes.

Experiment 5 was an investigation of induction of ovulation in female alpacas. This experiment was important as it determined the timing of ovulation. Ovulation in camelids is induced by the seminal plasma protein ovulation-inducing factor (OIF), recently identified as β-nerve growth factor (β-NGF; Kershaw-Young *et al.* 2012). The current study measured the total protein concentration in alpaca seminal plasma using a bicinchoninic acid (BCA) protein quantification assay and found it to be 22.2 ± 2.0 mg/mL. To measure the effect of varying doses of β-NGF on the incidence and timing of ovulation, corpus luteum (CL) size and plasma progesterone concentration, twenty-four female

alpacas were synchronised and treated with; (1) 1 mL 0.9 % saline (n = 5); (2) 4 µg buserelin (n = 5); (3) 1 mg β-NGF protein (n = 5), (4) 0.1 mg β-NGF (n = 5), (5) 0.01 mg β-NGF (n = 4). Females were examined by transrectal ultrasonography at 1-2 hour intervals between 20 and 45 hours post-treatment or until ovulation occurred, and at Day 8 to observe the size of the CL at which time blood was collected to measure plasma progesterone concentration. Ovulation was detected in 0/5, 5/5, 5/5, 3/5 and 0/4 females treated with saline, buserelin, 1 mg, 0.1 mg and 0.01 mg β-NGF, respectively. Mean ovulation interval (P = 0.76), CL diameter (P = 0.96) and plasma progesterone concentration (P = 0.96) did not differ between treatments. Mean ovulation interval overall was 26.2 ± 1.0 hours. In conclusion, buserelin and 1 mg β-NGF are equally effective at inducing ovulation in female alpacas, but at doses ≤ 0.1 mg, β-NGF is not a reliable method of ovulation induction.

The optimised cryopreservation protocol and effective ovulation induction technique can now be combined for application in alpaca artificial insemination programs. Artificial insemination trials using these techniques will be conducted over the coming twelve months with the expectation of producing the world's first birth of a live cria following artificial insemination with frozen semen. Following such successes, it is hoped that artificial insemination will be routinely applied for breeding within the alpaca industry.

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BREEDING PERFECTION

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After 35 years in the insurance & Marketing sector which included 25 years of running his own businesses with 250 staff and spreading to six countries across the globe, Ian turned his attention to one of his early ambitions, farming.

Whilst working in his early years in rural NSW he spent time working with sheep and cattle but ten years ago commenced his affair with alpacas and then started serious breeding and production 6 years ago.

Realising that commercialisation of the Alpaca Industry required total use of the animal, just not fleece & genetics. Ian commenced study of the alpaca meat and hide industry and where it could fit into the food chain. He has worked closely with Melanie Smith, a Phd candidate from Sydney University, RIRDC, and meat scientist Professor David Hopkins of the NSW DPI who combined to commence a Phd study in July 2012

Ian has also worked closely with chefs both internationally and local to develop recipes for this special meat.

Productivity of Alpaca meat now exceeds 20 ton per annum for the domestic market and orders are in hand for a further 38 ton for the export market.

Ian sees a big future for the Alpaca Industry across all three legs.

THE THIRD LEG – BRINGING STABILITY TO THE ALPACA INDUSTRY

Why farm alpaca for meat and hides? Why, because we have come of age and farming alpaca as a commercial sustainable industry is fact rather than fiction, and if we do not then our industry as it is known will not survive on fleece and genetics alone. The doors are now open to us to breed for the future meat industry alongside our fleece, and fleece products, sales of animals within our excellent genetic gene pool whether to existing breeders, new breeders both here at home or globally. The opportunity is there to breed seed stock for future meat breeders thus ensuring an ongoing industry and future prosperity. No longer will you need superannuation paddocks for those animals surplus to your breeding programmes with respect to genetics, show and fleece...now there is a market for

them, not just the diminishing pet or guard animal market, but a market of substance. There is now no reason to have empty females, as the progeny will fill a void This is why the meat and hide industry is so important, because it will become a dominant force in the future of the alpaca industry. But it won't be easy, as there is a science to this industry if we are going to get it right, and NOT A CULLING EXERCISE OF UNWANTED ANIMALS. We must plan for the future, ensure we have the markets and our public have been educated in the acceptance of our product.

We must firstly understand what product we have,before we even think of moving on. We know that the meat is lean.. 5.5 grams fat per 100 grams, i.e. 94% fat free, second only to emu (leave out the ultra lean beef)...but we must also understand the basic nutritional information. So its high in iron, high in protein and very low in cholesterol, so just on the first three its got to be good for you!! Presently we are conducting a PhD study in conjunction with RIRDC and Sydney University, into the qualities, sustainability and acceptance of alpaca meet and our PhD student Miss Melanie Smith will be talking with me today on what the study is about, where we are up to and the exciting results so far...well I think they are exciting.

So we understand the nutrition, and benefits of the meat, it has a taste between lamb and veal, we have dispelled the myth that it is gamey, but we must also understand the cuts from the carcass that our chefs and their patrons desire. The prime cuts are naturally what you would expect. The tenderloins / back-strap, racks, shoulders, short ribs, followed by shanks, neck rosettes, hind quarter for leg smoking and making jerky. Every part of the animal is used, kidneys are to die for and the liver grilled, or made into pate.

We then have to find an approved abattoir with a camelid processing licence. This is not daunting, it's just a matter of the abattoir applying for such a licence if not already having one. The big point is...do they want to do it! and will the volume be worth it. Can they do it on their sheep chain, or do they have to use the cattle chain. This totally depends on the individual abattoir. It is desirable to find such an abattoir as close as possible because the animals can stress on a long journey and have to be rested before slaughter. If they are stressed, like any animal the muscle and fibre tightens and the meat can become tougher..I think Mel will also elaborate on this point in reference to stimulation.

We have a situation where we are 45 mins away from our abattoir, the animals are first cab off the rank at 0700, hung for 24hrs then shipped to our butcher. Average carcass weight is 39/40 kilos which is approximately 59% of live weight. Your butcher should be well equipped with heat shrinking and cryovac etc. which eliminates the last of any bacteria and gives you an added two to three week shelf life in your cool room, resulting in always having stock on hand. If freezing same principal, except you will get minimum twelve month freezer life.

The average carcass will give you approximately:

7 kg of shoulder	3 kg strip loin
8 kg leg	1.5 kg fillet
1.5-2kg shanks	2 kg neck rosettes
3 kg prime mince	7 kg burger/sausage/kofta
2 kg racks(8 cutlets to rack)	1.5 kg short ribs

Now you have your cuts sorted, how about a chef? Highly advisable to befriend a chef and work with him to understand the cooking process and prepare recipes. You will need these recipes as you introduce the meat to other chefs and cooks. The majority will never have cooked or even seen alpaca before, Do not sell alpaca meat to supermarkets or butchers as the end user will not be aware of the cooking process and could end up having a bad experience. Burgers /Sausages by all means to

butchers and delis etc...very hard to ruin them...BUT SUPERMARKETS..NEVER. Actually, there is an excellent market alone in just burgers and sausages, easy to move, a differentiation in product and less hassle. We have one client who between Christmas and New year just past went through 150kg of burgers, nearly four animals in a week. Would you not like 52 clients like that?

Do not be afraid to invest in your chef, and take him to train other chefs. We need as many chefs as possible to enjoy this meat and understand it. Degustation nights are fun, introduce it to the slow food network , they are the foodies. Yes it may cost you a few dollars but a great investment in the future.

Once the word is out you can establish your markets. Have recipe brochures prepared, do sampling invite local press. We already have been on food safari, Paddock to plate in SA. Ambersun who in conjunction with Prados Alpacas run Fleurieu Prime Alpaca and have been on landline and then the ABC distributed the segment through all their cooking shows nationally. Great publicity but even greater were the orders that came through. This market being in its embryo stage is not just going to come to you...it is a business and you have to get off your bottom and make it successful. With a unique product its not really that hard.

Distribution...up to you..contract it out or do it yourself and get to your clients. There are onerous health dept requirements for the transport and sale of meat, so make sure you are aware of them

I mentioned earlier that the meat industry going forward was not about the culling of unwanted animals. We have to build breeding herds specifically for the meat like we have sort out the genetics for our fleece and show animals. There has to be a scientific approach to how we achieve this. We need robust males with excellent confirmation that can carry good condition. We need again robust females that are excellent mothers, produce good cria and have good supply of milk so we can wean earlier than usual and have them pregnant again asap. At present the average dressed carcass is 59% of live weight.. we need to increase that over the next five years to 65%, an automatic increase in bottom line profit 10%.

We need to experiment with nutrition, protein intake.. grasses that we feed the animals. Supplement feeding perhaps of grain without damaging the integrity of the meat (ie increasing fat levels).

If on supplement feeding will the percentage weight gain be greater over a shorter period? will it bring the animals to market quicker? but more importantly will it be cost effective? At present as you are all well aware we have 11.5 months gestation (342 days) and animals go to market at roughly 24 months of age. This means we are always working three years in advance and trying to judge what market will require in volume at the end of this three months. If just six months can be reduced off this timing then we will have made enormous progress and ensured sustainability. This is critical as the export market is waiting for us. We have orders that we cannot fill at present, due to a few hiccups. Firstly finding an export abattoir within close proximity that will process camelids. Secondly animal availability, putting together a 40ft container i.e. 500 carcasses or 20 ton can be done on a one off a basis but if repeat orders came in bi monthly it would hurt our domestic market which you can't afford to do, and therefore we could not guarantee supply and lose all credibility for the future....so we must crawl, before we walk, before we run. BUT IT IS OUT THERE.

A report published by RIRDC in 2001 stated that 11,250,000 kilos of alpaca meat was produced in Peru on an annual basis. That was 13 years ago, so lets imagine this has compounded at 5% per annum over the last thirteen years and now that figure 21,213,552 kilos, That is 21 thousand 213 tons!!

In comparison over a thirteen year period in Australia starting at say just 50 ton, the annual production would be 94.25 ton. easily manageable, just 2356 animals For 200 ton you would need 5000 animals. We can daily breed this. In round figures this would produce $2,800,000 in income on a whole carcass basis at $14 per kilo. On Peru's basis, if they exported you would be looking at $296,000,000. It is interesting to note also that Peru's average carcass weight in 2001 was 23kg, or 57% of the average Australian carcass, so therefore a lot more animals were slaughtered to reach this tonnage i.e. some 450,000, whereas on Australian figures this number would be 281,000. Quite a difference.

Fortunately for Australia, 70 to 80% of these animals were not killed in licensed abattoirs and used for local consumption, which gives us a huge advantage and opens the export market up for Australia. Hides wholesaled at $250 each would, on the 5000 alpacas for export, bring in a further $1.25m. The leather products from alpaca that I have seen so far have been magnificent, and cover, handbags, purses, mens wallets, gloves etc. Whilst we have not had time at this stage to venture into this area, it's certainly on the horizon.

So why farm alpacas for meat and hides?

Because it's the Third Leg ..The stool stays upright and we are finally looking at a financially sustainable commercial rural industry.

Remember in years to come we will always be farming two types of alpacas. Those that come with Pedigrees, and the others will come with Recipes.

Thanks ..will hand you over to Melanie we will both field any questions...and make sure you have some!!

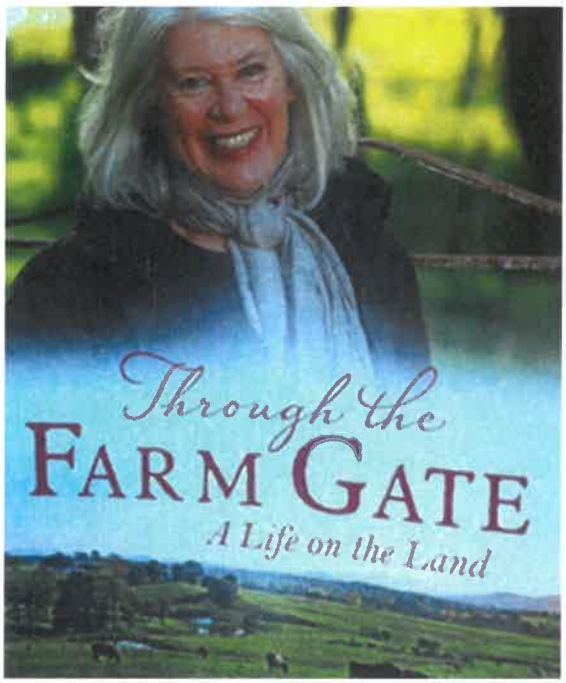
Melanie Smith's presentation can be found later in the proceedings.

Angela Goode

Angela Goode lives on a farm near Naracoorte, South Australia, where cattle graze and bush stone curlews call. She has spent her writing life bringing the images and issues of rural Australia to a wider audience in books, newspaper columns, and on radio.

Between 1981 and 2008 she wrote a country column for the Advertiser in Adelaide. The many editions of Great Working Dog Stories and Great Working Horse Stories, alongside For Love of the Land, celebrate working lives beyond the cities - where animals and humans are expected to pull their weight. In 2008 she was inducted by Rural Media South Australia as a Rural Media Icon.

THROUGH THE FARM GATE – A LIFE ON THE LAND



In December 2013 Angela released 'Through the Farm Gate A Life on the Land', told with wit and honesty this is a heart warming memoir that reveals the pain, the joys, the fears, dedication and complexity of life on the land.

Angela spent many years in the saddle-mustering buffalo, trail riding on bullock tracks, hunting, jumping logs, and sometimes falling off. Her horses know little about hard work these days.

Angela has been nominated for the Women in Australian Agribusiness 100 list.

Dr Laura Hardefeldt

BSc BVMS DACVIM (Large Animals)

Laura graduated from Murdoch University in 2003. She worked in mixed practice in Northern Victoria and then in Gippsland for 2 ½ years before completing an internship in Equine Medicine and Surgery at Sydney University. Following this, Laura successfully completed a 3 year residency in Large Animal Internal Medicine at the University of Wisconsin Madison.

It was here that she developed an interest in Camelid medicine, with a significant portion of the caseload being Alpacas and Llamas. After becoming a specialist, Laura returned to Australia and worked at Adelaide University as a Lecturer in Equine Medicine for 1 year. For the past 2 years she has managed the medicine department at Goulburn Valley Equine Hospital. After having 4 months overseas in 2013, Laura now runs a consulting service - Equine Medicine Consulting, but don't let the name fool you, she is still very interested in Camelid medicine!

NEONATAL CARE OF ALPACAS

Normal neonatal cria behaviour:

To recognise abnormal, a thorough understanding of normal cria behaviour is required.

Behaviour: The normal cria should stand and nurse within 2-4 hours and usually within 30-60 minutes. Colostrum is essential as the first feed, with ~80% of absorption occurring in the first 6 hours. Crias should nurse 1-4 times per hour, and typically nurse for a very short period of time each feeding.

Birth weight: Alpacas >5.5kg

Weight gain: 125-250g/day

Teeth: 6 erupted incisors

Day 1 routine procedures:

Weigh

Examine dam

Vital signs from cria – temperature, pulse and respiration rate

Check for congenital defects

Check teeth erupted

Dip umbilicus – 0.5% chlorhexidine

Day 2 routine procedures:

IgG testing

Other routine procedures:

Selenium deficient areas – Selenium supplementation

Crias born in winter, and those with dark fibre – Vitamin D supplementation

1500-2000 IU/kg once only (NEVER MORE THAN EVERY 2 MONTHS)

The most common problems occurring in neonatal crias (<2weeks) are prematurity / dysmaturity, sepsis following failure of passive transfer (failure to consume colostrum), and congenital abnormalities. Critically ill crias frequently require intensive management and the small size of crias adds a tier of complexity. Referral to a hospital that is able to offer 24-hour care and additional diagnostics should be strongly considered for valuable animals.

Clinical exam:

Historical information is very important. Specific questions that I always ask:

- Length of gestation (normal 330-352 days)
- Health of the hembra during pregnancy
- Number of previous pregnancies and the health of the crias from those pregnancies
- Duration of parturition
- Crias behaviour immediately after parturition
- Signs of disease
- Medications already administered

Crias are classified as "high risk" if:

- The dam has a history of sick crias from previous pregnancies
- Hembra sick during pregnancy
- Prolonged or short gestation
- Dystocia
- Meconium staining

A thorough physical examination should also be performed. Normal vital signs are listed below. Care should be taken to assess for any congenital defects such as:

Congenital defects	Presenting syndromes
Choanal atresia	Respiratory distress
Cleft palate	Milk coming from nose
Umbilical hernias	Incomplete body wall around umbilicus
Atresia ani	Failure to pass faeces, no rectum, colic
Atresia vulvi	Failure to pass urine, no vulva, colic

Congenital heart defects are also common, and may only be detected following examination by a veterinarian.

Pale, bright red, or purple mucous membranes (gums, vaginal walls, conjunctiva of eye), cold extremities and high or low temperatures are suggestive of sepsis in neonatal crias and should prompt examination by a veterinarian.

Premature/dysmature animals frequently will have tendon laxity, small body size and failure of eruption of incisor teeth.

Normal Vital Signs for Neonatal Crias

	Heart rate	Respiratory rate	Temperature
Neonatal crias	70 – 120bpm	20 – 30bpm	37.5 – 38.9°C

Common reasons for recumbency

- Low blood sugar
- Low blood pressure
- Acidosis (high acid level in blood)
- Electrolyte abnormalities
- Muscle disorders or fractures

Before a veterinarian arrives:

HYPOGLYCAEMIA: Rub honey on gums

SEVERE DEHYDRATION: Offer milk or electrolytes by bottle or tube feed. Sterile fluids can also be given under the skin if resources are available.

ELECTROLYTE ABNORMALITIES:

Electrolyte abnormalities are relatively common.

High sodium (salt) level in the blood is called hypernatraemia. In crias it is often found in combination with high blood sugar levels. Crias of all ages can be affected, and typically present with a dull demeanor, wide-base stance and an array of other signs (usually attributed to the primary disease process). Treatment revolves around resolving the high blood sugar levels with insulin.

Treatment of the primary disease is also important to address the stimulus for cortisol driven hyperglycaemia.

FAILURE OF PASSIVE TRANSFER

Failure of passive transfer (FPT) can occur for a range of reasons; failure of the dam to produce adequate colostrum, loss of colostrum or failure of the cria to nurse in the neonatal period. Agalactia (failure to produce milk) does occur in hembras, although seems infrequent. Premature lactation does not appear to be a problem in this species, however loss of colostrum often occurs when a previous cria is not weaned in a timely manner. The most frequent cause of FPT however, is failure of the cria to nurse.

Absorption of colostral antibodies by the gut is maximal at birth and starts to decline almost immediately. This process is hastened by the presence of anything within the mouth. In foals, there is ~80% decline in ability to absorb antibodies by 8 hours of life. This appears to be similar in crias, where nursing in the first 4-6 hours is most important.

Immunoglobulin G (IgG) measurement is only available by rapid immunodiffusion(RID), which takes ~12 hours to run. Total protein is not a good indicator of passive transfer in crias. Routine measurement of IgG is strongly recommended, especially for valuable animals. It is widely accepted than crias require an IgG level of >1500mg/dL to have a protective effect against neonatal sepsis.

FPT can be treated via plasma or whole blood transfusions. No commercial source of plasma exists in Australia for camelid plasma. Whole blood can be collected from an on-farm donor, hung (refrigerated) to allow gravity sedimentation of RBCs and then plasma decanted. Gravity sedimentation is recommended for 48 hours. Crias typically require 300-600ml of plasma to adequately address FPT. Given a typical PCV of 30%, 3 times as much blood needs to be collected as plasma required. It is generally safe to collect 1L of blood from a large adult male macho in good body condition, however heart rate and demeanor should be closely monitored during blood collection, and fluid support administered as required.

Whole blood can also be administered, and is less time consuming to process. However, administration of 1L of whole blood rapidly to neonatal cria will cause fluid overload, so slow administration is necessary.

Blood or plasma can be administered intravenously through an IV catheter, and this is the recommended route. The first 5 minutes of the transfusion should be slow, and the cria monitored for an adverse reaction, however these seem rare in camelids. Fluid rate can then be increased with the remaining transfusion administered over 1-2 hours. This time frame is often not practical for ambulatory practitioners, and plasma is administered intra-peritoneally. Using this method, the entire volume can be deposited into the peritoneal cavity rapidly. Whole blood **cannot** be administered via this method. Plasma is typically instilled through a catheter placed into the abdominal cavity in the right flank. Risks associated with this method include inadvertent puncture of the gastrointestinal tract, infection and colic, due to increased pressure.

SEPSIS

Sepsis is relatively common in neonatal crias, and also occurs in older crias. The infection is likely introduced through the gastrointestinal tract, however the umbilicus and respiratory routes are also possibilities. Crias usually present with signs of toxaemia (red/purple mucous membranes, cold extremities, high heart rates, high or low temperature), high or low blood sugar and dehydration.

Risk factors include failure to nurse, or other factors contributing to FPT, prematurity or dysmaturity, dystocia and diarrhoea. There has been 1 retrospective study looking at sepsis in crias. Crias should be treated with broad-spectrum antibiotics. I typically use ceftiofur at 5-10mg/kg q6-12 hours. Other treatments include fluid support, anti-endotoxic therapy and anti-inflammatories.

Blood analysis is useful in evaluating cases. In the retrospective study already mentioned, WBC count was not useful in diagnosing sepsis in crias. However, in a cohort of crias treated for sepsis at the University of Wisconsin by myself, low white cell count and immature white cells in circulation were both associated with sepsis (unpublished data). Blood cultures are often positive, and may allow more targeted antimicrobial therapy and detection of resistance.

NEONATAL ENCEPHALOPATHY

Neonatal encephalopathy is a syndrome of:

MILD — Loss of affinity for dam

Poor ability to find udder

Loss of suckle reflex

Recumbency

SEVERE — Seizures

Crias appear relatively resistant to neonatal encephalopathy, possibly due to their inherent resistance to hypoxia (low oxygenation). However, long births, and prolonged hypoxia, can lead to signs of neonatal encephalopathy. Crias typically present with lack of affinity for the hembra, poor ability to locate the udder, and, if not treated, dehydration and sepsis.

Similar to other species, prognosis is good if appropriate nursing care is instituted. A naso-oesophageal feeding tube can be placed, and alpaca milk/colostrum administered. A normal cria will nurse 10% of its body weight per day in milk by 4-5 days of age. I typically start with 6% on day 1 and increase by 1% per day until reaching maximal feeding, or until the cria starts to nurse on its own. Daily milk needs should be administered over 12 feeds (every 2 hours) for the first week of life. Prophylactic antibiotics should also be administered, as should anti-inflammatories. The prognosis is usually good, assuming that no secondary problems develop.

PREMATURITY/DYSMATURITY

Prematurity and dysmaturity are common in neonatal alpacas. Signs of non-readiness for birth include failure of the incisors to have erupted by birth, tendon laxity, and excessive curling of the ears due to soft pinnae cartilage. Crias often have trouble nursing due to weakness, and therefore are prone to FPT, dehydration and sepsis. Respiratory failure is uncommon and prognosis is good as long as nursing care is pursued. Placement of a naso-oesophageal feeding tube, treatment for FPT and prophylactic antibiotics are all recommended.

NEONATAL DIARRHOEA

Crias can develop diarrhoea from a number of aetiologies.

Bacterial	**Age group affected**	**Specific treatment**
Clostridium perfringens	All	Oral penicillins
Salmonella spp	All	None
Viral		
Rotavirus	7 days – 3 months	None
Coronavirus	7 days – 5 months	None
Bovine Viral Diarrhoea Virus		None
Protozoal		
Crytosporidium	1 week – 4 months	None
Giardia	2 weeks – 5 months	Fenbendazole
Eimeria	3 weeks – 4 months	Amprolium
Miscellaneous		
Sepsis	0 - 2 weeks	See elsewhere
Nutritional causes	All	None
Antibiotic induced	All	None

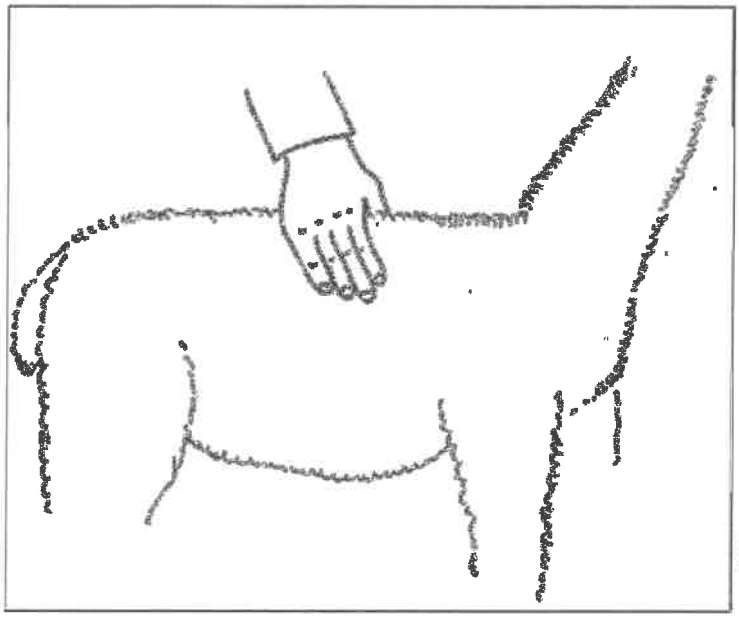
Clostridial diarrhoea is frequently associated with colic and rapid onset of toxaemia in crias of any age. Rota and coronaviral diarrhoeas typically occur in crias starting at 7-10 days, but more typically in those 2-3 weeks of age. Similarly, the protozoal diarrhoeas are more common in slightly older animals. Sepsis is also a common cause of diarrhoea, usually secondary to endotoxaemia.

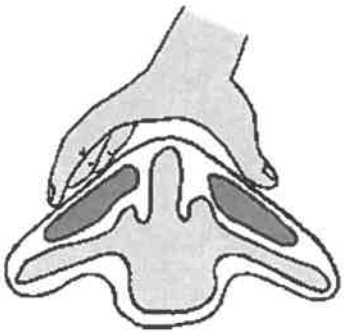
Nutritional causes of diarrhoea are also common and include feeding inappropriate milk replacer or overfeeding milk, and forestomach acidosis in older animals.

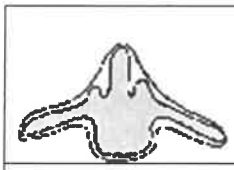
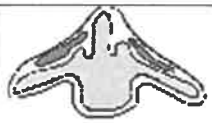
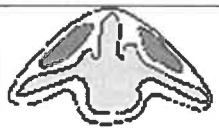
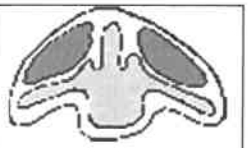
WEIGHT LOSS & ILL THRIFT IN YOUNG CAMELIDS

Camelids are commonly presented with a complaint of ill-thrift or failure to gain weight. Often owners will report a sudden onset of weight-loss, however lack of recognition of weight loss by farmers is common. The fleece type of alpacas makes observational assessment of body condition difficult, and unless owners are handling animals frequently, early weight loss may go unrecognised. For this reason, animals are often presented in very poor body condition.

Owners are encouraged to body condition score their animals at regular intervals using a standardised scoring scheme such as the one shown here





				
1 – Very thin, severely concave between ribs and vertebrae	2 – Moderately thin, slightly concave	3 – Good condition	4 – Overweight, slightly convex	5 – Obese, top of back is almost flat

*Australian Alpaca Association, 2008

Physical Examination

Physical examination is very important in the evaluation of ill thrift. Particular attention should be paid to dentition, presence or absence of bruxism, mucous membrane/conjunctival colour, and vital signs. In addition an evaluation of appetite should be made.

Normal Vital Signs	**Adult**	**Cria**
Temperature	37.5 – 38.3°C	37.5 – 38.9°C
Heart rate	60 – 90 bpm	80 – 120 bpm
Respiratory rate	10 – 30 bpm	20 – 40 bpm
Ruminations	2 – 4 per minute	NA

Differential diagnoses

Inadequate nutrition:

Weight loss in the face of a good appetite should always prompt the exclusion of inadequate caloric supply. Animals suffering from inadequate nutrition typically maintain a good appetite until terminally ill. Absolute starvation is fairly rare, however undernutrition is more common. Typically energy, protein, or both are deficient in the diet over a period of time. Body weight loss and a decline in body condition score are the most common finding. Growing animals may also show stunted growth. Pregnant and lactating females, and growing animals, may be prone to hepatic lipidosis (fatty liver).

Physiological conditions such as lactation, late term pregnancy and growth are commonly identified as requiring additional nutrition but other factors are likely to contribute. Environmental conditions may alter nutritional needs. Data from other species would suggest that for every 1°C increase or decrease from an animal's critical temperature increases energy demand by 1%. If animals are wet & exposed to wind/snow energy demands may be increased by as much as 75%.

Assessment of nutritional management should include stocking density, forage quality and supplemental feeds.

Vitamin D Deficiency (Rickets):

Camelids seem to be different compared to other ruminants in regards to vitamin D physiology. Alpacas between 3 and 6 months are seemingly susceptible to seasonal vitamin D deficiency resulting in a hypophosphataemic rickets syndrome. Vitamin D is required to facilitate intestinal absorption of phosphorus. Crias born in late autumn and winter are at highest risk, and those with black fibre also seem to have a higher prevalence. Vitamin D is synthesized in non-pigmented areas of skin upon exposure to sunlight, therefore more darkly coloured and heavily fleeced animals are at greater risk.

Clinical signs include decreased growth rates, reluctance to move, humped back stance and shifting leg lameness. On physical examination, joints are often enlarged with the carpus (knees) often the most severely affected. Xrays can be helpful in confirming the diagnosis and blood phosphate levels less than 3.0mg/dL is consistent with a diagnosis of Rickets.

Administration of vitamin D supplementation is both therapeutic and preventative. Parenteral administration of 1000-1500 IU/Kg maintains adequate vitamin D levels for up to 3 months. Oral dosing can also be successful but requires more frequent administration. Excessive vitamin D can be toxic, so care must be taken when treating animals. Toxic doses leads to acute kidney failure, which can be fatal.

Nematode Parasitism:

Parasitism in Camelids is a major health concern throughout the world. Growing populations of drug resistant, and often multi-drug resistant, parasites are challenging our ability to manage this problem.

Of the trichostrongyle-type nematodes, *Haemonchus contortus* is frequently incriminated in causing disease. Animals present with a history of weight loss and diarrhoea and are often anaemic. Due to the higher affinity of hemoglobin for oxygen in this species, the affects of chronic blood loss are not as clinically obvious until severe. This physiological adaptation to high altitudes reduces the need for blood transfusion in response to blood loss and many Camelids will tolerate a moderate anaemia remarkably well.

Other trichostrongyle-type nematodes can also cause disease. *Ostertagia, Teladorsagoa, Trichostrongylus* and *Cooperia* have been identified in the faeces of diseased animals. Animals tend to be unthrifty and may be anorexic with poor growth. Diarrhea is usually the most obvious clinical sign, but may be absent in early disease.

Other speakers at this conference will present further detail on this topic.

Coccidiosis:

Camelids appear to be susceptible to at least 5 species of *Eimeria*. These parasites appear to be camelid-specific and so transmission from other ruminants and wildlife is probably not important. In many species, coccidiosis is considered a disease of young animals, however in camelids adult morbidity and mortality is well recognised.

Other speakers at this conference will present further detail on this topic.

Other parasites:

Liver fluke: Camelids are susceptible to several species of fluke, including *Fasciola hepatica* and *Fasciola magna.* Animal typically present with ill-thrift, however blood analysis typically indicates liver involvement (increase in GGT, bilirubin).

Chronic infection:

Ill-thrift is a common presenting complaint for animals with chronic infections. Pneumonia can be bacterial or viral. Nasal discharge and coughing are uncommon, and mild changes in respiratory rate and effort often go unnoticed in animals with a heavy fleece. Auscultation can be useful in making a diagnosis, as can ultrasound, although presence of the fleece can make ultrasound challenging. Chest xrays can also assist in making a diagnosis of pneumonia. Treatment usually involves broad-spectrum antibiotics and anti-inflammatories.

Endocarditis (infection in the heart) can also cause ill-thrift. Endocarditis has been reported in a range of ages, however it tends to occur in younger animals, possibly following infection in the neonatal period. Animals tend to present recumbent and have a high heart rate (with a murmur) and high respiratory rate, however fever is not a consistent clinical finding. Signs of right-sided heart failure are often present, also.

Johnes disease has been reported in Australian alpacas but does not appear to be widespread. Unlike this disease in cattle, animals less than 2 years of age are frequently diagnosed. Animals typically presented for weight loss and ill thrift. Diagnosis can be made by PCR and culture, although the same issues arise with diagnosis as those found in other species. Necropsy typically reveals greatly enlarged mesenteric lymph nodes. Johnes is a reportable disease.

Persistent infection with Bovine Viral Diarrhoea Virus is a known cause of ill thrift (with or without diarrhea) in the US. The prevalence of disease has not been established in Australia, however widespread infection of the bovine population makes the presence of disease very likely in Australia. Among >60 herds tested in the US, 25% had seropositive crias and 4 herds had persistently infected crias. In addition to ill thrift, infertility, abortion, and congenital abnormalities have been reported. Testing procedures are similar as those used for cattle.

Third compartment ulcers:

Alpaca forestomachs compose 3 compartments. C1 is the largest, holding 83% of the stomach contents (15-25L). C2 holds ~1-2L (6%), however C1 and C2 contents intermix freely. Bicarbonate is secreted in the ventral sacules of C1 and C2 to buffer the acidity in these compartments. C3 is a tubular structure, the proximal ¾ of which has glandular epithelium and the distal ¼ is acid secreting. Very little is known about protective mechanisms of the lining of the compartments, although it is presumed to be similar to other species. Ulcers occur mostly in the acid-secreting zone of the distal C3 and the proximal duodenum. The incidence of third compartment ulcers (TCU) appears to be relatively high in the US (3.3-5.5%) and anectdotal evidence would suggest a similar incidence in Australia. Stress is probably the most important factor in the development of TCU in camelids. Glucocorticoid and NSAID use likely contribute. In my experience ulcers are usually secondary to physiological stress from another disease.

Other speakers at this conference will present further detail on this topic.

Perennial ryegrass staggers:

Neurological disorder caused by ingestion of a toxin produced by a fungus that grows on perennial ryegrass. Phalaris has also been incriminated as a cause. Disease is most common between January and March. There is strong evidence from overseas and limited Australian research to suggest that perennial ryegrass toxins, while not necessarily causing visual symptoms of staggers, can cause subclinical disease, significantly lowering feed intake, causing ill thrift and leading to lowered production and reproduction.

Neoplasia:

Cancer is relatively common in alpacas, however older animals are more often affected. Lymphoma can affect young animals, with animals <1 year of age often diagnosed.

Cameron Holt

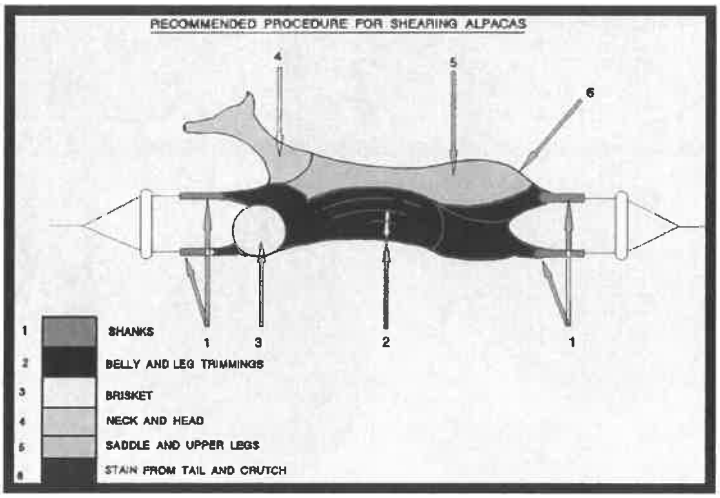
Cameron Holt, a leading alpaca fibre expert, has had some 50 years in the fibre industry, namely as a wool broker, sheep classer, judge, and educator. He first became involved with the Australian alpaca industry when working at the Melbourne Institute of Textiles. Because of his background and long association with wool, mohair and cashmere industries, Cameron was asked by the founding fathers of the Australian Alpaca Association to develop an education program and to advise and set up standards for both the testing and judging of alpaca fleece. Because there was little written about Alpacas in modern times, knowledge needed to be obtained, so a research program commenced in 1990.

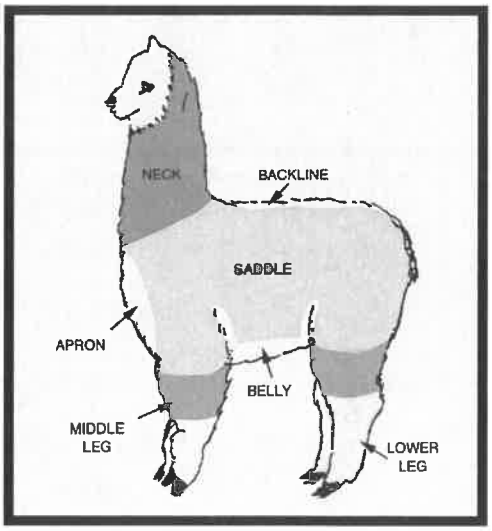
Cameron became Head of the Wool and Fibre studies department of R.M.I.T. University School of Textiles, where he was responsible for the delivery of the training for the Wool and allied fibres Industries. In 2012 Cameron was honoured for his work to the Alpaca Industries in Britain and Australia with Life Memberships to their organisations. In June 2013, he was awarded the "AOBA Distinguished Judge Award" to recognize the lasting and significant contributions to the alpaca industry he had made which was well beyond general expectation.

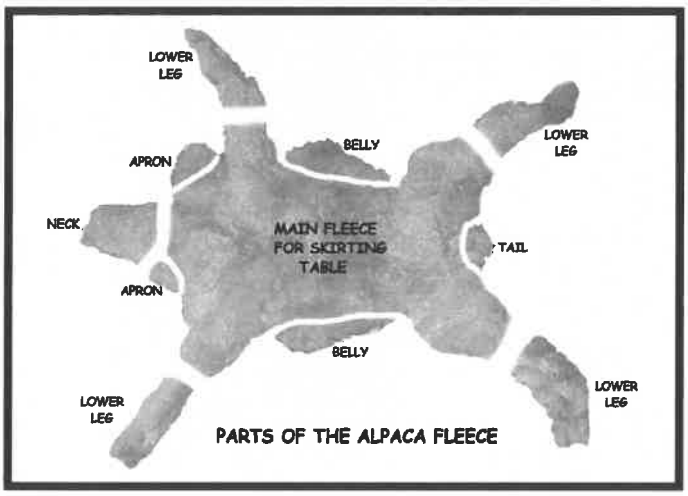
Cameron, continues to travel globally, judging for various alpaca groups. He also, along with judging, is currently training Judges for a number of International Alpaca Associations.

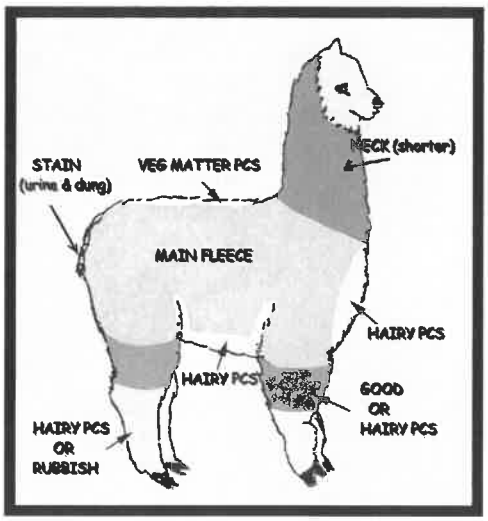
Cameron still continues his educational clinics , lectures and conference speaking throughout the world. In his semi retirement of grandchildren, golf, fishing and community support, still manages to find time to continue with publications and research into alpaca fibre characteristics and allied areas.

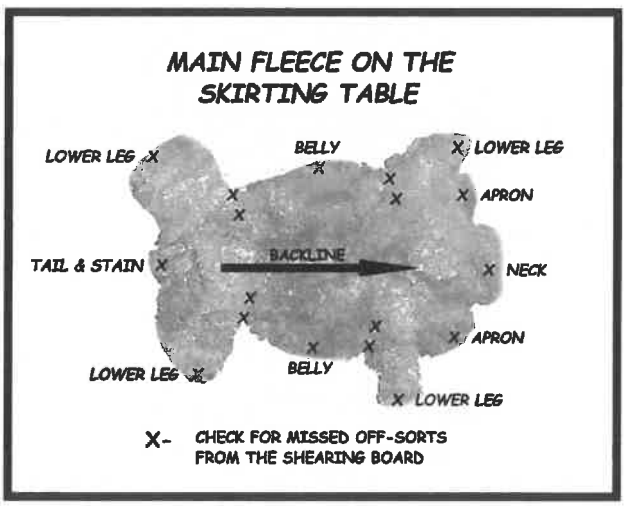
ALPACA SHED SORTING AND CLASSING







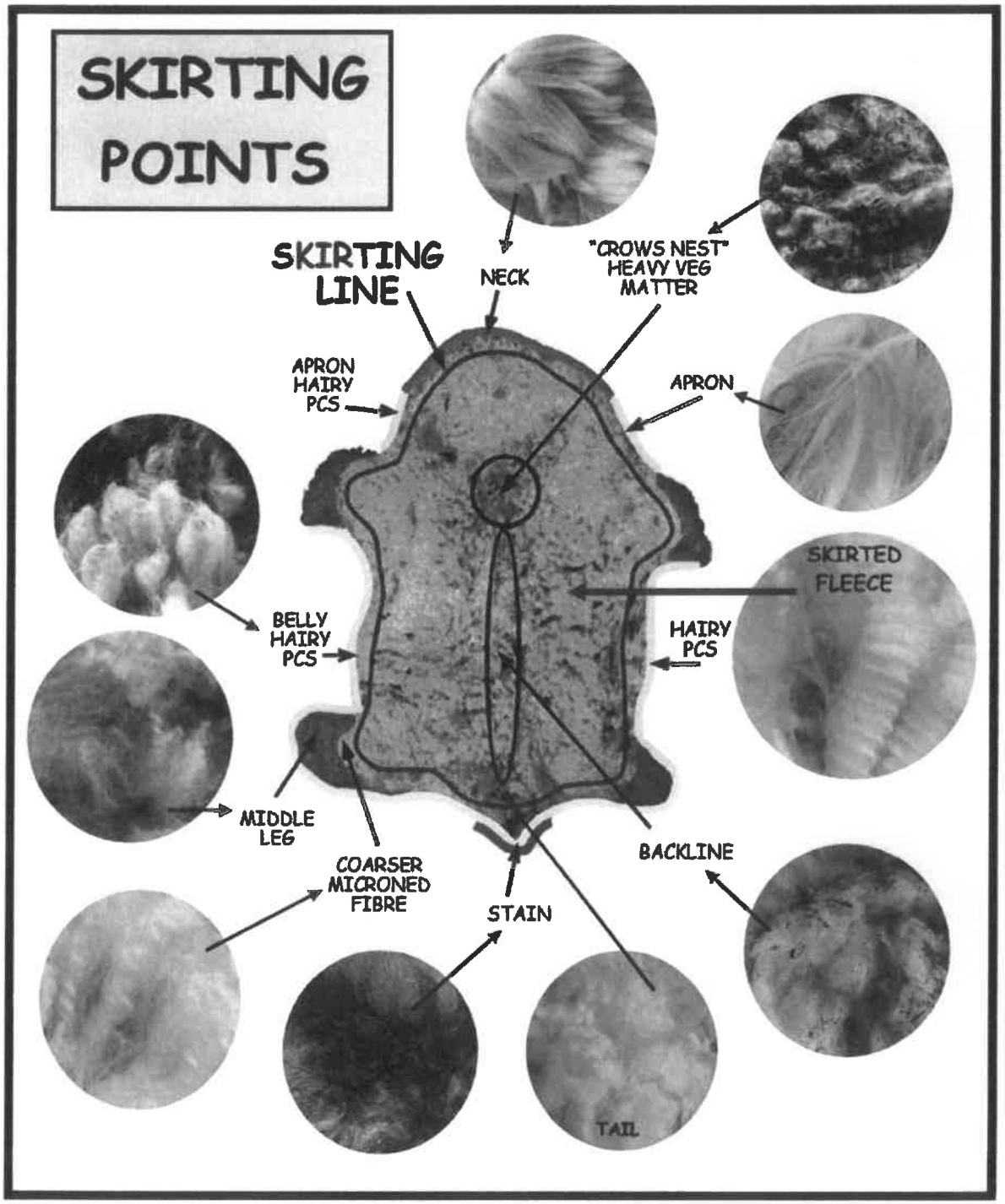




ODDMENT OFF-SORTS

- Neck Fibre
- Good PCS
- Good PCS VM
- Hairy PCS
- Heavy VM
- Shankings, stain and sweepings from floor
- Overgrown.

HUACAYA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SKIRTING LINE
NECK
"CROWS NEST" HEAVY VEG MATTER
APRON HAIRY PCS
APRON
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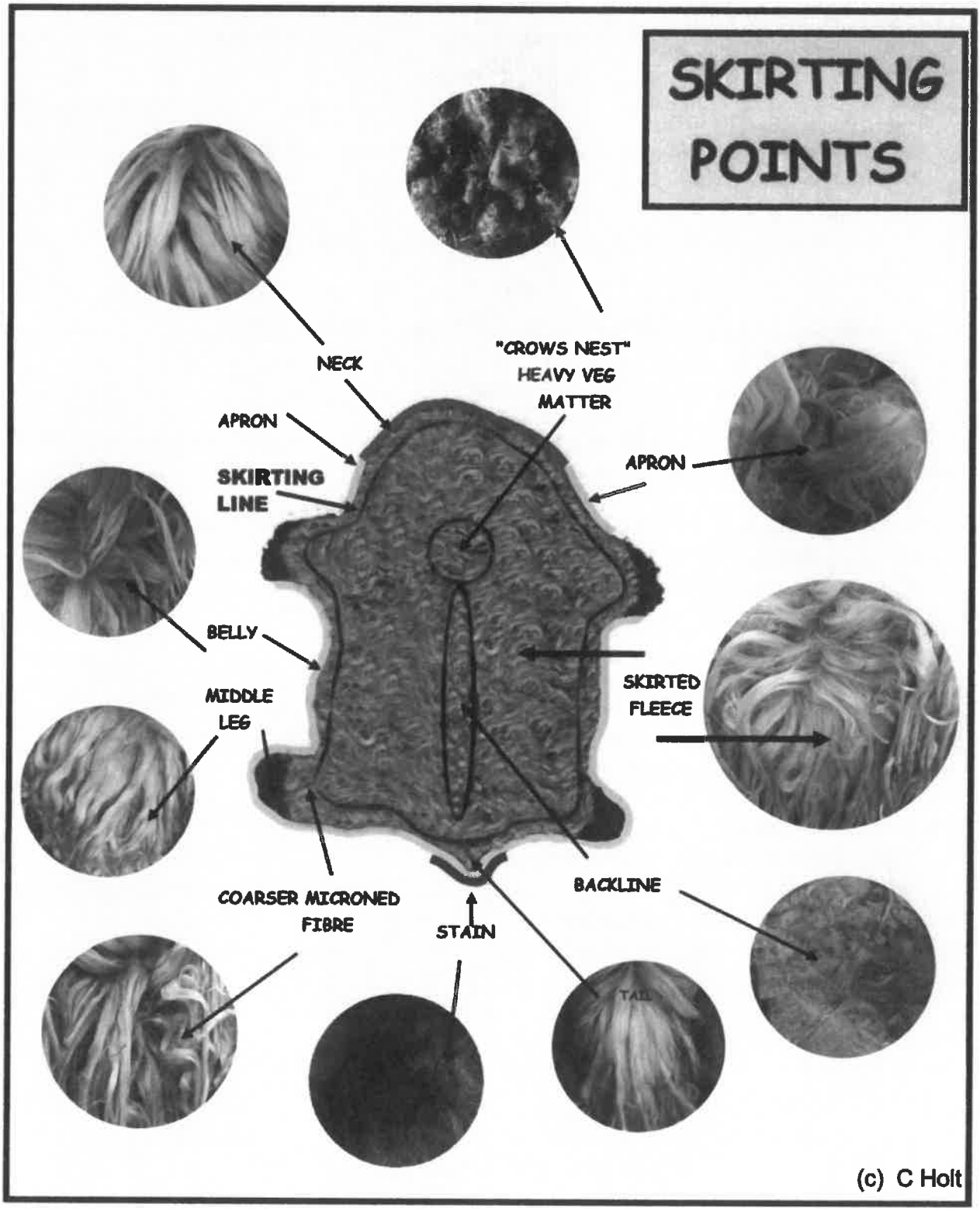
BELLY HAIRY PCS
HAIRY PCS
MIDDLE LEG
COARSER MICRONED FIBRE
STAIN
BACKLINE
TAIL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SKIRTING LINE
BELLY
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MIDDLE LEG
COARSER MICRONED FIBRE
STAIN
BACKLINE
TAIL
(c) C Holt

Alpaca Classers

Apart from the qualified Wool teachers, who were trained to teach the alpaca fibre classing course, a small number of wool classers have been trained as alpaca fibre classers and are now eligible to become registered alpaca classers with AWEX.

Cluster groups

A group of like minded alpaca breeders that together to class there their fibre. There are two main possible groups;

1. To send to a recognised Broker to have them class your groups fibre and sell it on their behalf, or

2. Form their own group, acquire a facility and employ a classer (see later).

Fleece Collection

In NSW arrangements have been made with Schute Bell – Wool Brokers, Yennora Wool Sales – to conduct a trial classing and interlotting of alpaca.

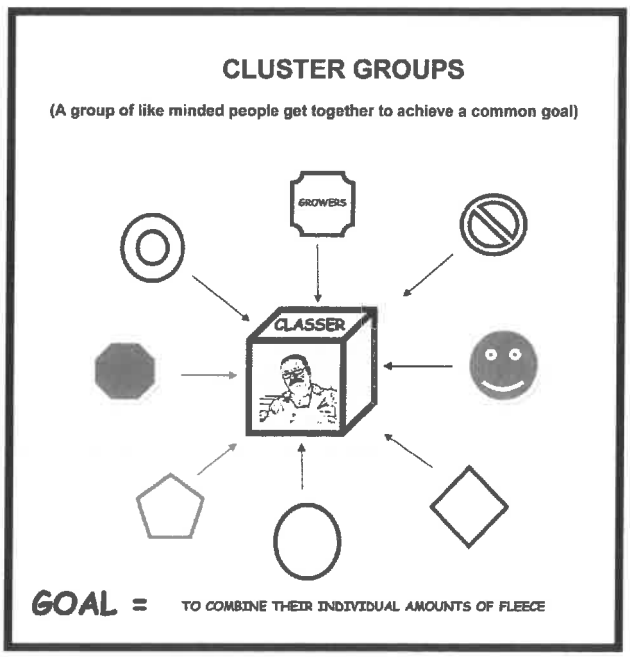
One of their operations is to interlot wool for small growers and sell through the wool auction system. Once classed, correctly branded, core tested and catalogued the fleece will be offered for sale through the auction system, unless sold by private treaty beforehand. Reserve prices have been established as is the norm for wool.

Primaries, Wool brokers from Western Australia have over the last two years sold alpaca fibre through their wool auction system, with the support of an Australian Wool Testing Authority certifying test. Some of this alpaca fibre was direct from growers and some from a cluster group, where the fibre was pooled together by various breeders.

There is no reason why other alpaca breeders cannot form cluster groups for the sale of their fibre. It only requires a group of breeders who know each other or might live in a similar area to get together and employ a registered alpaca classer to carry out the interlotting/blending of the fibre.

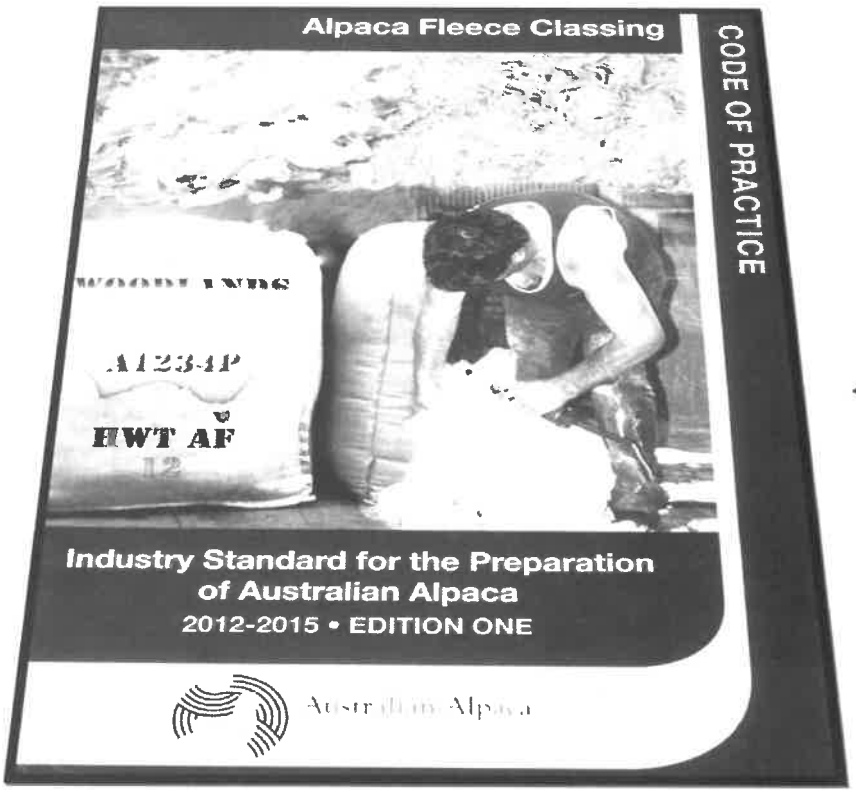
Before starting, the participants would need to decide on how the fibre is to be disposed e.g. to private buyers, directly to a processor or in the public arena at auction by one of the brokers selling alpaca fibre through the traditional auction system.

They would need a trading name (and bale "farm brand") and tax number etc.



Quality Assured

The Australian Alpaca Association is looking at licensing greasy fleece and products manufactured in Australia using a QA classing standard (Australian Alpaca Quality Assured).



CONTACT; A.A.A.L.

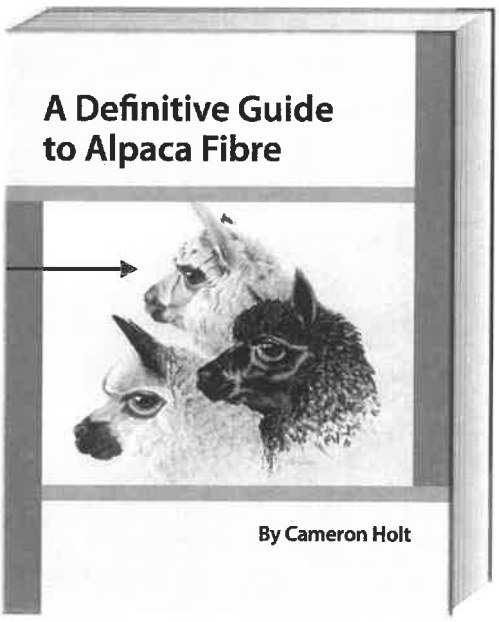
for a copy of this document,

"Alpaca Fleece Classing COP".

Cameron's new book is now available through the AAA office.

Author of "A Definitive Guide to Alpaca Fibre"

(Available from A.A.A.L.)



Dr Alison Lee

Alison has worked with the Victorian Department of Environment and Primary Industries since 1999. In her current position, she contributes to the development and management of national and state endemic disease control programs and disease surveillance projects, and undertakes animal health related data analysis and editorial activities. She holds degrees in both agriculture and veterinary science and is a Member of the Australian and New Zealand College of Veterinary Scientists (Epidemiology).

Alison has a specific interest in sheep and alpaca health and owns a property where she breeds alpacas and sheep.

Alison assists the Australian Alpaca Association analyse and report data obtained from the Q-Alpaca program.

DISEASE SURVEILLANCE WITHIN THE AUSTRALIAN ALPACA INDUSTRY

Australia's livestock industries currently claim a high animal health status. There is a growing need, however, for all industries to ensure effective disease surveillance programs are in place for increasing scrutiny by trading partners, and to ensure early detection of diseases that may impact on trade, public health and livestock productivity and welfare.

Q-Alpaca is a quality assurance program designed by the Australian Alpaca Association Ltd for voluntary use by its members. The program, which commenced in 2005, was developed to assist detect Johne's disease if it re-occurred in the Australian alpaca population, as well as to identify emergency animal diseases quickly and monitor endemic diseases effectively.

Information is **collated** at a national level and an annual report provided to the alpaca industry. Epidemiological analysis includes the spatial distribution of diseases and monitoring for disease trends.

Over recent years, the number of participants in this program has declined. Reasons for participants leaving the program include: (1) herd dispersals/alpaca owners retiring from the industry, (2) the costs associated with necropsies, particularly for aged animals and those that have died due to trauma (e.g. dog attack), (3) an inability to find accredited veterinarians in certain states/localities, and (4) a perceived idea that Johne's disease is no longer a risk to the Australian alpaca population.

AAA is currently undertaking a review of Q-Alpaca. The above-mentioned points are some of the issues being addressed, along with the potential to make the program a broader surveillance initiative, lessening the emphasis on Johne's disease and widening the scope of the program.

Proposed changes to the program will be discussed during this presentation, and feedback regarding the program will be welcomed.

Kris Lloyd

WOODSIDE
CHEESE WRIGHTS
ARTISAN CHEESEMAKER

Kris Lloyd is CEO and Head cheese maker of Woodside Cheese Wrights a specialist cheese maker of goat and cow cheeses, based in the Adelaide Hills in South Australia.

A range of around 15 different cheese styles are all made by hand at the factory in Woodside. Production is around 100 tonne per annum with distribution throughout Australia and export to Asian Countries.

Kris is a self taught cheese maker and has now been making cheese for around 15 years.

Kris is supported by a passionate team of cheese makers producing cheeses to traditional methods resulting in plenty of flavour and individual character. Kris has also pioneered a range of seasonal cheeses, with innovative packaging and presentation, to take advantage of seasonal variations in milk supply. Kris works closely with chefs around Australia to develop cheeses exclusively for their menus. Kris has grown the business tenfold since its purchase in 1999.

In 2004 Kris established the South Australian Specialist Cheese Association "Cheese SA" to promote specialty cheese and introduce industry driven education and training programs to South Australia. Kris is also the Director and Founder of CheeseFest, a Festival designed to raise awareness of specialty cheese and celebrate the Cheese Maker. CheeseFest has grown since the first festival in 2002 to be a nationally recognised event and 2013 saw CheeseFest run over 2 days attracting in excess of 15,000 visitors.

Woodside Cheese Wrights has won numerous cheese awards throughout Australia, including the Grand Dairy Award in 2004 and 2011 and has won the South Australian Premiers Food Award both for innovation and excellence in small business in 2002, 2005, 2006 and 2007. In late 2008 Kris was awarded the Zonta Womens Achievement Award for an Outstanding Entrepreneur. Over the years 2006 – 2009 Kris was appointed to the Premiers Food Council in South Australia which looked broadly at issues and improvements within the food industry in the State.

Woodside Cheese Wrights was awarded gold medals at the World Cheese Awards in 2008, 2009, 2011, 2012 an 201 3. Woodside was the only Australian cheese maker awarded a gold medal in 2008 and remains the only South Australian cheese maker to receive a gold medal at these awards, where over 2800 cheeses are judged from 30 countries around the world. 2012 Woodside was

awarded a Super Gold medal for the innovative Woodside Wakame Blue Cheese listing it as one of the best 55 cheeses in the World.

In 2008 Kris founded the Cheesemaker in Residence Program of which she was Chair. The program was awarded State Government funding to help in the skilling of current cheese makers and offer a pathway for new cheesemakers to learn and train. The program was highly successful and introduced French and American Cheese experts into the South Australian cheese making arena.

In 2010 Kris was the South Australian winner in the Telstra Business Women's Award in the Commonwealth Bank Business Owner category and The Nokia Business Innovation Award Catergory. She went on to win the 2010 Telstra Business Womens National Award for Business Innovation.

In 2010 Kris was also appointed to the Board the South Australian Tourism Commission.

2011 Kris was awarded The Adelaide Food Legend Award for her commitment to the South Australian food industry.

Kris has been the driving force behind the establishment of The Artisan Cheese Making Academy Australia, which is the only cheese making school in Australia to address Artisan style learning for cheese makers.

It offers both hands on and technical learning. She is chair of the Academy Steering Committee.

2012 Kris was one of three finalist in the Veuve Clicquot Business Women's Award and in the same year Kris was inducted into the prestigious International Guilde des Fromagers, France.

In 2013 Kris was inducted into the Australian Business Women's Hall of Fame and appointed one of South Australia's Ambassadors for Premium Food and Wine from our Clean Environment. Kris was also named as one of Australia's most influential women in the Westpac and Financial Review, 100 Women of Influence Awards.

Kris continues to be involved in many aspects of the cheese and food industry in South Australia and Australia. She writes a monthly cheese column for The Adelaide Review print media and is a fervent supporter of regional foods, farmers markets and producers.

Bryan McLeod

Email: proagcon@ozemail.com.au

Graduated Massey University, NZ 1962. Principal of Pro Ag Consulting Australia, established in 1987, and Pro Ag Associates NZ established in 1980. Pro Ag Consulting is now represented in Japan and New Zealand as well as Australia.

Bryan has been consulting for 35 years, specialising in identifying limiting factors in soil mineral balance, plant/pasture and animal production and nutrition. Not only the effects on pasture and fruit quality, but also the side effects on animal health and performance as a result of both poor soil and plant nutrition. Bryan has experience in most forms of agriculture, concentrating on soil nutrition, pasture nutrition and associated animal health issues. Pro Ag Consulting through APAL Laboratory, now offers a complete soil and plant analysis service with independent fertiliser programs and plans of action aimed not only for pasture production but also all aspects of stock health and production. Problem solving and identifying on farm productivity issues have been a big part of his consulting service

Bryan working with APAL Laboratory has as their objective not only the processing of soils, plant tissue, water etc, but also education in agriculture. This includes the teaching of farmers to understand their soils, their plants and animal requirements allowing them to make more educated evaluations and decisions on both fertiliser and animal health programmes.

Bryan is a much sought after presenter at agricultural seminars and educational workshops throughout Australia.

IMPLICATIONS OF SOIL BALANCE ON PASTURE PRODUCTION PASTURE PALATABILITY

ANIMAL HEALTH and PERFORMANCE

Power of Soil, plant, feed and blood analysis:
One must never under estimate the value found in information received from these four types of analyses, they hold information that gives you the power to control the nutritional status and productivity of your stock

I find that it is the interpretation of data that is often the limiting factor. So you also need understanding – not only understanding of the interpretation of data received but also

understanding its importance and application. Too often I see clients taking and using only sections of the data received based on not sure what?

The identification of all limiting factors in your operation is essential, whether it's in your soil, pasture, supplementary feed or mineral additives, once a deficiency or excess or otherwise has been identified and corrective treatment taken then you will start to benefit from increased productivity.

Too often we use shot gun mixes that may or may not correct our deficiencies. There are many mixes available that make excellent supplements but you still need to ensure they cover all the bases. Too often I see farmers listening to too many sales agents and changing to products that don't supply what is required

Productivity:
All aspects of animal productivity and health are determined by what goes into the stomach. And most can be prevented with understanding and planning.

These are:
Growth rates

Mineral deficiencies – Copper – Cobalt – Zinc – Magnesium – Calcium – Selenium – Manganese etc

Conception

Birthing problems - difficult birth or still births.

Metabolic Problems – milk fever – grass tetany (magnesium deficiency) – acidosis.

Lameness

New Born health – at birth and in the first few weeks after birth. Weakness in young can be related to the mother's diet before giving birth, eg high to excess protein

Bone development – in young stock. E.g. effects of copper deficiency during gestation.

and many more

All are preventable but first we need a total understanding of requirements, soil, pastures, supplements and diet.

E.g., I have seen a 300% increase in lamb growth rates when we helped a farmer understand his lamb requirements and the side effects of excess protein in the pasture in an irrigation situation

Identify limitations. Are they?

[a] Soil imbalances

[b] Pasture species or pasture production

[c] Pasture and supplement quality

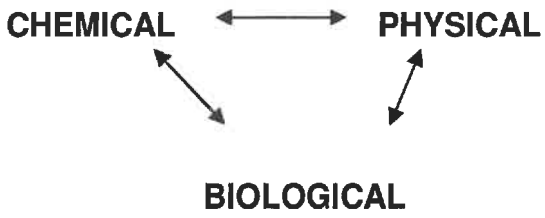
[d] Mineral deficiencies

[e] Misunderstanding of what and when to feed what

Let's first look at the soil

The three phases of the soil are interdependent

That is to say that you cannot change one phase of the soil without affecting the other phases of soils.



Each influences the other.

If pore space is increased by altering the chemical balance ➔ increased microbial activity ➔ improved soil structure ➔ more natural N ➔ more available P.

This refers to the minerals, including nutrients that make up the soil

This aspect of the soil is measured using soil analysis techniques.

Changes in the chemistry of soils affect

soil biology, organism populations and diversity.

soil structure, soils with adequate calcium are often well structured and friable whereas soils high in sodium have poor structure and are hard setting. Soils that are wet and sticky in the winter and hard setting in the summer are high Magnesium low calcium soils

Physical

Refers to the structure of a soil, hardness, friable nature, and porosity.

Changing the physical aspect of the soil

Affects nutrient availability

Changes the way water behaves in a soil. High Mg/K soils are impervious with limited water movement, high Ca soils have limited water holding capacity, ideally balanced soils generally hold optimum moisture

Affects the biology of the soil, tight compacted soils may not accommodate large soil organisms like worms and insects.

Biology

The biology of soils is one of the hardest aspects of the soil to measure and interpret. The simplest and most manageable way is by observation, the rate at which stubbles degrade, wormhole counts

etc. In recent time the biology of soils and its importance has attracted much attention and research activity.

Changing the biological aspect of the soil:
Affects nutrient weathering and nutrient availability. Soil organisms are involved in the decomposition of soil minerals and organic residues into soil and plant nutrients.

Affects the structure of the soil by the decomposition of organic residues. Organic cements are produced which help to glue soil particle together. Plant roots increase soil aggregate stability.

REMEMBER:
Plant/Animal deficiencies not only occur due to lack of a mineral but are... Also are created by excesses.

A nutrient excess in the soil will always result in the deficiency of another element in the plant, so when evaluating any analysis results not only look for deficiencies but also look for excesses. Eg, High soil Mo = low animal Cu, high soil Potassium = low plant Mg, Mn and B and low animal Mg and Na and there are many more examples

Nutrient interactions are complex and require consideration when planning fertiliser budgets for pasture and crops. The effect the interactions between soil elements can normally be seen in a tissue analysis. This it explains the need for a tissue analysis to help correct any nutrient disorder due to a deficiency or some other nutrient interaction.

What Is A Well-Balanced Soil?

- Contains good levels of sulphur, phosphorus, organic matter, calcium, magnesium, potassium, sodium and trace elements.
- Has a good soil structure
- Good water retention
- Good water movement both up and down
- Allows for good root development.

How Soil Balance Affects Pasture Palatability & Animal Health

Soil Balance and It's Importance

Pasture species are very versatile and tolerant to various soil conditions and will grow in a wide variation of soils that contain different nutrient balances. For optimum stock performance we need to produce both a palatable and nutritious pasture for our stock as well as achieving a reasonable pasture volume. As the levels of all nutrients are critical for healthy pasture and stock performances we need to look for any limiting factor that is adversely affecting either pasture or animal productivity. As the ratios of soil calcium to magnesium to potassium to sodium change so does pasture palatability. As we change palatability we change productivity.

MAGNESIUM: Soils with excess magnesium are wet and sticky winter & hard setting summer soils. Calcium is low and they lack air space, microbial activity and nitrogen production. They have very poor water movement both up and down. This can mean a lack of winter / early spring growth

due to wetness and poorer late spring / early summer growth due to poorer water movement up through the soil. Excess soil magnesium will suppress plant potassium and reduce pasture production.

High magnesium soils are characterised by their ability to stick to boots, machinery and everything that passes through them.

The lack of air space means plants growing in these soils generally have very shallow root systems. A stressed root system will cause the plant to accumulate higher levels of Non Protein Nitrogen. So we often see a higher incidence of lameness, mastitis and grass tetany on these soils. Pastures are often unpalatable which reduces dry matter intake and animal performance.

An interesting point with high magnesium soils is that the pH doesn't indicate a lack of calcium.

If we add calcium in the form of lime, we increase air space by altering the chemical balance ➔ increased microbial activity ➔ more natural N ➔ more availability of P.

Increasing the Ca:Mg ratio (Ideal 5.5:1) ➔ less compaction ➔ greater soil water movement, both down and up and a reduction in soil sodium. This means a greater utilisation of water.

CALCIUM controls the plant absorption of all other minerals. Plants growing on low calcium soils will absorb higher levels of iron and aluminum, which can be toxic to growth. Increasing soil calcium to the ideal will negate the adverse effects of excess soil aluminum and iron.

Effects on plants are an increase in pasture growth due greater root development and an increase in pasture palatability. This enables an increase in stock performance and a greater long term establishment of good pasture.

High calcium soils have poor water holding capacity; poor late spring / early summer growth; increased trace element deficiencies; and decreased P recovery. This means a lower percentage of applied P is available to the plant.

POTASSIUM One of the confusing issues with potassium is that pasture requirements are totally different to animal requirements. For example, pastures species require 2.5% to 3.5% leaf potassium for optimal growth. Animals only require approximately 1.2% potassium in their diet. Pasture samples will often show a level of potassium that will be limiting pasture production but will in excess for stock. The reason why many farmers are reluctant to apply soil potassium due to high pasture potassium levels, even when there is a soil deficiency. You must always address a soil deficiency before considering pasture levels, remember you are growing a crop and it is in the form of pasture, it is still being harvested but being harvested by your stock, for optimum pasture production you must think of it as growing a crop. On the other hand excess soil potassium can suppress plant magnesium, boron and manganese resulting in unpalatable pastures. This demonstrates the importance of taking both soil and pasture for analysis. When soil potassium is high I would expect to see an increase in metabolic problems. High soil potassium can be a natural occurrence eg NSW soils where we often see soil K higher than soil Mg, or areas where there is high stock concentrations or where high rates of animal manures have been applied. Stock grazing areas

where there is high soil potassium will require a higher intake of sodium so ensure salt is available at all times

SULPHUR This is critical for nitrogen utilisation by plants and animals. It is essential for healthy stock, meat and wool production. Low soil sulphur = poor pasture production with reduced animal performance, excess soil sulphur may suppress animal copper and selenium. Super phosphate contains 11% sulphur, the value of which is often overlooked when applying this product.

BORON Essential for plant reproduction, pasture energy, nitrate reduction and calcium absorption. When grazing fresh pasture, maintaining a good boron status in your pastures will increase pasture palatability and reduce metabolic problems. Essential for all plants, legumes and woody species have a high boron requirement

Trace elements Copper, Manganese, Zinc and Cobalt

All are essential for both plant and animal health. Copper deficiency is often a problem where soil molybdenum is high, never apply fertiliser Mo unless you know the soils Mo status. Zinc deficiency is common when stock is grazing fresh high protein pastures. Here we often see skin lesions which will clear as the pasture matures and protein levels decrease

What Consideration is pH?

PH - Soil Acidity: Ideal 6.0 to 6.5 (in water)

PH is one of the most misunderstood factors on our soil reports.

SOIL PH IS ONLY A MEASUREMENT OF THE HYDROGEN, NOT AN INDICATION OF SOIL NUTRITION OR MINERAL BALANCE.

PH gives no suggestion as to which nutrients are grossly deficient or to what degree the nutrients are unbalanced

The balance or levels of the four major cations calcium, magnesium, potassium and sodium are what influences pH. Soil acidity (or the alternative soil reaction) refers to the concentration of hydrogen ions (H+) in the soil solution, or more correctly the relative concentrations of H+ and hydroxyl (OH-) ions. The degree of acidity (or alkalinity) of a soil, or of any other systems, is expressed by means of the pH scale.

PH can be looked upon as displacement. Hydrogen is displaced as cations are added to the soil complex. This causes the pH to increase. As plants use the cations (Calcium, Magnesium, Potassium and sodium), they are replaced by hydrogen and the pH goes down.

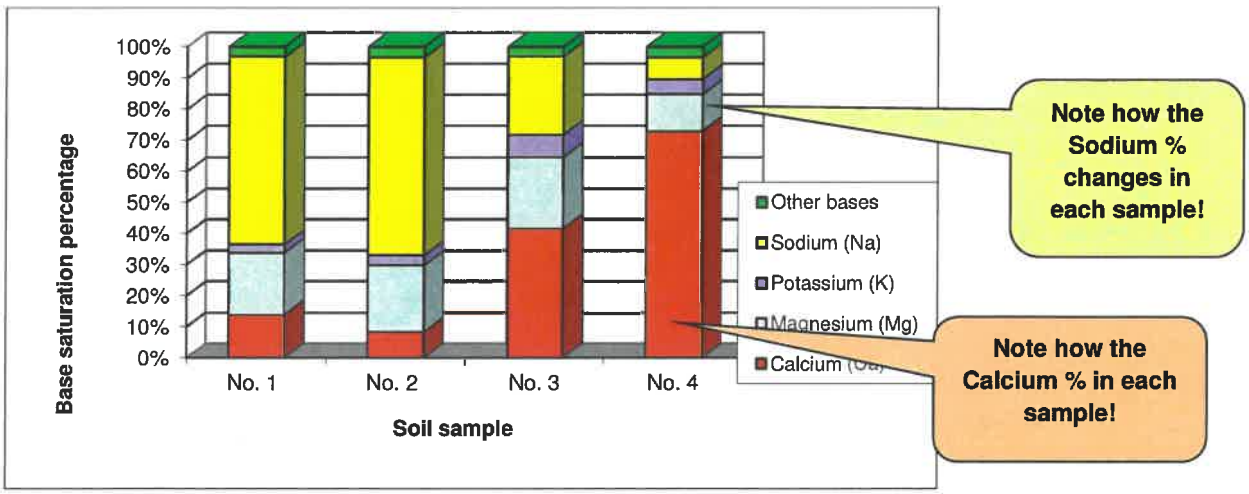
The pH of a soil is constructed with all the major cations (Positively charged elements) Calcium, Magnesium, Potassium and Sodium. Ph levels in NSW soils are often formed with high K, SA soils with Mg and Ca, Northern Victorian soils with high Mg. It is important to know just how the pH of your soil is constructed

Base Saturation Percentage and pH Values

The following chart shows how the wrong information can be gained from using pH to establish the fertility or balance of a soil.

The 1st sample taken from the centre of a salt pan, 2nd edge, 3rd 50mtres from edge and 4th 100metres out,

8.3 pH 8.1 pH 8.4 pH 8.1 pH



The above chart shows similar pH values but with different makeup

No 1 Middle salt scald – 2 Edge of scald – 3 -50mtres into crop – 4 -100mtres into crop

PASTURE ANALYSIS:

An analysis tells you directly the quality and nutrient levels that your animals are consuming. A pasture analysis does not give you a good indication of soil levels as the soils mineral status will control what a plant takes up. Eg High Ca soils = low plant trace elements, phosphorus and magnesium, high soil potassium = low plant magnesium, boron and manganese.

A pasture analysis will help to complete the picture of what is happening in your soil and what effect the soil is having on plant uptake

Pasture Species: On the same soil different pasture species will extract various levels of the same element, so it is advantageous to have a variety of species in your pastures. Eg Grasses are generally low in magnesium, clovers are higher in magnesium. NZ trials with growth rates in lambs showed a difference from 23kgs to 37kgs with the same dry matter intake

So the challenge for you is to recognise the changes in pasture nutrition and supplement accordingly to meet your animals requirements for optimum production.

Alpacas are out of their natural environment and comfort zone

We must recognise and understand their natural environment and feed source. They graze on slow growing species that will be naturally low in protein and contain high levels of nutrients.
Alternatively faster growing immature species are high protein and generally lower in essential nutrients.

All high country animal species require lower protein diets for optimum production – Deer, Merinos, Goats and Alpacas. For optimum production we need to understand their basic requirements and how can we replicate their natural environment or negate the side effects created in a different environment?

Salivation time: This is the most critical aspect of helping alpacas adapt to your environment this is the time taken chewing and producing saliva. The amount of saliva produced is an essential and critical part for the efficiency of every of every animal's digestive system. In their natural habitat Alpacas produce maximum saliva as they have a high fibre low protein intake – This means a lot of chewing and optimum saliva production = optimum digestive pH, see below how as the eating rate decreases saliva production increases. As we increase dietary protein, digestion time and chewing (rumination) time is reduced stomach pH stays lower and we see various levels of acidosis subclinical to clinical and naturally lower productivity

Effect of Ration on Eating Rate & on Saliva Production

Feed	Eating Rate of lbs of feed/min	Salivary Production Teaspoons/Pound of Feed
Pelleted	0.79	1.0
Fresh Grass	0.62	1.5
Silage	0.55	2.0
Dried Grass	0.18	5.0
Hay	0.15	6.0

Source: Bailey, C.R. 1958. The role of secretion of mixed saliva in the cow.

Alpacas have a high fibre requirement – see above the difference in saliva production between dry feed and fresh green pasture
Ask yourself the question: Are your animals producing the optimum amount of saliva for optimum production?

FEED ANALYSIS

VALUE and TIMING of SUPPLEMENTS to improve PRODUCTIVITY

The first point to make is that many supplements and animal treatments are wasted due to:

- Poor timing of supplementation or animal treatment;
- Misunderstanding of animal requirements: All have different daily needs.
- Lack of knowledge as to what the nutritional status is of various supplements.

What are the basics of a feed?

- Protein
- Energy (measured as ME / kg DM)
- Minerals
- Vitamins
- Water

How do these basics match the requirements of your stock?

A supplement must be a feed that complements the main diet or paddock feed. The idea of a supplement is to provide nutrients that are deficient or to provide extra feed to improve stock performance.

It must balance the main diet to the ideal. Eg if stock are grazing high protein pasture then a high energy high saliva producing feed is the ideal supplement. In cases of a severe feed shortage the supplement maybe the complete diet.

If you are providing an additional feed that has a similar composition to the feed already available in the paddock. Then you are not providing a supplement. Any supplement you provide must complete the diet necessary to maintain or improve productivity.

Protein/Carbohydrate Balance

This is the most important starting point when evaluating a diet for any class of livestock, get this right and the mineral vitamin content becomes less critical.

Excess protein increases requirements for both minerals and vitamins and also reduces salivation time which is critical for all species. Once salivation time is reduced intestinal pH is reduced and in severe case we see an acidotic condition

Alpaca Protein Requirements

Maintenance	8% to 10% Crude Protein
Rapid Growth from weaning	16% Crude protein
Pregnancy and Lactation	12% to 14.5% Crude Protein

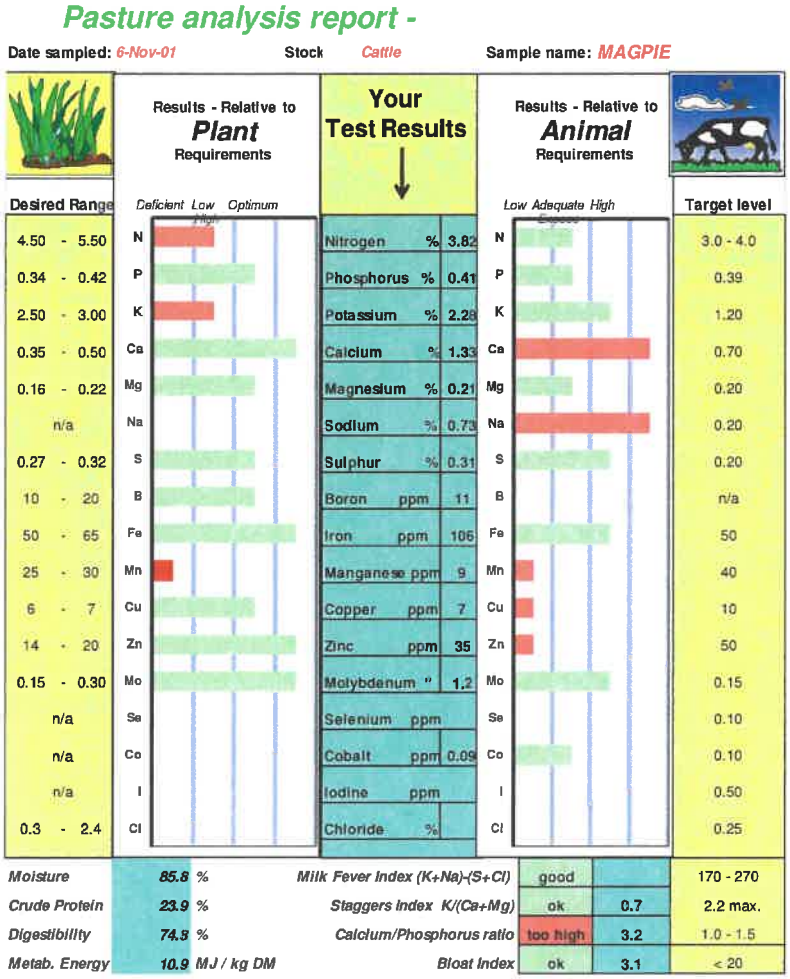
Impact of Excess Pasture Nitrogen (Crude Protein) On Animal Performance

If the diet is protein rich to the point where it is energy deficient there is a damaging effect on the liver.

Side Effects of Excess Protein & low energy

- Unnecessary expense
- Increased energy requirements
- Scouring
- Increases requirements for trace elements
- Weight loss
- Vitamin A reduction
- Early deaths at 7 to 10 days of age
- Mis-mothering
- Pasture burns
- Magnesium suppression
- Lameness
- Poor conceptions
- Full term young born dead.
- Weakness at birth

Pasture Analysis showing the different requirements between pasture and stock. See how pasture requirements differ from animal requirements



Pasture analysis report -

Date sampled: 6-Nov-01 Stock Cattle Sample name: MAGPIE

Desired Range		Results - Relative to Plant Requirements (Deficient / Low / Optimum)	Your Test Results		Results - Relative to Animal Requirements (Low / Adequate / High)	Target level
4.50 - 5.50	N		Nitrogen %	3.82		3.0 - 4.0
0.34 - 0.42	P		Phosphorus %	0.41		0.39
2.50 - 3.00	K		Potassium %	2.28		1.20
0.35 - 0.50	Ca		Calcium %	1.33		0.70
0.16 - 0.22	Mg		Magnesium %	0.21		0.20
n/a	Na		Sodium %	0.73		0.20
0.27 - 0.32	S		Sulphur %	0.31		0.20
10 - 20	B		Boron ppm	11		n/a
50 - 65	Fe		Iron ppm	106		50
25 - 30	Mn		Manganese ppm	9		40
6 - 7	Cu		Copper ppm	7		10
14 - 20	Zn		Zinc ppm	35		50
0.15 - 0.30	Mo		Molybdenum "	1.2		0.15
n/a	Se		Selenium ppm			0.10
n/a	Co		Cobalt ppm	0.09		0.10
n/a	I		Iodine ppm			0.50
0.3 - 2.4	Cl		Chloride %			0.25

Moisture	85.8 %	Milk Fever Index (K+Na)-(S+Cl)	good		170 - 270
Crude Protein	23.9 %	Staggers Index K/(Ca+Mg)	ok	0.7	2.2 max.
Digestibility	74.5 %	Calcium/Phosphorus ratio	too high	3.2	1.0 - 1.5
Metab. Energy	10.9 MJ / kg DM	Bloat Index	ok	3.1	< 20

Blood analysis:

Use to identify mineral deficiencies in livestock. Test for Vitamin B12 (Cobalt), Vitamin D, Selenium, Copper and Zinc. With this information you can complete the nutritional picture, it confirms whether or not you are supplying the optimum for optimum health and productivity.

Vitamin A We often hear how much vitamin A there is in green plants and the belief that pasture contains high levels of this essential vitamin, but in fact green plants contain only carotene which is the precursor of vitamin A. Carotene is then converted to vitamin A in the body but if protein (Nitrogen) intake is high to excess then this conversion is limited and the animal becomes vitamin A deficient.

Alpacas in their natural environment consume low protein plants, vitamin A deficiency wouldn't be an issue, however when consuming high protein species vitamin A deficiency would become an issue, this problem is not only limited to alpacas but to all livestock, in my consulting I also ensure there are good supplies of A.

You all have total control of your animal's health and productivity, if you have an issue then any or all of the above will help you solve the problem. To do this you may need to step outside the square and look at the wider picture.

Alpacas come from a different environment, you don't need to replicate this environment but you can effectively counter the side effects they will experience of being in a different place.

David Michell

Owner Michell Wool

David Michell is Chairman of Michell Wool Pty Ltd (Michell) – a South Australian-based global Wool company, sourcing, processing and supplying the international textile industry.

Michell was born in June 2004 when Michell Australia Pty Ltd – a company founded in 1870 with diverse interests across Wool, Leather, Agriculture and Commercial property – sold its wool processing and trading business, to brothers David and Peter – both fifth generation Michell family members. The brothers drove the company's restructure, sharing the Managing Director role of the new venture, Michell Wool Pty Ltd, with their late father John Michell as Chairman.

In February 2008, John Michell retired (but remained as Director until 2013) and David stepped into the Chairman's role – with Peter now the sole Managing Director of Michell.

David commenced his career as a cadet wool buyer with Michell Australia – joining the family business in 1977. After 3 years as a Wool Buyer in Adelaide and Melbourne he joined the Merchant Banking industry working in the JV between Michell and the National Bank of Detroit in Detroit as a currency trader and Financial Risk consultant. This experience lasted for 10 years after which David re-joined Michell Australia as Treasurer returning to the company's headquarters at Salisbury in Adelaide. In 1997 he took on the position of General Manager - Wool which he held until the 2004 restructure.

Today, Michell Wool Pty Ltd remains the world's largest Carboniser of wool (approx. 15million Kgs pa), a significant buyer, processor and exporter of Australian wool. The company's primary headquarters and processing facilities remain at Salisbury but are now complemented by Michell Suzhou, a green fields wool processing plant built in China in 2006.

Michell has invested in a number of cutting edge, wool centric, start-up businesses since its inception. These include FRAM Technologies (www.framglobal.com) - a fire retardant technology business, p_9 (www.p9wool.com) – a performance fabric business, Nexpoint (www.nexpoint.com) – a textile supply chain management business and the Michell development brand, I/O Merino (www.iomerino.com) - a performance base layer garment business specialising in wool.

In addition to the textile-related investments, the Michell Family group diversified its investments into the Wine Industry with an investment in Wirra Wirra – a prominent South Australian winery based in McLaren Vale. David currently chairs the Board of Directors.

As Michell Wool Pty Ltd's Chairman, David's main objective is to keep the family business at the forefront of the international textile industry – by developing and implementing key business strategies designed to expand production capabilities and encourage the creation of innovative new fibre products while focussing on the organisation's long-term viability.

In addition to his role at Wirra Wirra, David also Chaired Nankai Worsted Top Making (Terangganu, Malaysia) until sold in 2012, a former Director of AWTA Pty Ltd, a Fellow of the Finance & Treasury Association and a Past President of the Australian Wool Processors Council.

Married with three adult children, David enjoys spending time with his family as well as at the gym, snow skiing and cycling.

Dr Stephen Mulholland

Stephen Mulholland originally trained as a laboratory scientist, and holds a Ph.D. in Biochemistry and Molecular Biophysics. In 2003 he moved to NZ, purchased a small farm, and started raising alpaca.

When the first of those animals died in 2004, Stephen began investigating the available information on camelid morbidity and mortality, and was disappointed with the results. In 2005 he began, with the assistance of the AANZ and the NZLA, to run health surveys of the llama and alpaca populations of NZ. To date he has collected more than 15,000 animal-years of data on their morbidity, mortality and management.

Stephen also works closely with the Animal Welfare Directorate of the Ministry for Primary Industries. He led the team which produced the final draft of the Code of Welfare: Llamas and Alpacas, presented that draft code to the National Animal Welfare Advisory Committee, and consulted with the ministry throughout the further development of the Code up to its launch in April 2013.

As an offshoot of his work with MPI, Stephen joined the Johne's Management Limited consultant network in 2012 and now acts as a contact point for discussions involving the disease in camelids in NZ.

In 2013 he started a collaboration with Dr Kylie Munyard of Curtin University to study the genetic underpinnings of dwarfism in alpacas, and he is in talks with Massey University to launch an in-depth epidemiological analysis of the eight years of accumulated health survey data.

Stephen has written dozens of articles for the trade magazines of the llama and alpaca associations. Keen to increase the general literacy of owners as to what they can do to improve animal welfare, improve management practices, and make better-informed breeding decisions, in June 2013 he founded a camelid health and welfare charitable trust with other interested llama and alpaca owners: www.camelidhealth.org

He also works with, and helps do fund-raising for, local SPCA chapters.

THE NECESSITY OF SCIENCE AND STATISTICS IN A SUCCESSFUL ALPACA INDUSTRY

Running a business means making decisions every day. Buying, selling, money and marketing; these are the common threads that link all businesses.

But when you're breeding alpacas, you need to start making other more complicated decisions involving nutrition, genetics, parasite control and a host of other quite technical issues. Since many alpaca owners don't have a strong science background, they must make do with the information available from web sites, books, other breeders, and conferences like this one. But this leads to the old joke in the industry- ask five breeders a question, and you'll get at least six different answers. How can you make an informed decision in such a confusing environment? Think in a more scientific manner.

I'm going to show you a few methodological techniques that can improve your decision making, and then I'm going to give you some examples to hopefully make you think.

But first, a disclaimer. It is highly probable that at least one thing I am about to tell you is wrong. Furthermore, it is also highly probably that every other speaker at the conference has told you something that is wrong.

Welcome to science, and specifically the science of camelids. We still have a lot to learn. Best knowledge and best practice keeps evolving and improving. What the speakers (myself included) are telling you is our best understanding of the situation based on the current evidence available. But each year we learn more, and sometimes what we learn makes us have to change our previously held assumptions.

This is why "Answers" are dangerous. When people have an Answer they happily turn off their brain, and never think about the issue again. We should always be asking questions, and challenging the Answers we've been given. Channel your inner sever-year-old. Why? How does that work? Does that make sense? Can you prove it?

Answering these questions will take you in the right direction. (Of course, to do so with "scientific rigor" requires a heap of knowledge about experiment design, statistics, and the other drudgery you slog through on your way to a doctorate.)

A common mistake many people make when trying to find the answer to a question is confirmation bias. This is where you look for (or only see) the facts that support your pre-conceived notion, while ignoring everything that disagrees. The internet makes this easy. No matter how crazy a notion, you are sure to find at least a few people out on the web who agree with you. "Someone agrees with me" is not a valid defence of a thesis. You need to back-up your assertion with either facts (evidence collected in a systematic and unbiased fashion) or logic (making reasonable comparisons to systems or evidence sets for which we have a good understanding- this is how we work out drench dosing by comparing alpacas to other farmed species for which there is testing data).

An example of confirmation bias can be found in the way some people collect fleece samples for testing. While ideally you should take a representative sample from a fixed location (commonly mid-side), some people search out a "nice, clean looking staple" to send to the lab. In doing so they confirm their pre-existing bias about that animal's "good quality" fleece by carefully eliminating results that might contradict that notion.

Another concept you need to keep in mind is falsifiability. This simply means that beliefs can be disproven. The strength of the scientific method arises because every theory is open to continual challenge. New evidence, if it is sufficiently compelling, can make us reconsider. In areas where the depth of knowledge is not very great, there is rapid turnover as new evidence accumulates, and old theories either stand or fall in response to them. So if I said "alpaca don't have live triplets" I think most people would agree this is correct. However, if an alpaca did ever give birth then it would falsify my statement, and I'd have to amend it to "it's extremely rare for alpacas to have live triplets."

You must be willing to change your mind, if new facts come to light. Feel free to questions those facts. As I said above, you always need to ask questions. But don't cling to old beliefs just because they are more convenient or more comfortable.

Science is testable, observable, and repeatable.

In science, "We don't know yet" is a perfectly acceptable answer.

Taking an analytical, systematic mindset can help you distinguish the Snake Oil from the Science. Be swayed by evidence, not simple proclamations. That includes what I'm saying here.

Where do statistics fit into all of this? (I should really deal with both science and statistics, since both were mentioned in the title.) Wikipedia tells us that statistics is "The study of the collection, organization, analysis, interpretation, and presentation of data. It deals with all aspects of data including the planning of data collection in terms of design of surveys and experiments." And if you think it's complicated and boring, you're right. It is. But what you need to take away from this is the habit of thinking numerically.

Numbers are good. You should use them more often.

If I say "my alpaca is big" it tells you very little. If I say "my alpaca weighs 95 kg, is body condition score 3, and stands 88 centimeters at the withers" it tells you a great deal. The alpaca industry is full of superlative adjectives, too often with no factual measures to support them. These sorts of adjectives make it nearly impossible to fairly compare the actual virtues of any given alpaca.

Fleece statistics (mean micron, SD, staple, fleece weight) are the most commonly collected numbers when it comes to alpaca breeding, but there is much more we can measure, and improve.

Remember: you can only successfully breed for something if you measure it! Conversely, you can only avoid bad outcomes if you are measuring and monitoring. Recording a wide set of traits in all your animals can let you spot surprises, both good and bad, that otherwise might have slipped by.

This of course leads to a discussion of the Spanish Habsburgs. Why? They are an interesting test case which we can use to understand the role of increasing inbreeding on the health and fitness of a population. (See: "The role of Inbreeding in the Extinction of a European Royal Dynasty" by Alvarez *et al*. 2009) This paper provides not only a warning of what can go wrong, it provides some insights on how we can use numerical tools to look for potential problems in our own breeding populations.

Too many people think that inbreeding means you start getting cria with two heads. The negative effects of inbreeding can be very subtle, yet simultaneously very damaging.

There are three take away lessons we can apply to our alpaca breeding.

1.To properly calculate the cumulative increase in inbreeding coefficients you have to look back at least 5 generations. Lots of distant-relationships can add up to a very high level of inbreeding. In the case of Charles II, the last King of the line, his inbreeding coefficient was .254, greater than what you would get from a brother-sister pairing. Charles II also had two genetic diseases (combined pituitary hormone deficiency and distal renal tubular acidosis), and was insane and impotent. And the last of his line. How many generations back does the inbreeding tool on the AAA site go back? You need to go back 4 generations to see the true extent of Charles II inbreeding.

2.Inbreeding is not just about gross defects. A broad weakness in the population is expressed by declining survival rates. The survival rate of Habsburg royal children reaching 10 years of age with high inbreeding coefficients was significantly lower than the general population at the time. You might expect royal children to have had better survival rates, not worse, as they had the best nutrition, housing and health care. What is the overall survival rate to weaning for cria? How is it affected by inbreeding coefficient? We don't know, because we haven't measured.

3.Decreases in overall fertility is another common affect of inbreeding. How many matings does it take your 'highly bred' stud male on average to get a female pregnant? 1.3? 1.5? 2.5? How has this changed over the years as your breeding system 'progressed'? You can do the same calculations for females, but it is more error prone (statistically uncertain) because females have few pregnancies individually, where a stud male can generate hundreds.

4.Due to a lack of systematic data collection, it's impossible to tell if inbreeding is causing problems in the alpaca industry. We don't know if we're speeding towards a cliff or not.

Now for a specific example of trait you may not be measuring, but should be. Milk production.

No, I'm not expecting some weird camelid-dairy industry to develop. Milking alpacas is no fun at all. But milk production is absolutely vital to cria survival rates. Marginal milkers, i.e. those dams whose cria need supplementary feeding in order to put on weight, are a drag on herd productivity, and yours. Milk production is quite heritable (0.3 to 0.35 in dairy cattle). Which means you can quickly change the average milk production levels in your herd, up or down.

From the data we've collected over the last decade, we know we have dams that can put more than 300 grams a day onto their cria for the first month, and 200 grams a day for the months that follow. Such cria are big, strong, and vigorous. And when the dam produce such prodigious quantities of milk, get pregnant again, and maintain a body score of 4+ through the lactation period without supplementation, you have a winner that needs to be identified and bred from.

100 grams per day is "enough," and dams that provide a 100 grams a day weight gain in their cria are common. But by three months of age there can be a 7 (or more) kilo different between cria from strong-milking mothers and weak milkers. Weak milkers get culled from our herd.

Fat, fast-growing cria are what we want. If an alpaca is to grow to its full genetic potential, it must have good nutrition. If we had paddocks full of 350 g/day mothers, more cria would survive, and we would see those cria fully express their potential. They are also better suited to survive harsh winters, something we have to consider in Wellington where the rain tends to fall sideways.

There are lots of males who advertise their ability to improve a fleece. Who can point me to a stud male with a proven ability to improve milk production? Because we need some of them. There are plenty of "fine fleeced" dams out there with mediocre to marginal milk production.

Measuring traits and keeping accurate numerical records can help you spot all sorts of things. It also helped us spot a stud that was throwing 50% miniature offspring. What weirdness is lurking in your herd, ready to be discovered?

To make informed decisions you need facts. You need to be measuring many traits. Large farms here in Australia have the advantage of scale, just their own herd provides a large enough data set for meaningful analysis. But they wouldn't know how well (or poorly) they are doing compared to the national average. There needs to be continuous collection of data on key performance (health, management, fertility) traits so the future of the alpaca industry can be planned, and not stumbled into. In New Zealand I've been running a Health Survey for the last 8 years, trying to form the basis of such an analysis for both llamas and alpacas. I'd be happy to help you set one up in Australia. The Australian and NZ surveys could even be fused to provide more potential insights into how climate and management affect outcomes.

Health and productivity are fundamental to any animal breeding. With selective breeding it is possible to improve a vast array of traits, from fleece quality to milk production. But there are two things breeding can't fix- infertile and dead. You don't have to look long to find alpaca with fantastic fleeces that can't get pregnant. At that point the fleece is irrelevant, as that animal is a failure and the end of its line.

Remember the simple equation of profits = outputs – inputs. One of the greatest potential advantages to alpaca is that they can be very low maintenance, especially compared to sheep. No need to crutch, dag or dock. Easy handling, strong constitution, and the ability to develop a natural resistance to parasites. If they are highly-fertile, good-milkers that birth easily and maintain a healthy body score whilst lactating, then they need only minimal handling. And since time is money, the less you have to do, the more profitable they are. Sickly, finicky, high-maintenance animals will be worth less, and if we're not careful they will be worthless.

A single vet call can cost the lifetime-fleece-value of an animal. Every time you administer a drug, or feed a supplement, the cost comes straight off the bottom line.

Controversy can Make you Think. (Hopefully)

And now to say some controversial things, in the hope that it will make you think. I may well be wrong. And if I am wrong, I invite each of you to try and prove it. Factually falsify my assertions. That is how science works, and that is how we progress the body of knowledge about camelids.

Example 1- How we often talk about breeding and alpaca genetics is wrong

I'll start with the problem of conflation of breeding traits; that is where two separate traits are treated like they are linked. For example, if I told you "For the last 20 years we've been breeding for tall, brown alpacas. Now we have a herd of tall, brown alpacas. Therefore colour and height are genetically linked" what would you think?

I hope you would simply laugh at the above statement. If you breed for both A (colour) and B (height), why should we be surprised when you get both? That doesn't mean they're related, or linked. If, on the other hand, I was breeding only for colour, independent of all other traits, and every animal with that colour was taller than average, then there might be something interesting going on.

Why do I mention this? Because this sort of conflation goes on all the time. If I said "For the last 20 years we've been breeding for fine, crimpy fleeces. Now we have a herd of animals with fine, crimpy fleeces. Therefore fineness and crimp are genetically linked." what would you think? That comparison is as much a fallacy as height and colour. I find many people apply the same false association to density and coverage.

Example 2- Being too clever for our own good

Did you know that many leading farms in Australia and New Zealand have a systematic program to identify their best alpaca-mothers? They find the dams with a proven ability to carry pregnancies to term, that birth easily, have a strong mothering instinct, and that produce large quantities of milk. And then those dams are labelled "ET recipients" and culled from the herd. Their genetics will not be passed on.

I know ET donors must be at least adequate mothers. But consider the milk production example above, if we start propagating lots of 100g/day mothers at the expense of the 350 g/day mothers, we will see an inexorable decline in the average dam milk production in the national herd.

What happens after generation upon generation of this practice? If our selection criteria is overly focused on fleece, are we going to breed a line of animals that can no longer reproduce on their own? That would be a catastrophic failure.

Example 3- When "desirable" traits harm health and profitability

What is good fleece coverage? Some breeders talking glowingly of their animals with massive "hair helmets" and thick leg fleece that goes right down to the toes. But those traits actually lower the value of the animal.

Face fleece has no economic value. It takes time to shear, and time = money (fewer alpacas shorn per hour). And a shearing mishap around the eyes, lips or ears can be nasty and expensive. Furthermore, thick face fleece can result in wool-blindness, which would be a big management/welfare issue in an extensive farming situation.

Animals where the fleece stops behind the ears are derided as looking llama-like. But they're more economical. Is this preference economic, or just fashion?

Likewise with leg fleece. After damp Wellington winters we see animals with fungal skin conditions on thickly fleeced legs. It clears up after shearing, but that means by definition the legs require shearing for health reasons. Which takes time, can be fiddly work, and produces very little fiber of value. Again "llama like" clean legs can solve the problem.

And I have yet to see good evidence that "coverage" is linked to follicle density (and thus the weight of high-value fleece per animal). See the conflation example above.

An extreme case of where breeding for coverage and density can lead to came onto our farm a few years ago. The animal was not ours, but he was in our care for 6 months. He had an incredibly thick fleece, he had fleece everywhere (amazing coverage!), and as you would guess he was a certified stud. He also had soft skin that lay in rolls down his neck. And because of this he suffered from persistent, extensive fungal infections. We spent upwards of half an hour a day just trying to manage his condition. Eventually he had to be destroyed for humane reasons. Yet too many people would consider him to have held very advanced and desirable fleece traits.

The most amazing fleece is of little value on a dead animal.

Example 4- Road to ruination

It is important to remember that with an aggressive breeding program focused on a small number of heritable traits, it is very easy, in only a few generations, to completely ruin a herd/breed. One of the tools that best speeds this process of ruination is the show ring.

Don't believe me? Look at what we have done to too many dog breeds over the last century. Once healthy, productive dogs are now unhealthy, infertile, sickly beasts incapable of the tasks for which the breed was originally created. These breeding choices were made with "breed standards" firmly in mind, which further accelerated the damage. The german Shepard is a commonly cited example. One hundred years ago the German Shepard was a 25 kg dog that could leap a 2.5 m high wall. Those days are long gone. German Shepard in today's conformation ring are 38 kg angulated, barrel-chested, sloping backed, ataxic beasts famous for their hip dysplasia issues. Too many dog (and an increasing number of cat) breeds have been reduced to sad, sickly parodies of once productive and distinctive breeds after a hundred years of showing.

This process of selecting for vanity traits amplifies generation on generation, as to achieve a "win" in the show ring you have to be a more extreme expression of the "standard" than your competitors.

Show judges can't know if the competing animals are fertile. They can't spot studs that throw large numbers of deformed cria. The notoriously stoic alpaca can walk proudly around the show ring merrily concealing a host of metabolic and chromosomal abnormalities.

At the end of the day we need to remember the three main traits we are looking for. Is the animal healthy? Is it fertile? And, once those two criteria are met, does it have a nice fleece?

If an animal fulfills these three criteria, why do we get upset if its snout is "too long"? That's vanity, pure and simple. And vanity-driven breeding decisions can rapidly ruin a breed.

Example 5- AGE, or Pretty graphs does not make it science.

I was excited when the Across Herd Genetic Evaluation scheme was first launched. The sort of quantitative genetics it promised can quickly result in significant genetic advancement. AGE also inspired people to start measuring and recording more traits about their animals, and that's wonderful, too.

But unfortunately the way AGE was set up means that it generated meaningless results. Because those results come in the form of very official-looking graphs and tables, they are mistaken for being scientifically valid. Sadly, they're not.

The principle behind AGE (and other breed value systems) is to measure productive traits in offspring (cria), and then use statistical tools to determine the genetic contribution of the sire and dam to those traits. In this way you can rate the strengths and weaknesses for such factors as mean micron, fibre variability, milk production, and any other measurable, heritable trait. Measuring only the traits of the parent (sire) only tells you the phenotype. It is through the offspring that you get a window onto a given animal's genotype.

The AGE system has two fatal flaws, unfortunately. First, most of the fleece data entered is for first fleeces. The first (cria) fleece is quite variable, many cria coarsen significantly in their first year or two of life, so age of sampling can introduce considerable variability. (Was the cria 6 or 16 months old at time of sampling? That can result in many microns difference on the same animal.)

Second, and perhaps more importantly, the contribution of the dam is not entered. How can you know what the sire contributed, if you have no idea if the dam was 18 micron or 35 micron? You can't. And thus, sadly, the AGE system is rendered meaningless. We could fix it, fairly easily. I don't know why we haven't. (Though I suspect it is a combination of ignorance- not many people understand quantitative genetics- combined with a conflict of interest of those who use the current broken system for marketing purposes and would not like the actual worth of their stud males revealed.)

Also, if you only send in a subset of cria samples, your AGE results can be skewed by confirmation bias. It's too easy for a breeder to self-justify dropping the 'not so good' cria to make sure his or her super-stud gets a 'fair' analysis in AGE. All this does in reality is make the entire AGE system meaningless.

Example 6- Do we know what we're doing? Do we know where we're going?

As I said previously- if you don't measure it, you can't breed for it. And if you're driving in the dark, you never know when a cliff might be ahead. The same logic applies to an industry-wide standard.

A question I've asked a few times in NZ, but never gotten an answer to is: "what is the mean micron of a 3-year old white male." Meaning, what is the national-herd average of the sort of alpaca that would form the backbone of a large scale fleece industry. What was that average 5 years ago? 10? 20? We know that the "top end" alpacas have really improved over the last two decades, but the industry is built from the average, not the top end.

And there are many questions that I can't answer right now, and that makes me nervous.

How have the following changed over the last 20 years (as a national average)?

-Incidence of wry face?
-Milk production/Live-weight gain?
-Male fertility (matings per pregnancy)?
-Dam fertility (quickly pregnant and carries to term)?
-Cria mortality to 6 months?
-Rate of choanal atresia?

And I could easily add another dozen important heritable traits to that list. We're in the dark as to many fundamental aspects of health and productivity. How many people have the policy of "shoot, shovel and shut up" when something goes wrong? The merciless hand of probability indicates that the larger alpaca farms probably get multiple deformed cria each year, yet it can be very hard to get them to talk about it. Such secrecy may be good for their businesses individually in the short term, but it can be very bad for the industry overall (and themselves) in the long run.

I've been trying to remedy this situation in New Zealand by running anonymous, privacy-protected annual health surveys of the national camelid population. In the last 8 years we've accumulated about 17,000 animal-years' worth of data. I've helped owners understand the rarity (or not) of conditions affected their animals, and helped them (and their vet) make more informed decisions about breeding, culling, and animal management.

Australia should be collecting the same sort of data. I'd be happy to help.

HOW DENSE ARE YOU?

Generating a fair assessment of alpaca fleece density.

Alpacas have the capacity to produce truly wonderful fleece- soft, lustrous, and in a panoply of colours. To maximize the profitability of alpacas, people are naturally pursuing breeding goals the maximize the output of fleece. In this pursuit the trait called "density" is seen as a worthy objective.

But what is "density", how do we measure it, and how do we fool ourselves? And perhaps most importantly, how can we make better-informed decisions when it comes to breeding programs seeking to increase density.

The scientific definition of density (in regard to alpaca fiber) is a measure of how many hairs grow per unit area of skin (usually measure in follicles per square millimeter). This can be measured directly by taking a skin biopsy, and stdying it upder a microscope to count the number of hair follicles. This also give a measure of the number of primary fibers (guard hairs) compared to secondary fibers.

There are a couple of practical problems with this direct biopsy measurement. Most obviously, this is not something that you can do on your own (unless you happen to be a veterinarian with access to a good histology setup). So there is a cost to such sampling. A single biopsy also gives you one "data point", it is a measure from a single area on the blanket region of the fleece.

When you are looking at large numbers of animals for sale (or for use as a stud), it is quite difficult (expensive) to get that many samples collected and analyzed, and some owners may not want a skin punches taken from their animals by a possible tire-kicker. The single-point-in-time measure of density is also problematic on young, growing animals that have not yet come into their adult fleece, which could be higher or lower density than their cria fleece.

One of the common techniques I see people applying is what I've terms "a good grope". While examining the fleece to look for colour, staple, crimp they will at some point give at a good squeeze to "see how dense it is". I do this myself.

But just what are we measuring when we give the fleece a squeeze? Density? Nope. What we're measuring is the resistance to mechanical compression.

Now, the resistance to mechanical compression has a relationship (correlation) to fleece density, but it is driven by many variables, is more complex than you might first think, and is very susceptible to incorrect assessment of fleece density.

Yes, more fleece is harder to compress than less fleece, but that is only one aspect that affects your perception.

1. Thick fibers are stiffer than thin fibers. (An everyday example is tree limbs. The little branches bend out of your way when you apply pressure, but the same amount of wood in a thick branch would resist that same pressure. Make a bit of wood a bit thicker makes it much stiffer.) What this means is that a bunch of 25 micron fibers will be much better at resisting the compressive load than 15 micron fibers.

2. Bundled staples let fibers support one another. Like coiled bed springs, an "organized" fleece where the fibers are bundled together will be much stiffer, and thus resist your grasp.

3. Longer fibers bend more easily. The tactile response to hair varies with length. Short hairs can feel stiff (think of rubbing the head of a freshly crew-cut friend), while the same hair a few centimeters long feels completely different. The same applies to alpaca.

4. You can end up confusing the amount hair-mass per unit area from the number of hair follicles. If all else was equal (and it's generally not, as the point above show), one 30 micron fiber has as much hair-mass as four 15 micron fibers. First second fleece has four times the density (follicles per square mm) as the first.

5. Environmental factors can change the feel of a fleece, from humidity to the nature of the soil that the animal dust bathes in.

It is possible to account for all of these factors and make a good estimate of density. You would need to estimate the micron, look at the fleece structure, and examine the staple lengths. Then you juggle these variables together in your head, and make the final calculation. It takes time, and lots of practice.

Of course, there is a difference between thinking you know how to estimate density by-feel, and actually being able to do so. Just because you've had lots of practice, it doesn't mean you;re doing it right.

The way to measure you effectiveness is a "blind" test. This doesn't mean you work with your eyes closed! Rather, you are presented with a group of animals with known density measurements (either though skin samples, or by using the math I'll get into shortly). But the key here is that you don't know how dense they are. You study each animal, and make your results known. Then the true values are revealed, and you see how close (or not) your predictions were.

Training in this process can be helpful- but only if the person training you knows what they are doing. A wrong-method can be dutifully passed on from teacher to student just as readily as a correct method.

Of course, when you assessing the density of animals for use (studs) or purchase, getting out to get a feel for the fleece may not be possible. Maybe they were just shorn. Maybe they are thousands of kilometers away, and you can't justify the cost just to look at one or two animals. Or maybe you're surfing the web and have assembled a list of 200 animals across dozens of different farms you're interested in, you'd never have the time to go see them all.

I'm going to show you a technique called 'Normalization', this is how you can convert numbers so that when you compare two different alpacas you are, in a sense, comparing apples to apples. It does not provide a definitive answer as to the exact fleece density of an animal, rather it allows a large pool of animals to be compared to one another determine which are better and which are worse.

The first and most important factor to correct for is the mean micron. It takes four times as many 15 micron fibers to make a kilogram of fleece as it does 30 micron fibers. (So if animal A had 1 kilo of 15-micron fleece, and animal B had 1 kilo of 30 micron fleece, then animal A would have 4-times the fleece density.

The math behind this calculation is quite simple- I'm just taking the cross sectional area of a round fiber- πr^2. The table below lets you compare the relative weights of different finenesses.

1 kg of	Is equal to this many kg of fleece of a different micron (μ)								
	14μ	16μ	18μ	20μ	22μ	24μ	26μ	30μ	35μ
14μ	1.0 kg	1.15 kg	1.65 kg	2.05 kg	2.47 kg	2.94 kg	3.45 kg	4.6 kg	6.24 kg
16μ	0.76 kg	1.0 kg	1.27 kg	1.56 kg	1.89 kg	2.25 kg	2.64 kg	3.52 kg	4.78 kg
18μ	0.6 kg	0.79 kg	1.0 kg	1.23 kg	1.49 kg	1.78 kg	2.08 kg	2.78 kg	3.78 kg
20μ	0.49 kg	0.64 kg	0.81 kg	1.0 kg	1.21 kg	1.44 kg	1.69 kg	2.25 kg	3.06 kg
22μ	0.40 kg	0.53 kg	0.67 kg	0.82 kg	1.0 kg	1.19 kg	1.40 kg	1.86 kg	2.53 kg
24μ	0.34 kg	0.44 kg	0.56 kg	0.69 kg	0.84 kg	1.0 kg	1.17 kg	1.56 kg	2.13 kg
26μ	0.28 kg	0.38 kg	0.48 kg	0.59 kg	0.72 kg	0.85 kg	1.0 kg	1.56 kg	1.81 kg
30μ	0.22 kg	0.28 kg	0.36 kg	0.44 kg	0.54 kg	0.64 kg	0.75 kg	1.0 kg	1.36 kg
35μ	0.16 kg	0.21 kg	0.26 kg	0.33 kg	0.40 kg	0.47 kg	0.55 kg	0.73 kg	1.0 kg

Example: After years of use, it's time to replace you're stud 'Super-Duper.' You want boy who is a bit finer, but has at least the same density. Super-Duper clips 2.5 kg of 22μ blanket fleece. So all else

being equal (we'll get to staple length and body size later), how much blanket fleece would you need on a finer animal to be the same density?

For a 14µ stud you'd need a fleece weight of at least 2.5 kg * 0.40 = 1.00 kg

For a 16µ stud you'd need a fleece weight of at least 2.5 kg * 0.53 = 1.33 kg

For a 18µ stud you'd need a fleece weight of at least 2.5 kg * 0.67 = 1.68 kg

For a 20µ stud you'd need a fleece weight of at least 2.5 kg * 0.82 = 2.05 kg

So if you find a 16µ stud, he only needs a 1.33 kg blanket fleece to equal the density of 22µ 'Super-Duper'.

Next we need to consider staple length. Obviously a longer fleece weighs more. This is great for increasing total fleece yield, but can confuse matters when considering density. Simple example, if you had two animals (A and B) with the same micron, and the same fleece weight, but A had a 12 cm staple and B had a 6 cm staple, then B has twice the follicle density.

Don't necessarily discount the density of low fleece-weight animals. Their density may be fine, they may just need to be bred to an animal that will improve staple length.

Staple length consideration is especially important when considering first fleeces (which can be 15 cm or more) to the fleece of an older animal that is only cutting 6cm a year. Once you account for staple length you'll be surprised just who is the densest!

This leads of course to body size. When you've just shorn a 2-month old, 20kg cria, how can you fairly compare its density to a fully adult 75 stud male?

Micron and staple length play a major role, but you can correct for body size by measuring back length (from the bottom of the neck to the top of the tail). This defines the area that is clipped as the blanket. I know it is a linear approximation for a what is a changing 2-dimensional shape which is the region we call the 'blanket', but it will produce at least a ball park figure.

Adult animals generally have similar back lengths, so this correction factor can often be ignored. If you have an especially long-backed animal, it is probably worth measuring.

It is also worthwhile to measure (calculate) the density of the animal each year, as it changes over time.

A cria is not born with all of its adult hair follicles- they physically couldn't fit! Instead the skin of a cria has thousands and thousand of 'placodes', these are biochemical markers which say 'a hair can grow here'. That doesn't mean a hair will grow there, it is simply a limit on the maximum potential fiber density. A cria that is sick, injured, or underfed may never reach its full genetic potential, and not every placode will turn into a hair follicle.

Likewise older animals can suffer from declining density. We all know what its like when you get older and your hair starts getting a bit thin. But if you measure the density (correcting for micron and staple) you can then spot those animals that maintain good density later in life, a useful (and probably hertiable) trait you can use to add value.

Finally, a quick warning about breeding for any trait. Any trait, bred to an extreme, can be detrimental. It is possible that super-high-fleece density could lead to other health problems. Balance your goal of breeding for improved density with the overall health and welfare of your alpaca. More doesn't always mean better.

Dr Kylie Munyard

B. Sc. (Hons) PhD

Dr Kylie Munyard earned a B.Sc. (Hons) in Biology & Veterinary Biology from Murdoch University and her Honours thesis reported on her work at linking molecular markers to drug resistance in *Giardia*. She earned a Ph.D. in Agriculture from The University of Western Australia in 2000, where she studied the ecology of methanogens in the sheep rumen. After a hiatus from research, she commenced postdoctoral research at Murdoch University, in the laboratory of The Centre for High-throughput Agricultural Genetic Analysis. She was instrumental in developing molecular markers for sheep, and adapting standard and novel genetic tests to high-throughput format for sheep and cattle. She was also part of a team that developed and commercialised a rapid, high-throughput DNA extraction method. Along with two colleagues, she holds a patent covering the use of microsatellite markers in sheep.

Kylie is currently a Senior Lecturer/Researcher in Molecular Biology at the School of Biomedical Sciences at Curtin University, and has been there since mid-2005. Her main areas of research are alpaca genetics; quail conservation genetics, and diabetes. In 2006, with funding from the AAA, she worked with alpaca breeders to perform a survey of the incidence of staggers in Australian alpacas. Another project, investigating microsatellite markers in alpacas (funded by Morris Animal Foundation) has also been successfully completed. More recently two RIRDC funded projects have also been completed, the first looked at the genetics of colour in alpacas, and the second examined the extent of genetic contribution to micron blowout in alpacas. A project investigating the genetic cause of random coloured spots in white alpacas (funded by COALAR and supported by Pacomarca S.A.) is in its final stages. Also associated with these alpaca projects are a number of publications already in print or in preparation, two PhD students nearing completion and an Honours student. Student presentations of the alpaca research results have garnered prizes at National and International conferences.

ALPACA COLOUR GENETICS: MENDEL MEETS MOLECULAR

Over the past 7 years the team at the Alpaca Molecular Research group at Curtin University has been researching the inheritance patterns and molecular causes of colour in alpacas. Using a combination of Mendelian genetics principles, molecular genetics techniques, objective chemical analysis of the fibre and observation of skin and nail colour we have been able to arrive at a model that, we think, describes most of the colour variation in alpacas. The current nomenclature for alpaca colours contributes to the confusion. One person's fawn is another's light brown, and one person's mid-brown is another's red-brown. We therefore also propose a new set of names for base colour varieties that reflects the genetic basis of the colour.

Alpaca colour genetics can be broken down into two parts, base colour and pattern. There are only two genes that control the base colour of the animal, MC1R and agouti. However, there are several genes that control the many patterns that are possible (e.g. classic grey, roan, greying, tuxedo, piebald, appaloosa, vicuna, dilution). Any base colour can co-exist with any pattern, more than one pattern, or none of the patterns.

Base Colour

The base colour in alpacas ranges from white to black, through fawn and brown, with or without black on the extremities. The base colour arises because the genes MC1R and agouti work together in the pigment producing cells to tell the cells what colour pigment to produce. Mammals can only produce two types of pigment, yellow and black, and all the variety of colour is produced by differing amounts and locations of these two pigments.

Agouti variants are probably responsible for most colours in alpacas. Our data suggests that agouti has four variants in alpacas, each of which leads to a different colour outcome. The difficulty in assigning an accurate colour classification (that is, determining which agouti variant is present) is that there is a range of colour intensity for each variant. The most dominant agouti variant, "A" produces white through to fawn fibre. The next most dominant variant, "A^{b}" which we propose to call "bay" is characterised by a tan/brown body with black on the extremities (the same as bay in horses). Next in the hierarchy is "a^{t}", which we propose to call "black & tan". This one produces a black body with tan on the undersides, similar to a Doberman dog, and could be considered to be a reverse of bay. Finally, the most recessive agouti variant is "a". An alpaca with only "a" present will be black, and should more correctly, from a scientific point of view, be called recessive black. All animals with an Agouti base colour have black skin regardless of the colour of their fibre. Because each individual has two copies of its genome, each gene can have up to two variants in a single animal. Therefore, these four variants can occur in 10 different combinations, leading to the huge range of different shades of base colour. The use of the term "brown" to describe any alpaca colour is very misleading. From a scientific/ genetic point of view, "brown" describes a colour that is caused by a defect in black pigment, which makes the black pigment look brown. Our research has shown that the vast majority of alpacas described as brown are actually different shades of yellow. In fact we have not found ANY true brown alpacas, but we haven't tested them all, so we are being in cautious in saying "most". The darker "brown" alpacas are actually yellow with differing amounts of black mixed in. We have shown that:

- White and fawn alpacas have only small amounts of the yellow pigment, and negligible amounts of black pigment;
- Brown, dark brown and black brown alpacas have mixed yellow and black pigment in different proportions, and
- Black alpacas have about the same amount of yellow pigment as fawns, but have much more black pigment, so that the black pigment masks the presence of the yellow pigment.

MC1R is a relatively simple gene, it either allows (via the dominant wild-type variant "E") or prevents (via the recessive variant "e") the production of black pigment. So, agouti sets the base colour, then MC1R variants determine if the black part of the agouti colour will be allowed to occur or not. With white (AA) and fawn (AA^{b}), the fibre contains only negligible traces of black, so the only visible effect

of the preventative MC1R variant is on the skin, this is how you geet a pink-skinned white alpacas. Preventative MC1R variants have a greater effect on bay (A^bA^b), black-bay (A^ba or A^ba^t), black & tan (a^ta^t or A^ta) and black (aa) alpacas. Bay coloured alpacas become chestnut (A^bA^b ee), just like in horses. Black-bay (A^ba ee or A^ba^t ee) could be anything from chestnut to fawn in colour. Black, when accompanied by preventative MC1R variants (aa ee), becomes chestnut through to white, depending on how much yellow pigment was hidden by the black pigment. All of these dark base outcomes are determined by how much of the yellow pigment is present, the more yellow pigment, the darker the fibre colour. These darker "ee" alpacas will have dark skin, but it will be only as dark as the fibre, and will not be black. Nature has added a twist to this story. Some animals with pink skin (genetically) will develop black pigment as they age, in response to sun exposure. So, it can be hard to tell if an older animal has a) skin the same colour as its fibre, b) pink skin, or c) black skin.

If we add the two MC1R variants, which can occur in three different combinations, to the 10 agouti variant combinations, we now get 30 different colour outcomes possible from just these two genes. That is more than enough to explain all of the normal base colours in alpacas. Our research has identified the DNA signatures of the two different MC1R variants, and two of the four agouti variants.

Patterns

All of the patterns in alpacas are caused by genes creating a variation to the base colour. All of the pattern genes have a wild-type variant that does nothing, plus one or more variants that cause the pattern. Each alpaca will have two copies of every pattern gene, this could be two wild-type (do nothing) variants, or one each of wild-type and pattern-causing, or two pattern-causing copies. The patterns classic grey, roan, greying, appaloosa, vicuna, tuxedo, piebald, blue-eyed white and dilution will be discussed herein.

appaloosa, vicuna, tuxedo, piebald, blue-eyed white and dilution will be discussed here.

Classic grey (M)

Silvergrey and rosegrey are the result of a single pattern variant acting on different base colours. In our lab we call this pattern classic grey and the gene symbol is "M" (named after the pattern "merle" that it resembles) until the gene identity is confirmed. Classic grey is used as a term as opposed to simply "grey" to differentiate it from the well-known greying characteristic found in many other species (and possibly in alpacas too). Silvergrey (aa E- Mm) is a classic grey variant on a black base colour. Rosegrey is a classic grey variant on any other base colour. This explains the huge variety of different rosegreys that are seen. The typical signs of a classic grey are that the neck and legs are paler than the body, the body is a diluted version of the base colour, and that the overall depth of colour of the animal often increases with age. They also commonly have a pale face, and a non-diluted bonnet of colour on the head. When you examine the fibres under a microscope they are not a mixture of white and black fibres, instead the fibres are diluted to different degrees from white to the fully intense base colour. Some classic greys have spots or patches of undiluted fibre in random places. The classic grey variant is easy to see on a dark background, but can be very hard to see on a pale background, and impossible to see on a white. Genetically one copy of the classic grey variant is required to be present to cause the classic grey pattern (Mm). In genetic terms it is an incomplete dominant. Pedigree analysis by Elizabeth Paul has shown that the presence of two

copies of classic grey (MM) is lethal at the embryo stage of development. Hence, you don't get any "true breeding" classic greys. Our analysis of pedigrees, combined with work done by Dr Belinda Appleton suggest that there may be at least three different classic grey variants, each of which produces a different version of the pattern.

Roan (Rn)

Roan is in many ways the reverse of classic grey pattern. Roan animals have a diluted body with undiluted neck and legs. Microscopic examination of the fibre shows that the roan pattern is caused by a mixture of fully pigmented and white fibres. In contrast to classic grey, a roan will get paler with age, and the body may end up almost completely white. Roan animals are rarely born with the pattern showing, that is, a black roan will be born black, and will develop the typical dilution effect over time. The roan pattern can occur on top of any base colour. Similar to classic grey, the roan pattern is most easily seen on a dark background, and in our experience, white and fawn roans may not even be recognised as such. Roan is not the same as progressive greying, although it can be hard to tell these two patterns apart. Roan is also an incomplete dominant. Only one roan variant is needed to cause the pattern (Rnrn). However, preliminary data suggests that when two roan variants are present the pattern progresses more quickly, and is not lethal (in some species homozygous roan is embryonic lethal). Breeders wishing to get "whiter than white" fibre could introduce the roan pattern into their herd to remove any traces of pigment from the fibre.

Greying (G)

Age-related greying occurs in most species, and is caused by the premature death of stem-cells in hair follicles. It is not clear whether this kind of grey occurs in alpacas as a distinct separate pattern, or if the milder forms of greying are a third variant of the roan pattern. The physical attributes are similar to roan, except for the differentiation of effect between the body and legs. The inheritance pattern is unknown.

Appaloosa (Lp)

Appaloosa is not, as most people think, a pale background with coloured spots. It's a dark background (what is perceived as the spots) with pale spots (what is perceived as the background). Appaloosa can also occur on top of any base colour, and you can clearly see the bay base colour distribution of black and yellow pigment in some appaloosas. The pattern of inheritance has not been proven, but it is probably dominant or incomplete dominant. That is, one copy of the appaloosa variant is enough to cause the pattern (Lplp), and two copies will cause the same pattern (LpLp). Similar to all of the other patterns, appaloosa can't be seen on a white alpaca.

Vicuna

Vicuna is an intriguing pattern. This is the pattern where a fawn alpaca has white undersides with white extending onto the body behind the front legs. We have not completed a thorough analysis of this colour, and are hoping to do so as soon as possible. Two hypotheses to explain this pattern are currently under consideration. The first is that vicuna is a separate pattern in its own right, and the second is that it is simply a manifestation of the black & tan agouti base colour with non-permissive MC1R variants.

Tuxedo/Piebald

Animals with white patches are tuxedo or piebald. This is the only pattern that is localised to specific regions of the body. In effect the presence of a tuxedo or piebald variant leads to white spots of varying number, size and shape. It is not definitively known if tuxedo and piebald are caused by different genes, or by different variants of the same gene. It is also assumed that these two are distinct patterns. However, the evidence suggests that the tuxedo pattern is restricted to the head, neck and legs, while the piebald pattern occurs on the body as well, and tends to cross the dorsal mid-line. Both tuxedo and piebald are dominant, that is, only one copy of the pattern variant needs to be present to cause the white pattern. Therefore, in any mating where one parent is white and the other is a solid colour and the cria is tuxedo or piebald, it is probably the white parent that has contributed the white spotting pattern. However, even a tiny amount of white on a solid animal is an indication that it is actually tuxedo or piebald.

Blue-eyed white

Blue-eyed white (BEW) is the most controversial of all alpaca patterns. The evidence indicates that classic grey is strongly implicated in this pattern. If a BEW is mated to a solid dark colour, the most common outcomes are classic grey or tuxedo, which suggests that BEW is a combination of two pattern variants that leeches all of the colour from the animal.

Dilution

The final pattern being discussed is not really a pattern at all, but it does affect the base colour of the animal, so it fits in this section. Our research has shown that animals with the exact same gene variants present at A and E can be different colours. For example, fawn versus dark fawn. Therefore, there must be other genes acting to dilute out the colour in a uniform way. These genes are known to occur in other species, "D" in dogs causes black to appear steel grey, and brown (real genetic brown) to appear milk chocolate coloured, and red to appear champagne. In horses you see the very striking palomino and silver dilutions. Each of these dog and horse dilutions is caused by a variant of a single gene. We are currently analysing gigabases (i.e. billions of bases) of alpaca RNA sequence to try to find a gene or genes that have a similar effect in alpacas.

Summary

The colour of an alpaca is controlled by its genes. If you evaluate colour in an objective way, you can usually work out which genes, and which variants of those genes, are creating the colour you see. If you also include information about an animal's parents and offspring, the success in predicting colour will increase markedly. DNA tests for these genes can be used to determine the genetic potential (in terms of colour) of an animal, and therefore allow a breeder to plan matings to produce (or not produce) a particular colour. We suggest that the names used to describe alpaca colours should reflect the genetics of those colours, so that there is more effective communication and more precise records, leading to higher predictability in breeding outcomes.

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Andrew Padula is a veterinarian based in Bairnsdale, Victoria. Andrew has a PhD from the University of Melbourne and has European Veterinary Specialist qualification in ruminant herd health and reproduction. He has worked in academia, industry issues management and private veterinary practice. He has a driving interest in applications that utilize animals to improve the lives of humans. Andrew is also an honorary senior research fellow of the Faculty of Medicine, University of Melbourne.

IMMUNOGLOBULINS

Immunoglobulins, more commonly known as antibodies, are an essential molecule in the mammalian immune system. This RIRDC funded project has explored the use of alpaca for the production of medical grade therapeutic antibody products. This report is a brief overview of the opportunities and processes involved in production of therapeutic antibodies.

All members of the camelid family have been shown to produce a unique class of immunoglobulin molecules in their blood stream (Hamers-Casterman *et al.* 1993). These molecules can be refined and harvested for production of specialised therapeutic medical products. The unique properties postulated for the camelid antibodies include reduced allergenic potential, greater heat stability and greater capacity for inactivating certain enzymes.

The use of alpaca for therapeutic antibody production has potential to create a high value niche market. This would expand the opportunities much further than the traditional meat and fibre markets. Australia is recognised internationally as one of the lowest disease risk countries in the world for producing products derived from animals. A low zoonotic disease risk is the essential basis of production of therapeutic antibodies. There is now over 150,000 alpaca currently in Australia and prices per head have fallen to very affordable levels. Thus alpaca present an attractive proposition for production of medical grade therapeutic antibodies.

Therapeutic antibodies are molecules found in the bloodstream that can bind to, and inactivate, a wide range of chemical substances. There is a billion dollar global demand for therapeutic antibodies. A diverse range of medical products with well-defined markets already exists. Amongst these include various anti-toxin serums for snakes, spiders, bacterial infections (tetanus, rabies, botulism, anthrax), emerging anti-cancer therapies and a host of other applications. Currently these products are produced primarily in horses, sheep, rabbits and a small range of other animal species. Camelid antibodies have exciting potential to improve the quality of products available in this existing product marketplace.

The other substantial market for antibodies is in diagnostic test kits. Perhaps the most well-known of these, at the everyday consumer level, are human pregnancy tests available over the counter at chemists. The test is usually performed on urine samples. At the core of these simple immunological kits are antibodies that capture and bind the molecule of interest. For human pregnancy diagnosis the molecule associated with early pregnancy is human chorionic gonadotrophin. When the capture antibody in the test kit binds to the target molecule a colour change occurs in the test strip. The better the quality of the antibody the more sensitive and specific these tests can be made. The same principle is also used for many drug detection rapid tests. In fact the range of diseases and molecules that can be tested for is almost limitless. In parts of the world with malaria the rapid test kits are now widely used for diagnosing the condition. The market for rapid tests kits has expanded enormously in recent years. Typically rabbits, goats and chickens are used to make the antibodies. The alpaca has potential to replace those animals for commercial scale production.

The production of antibodies requires stimulating the immune system with the product (known as the antigen) that the antibodies are desired for. Artificially high levels of antibody result from this hyperimmunisation process. This process takes time for the animal's immune system to respond with high levels of specific antibody. Blood is then collected from the animal and processed to concentrate the specific antibody fraction. The product is then tested for its potency and a range of other physiochemical parameters. Typically these products are final-packaged in sterile glass vials for human or veterinary medical use. Our work has shown that alpaca can respond to artificial hyperimmunisation regimes with similar responses to sheep, goats and horses. We have also demonstrated that serum can be repeatedly harvested from alpaca without harming the animal. We have also developed methods of monitoring the immune response in alpaca. Our results have shown that there is wide variation between alpaca in how they respond to immunisation. This presents an opportunity to screen animals and select the best responders to work with.

This project has specifically explored the potential for alpaca to make therapeutic antibodies against snake venom toxins. Snake venom toxins were chosen because, for an antibody to be truly effective, it must neutralise the actions of the venom. This provides a very robust demonstration (ie prevention of death in a mouse model) that all of the steps required to produce the neutralising antibody are working efficiently. Snake venom is a complex pharmacological substance with many different components. This project has also examined the antibody response of alpaca to different venom components. Alpaca respond to venom immunisation with rapid development of neutralising antibodies. Extremely low doses of venom are used which do not harm the animals. The alpaca serum can be processed with modification to existing protocols used for other species. A very pure and concentrated antibody product is then produced from alpaca serum. When tested for venom neutralising capacity the experimental products were highly effective. However the serum processing appears to be less efficient than with other species but more work is required to optimise the methodology.

This project has demonstrated that alpaca can be used for efficient production of camelid-type antibodies. The abundance of alpaca in Australia and their affordable price presents many opportunities for using this species for antibody production. Therapeutic antibodies are a high value-niche market industry. Australia is placed very favourably in terms of low disease risks for using alpaca to produce various antibody products for use in Australia and internationally. Aside

from existing markets, new product applications are constantly emerging. This proof-of-concept study has demonstrated that alpaca can be used successfully to make traditionally difficult to make therapeutic antibody products. The next steps will require further investment to explore and expand into the various potential market opportunities.

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Elizabeth Paul

Photo not available

As a biologist, Elizabeth Paul has always had a deep interest in various aspects of alpaca biology. As a craftmaker she has, in conjunction with her family, run craft stalls for many years making and selling many different types of craft work. From the very start of her involvement in the alpaca industry, Elizabeth has been working with and selling alpaca fibre both as fibre, and as value added items, with particular emphasis on using the natural colours to best advantage. She believes that breeders who actually work with their own fleece, will not only have a better understanding of their breeding goals, but can also improve their return by improving their fleece presentation, whether for their own use or for further processing.

My aim in running this workshop is to help other breeders to work with and use the fleece that they already have. From a craft point of view, the important parameters are handle, cleanliness, strength (tensile, not micron) and colour. What is even more important is how easily good fleece can be degraded, by inattention to preparation. Topics will include fleece selection, preparation such as skirting, washing and picking, and presentation for use in craft work, or for further commercial processing. I will also discuss how to improve cria fleeces, including clipping; tips on using fleece in various crafts; and there will be samples of various stages of fleece from the raw state to finished products.

WORKSHOP: USE OF COLOURED FLEECE

Introduction

My aim in this workshop is help breeders utilise the fibre that they have. It was the stunning range of natural colours that attracted me to alpaca fibre in the first place, and I have used all colours, with more emphasis on grey shades. My family and I have been making and selling items on craft stalls for many years, and in the last 5 years I have made mine exclusively an alpaca stall.

I also place a great deal of importance on having clean, well presented fibre whether I am sending it for commercial processing or for onselling to other craft workers. If a fleece is matted with clover burr, or heavily contaminated with short hairy bits, processing is more difficult, the end product will not be as good, the return will be less, and the whole exercise will have cost you, the fibre producer, more. As a member of the craft community, I have also heard some very negative comments from other craft workers who have already been sold or given very poorly presented alpaca fibre.

Selection

The most useable fleeces are those shorn between the ages of 2 and 5 years old, assuming annual shearing. I have used fleeces of up to 10 year old animals. Parameters of importance are handle, length, tenderness (breaks), and above all, as free as possible of **vegetable matter.**

Micron

I prefer a micron range of 22-30u, average about 25u, with the handle assuming more importance in broader micron fleece. The fibre is physically more robust and more easily worked. Given a choice, I would prefer an older, higher micron, cleaner fleece, to a softer, very tippy cria fleece.

Preparation

Preparation of fibre is all important. Even average grade fibre can be improved with good preparation. On the other hand, good quality fibre can be seriously degraded, due to lack of attention before processing.

Vegetable Matter or VM

A fleece heavily matted with vegetable matter is almost useless. If the VM can't be removed by hand, it will not be removed in processing, but will become evenly distributed throughout the finished product. Early shearing, before seed set, is the first and most useful thing breeders can do, to help with this problem.

Fleece Separation

There are three parts to a shorn fleece, the saddle or the main part of the fleece; the neck, which is shorter; and the leggings or pieces, which are all the really short hairy bits. These three groups need to bagged separately at shearing. Remember that commercial fleece processing is usually charged on weight in. The more rubbish is in the fleece, the lower the return ie it's costing you money.

Handle and Strength

Handle or feel of the fleece, can be affected by dust levels, vegetable matter, dampness, etc. besides the actual micron. A more uniform fleece will have better handle. A tender fleece is one which will break somewhere along the lock, thus reducing the length of fibre and causing greater loss. Breaking close to one end is less of a problem than breaking in the middle. To test a fleece for tenderness, remove a whole lock and hold taut between two fingers, and flick with another finger, or try to pull the two ends apart. Also reject a fleece that feels chalky or hard, remembering that in a drought year, a fleece can be stiff with dirt, which will just wash out. It has been my impression that fleeces from last year have been softer, cleaner and longer, probably due to the extra rainfall.

Skirting

Skirting a fleece is the removal by hand of all the matted dags or really hairy bits, dung clods, and vegetable matter that accumulate over a year on any fleece. Often the fleece is shorn off in two halves, so spread just one out on to a large flat table.

The first part of the fleece to skirt off, is the **bird's nest.** This is the point on the alpaca where the neck joins the backbone, and where the animal bends its head up and down. This bending constantly opens and closes the fleece, leading to a big concentration of vegetable matter. This fleece is usually very fine, but useless. Then go round the edge of the fleece, picking off the matted and cloddy bits, also check for wire pieces or other rubbish. The more rubbish that you can pick off the fleece, the better. In a dark fleece, also look for lice eggs, often on the shoulder fleece.

While there have been great improvements in breeding, it would be fair to say that the average coloured alpaca fleece is still quite variable in type. The best ie the finest fibre with the least hair will be found on the saddle, that is over the higher part of the flank from the neck, about half way along the back, but more towards the front. The saddle fibre is also shorter. The rear and lower parts are often longer, and hairier, but if they still have good handle, by no means useless. I usually end up with good saddle, and longer bits put aside.

Long fleece

Long fleece for handspinning is not a problem. Really overlong fleece wraps itself around machinery. Overlong fleece would be the wether that hasn't been shorn for two years and is quite likely matted with dirt, straw etc. Bin it.

Short fleece

Short fleece is more of a problem, as the shorter the fleece the more is lost in processing and the harder it is to spin. If too short for spinning, it can still be used for felting or stuffing. Check with your processor beforehand, what their preferred limits are.

Picking

The purpose of a picker is to break up the locks of fleece; it also blends the fibre and helps to remove some of the hair and cloddy bits. The hand picker that I use is a rocking cradle with two sets of long steel spikes, and looks like a weird mediaeval torture device.

Real fibre is broken up into individual fibres, floats up and drifts down into the bag. Hairy and cloddy bits tend to stay together so they are more easily picked up at this point. The rocking picker does not remove small vegetable matter, the tiny bits will be nicely blended all the way through. I only put a skirted fleece free of vegetable matter, through the picker. I discard the fibre and bits caught in the bottom of the picker. The fibre caught in the top half, can be pulled off (carefully) and added to the bag, as it will mostly be good.

From a colour point of view, as I have mostly grey and black fleeces, I start with a cleaned picker and put the best black through first. A bit of extra black in a grey fleece is no problem, but not the other way round. If I was using white and fawn, then the best white would go through first, then the lightest fawns, darkest fawn or brown next, and a rosegrey would go through last of all.

Dehairing

Large commercial wool processors most likely do not have or use dehairing machines for two reasons: if they are mostly processing fine merino wool, there should be little need for a dehairer, because the merino industry has been breeding hair out. Also dehairing takes a lot of time, and therefore adds extra cost to the processing.

Mini mills do have small dehairing machines, and this has made a great improvement to the quality of the finished alpaca yarn. The downside is that the return on very hairy fleece will be reduced. If breeders want to maximize their return, they need to skirt out (or breed out) as much coarse hair as possible.

Carding

Carding involves combing the fleece usually around a small rotating drum to make the fibres parallel. After several turns, the fibre is lifted off the drum in one piece called a batt. Hand drum carders are very useful to process small amounts of fleece, and for blending. In my opinion, the pins should be of finer wire, and a little closer together, than the short, fat, wide apart pins that are in normal use for sheeps' wool. I have not used flat carders much, as the length of alpaca fleece is a problem for these, and to me it just looks like a tangle.

I have used an electric drum carder but found it almost impossible to turn back the drum against the weight of the engine, to get the fibre off. Also it had a back roller, which for sheeps' wool combs the fibre down, but just seemed to pull the alpaca fibre up again. I sold it, my old drum carder broke, and now I just use the picker, it's quicker.

Coloured Fleece

Processing of any kind tends to blend all the fibre colours together. Different colours from one fleece can be separated at skirting. On a silver grey fleece, I leave black spots in, but skirt brown spots out. On a rosegrey fleece, I leave all the spots in unless there is one very large spot. If you want to see the variations in colour of an appaloosa fleece in a yarn, it should be handspun from the raw fleece, without carding or picking. (No it won't be spotty!)

Batching

In order to get a bigger lot for processing I often batch several fleeces that are similar regarding micron, length, handle, colour ie if they are most uniform. I also batch different colours in order to make another colour, or I combine leftovers to make a pieces batch. Depending on the quality of the pieces this can turn out quite useable yarn. As a general rule, I do not batch fine micron with broad micron to try and soften the broad fleece. Better products of both are obtained by keeping fleece of widely dissimilar microns separate.

Herd vs Yarn Colours

See Table 1 for a comparison of the cria colours from approximately 145,000 matings recorded in AAA Inc Herd Books 4-21, and the yarn colours that I use. Of 100 kg of spun yarn, 60% was a shade of grey.

Black fleece

I only use the blackest of black fleece, for black yarn. Any black with tips goes into bay black or dark chocolate brown; a dull or dusty black, or black roan would go into a dark grey batch. Often black fleeces have better handle than other colours, even at higher micron. I tend to make smaller batches of black, or process individual black fleeces, to maximize the handle.

Brown Fleece

Browns often have finer fleece than other dark alpacas. I use the bulk of my browns batched with white and grey, to create various shades of darker rosegrey.

Greys

Grey from a silver grey alpaca is a mix of grey, white and black fibres. However batching black and white together makes a good substitute grey. I usually batch several silver grey fleeces matched in shade together to make a bigger lot. To make a charcoal grey I prefer to add extra black to a real grey. Brown and white can make a darker rosegrey, but lavender rosegrey is difficult to reproduce by batching colours.

Washing

After trying to spin my first raw fleece, sitting in the attendant cloud of dust for ten minutes, I gave up on that idea forever. When I was handspinning I washed all the fleece before putting it through the carder, and then I would wash the batts again before spinning. Now I put it all through the picker first without washing, before sending for processing to skeins, and then I wash those to remove the added conditioner.

Use warm to quite warm water with a drop of shampoo (any). Put handfuls of the fleece into net bags, push down into the water several time to expel the air. Even though it doesn't have lanolin, alpaca fleece is not very wettable, and there will be pockets of air where the fleece doesn't get wet. Leave for 15/20 minutes. Drain and squeeze out the water, then place the bags in the washing machine on spin cycle to get the excess water out. Rinse in clean water and spin again. Then the bags can be shaken up and put outside for drying. You will need somewhere to contain the drying fleece, or it will all end up down the paddock.

For skeins, unwind the skein from its figure 8 shape and immediately tie a piece of waste wool around the other end from the tie. Then place eg two skeins in the net bag, wash as above. The bags can be wrung out, as long as you don't rub the wet fleece or skeins. Spin the excess water out, rinse and spin again. Remove the skein, hold by the tie end and flick out to straighten the wet yarn, and drape the skeins over a clothes line/stand for drying in the sun. Turn the skeins over at least once to dry the other side. A warm windy day is the best day for drying.

Alpaca fleece/yarn must be fully dry before storing, as wet fleece will go mouldy quickly and the smell is almost impossible to remove. Shorn fleece wet with urine must be dried or washed immediately, as if left bagged, it will have to be binned. I dry skeins over at least two days to be sure, or put them in front of a heater. Vegetable matter can also be picked out of the skeins before using or balling up.

Cria Clipping

Cria fleece may be the finest fleece that an alpaca will ever produce, but for what I make, it is less useable than older fleeces. The main reason for this is the cria tips, which can accumulate a large amount of rubbish before first shearing. Crias born a few weeks prior to shearing, can be shorn by the shearer. Crias born post shearing can be clipped using small animal clippers with a skip tooth blade. I prefer a No 4 or No 5 blade. The best age to clip a cria is 1-2 weeks if the cria is strong and healthy. A very young cria is smaller, and the fleece is shorter and cleaner, all taking less time and effort. The cria can be sat in cush, stood up or even held by someone else. The clippers are used in short bursts, rather than a long pass. I start at the rump, and turn the clippers to one side frequently to get rid of accumulated fleece. Very young crias may need a cria coat for a couple of days.

Colourwise, blacks and greys show the most benefit from cria clipping. I usually have at least two mums and crias in, so that the mother is not too anxious while bub is gone. Overall I have had very few problems with cria rejection, but breeders know their own females best, and obviously a cria with a nervous or stressy mum should be left alone until shearing. Clipping notes of course only relate to huacaya crias.

Combing Fleece

If not too heavily tipped, shorn cria fleece can also be retrieved, by combing out the tips of each lock with a dog comb.

Crafts

Knitting/Crochet

Most of my fleece is processed into 8 ply knitting yarn, for handknitting and/or crochet work. See Table 2 for yarn production. I use 90% of my own yarn for knitting small items. I prefer to knit alpaca into simple rib or cable patterns, which help give springiness to the fabric and still reflect the lovely handle. I do sell the yarn as well, but far fewer people knit these days. Alpaca also comes up beautifully in crochet work. I have used 8 and 5 ply in a handknitting machine, but electric knitting machines require finer plys on cones.

Weaving

Alpaca was made to be woven, because it drapes beautifully and the colours can be seen to best advantage. I use commercially processed yarn for more classic designs, and handspun or even carded fibre for a more rustic look.

Handspinning/Felting

I usually have a small amount of washed and carded/picked fibre on hand for for handspinning and felting. I also have skirted fleece, and clean pieces for stuffing or felting. I make a point of having all these as clean and free of VM as if I was using them myself, although I don't necessarily wash them.

Adding Other Fibres

Apart from silk, I feel that adding other fibres to alpaca detracts from the handle. Between 5-10% wool is usually added to commercially sold alpaca yarn to improve the processing and memory of the yarn, and there are cotton and acrylic blends as well. Woollen yarn made with 5% alpaca, is pointless.

Dyeing

My emphasis is on using the natural colours. Obviously white alpaca is dyed commercially, however for those interested, both fleece and yarn can also be dyed with natural dye materials such as boiling concoctions of dahlia flowers, birch bark, onion skins, lichen, berries etc. Light fawn and grey can also be overdyed. Alpaca will take a more muted, softer version of wool colour in the same dye bath. Note that dyeing, whether with natural or commercial dyes, plus heating and added mordants (colour fixing agents) will harden the fibre. Fading could also be an issue.

Summary

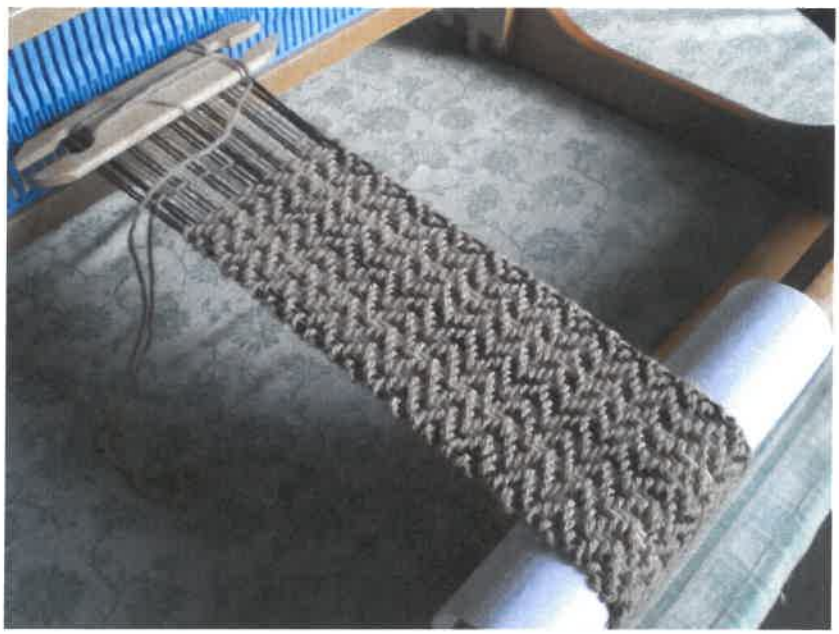
Whatever is made with alpaca should reflect the two best aspects of the fibre, namely the colour and the handle. It is really up to us, the breeders, handlers and workers of this wonderful fleece, to use and present it in the best possible way, to get the most positive feedback.

Table 1: Colour Comparison

Colours	**White**	**Fawn/Brown**	**Black**	**RG**	**SG**
HB 4-21	33%	48%	13%	3%	3%
Yarns	6%	23%	12%	21%	38%

Table 2: Yarn Production

Year	**2008**	**2009/10**	**2012**	**2013**	**2014**	**Totals**
Kg In	10	37	28	27.3	34 (+10)	136
Kg Out	8	28	21.4	19	25.5	102
Return	80%	76%	76%	70%	74%	75%
Balls 50g	147	670	382	384	470	2000
Used	100%	100%	80%	25%	4%	40%



Allison Quagliani

Alpaca Dental Services
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Allison's experience with Alpacas began in 1996 when she and her husband Danny bought their first two alpacas. They live in Toomuc Valley, south east of Melbourne on their small farm and run a herd of around twenty alpacas.

Allison first became interested in Alpaca Dentistry when one of her own animals developed a tooth problem. Allison had trouble finding someone who could fix the problem and realised there was a need for a practitioner who specialised in dental care for alpacas. Allison decided to address this gap and in 2004 commenced an apprenticeship with a Master Equine Dentist who had experience working with alpacas. Allison carried out extensive study and research covering all aspects of alpaca dental care. She also developed and had manufactured specialised instruments and equipment to make the inspection and treatment of alpacas as safe and stress free as possible.

Alpaca Dental Services started providing dental care to the alpaca industry in January 2006. Today Allison travels extensively throughout her home state of Victoria attending to her many clients. Allison has written a number of papers and regularly speaks at conferences and events on the subject of alpaca dentistry.

TALKING TEETH

Abstract

Alpacas spend more than half of their lives eating and chewing. It is essential as their carers that we understand the basics of alpaca dental anatomy and the effects dental problems can have on the overall health and welfare of our alpacas. This paper will provide an overview of alpaca dental anatomy, discuss the most common problems alpacas have with their teeth and provide owners with a guide to identifying individuals within their herds, that may need dental attention.

Introduction

As in other animals, the mouth is the first stage of the digestive tract. A correctly balanced, well functioning mouth allows the alpaca to graze and chew both comfortably and efficiently -essential for the overall health and wellbeing of the animal. Good teeth play an important role in this process. Dental disorders are not only painful for the animal, (to which many humans can relate) but left untreated will have a detrimental effect in many areas including body weight, fibre quality and the ability to reproduce.

Dental Anatomy

Alpacas have a total of 30-32 teeth comprising of six incisors, six fighting teeth and 18-20 cheek teeth (Fig 1).

During their lifetime alpacas will have two sets of teeth. The first teeth, known as deciduous or baby teeth, are temporary and will be replaced between two and four years of age with permanent teeth.

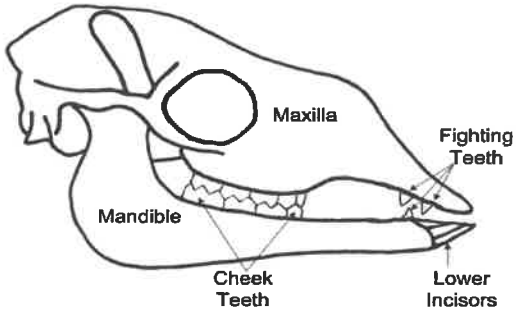

Fig.1 Alpaca skull showing dental anatomy

Incisors

The incisors are the six teeth in the lower jaw at the front of an alpaca's mouth (Fig 2). They are designed to fit snugly against the dental pad of the upper jaw. They are used to grasp and cut grass, leaves and shrubs.

Cria are usually born with their front two incisors and by six months of age will have all six. These first incisors are deciduous teeth and they will be replaced commencing at around two years of age with permanent teeth. By the time the alpaca has reached three and a half years all six deciduous incisors will have been replaced.

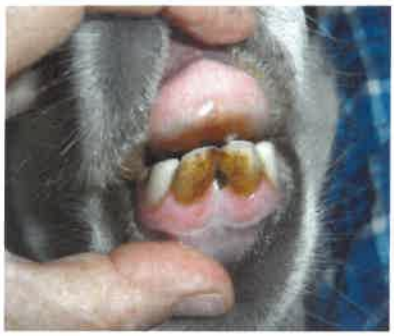

Fig.2 Incisors

Fighting teeth

The fighting teeth of an alpaca are located behind the incisors (Fig 3 and Fig.4). There are two in the top jaw and one in the bottom jaw on both sides of the mouth. These teeth are very sharp, curve towards the back of the mouth and are well designed to rip and tear. Males have well developed fighting teeth and can cause serious injuries to their paddock companions during fights for dominance.

Females usually have fighting teeth but they are much smaller than those of a male.

Fighting teeth erupt at around three years of age and should be trimmed to prevent serious injury to other alpacas.

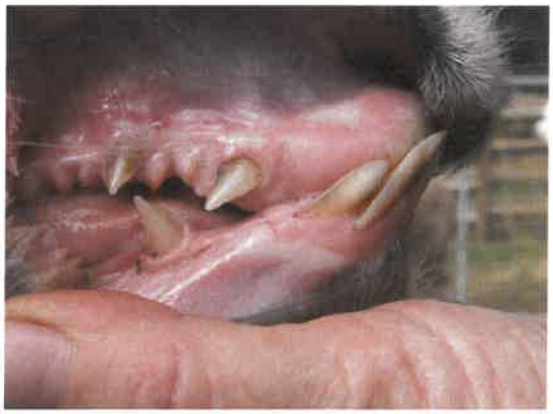

Fig.3 Fighting Teeth of a Mature Male

Cheek Teeth

At the back of the mouth in both the upper and lower jaws are the premolars and molars, usually referred to as the cheek teeth (Fig.4). The cheek teeth are arranged so the upper and lower arcades (rows of teeth) mesh together to produce an efficient grinding surface. These teeth do all the hard work grinding the food to a consistency suitable for swallowing.

Fig.4 Skull Showing the Cheek Teeth and Well Developed Fighting Teeth

Occlusion and Malocclusion

The meeting together of the upper and lower teeth and the dental pad is referred to as occlusion. If the teeth do not occlude correctly then this is referred to as malocclusion. The teeth of an alpaca form and grow in the jawbones beneath the gums. These teeth erupt into the alpaca's mouth and wear away against the opposing teeth/ dental pad as the animal bites and chews. This process of continual wearing and eruption continues throughout the life of the alpaca until the teeth are worn out. Any uneven wear will cause a malocclusion and hamper the animals' ability to graze and chew correctly.

Malocclusions cause varying degrees of discomfort or pain to the animal. While times are good and food is plentiful the discomfort may not seem so obvious. When times are tougher, for example during times of drought, late pregnancy or lactation the effects of malocclusions become more pronounced.

Incisor Malocclusions

Undershot jaw (Fig.5A and Fig.5B) is one of the easiest malocclusions to recognise as the lower incisors protrude beyond the dental pad. Overshot jaw (Fig.6) is when the dental pad protrudes beyond the lower incisors.

Both of these malocclusions can easily be seen by parting the lips and looking at the conformation of the mouth. These problems have been proven to be hereditary in other species (Fowler, 1998, p. 483)

Fig 5A. Undershot jaw front view

Fig 5B. Undershot jaw side view

Fig 6. Overshot jaw, side view

These alpacas can still hold good condition on long pasture or with supplementary feeding. When pasture is in short supply and these animals have to forage for themselves their ability to hold condition is compromised.

Molar Malocclusions

Between birth and four years of age an alpaca will replace its deciduous premolars and twelve permanent molars will also erupt at the back of its mouth, three on each side, in both the upper and lower jaws. As you can imagine this eruption pattern doesn't always go to plan.

When the upper and lower cheek teeth do not occlude correctly the unworn teeth become so long that they grow into the opposing gum and in extreme cases the bone is also damaged (Fig.7).

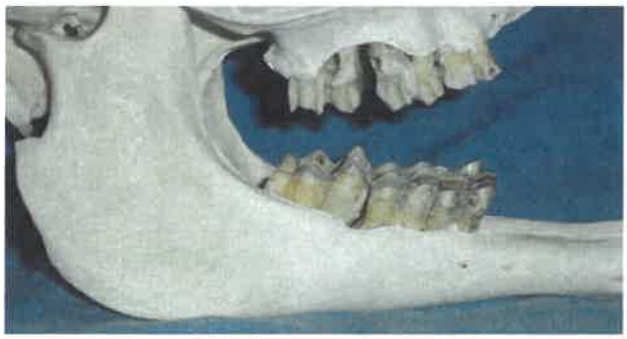

Fig 7. An overlong molar in the lower jaw has worn a gap between two of the top teeth. When this alpaca's mouth was closed the long tooth was gouging the gums and has also damaged the bone.

Recognising Malocclusions

A dental abnormality will manifest itself with one or more visible signs. Regular observation of your alpacas will identify small problems before they become major challenges. Animal age, conformation, seasonal conditions, diet and regional soil variations can all have an impact on dental health. Alpacas should chew evenly on both sides of their mouth without dropping food to the ground.

Often loss of body condition is the first symptom owners become aware of. Some animals may show a reluctance to eat, dribbling, an abscess or swelling in the cheek area and obvious pain whilst chewing.

Fig.8 A 'quid' held in the cheek area. A sign of serious tooth problems

Swelling in the cheek area may be due to the holding of grass or hay in the side of the mouth referred to as quidding (Fig.8). Alpacas with overgrown or very sharp molars use this ball of food to protect their cheeks and gums from the pain incurred while chewing their food. The quid is not swallowed but spat out and you may find them on the ground in the area where the alpacas live.

Conclusion

A correctly functioning mouth is an integral part of alpaca health and well being. Malocclusions can have significant negative impacts but can usually be corrected to a degree sufficient to restore animal health.

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INFANT ANIMAL NUTRITION – HOW DO WE GET IT RIGHT?

Introduction

The challenges we face with the neo-natal digestive system of any animal are the same. Cria, like all infants, are born with a sterile gastro-intestinal tract (GIT) and, as such, it is important to remember that the environment, either in the paddock or the barn, into which they are born, lends itself to them being exposed to more pathogenic organisms than beneficial ones, post-parturition/birthing.

The question we need to address is: What can we do in our husbandry practices to ensure that we maintain a healthy infant and, more importantly, one with a GIT that is optimally functional as soon as possible post-birthing?

This talk is not intended to break any startling new ground in nutrition but will rather cover some of the basics that we need to get right to address the question posed above.

Hygiene

The world renowned animal scientist and animal behaviour expert, Professor Temple Grandin, when asked at the Professional Calf Rearers Association meeting a few years ago what the 3 most important things were to consider to successfully rear calves, responded:

"Hygiene, Hygiene and Hygiene!"

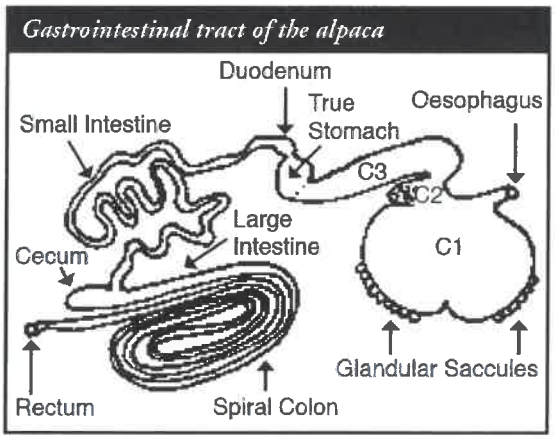
What would hygiene have to do with cria nutrition? Simply put, the environment into which our progeny are born needs to be as clean as possible, for long enough, for us to be able to positively influence the microbial population of the GIT, such that the cria can get the most, nutritionally, out of its colostrum feeding and subsequent milk or milk replacer.

We are fortunate in this country to have access to good quality, highly effective and, above all, SAFE disinfectants for use in our rearing operations. Too often the cleaning out and preparation of the birthing area is only done at the start of the season. While this is fine for the first few cria born into that birthing area, over time the build-up of pathogenic organisms results in the cria born later in the

season being exposed to significant pathogenic load and, eventually, to the point where the pathogens overwhelm the passive immune system of cria within 24-72 hours after birthing. The efficacy of a top biosecurity programme can be measured by the severity and frequency of pathogenic scour incidents in the birthing paddock/housing. It is advised that disinfectants (both liquid spraying and the use of dry, spreadable powders), that are non-toxic to the cria and the staff working with them, be used regularly (every week to 2 weeks depending on the season) and that the housing environment be kept as dry and draught free as possible to reduce the stress placed on the newborn cria. Apart from regular pen disinfection, good biosecurity practices such as washing or disinfecting hands and footwear before handling cria, only ever handling them from youngest to oldest, restricting access to the cria rearing area to only those responsible for their rearing and cleaning and disinfecting the trailers or utes used to transport cria should be implemented.

Digestive physiology

The GIT of the alpaca comprises the 3 compartment forestomach (C1, C2 & C3), the small intestine including the duodenum and the large intestine comprising the caecum and very long spiral colon. The last 20% of C3 in adult alpacas comprises the true stomach which contains the gastric glands responsible for producing acids and enzymes. In the newborn cria, the true stomach dominates in size with a poorly developed C1. Liquid feed enters via the oesophagus and progresses directly past C1 via an oesophageal groove where it enters the true stomach. It is this true stomach and the progression of digesta from there into the duodenum and beyond that concerns us in the pre-weaned cria.



Colostrum

In addition to being born with a sterile gut, the cria is also born with no built-in (termed humoral), immunity. For the first 3 weeks of life the cria relies almost entirely on the antibodies it receives in the first 18 hours of life from its mother to protect it from pathogenic infection. These antibodies (immunoglobulins) are in abundance in the first milk or colostrum of the mother but their ability to be absorbed by the GIT of the cria is determined by a number of factors. These include, but are not restricted to:

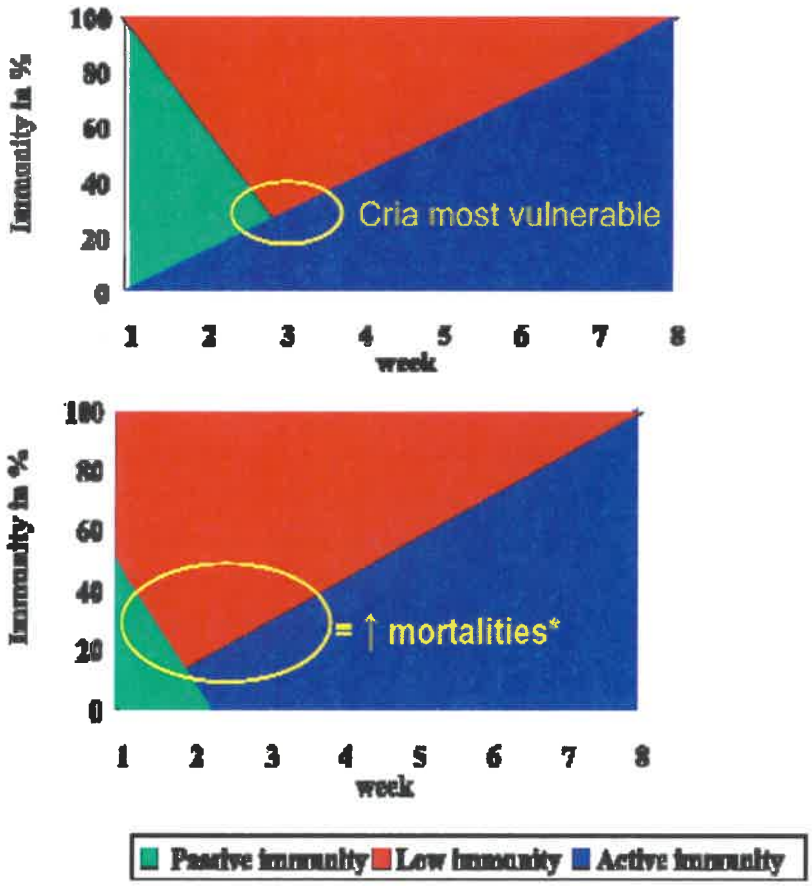
- **The quality of the colostrum produced by the mother**
  This is the number of immunoglobulins present/mL of colostrum. The most important of these for newborn protection is IgG. The minimum level of IgG for a healthy cria is ~500mg/dL. The quality of colostrum can be measured using a Brix Refractometer. This measures the density of the colostrum as an indicator of its quality, with the result being visually displayed in the eyepiece of the unit, on a scale that measures the sugar (lactose) reading present. In cow colostrum, good quality is a reading of +22% on the scale. It is not known by the presenter whether this technology is being used within the alpaca industry and, if so, what comparable Brix reading would be deemed "good quality" in the colostrum of alpacas.

- **The quantity of immunoglobulins ingested by the cria**

  Cria that get insufficient colostrum, have poor colostrum quality or who are unable to absorb the antibodies in sufficient quantity are at far higher risk of dying. Cria should receive a minimum of 10% of its bodyweight in colostrum in the first 12 hours to ensure maximum antibody uptake. It is recommended that 115mL be fed every 2 hours to meet this intake level.

  In a presentation by Dr. Geoff Neal of Manawatu Veterinary Services in New Zealand, to the Central Region Alpaca owners, he reported that the average death rate of alpaca cria in the US is 10% in the first 3 weeks. He also reported that 10% of all alpaca cria have insufficient colostrum absorption in the first 24 hours of life. He legitimately questioned whether the similarity between these two figures is purely coincidental. Further, he stated that cria born prematurely, or cria exposed to cold or other stresses may have antibody absorption as low as 2% in the first 0-6 hours of life.

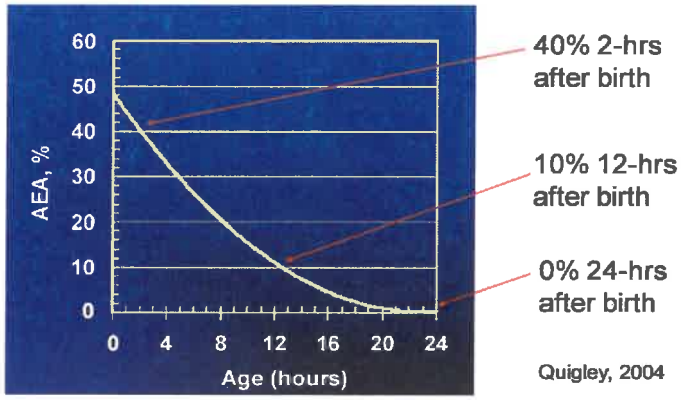
  The effect of this variability in antibody intake increases the risk of neo-natal mortality. The passive immunity acquired through colostrum has to protect the cria until the humoral immunity of the cria develops. The diagrams below illustrate this. As the intake of antibodies decreases as a % from the ideal, the period the cria spends "at risk" increases.



- **The speed with which the first microbes arrive at, and start multiplying on, the wall of the duodenum or small intestine**

  Immunoglobulins are the largest known protein molecules in nature and their size is one of the main reasons why their ability to be absorbed is compromised the longer the time from birth to digestion of the antibodies. As the duodenal wall becomes "cluttered" by bacteria, mucous, viruses etc, so the physical and chemical absorption of the antibodies declines rapidly.

Research conducted in the US by Dr James Quigley (2004) showed the following decline in the absorption rate of IgG in calves over time post-calving:



Quigley, 2004

Dr Neal from New Zealand reported that the absorption rate of antibodies in normal healthy cria to be "about 26% from 0-6 hours post delivery. This figure decreases to around 18% from 6-18 hours post birth, and by 24 hours post-delivery, the absorption rate is well below 10%". Thereafter, the antibodies are no longer absorbed as whole units into the blood stream but are broken down into amino acids and energy and absorbed in these forms.

- **The level of "risk" that the cria faces at birth**
  These include the premature births, cold-exposed and otherwise stressed cria mentioned above as well as cria born weak, which fail to stand and suckle in the first 3 hours. Also included in this list are: assisted births; caesarean section births; cria born to females with a history of dystocia or poor mothering; poor body condition; sick females including mastitis; weak or old females.

There is no substitute for cria getting the 3 "Q's" of colostrum management, being Quality colostrum, in the right Quantity (min. 10% of bodyweight) and Quickly (within the first 6 hours post-birth) to set them up for the best start to life. If there is no certainty that this has happened, the only direct antibody intervention is to use plasma infusions into the abdomen of the cria. According to Dr Neal, this intervention can raise the IgG level in "at-risk" cria from 200mg/mL to 800mg/mL within an hour of infusion. The benefits of this are obvious, however, this will require veterinary oversight so interested breeders should consult their vet and also consider the cost of the therapy.

Beneficial GI microbes

The best indirect intervention in an attempt to counter any inefficient colostrum absorption is to ensure that the duodenal region of the cria gut is exposed to beneficial microbial species as soon after 24 hours post-birthing as possible. Once again, the speed of intervention is key. This should involve the dosing of an oral gel containing beneficial bacteria and, preferably, a live yeast to assist in creating a duodenal environment (lowering the pH of the small intestine) hostile to the proliferation of pathogenic bacteria such as E. coli, Salmonella and Clostridia spp. A good probiotic gel will deliver a viable population of both bacteria and yeast such that they "crowd out" the pathogenic organisms in the hind gut region. This is known as competitive exclusion.

The use of a additive containing high levels of probiotic bacteria, yeasts and anti-oxidants into the milk of the cria, if being hand reared, is a further method of ensuring that, having set the gut up initially with beneficial bacteria and yeasts, the cria continues to get a prophylactic dose of the these "good bugs" daily to counter the natural influx of pathogenic organisms from their environment.

If the cria is being fed on milk replacer powder, ensure that the powder used has the best level of probiotic in it, as well as high levels of all the vitamins and trace minerals required to promote the rapid development of the cria's own immune system. Under normal conditions of suckling off the mother, the cria will be exposed to a wide range of bacteria, both good and bad. It is this uncertainty of the status of the cria gut health that leads many breeders to prefer to rear cria on other milk sources. If milk of another species is being used such as cow's milk, it is strongly advised to use a probiotic milk additive daily to counter any negative effects of either pathogenic contamination of the milk (e.g.: from mastitis milk) or the presence of anti-biotic residues from the "waste milk" of cows treated for infection.

Pre-weaning: Cria daily nutrient intake

- Daily liquid feed intakes should, ideally, be divided into 6 feeds for the first 1-2 weeks. Reduce the frequency down to 4 feeds by 3 weeks then further down to 3 feeds until weaning. By reducing the frequency of feeding, the aim is to encourage the cria to begin dry feed intake to drive the development of C1 in the forestomach as early as possible.
- Introduce a good quality creep feed with high digestibility and access to forage (either pasture grazing of good quality hay) from 1-2 weeks of age. The gut development of the cria, if not compromised, should see the development of the C1 section of the forestomach to almost adult proportions (~80% of forestomach volume) by 2 months of age and to be able to effectively digest roughage as a significant portion of its diet by 3-4 months of age. At this time, weaning should be completed.
- Use visual assessment of the dung of the cria, from birth, to get an insight as to the health and development of the cria GIT. Ensure that fresh, clean water is available to the cria at all times. Dry, sticky dung in healthy cria is often an indication of insufficient hydration and stock watering should be checked.
- As with any livestock enterprise, the best way to determine the success of a pre-weaning diet is to measure the weight gain of the cria at regular intervals (weekly, fortnightly or monthly). Average daily gain (ADG) in cria should be in the range of 100-250g/day from at least a week of age for small and large breeds respectively. Optimum gains should increase to 250-450g/day respectively. The first target to achieve is for the cria to double its birth weight by 4 weeks of age. The better the early gut health and development of the cria, the more likely it is that this target will be reached.

The table below shows the daily nutrient intakes of DM, Protein, Fat and Lactose by cria of varying sizes and, therefore, daily liquid feed intakes. As shown, all intakes are calculated using a liquid feed intake equal to 10% of the live weight of the cria. The proximate analyses of both the Alpaca Colostrum and Milk are shown as averages. The milk composition can vary from these averages according to a number of factors. These include:

- The protein content of the milk will vary according to the quality of the diet and, in particular, the grazing component thereof;
- The body condition of the female will have an effect on the fat content of her milk;
- Any udder infection such as mastitis will have a negative effect on the composition of the milk solids as well as introducing pathogenic bacteria in the milk;

Cria Nutrient Intake: Daily Intake of Nutrients for Alpaca Colostrum, Alpaca Milk and a commercial Milk Replacer

Colostrum Nutrient Intake (g)	Colostrum %	Colostrum g
Liveweight (kg)		7.0
Daily Intake (mL)		700
DM	20.0%	140.0
Protein	10.0%	70.0
Fat	4.5%	31.5
Lactose	5.0%	35.0

Milk Nutrient Intake (g)	Milk %	*Daily Milk Intake is calculated at a minimum of 10% of Liveweight*				
Liveweight (kg)		9.0	11.0	14.0	16.0	18.0
Daily Intake (mL)		900	1,100	1,400	1,600	1,800
DM	16.0%	144.0	176.0	224.0	256.0	288.0
Protein	6.5%	58.5	71.5	91.0	104.0	117.0
Fat	4.0%	36.0	44.0	56.0	64.0	72.0
Lactose	5.0%	45.0	55.0	70.0	80.0	90.0

The comparative table below is for a Milk Replacer with a 25% Protein, 30% Fat and 30% Lactose content (DM Basis) fed at 180g/L (18% DM).

Liveweight (kg)		7.0	9.0	11.0	14.0	16.0	18.0
Daily Intake (mL)		700	900	1,100	1,400	1,600	1,800
DM	18.0%	126.0	162.0	198.0	252.0	288.0	324.0
Protein	4.5%	31.5	40.5	49.5	63.0	72.0	81.0
Fat	5.4%	37.8	48.6	59.4	75.6	86.4	97.2
Lactose	5.4%	37.8	48.6	59.4	75.6	86.4	97.2

Figures shown in RED in the tables above differ by >20% in daily intake.

Factors affecting the intake of nutrients

The intake of nutrients, as shown above, requires a balance between quality of the nutrient source and the volume ingested. In a healthy cria, we should be challenging the daily intake to optimise its growth potential. There are a number of factors that we need to control to ensure that there is not a disruption to this intake pattern.

Stress reduction:

Cria are very sensitive to stresses in early life. These include the stress of birth, the environment (noise, temperature, draughts etc.), pathogenic infection, irregular feeding and excess or insufficient liquid feed. Any stress that results in a decrease in daily energy intake puts the cria at risk of hypoglycaemia, a condition to which they are particularly prone. This condition is exacerbated under cold conditions and present as hypothermia. Hypoglycaemia also predisposes the cria to infection and the risk of sepsis needs to be well managed.

Clotting in the true stomach:

Another complicating factor of stress is the negative effect that it has on the production of the required quantity of acids and enzymes in the true stomach of the cria to ensure that the best clotting of the milk consumed. Without the formation of a good curd in the true stomach to slow

down rate of passage, and allow a controlled digestion and absorption of the nutrients, there is a real risk of excess fluids travelling through into the duodenum and overwhelming the digestive capacity of the microorganisms, enzymes and gastric juices. The result is the undigested carbohydrates in the overflow milk begin to ferment in the lower gut. This supplies nutrients to pathogens in the lumen of the small intestine which begin to grow in population leading to further chemical toxicity and physical complications in the gut. The end result is digestive or nutritional scours which are termed non-infectious scours.

Any scour event is undesirable since it disrupts the pH of the small intestine and this can result in irritation and inflammation of the intestinal wall. This makes the cria more susceptible to pathogenic infections and can result in high mortality rates, especially within the first week of life.

If scouring is a persistent problem, it is best to work with the consulting veterinarian to institute protocols around hygiene practices as previously discussed, the type and frequency of electrolyte treatment of which there are a number of new and improved treatments on the Australian market, the body temperature levels at which anti-biotic treatments will be required and which to use, and finally, the protocols to follow to restore the beneficial microbial population of the GIT after an anti-biotic treatment. The latter is very important since anti-biotics are indiscriminate as to which bacteria they kill off so the population of the "good" bugs will decrease as much as the "bad" bugs that were targeted. While the infection may be treated, the reduction in digestive bacteria will put a break on development and growth. This is turn increases stress in the cria and can make it more susceptible to re-infection and so the cycle begins again.

As mentioned previously, knowing the growth target for the cria and measuring the ADG is important to evaluate to overall progress of the cria.

Conclusion

Mortality in cria pre-weaning can be extremely high, resulting in a waste of resources, a slowing down in the genetic progress of the enterprise and welfare issues on-farm that distracts the management. The importance of attention to this part of the enterprise has been highlighted by what needs to be done to get the young alpaca from a pre-weaned state to its role as a pseudo-ruminant as quickly as possible. Not only will this ensure the health of the cria as they develop, but it will reduce the overall cost of the enterprise since it is cheaper for the young alpaca to be getting its nutrient requirements from the grazing roughage on-farm rather than relying on on-going liquid feeding.

PASSION FOR ALPACA

Portraying your animals in visually stunning images is one of the most effective marketing tools for alpaca breeders.
Anne Rogers (Specialist Alpaca Photographer) excels in attention to detail, listening to her client's brief and designing portfolios with an exclusive and customised point of difference.

"Whilst I have been a photographer for many years before owning alpaca, "Passion for Alpaca" became my catch phrase - I adore these gorgeous, majestic and sensitive animals and respect the patience needed for capturing them at their best.

Whether you want a 'punchy-in-your face' charismatic macho photo, a dramatic picturesque landscape, a soft, cute melting moment, a technical fleece shot, or ... something completely out of left field! I'm up for the challenge and available for consultation and assignments in Australia and New Zealand"

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Greg Rundle is a director and senior adviser at 360Private Pty Ltd. As a chartered accountant and financial planner he specialises in providing advice to small and medium size organisations in the areas of taxation, structuring and high level business advice.

After 18 years in practice, Greg has advised various types of primary producers, including those associated with the alpacas. In this respect he has helped arrange structures and business plans for the successful establishment and running of alpaca ventures.

Greg also concentrates on family groups so that he can fully understand the impact that business and professional life has on the financial status of the broader family. This helps link superannuation, investments and insurance advice to client's affairs as these are critical for long term planning as well as protecting valuable assets.

Greg is the chairperson of the 360Private Investment committee which is charged with ensuring that a rigorous process in the selection of stocks is undertaken. This allows the firms strong ideal of value investing and capital protection to be front of mind at all times.

ALPACA FARMING – BUSINESS OR PLEASURE?

Guidance on the Australian Taxation Office Classification for your Primary Industry Activity

The increasing demand for exposure to a rural lifestyle has seen an increase in 'hobby farming' activities. Here we investigate the potential taxation implications of these activities and considerations to be made when distinguishing between a business activity and an enjoyable (albeit challenging) past-time.

Australia's taxation system revolves around the concept of self-assessment. The onus is placed on the taxpayer to assess their personal affairs and comply with the relevant taxation laws accordingly. Whilst this system provides flexibility and increased efficiencies, there are numerous grey areas in

taxation legislation which the average taxpayer can find difficult to navigate. The classification of income producing activities as a hobby or business is one such area.

Where a taxpayer determines that their activity constitutes a business of primary production, the money earned from the activity is generally assessable income for taxation purposes, with expenses incurred in earning the income allowable deductions. In financial periods where a loss arises, provided that the non-commercial loss rules are satisfied a sole-trader or partner of a primary production partnership could utilise the loss against their other assessable income.

In contrast, if the primary production activity was classified as a hobby, the points mentioned prior do not apply. The transactions relating to this activity will generally have no taxation consequences.

So, how do you make a determination regarding your own activity? *Taxation Ruling 97/11* is a good place to start, but for those who prefer to count alpacas when falling asleep at night we have spared you the mind-numbing taxation language and have summarised the key concepts below.

The Australian Taxation Office (ATO) explains there are a number of factors to consider when determining whether you are running a business or a hobby, being:

- Does your activity have a significant commercial character?
- Is there more than just an intention to engage in business?
- Do you have the purpose of profit as well as the prospect that you will make a profit, even if you are unlikely to do so in the short term?
- Is there repetition and regularity to your activity?
- Is your business similar to other businesses in your industry and is the way you operate consistent with industry norms?
- What is the size, scale and permanency of your activity? Is it sufficient to allow you to make a sustainable profit?
- Is your activity planned, organised and carried on in a business-like manner?

The ATO suggests that as a start, significant commercial purpose or character could be evidenced by a documented business plan, advice sought from experienced farmers, analysis of land suitability, investigations into market sustainability or research into profitability based on market prospects.

An intention to engage in business involves taking action. The extent of activity will determine whether the business is carried on. If preparations are still being made then you may not have commenced business.

The prospect of a profit is considered an important factor by the ATO. Business activities are carried on for the purpose of profit on a continuous and repetitive basis. Whilst it is not necessary for a business activity to produce a profit, there should be a reasonable expectation of a profit being achievable and this could be evidenced by research or consultancy provided by experts. If the activity continued unprofitably, the taxpayer would need to show that other indicators of business were present that outweighed the objective view that the activity was inherently unprofitable.

TR 97/11 explains a feature of business is that similar sorts of activities are repeated on a regular basis. This repetition of activities helps to determine whether there is the carrying on of a business. Regular purchases, sales and seasonal variability's would be present in the financial transactions recorded for alpaca farmers carrying on a business of primary production.

Operation in line with industry practices is a strong indication of business activity. The ATO would expect that the volumes of sales, types of customers, marketing methods, expenses incurred, capital invested and past experience of the operator would be similar to other businesses in the industry. A key point is that the activities should be compared with that of a keen amateur. Sales by an amateur may just be a way of continuing on with the activity rather than representing a commercial purpose.

Whilst a large operation would be suggestive of a primary production business, small scale transactions are not excluded from classification as a commercial activity. The case of *JR Walker* involved five Angora goats, two of which died. Despite the scale of operation being small, the court held that a goat breeding business was being carried on because of the profit making motive and the regularity of activities. Research had been undertaken by the taxpayer that showed profit could be made from the capital allocated to breeding stock.

Whether your activity is carried on in an organised business-like manner is another important consideration. The maintenance of business records and accounts, separate bank accounts, business premises, licences or a registered business name would all be suggestive of business activity.

Each taxpayer's activities must be considered separately from others when a determination between hobby and business is made. A single indicator may be all that is needed when making the determination regarding classification but generally all relevant indicators will be considered as part of the decision making process. The indicators provide general guidance rather than a conclusive test.

Where uncertainty exists regarding your activities, a Private Ruling can be obtained from the ATO. As part of this process the taxpayer will be required to provide information regarding the indicators and the ATO will then make the determination based on their consideration of this information. We suggest that you first contact our office as it may be possible to avoid this process through more rigorous investigation into the activity.

Non-commercial Losses

In circumstances where an activity is classified as a primary production business, the legislation regarding non-commercial losses becomes relevant and important.

The non-commercial loss rules apply to individuals and partnerships. Basically, where the loss tests are passed the loss from the primary production activity will be available to reduce other assessable income.

Generally, you can offset a loss from your business activity against other income where your income for non-commercial loss purposes is less than $250,000, and your business activity passes one of the following tests:

- The activity produced assessable income (turnover) of at least $20,000.
- The business activity has produced a profit in three of the past five years (including the current year).
- The business uses real property (land and buildings) or an interest in real property worth at least $500,000 on a continuing basis.
- The business uses other assets (machinery, equipment etc.) worth at least $100,000 on a continuing basis.

Despite failing the tests above, primary producers have access to another concessional test which could allow for the application of losses against other income. This test means that losses will still be available where the business tests are failed, if:

- The business is a primary production business and your other assessable income is less than $40,000.
- The loss is due to amounts claimed under the small business tax breaks allowed during the 2010 and 2011 financial years.

Furthermore, where any of the above points can not be satisfied, a taxpayer retains the ability to apply to the Commissioner of Taxation for discretion which will allow the loss to be claimed. Please contact our office if you believe your circumstances warrant this discretion.

Melanie Smith

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Melanie graduated from The University of Sydney in 2011 with a Bachelor of Animal and Veterinary Bioscience, receiving first class honours in her project on sheep productivity. Her main interests lie within the areas of meat science, and animal production. In 2012 she started her PhD titled *Investigation of quality attributes and consumer acceptance of Australian Alpaca meat.*

INVESTIGATING MEAT QUALITY ATTRIBUTES OF ALPACA MEAT IN AUSTRALIA

The Australian Alpaca industry is at a pivotal stage of its transformation in becoming a commercial entity beyond its reliance on fibre as a product. Traditionally alpacas have been farmed for their fleece with advancements in fibre production and reproductive performance being successful. However, to promote the longer-term viability of the industry there needs to be a commercial market for Alpaca meat. Benefits for Alpaca producers of establishing a quality meat industry include the utilisation and monetary returns generated from otherwise unprofitable animals that are not suited for fibre production and improvement in genetic gains.

Currently there is a paucity of local and international scientific information available on Alpaca meat quality and production. This includes an absence of information on the impact on carcass quality of meat processing technology such as electrical stimulation, tenderstretching and ageing. The majority of the available scientific information is inconsistent and not entirely relevant to Australian producers. These knowledge gaps need to be addressed for optimal production of quality alpaca meat.

To address this, research is currently being conducted by The University of Sydney and the NSW Department of Primary Industries, Centre for Red Meat and Sheep Development. The main objectives of this research are to provide alpaca producers and processors with information on key meat production parameters including optimal slaughter age, carcass composition, and meat quality attributes of alpaca meat.

Preliminary results are very encouraging from the first of two experiments which investigated important carcass and meat quality traits, between different age groups (18, 24 and 36 months) and genders (castrated males vs. females). These traits included; dressing percentages, carcass yield

breakdown, processing techniques (applying electrical stimulation), tenderness, fresh colour, colour stability, nutrient value, and a consumer sensory evaluation.

The second and final experiment will be investigating the effects of on farm finishing methods by comparing the differences between animals grazed on pasture only compared with animals grazing pasture and provided with a grain supplement. The flow on effects to carcass and meat quality traits will be investigated, along with the benefits of tenderstretching. It is anticipated results from this research will be distributed over the next 12 months.

Acknowledgement:

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Fiona Vanderbeek

Fiona was raised in England, and pursued a career in medical management, research and education, until she married Ian and migrated to Australia in 2001. They settled in Canyonleigh in the Southern Highlands of NSW and bought their first suri alpacas in 2002.

Many of the foundation animals were suri crosses and Fiona used her understanding of genetics to advance the quality of the Birrong Suri Alpaca herd. Fiona was the technical adviser for the NSW DPI's publication "Alpaca AgSkills" published in 2011 and wrote the companion volume "Alpaca AgGuide – Farming Alpacas", published in 2012. In addition to her love of genetics, Fiona is passionate about grazing management and alpaca nutrition and is also the AAA's representative on the Rural Industries Research & Development Corporation (RIRDC).

"Understanding Suri Genetics... or, How to Win at Cards"

This session is aimed at those who feel overwhelmed with the terminology and concepts surrounding suri genetics. It is an interactive, hands-on session which will demystify terms like "homozygous", "heterozygous", "dominant" and "recessive" – to name but a few. Fiona devised this very visual representation of what happens genetically when we mate suris or huacayas, or cross the two together, to explain some complex concepts in a way that is unforgettable – a previous participant once described it as "a light bulb moment".

WHAT IS GENETICS?

Genetics is the study of heredity and the units of inheritance – called "genes".

Genetics is the science behind the art of livestock breeding and will help you to:

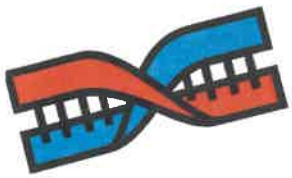
- Choose the right animals to buy
- Improve your herd through selective breeding
- Reduce the risk of breeding animals that do not match your breeding goals
- Reduce the risk of breeding animals that have inherited defects

The terminology can be rather baffling... but is important... so don't put it in the "too hard" basket...

Terminology

DNA and Chromosomes

- Your DNA contains all your genetic information – DNA occurs in every body cell as a "double helix"



- Chromosomes are sections of DNA and occur in pairs – alpacas have 37 pairs, humans have 23 pairs
- Each parent contributes one member of the pair

Gene

- Part of a chromosome responsible for a particular characteristic (eye colour, fleece type etc)
- There are always two copies of each gene – one inherited from each parent
- These two copies may be the same or different

Alleles

- When the two copies of a gene are different from each other, they are referred to as different alleles
- These are usually written as upper and lower case letters... B (for brown eyed gene) and b (for blue eyed gene) or S (for suri fleece) and s (for huacaya fleece)

Homozygosity

- If the two genes in a pair are the same, the individual is homozygous for that gene
- A person who has two genes for blue eyes is "homozygous for the blue eyed gene"
- This is written as "bb"

Heterozygosity

- If the two genes in a pair are different (different alleles of the gene), the individual is heterozygous for that gene
- A person who has one allele for blue eyes and one for brown eyes is "heterozygous for the eye colour gene"
- This is written as "Bb"

Dominance

- *THE MOST IMPORTANT CONCEPT IN GENETICS...*
- Some traits, caused by different alleles of the same gene, will always show dominance over others
- In humans the allele for brown eyes (B) is dominant over the allele for blue eyes (b)
- A person who is heterozygous for the eye colour gene (Bb) will have brown eyes, even though they are also carrying a blue eyed allele, which remains "hidden"
- So, a person who is Bb will always have brown eyes

Recessive traits

- For a recessive trait to show itself in the appearance of an individual, they must be homozygous for that gene
- As the blue eyed allele (b) is recessive, for a person to have blue eyes they must be "bb" – homozygous for the blue eyed gene

Genotype

- The genetic make-up of an individual
- An individual's genotype can never be changed – you are born with a set genetic blueprint
- You cannot *see* a genotype but you may be able to deduce something about it by looking at appearance or studying a family tree or pedigree

Phenotype

- What you actually see in front of you... the person with blue eyes or the alpaca with suri fleece.
- Phenotype cannot tell you what the genotype is because recessive alleles will be masked by dominant ones.

Sexual Reproduction

- Egg and sperm cells are different to most cells in our bodies in that they carry only one member of each chromosome pair
- If the parent is homozygous (bb or BB) then all their reproductive cells will be the same (b or B).
- If the parent is heterozygous (Bb) then half their reproductive cells will be B and half will be b
- When egg and sperm fuse to create an embryo, the two half sets of chromosomes join and the offspring has its own unique set of genetic material inherited in exactly equal amounts from each parent

Suri and huacaya genetics

- Suri fleece is an example of a dominant trait, and the suri allele is written as "S"
- Huacaya fleece is an example of a recessive trait, and the huacaya allele is written as "s"
- As it is a recessive trait, all huacaya are "ss" – the are homozygous for the huacaya gene
- As it is a dominant trait, suri may be "Ss" (heterozygous) or "SS" (homozygous) – the phenotype (suri fleece) is the same, but the genotype ("Ss" or "SS) may be different"

Terms used in suri/huacaya cross-breeding

- First Cross or F1 suri: This is the progeny of a huacaya and a suri. The phenotype is suri, and the genotype must be Ss.
- Back-cross One; BC1; Second cross; Second generation suri. All these terms are sometimes used to describe the offspring produced from an F1 mated to a suri. The phenotype is suri, but the genotype could be Ss or SS (heterozygous or homozygous)
- Back-cross Two; BC2; Third cross; Third generation suri. All these terms are sometimes used to describe the offspring produced from a BC1 mated to a suri. The phenotype is suri, but the genotype could be Ss or SS (heterozygous or homozygous)
- Back-cross Three; BC3; Fourth generation suri. The phenotype is suri and they can be considered "pure suri" having 93.7% suri blood and an 87.5% chance of being homozygous

Maximising the success of a cross-breeding program

- Cross breeding is generally used as a means of reducing the cost of producing suri, to improve the quality/quantity of coloured suri and sometimes with a view to commercial fibre production
- There are disadvantages – the time taken to produce "pure" suri from cross-breeding; the risk of producing unwanted huacaya; what to do with cross-bred males
- You can maximise the success of a cross-breeding program by researching before you buy and using only homozygous males... Understand the rules of the game!
- The IAR can be some help in checking the homozygosity/heterozygosity of a suri. Mating a male to 10 huacaya females will test his homozygosity... if all ten progeny are suri, he is almost certainly homozygous... as soon as one huacaya offspring is produced, you know he is heterozygous. A DNA test for heterozygosity should soon be available.

.PDF ARTWORK ON THE NEXT PAGE

Drawn by Dean Morris and reproduced courtesy of the NSW Department of Primary Industries

Huacaya, ss

Looks like a huacaya. Carries only recessive ss genes for fleece type

Each parent contributes recessive s gene for body type.

Homozygous suri, SS

Looks like a suri. Carries only dominant SS genes for fleece type

Each parent contributes dominant S gene for body type.

Heterozygous suri, Ss

Looks like a suri because S is the dominant gene for fleece type, but also carries hidden s gene.

Each parent contributes different gene for body type. One S, one s.

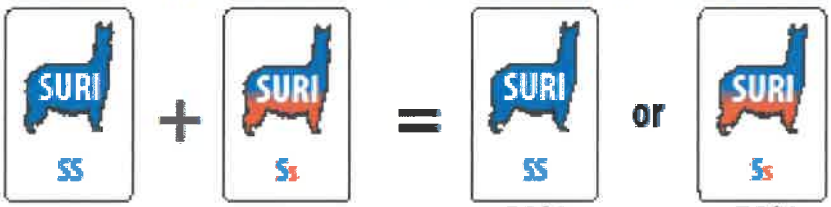
What happens when we mate two huacayas? As all huacayas are homozygous, ss, then the offspring will, of course, also be homozygous huacayas because there is no other allele to inherit.

100%

What happens when we mate two suris? If both parent animals are homozygous, SS, then the offspring will, of course, also be homozygous suris because there is no other allele to inherit.

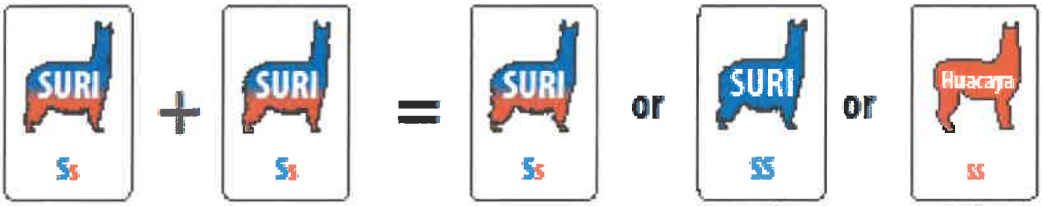
100%

If you mate a homozygous suri, SS with a heterozygous suri, Ss, you will also always get a cria with a suri phenotype… but half of them will be heterozygous, Ss, having a "hidden" huacaya gene!



50% 50%

If you mate two heterozygous suris, Ss and Ss, however, you have a 50 per cent chance of producing a heterozygous suri, a 25 per cent chance of producing a homozygous suri but also a 25 per cent chance of actually producing a huacaya!



50% 25% 25%

SURI – THE COMMERCIAL REALITY

Anyone who has seen and felt the fleece of a top quality suri is blown away by its unique qualities of lustre and silky, slippery handle – what we often hear referred to as the "WOW factor" of suri. Why then, more than twenty years after this rarer type of alpaca first arrived in Australia are we still grappling with converting this magnificent raw fibre into an equally sumptuous fabric? In this presentation I will review previous research into fabric processing, current projects that are underway and look at what we hope to see develop in the future.

The "Surissimo" Project

In the early days of the Australian suri, it was often said that suri simply could not be processed; it was thanks to Julienne Gelber of Bumble Hill Alpacas, and some like-minded suri breeders in New Zealand, that this myth was finally debunked.

It took Julienne fourteen years from the time she first set eyes on the silken locks of a suri at sunset, to the achievement of her dream. Using her wool and textiles background to understand what high-end processing would demand of the fibre, she embarked on a ten-year breeding program with the objective of producing a luxurious, lustrous suri fibre full of drape and with exquisite handle.

Working with breeders in New Zealand, an approach was made to the government's experimental mill at Lincoln University (at the time called WRONZ and later renamed AgResearch) to produce a sample range of high quality/high value 100% suri woven fabrics which would exhibit the natural lustre and soft handle of the suri fibre. It took two years to convince the mill's technical team that the project was feasible, as they initially believed the micron range proposed (20-22 micron) was too fine and that the fibre available would lack sufficient uniformity. Working with like-minded breeders in Australia and New Zealand, genetics were further refined to produce a style of suri that had the desired fibre characteristics, together with husbandry and classing modifications which ultimately produced a fibre that was shorter, had a tighter micron range and lower SD of length, with classing selecting only the softest and most lustrous fleeces. By 2007 the research team were won over by the quality, and the breeders were able to provide sufficient quantity, of raw fibre for the trial to commence.

The end result of this trial, in 2010, was six fully finished fabric pieces, in a variety of finishes, but all of which exhibited the high lustre and glossy, sleek handle unique to suri. Despite their initial scepticism, the mill's technicians had encountered no significant problems in the way the suri fibre had scoured, gilled and converted to yarn, and there were also no issues with the weaving process.

The twelve years' work behind this achievement adopted an integrated approach including genetics (to select and breed animals with the desired fleece characteristics), management practices (such as time of shearing to achieve desired fleece length), nutrition and farm environment (to ensure fibre is harvested in peak condition) and a total quality management approach from farm to mill.

The "Rumplestiltskin" Trial

Following on from the work achieved in the Surissimo trial, in 2012 two breeders in New Zealand (Surico and Thistledown) received a grant from the Sustainable Farming Fund to undertake more

research into the commercial processing options for suri. Unfortunately the AgResearch facility previously used by Surissimo had been closed and alternative commercial processors needed to be found. The approach taken this time was a little different in that fleeces were sourced from breeders across New Zealand and, when additional quantities were required, Australian breeders were also invited to join the project. The involvement of Australian suri breeders was initially spearheaded by Paul Vallely of Australian Alpaca Fibre Testing, who asked me to assist him in co-ordinating the collection of suri fibre on this side of the Tasman.

In 2012/13 the focus was on collecting a sufficient quantity of white suri fibre, with a micron range of 22-23.9, and a SD of <5.0, together with length being between 80-140mm. These were the stipulated requirements of the trial's processor, who need uniformity of micron and length for successful processing. An initial collection day in New Zealand had yielded around 60kg of fibre that met all these requirements and so the trial coordinators were delighted to work with the Australians to boost quantity. A nationwide call for suri fibre that met the requirements yielded a bale of 110kg which was exported to New Zealand in March 2013.

The New Zealand and Australian fibre was combined and scoured in July 2013, remaining under quarantine restrictions until after carding. The normal speed of the scour is 500kg per six minutes, which had to be slowed to a minimum to deal with the small batch of around 170kg of suri. This stage of the process highlighted the absolute importance of fibre length – every bag of fibre was inspected before being put in the scour, as over-length fibre would tangle in the machinery. Fortunately the strict collection criteria ensured that the fibre was of suitable length and the scour was generally happy with the process and prepared to undertake further work with suri.

The next stage was carding, combing and gilling into tops, before spinning into yarn. In New Zealand, as in Australia, many mills have closed in recent years and the machinery been sold overseas or scrapped. It has therefore taken longer than hoped to find a mill keen to work with suri and with the necessary expertise to take the fibre through to yarn. In March 2014 a commercial mill in New Zealand was finally identified and the fibre will be processed into a worsted yarn with capability for both knitting and weaving.

While work is continuing in New Zealand, here in Australia we are in to a second round of fibre collection, as the coordinators are keen to progress their work with suri fibre, including breeder education and information sharing across the Tasman. However, for this phase of the work there will be no available grant funding and the New Zealand breeders will be taking over the commercial risk. The 2013/14 fibre criteria have been extended, to include three micron ranges (<19; 19-21.9; 22-23.9) and some colours (white; light fawn; medium fawn; black), with SD<5.0 (or 5.5 for black) and length between 80-140mm still being critical. In addition, in recognition of the previous generosity of Australian suri breeders in donating their fleeces, and to offer a contribution to the costs of fibre harvesting, a small financial contribution is being offered – whilst recognizing that this is in no way to be seen as a true market value of the fibre.

Once again, in Australia, fleece collection/baling days have been designed to be educational in an effort to drive continuous improvement in the quality of fleeces being put forward for processing. Regional Coordinators have been identified for Victoria, New South Wales, Queensland, South Australia and Tasmania and baling days have already been held in Victoria and New South Wales.

The selection of Australian fleeces for the trial has been rigorous. Not only must criteria for length, contamination (VM or colour fibres), tenderness or chalkiness be met, but every fleece has been 3-point tested to ensure it meets the relevant classing line, both in terms of micron and SD. From two collection days, held in Victoria and Southern NSW, a total of 112 fleeces (83.2kg) were baled, with the distribution across lines and colours shown below:

Victoria and SNSW Baling days

Colour	Micron	Number of fleeces	Micron	Number of fleeces	Micron	Number of fleeces
White	< 19	17	19-21.9	44	22-23.9	32
Light Fawn	< 19	-	19-21.9	1	22-23.9	2
Med Fawn	< 19	-	19-21.9	8	22-23.9	3
Bay Black &True Black	< 19	-	19-21.9	1	22-23.9	1

A total of 211 fleeces were brought to the two baling days, of which 99 did not make the grade. An analysis of the reasons why fleeces submitted (which had met the initial micron/SD criteria based on a midside sample) did not make the grade is given below:

Reasons for rejection: Victoria and SNSW Baling days

Reason	Number	Percentage
Length (too long/too variable)	32	32%
3-point FD too high	20	20%
3-point SD too high	18	18%
3-point FD and SD too high	13	13%
Tender	6	6%
Coarse fibre contamination	5	5%
Colour contamination	1	1%
VM/Stain	1	1%
Other	3	3%

Key Learning Points from "Surissimo" and "Rumplestiltskin"

Whilst the work of Rumplestiltskin is still ongoing, already there are many lessons learned and key points to help guide us towards the suri processing future:

1. Herd management

Perhaps the most important lesson highlighted by both projects to date is the importance of *fleece length*. For successful commercial processing fleece needs to be between 80-140mm in length. If this is what the processors want, then this is what the grower must provide. To ensure that fleeces are not over-length, breeders may need to consider changes to their birthing or shearing programs. For example, autumn-born cria can be shorn with the main herd, when they are around six months of age. Cria born in spring will need to be shorn in autumn, otherwise

their fleece will be too long if left until they are a year old for first shearing. Our strategy is to have cria born between July and October (which best suits our climatic conditions and prime feed availability). Most cria born in July/August are shorn with the main herd in early November, when their fleeces are usually around 90mm in length. Those born in September/October are shorn in early February, when their fleece is also of good length, and by the following November they will have produced a second fleece of processable length – thereby giving us two prime fleeces by the time they are 12-14 months of age.

Excessive vegetable matter (VM) or cotted fleeces can also be minimised by consideration of time of shearing (ideally before seed-set on grasses) and by paddock maintenance – control of weeds with burrs, annual grasses such as barley grass etc. Shearing of cria with very fine fleeces will often save a fleece which would otherwise become contaminated and cotted – regrowth fleeces are generally easier to keep free of excessive VM.

2. Management at shearing

Management at the time of shearing is of critical importance in maximising the value of fleece.

Always *shear white animals first* and progress through the colours from lighter to darker shades. Be absolutely sure the floor is swept clean between animals, particularly when moving to a darker colour. Also ensure there is no possibility of fibre contamination in the area of the fleece skirting table. The contamination of a white fleece with even a couple of coloured fibres will render it almost worthless.

Consider modifications to *shearing technique* – such as "saddle first" – to minimise the risk of coarse fibres contaminating the prime saddle area of the fleece. Whatever shearing technique is used, ensure that the hairiest parts of the fleece (belly and lower legs) are removed on the shearing floor and separated from the saddle before it is taken to the skirting table.

Ensure that a suitably trained and competent person has responsibility for skirting each fleece as it is placed on the skirting table. *An initial, thorough skirting must take place at shearing* to avoid contamination of the fleece with coarse fibres. Areas of the fleece that are heavily contaminated with VM should be totally removed; the occasional small pieces of VM will be removed in the scouring process. *Never place a fleece in a bag unskirted* – once the coarse edge fibres have contaminated the prime saddle fleece they can never be removed.

3. Breeding program

While some issues in producing top quality suri fibre for processing can be relatively simply remedied by modifications to herd management and shearing, there are other critical factors which can only be improved by longer term measures to modify the fleece qualities within a herd. The "Surissimo" project was the result of twelve years of selective breeding to produce the desired style of fleece.

Lustre and handle are of paramount importance. One fleece with a dry, chalky handle within a bale of otherwise high quality fleeces will have a detrimental effect on the performance of the entire bale.

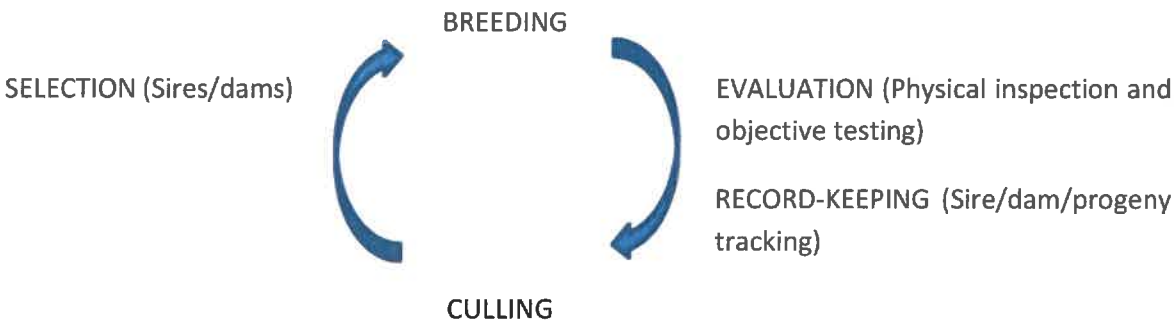
Fibre diameter (micron) is critical to maximising fleece value. Whilst it does not necessarily need to be ultrafine, like any natural fibre, coarse fleece is not going to have value to the top-end processor. It is also important to monitor the increase of fibre diameter with age of the animal – if animals produce one good fleece and then "blow out" then their value in the herd is significantly reduced.

Uniformity is often overlooked by breeders not focussed on fibre production. Standard deviation (SD) – which is a measure of the uniformity of the fleece – is at least as important, if not more important, than fibre diameter. Fleeces which have coarse fibres scattered throughout them will have a higher SD and are not desirable for high-end commercial processing. Selecting animals with low SD (<4.5), particularly as the animal ages, will greatly enhance fleece quality.

Style is also important, particularly in terms of producing fleeces which remain well-aligned and avoid cotting both on the animal and during processing. The flatter style of lock generally seen in the show ring these days is considered preferable to the older style of tightly twisted lock, which do not go through a scour as well (tightly twisted locks will tend to fall through the machinery and cause excessive wastage). Some wave along the lock also helps to maintain the fibres in alignment.

Density is obviously important to maximise the quantity of useable fleece cut from each animal. A dense, solid structure to each lock is also helpful in repelling VM and reducing cotting of the fibre.

A breeding program to produce top quality fibre producing suri must involve a constant cycle of breeding, evaluation, record-keeping and culling to drive the quality of suri higher.



Where to from here?

Early in 2014, Paul Vallely decided it was time to hand over responsibility for co-ordination of the Australian end of the Rumplestiltskin project, together with steering the suri fibre industry in general, to the breeders themselves.

A team of eight suri breeders, all with a passion for suri fibre, and spread geographically across Victoria, New South Wales and Queensland, met for the first time in March 2014. At the time of writing, the first meeting had only just been held, and whilst recognizing that there is much to be done, the team is committed to the task.

With the over-arching focus being "suri fibre", the team has started work under the title of the "Suri Fibre Development Group" and will initially focus attention on one or two challenging, but achievable, goals – including a system to collect and class suri fibre, and liaison with potential commercial partners.

Some of the challenges that will need to be tackled going forward include:

- The lack of volume of high quality suri fibre. Should we be considering the blending of suri with other natural fibres?
- The need for a cyclical quality-driven approach to breeding that pushes the quality of suri fibre available to the market to a higher and more consistent level. This will require education on required traits and how to achieve genetic gain, together with training in the assessment of fleece quality in a suri and in the classing of shorn fibre.
- The lack of awareness by processors of this rare natural fibre - "suri" - and a lack of understanding by the breeders as to what the processor wants. Market intelligence and sensitivity though commercial partnerships are going to be key to success.
- The currently fragmented nature of the suri industry with many small owners/breeders who are not working together to drive the industry forward.
- Funding will be required, both to support the collection/classing process and to facilitate suri fibre-related projects.

As suri owners we are privileged to be in possession of an animal with the potential to produce a luxurious and sought after rare natural fibre – but this can only be achieved by an integrated approach to quality – encompassing genetics, animal selection and culling, together with high standards of farm management, husbandry and fibre harvesting. If the suri industry is prepared to work collaboratively and to have a real desire to take the sometimes hard decisions necessary to drive the quality of their fibre higher, and if commercial partners prepared to work with suri can be identified, then the future for the suri industry remains bright.

Dr Jane Vaughan

BVSc(Hons), PhD, MACVSc (Ruminant Nutrition)

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Jane graduated with a Bachelor of Veterinary Science with honours from the University of Melbourne in 1988. She spent 7 years working in rural practice around Australia and began working with alpacas in 1991 whilst working in dairy practice in south-eastern Australia. In 1996, Jane worked on Cocos Islands Quarantine Station for 12 months with more than 600 Peruvian alpacas during their importation to Australia. She then spent a year at Murdoch University, Western Australia, doing a Residency in Food Animal Medicine and passed examinations for membership to the Australian College of Veterinary Scientists in the Ruminant Nutrition Chapter. Between 1999 and 2001 she studied the control of ovarian follicular growth in alpacas as part of a government-funded Doctor of Philosophy at Central Queensland University. Jane now performs commercial embryo collection and transfer in alpacas throughout Australia, New Zealand and the United Kingdom. She also provides nutritional advice and Johne's disease Market Assurance Programs to alpaca breeders in Australia.

PRACTICAL NUTRITION IN ALPACAS

Stages of gastrointestinal development

Crias are born with a sterile gastrointestinal tract and the third compartment of the stomach (C3) is proportionally larger than in the adult.

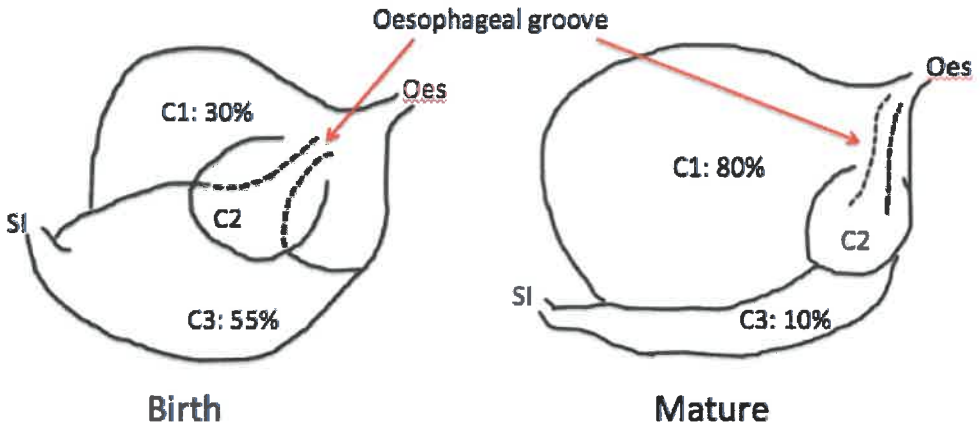
The first, or pre-ruminant phase of gut development is when the cria is totally dependent on milk. Suckling from the udder elicits the oesophageal groove reflex that allows milk to by-pass C1 and C2, and enter C3 directly, so crias digest milk as a monogastric animal would. This stage generally lasts until 2 weeks of age. Crias may start nibbling solid feed in their first week. This phase will last longer if no dry feed is offered. Crias require access to clean fresh water at all times to ensure normal first compartment development.

The second, or transition, phase of development begins at about 2-3 weeks of age, and lasts until weaning. The first compartment reaches adult proportions by 8 weeks of age, but takes about 12 weeks to reach full adult capacity. Phase two continues as long as milk is offered to the cria. Crias pick up microbes from the environment to populate C1-2 and start drinking water. Fermentation products produced in the first compartment of the stomach are responsible for the rate of C1-2

development, so access to forage and water during the transitional period is essential to ensure normal development of the stomach.

The third, or ruminant phase is from weaning onwards and alpacas rely solely on pasture (and supplements where necessary) and water for nutrients. Weanlings require access to fresh water and a good quality ration containing adequate energy, protein, fibre, vitamins and minerals to support growth.

Aim for a smooth transition between the 3 stages to maximise cria growth and development. A sound feeding program ensures immune system can start functioning normally, supplies nutrients for optimum growth and optimises long-term fertility.



Proportional differences in size of first (C1) and third (C3) compartments of the stomach in neonatal and mature alpacas (Oes = oesophagus, SI = small intestine).

Rules of thumb for supplementing crias

- Crias should stand and begin nursing within 1-2 hours of birth. Give the cria time to bond and suckle.
- If the dam has no milk or the cria too weak to nurse, feed colostrum (frozen and stored from another disease-free alpaca/cow/goat or powdered colostrum) via teat (preferably) or stomach tube for the first 3 days of life to maximise antibody uptake into blood (first 12 hours of life) and to provide local gut protection from microbes.
- If no colostrum available, use milk replacer and strongly consider intravenous plasma transfusion for antibody transfer (speak to your vet about plasma use). Use milk in preference to oral glucose/honey and water.

Choice and use of milk replacer

- It does not matter which brand or type of milk replacer you use, but do not vary the brand of milk replacer once you have made your selection. The way it is fed determines success or failure of bottle-rearing.
- Store milk powder below 30° C and protect from moisture.
- Use clean bottles/teats/feeders, thoroughly clean afterwards.
- Warm milk to 35° C before feeding.

Types of milk replacer available for crias

Milk type	Per litre of reconstituted milk			
	% Solids	% Protein	% Fat	% Sugars
ProfeLAC Shepherd Premium Lamb-Kid-Cria Milk Replacer®	18	4.5	5.4	5.4
Di-Vetelact®	13.5	3.2	4.1	6.3
Palastart Lamb & Kid Milk Replacer®	22	5.3	5.5	
Wombaroo Cria Milk®	17	5.9	2.7	6.2
Full cream cows' milk from supermarket	12-12.5	3.2-3.6	3.8-3.9	4.8-5.2
Full cream cows' milk powder	13	3.2	3.7	4.9

Frequency of feeding

- Aim to feed 10-15% of body weight as milk per day, to demand, as 6 feeds/d initially, decreasing to 4 feeds/d.
    - E.g. 10 kg cria requires 1-1.5 L/day, equivalent to 250-375 mL per each of 4 feeds.
    - Feed first thing in the morning, mid-morning, mid-afternoon, last thing at night. Wait-times in between feeds encourage the cria to develop an appetite to:
        - Nurse from dam as much as possible.
        - Start picking at grass/hay/supplements and develop 1st compartment of stomach.
        - Drink enthusiastically from teat when offered.
- Crias should double birth weight by 4-6 weeks of age, gaining at a rate of about 200 g/d.
    - Weigh cria regularly to monitor weight gain. Keep good records of weight gain and milk intake.
- Reduce frequency of bottle-feeding from 4 weeks of age.
- Aim to wean from milk replacer at 12-16 weeks of age when the cria is eating substantial amounts of forage.

Method of feeding

- Tube-feed cria (in consultation with your vet) if animal is moribund.
- Bottle-feed cria with teat, holding bottle vertical and ensuring nose of cria pointing skywards to simulate cria suckling from udder. This posture elicits a reflex in the gut, which allows passage of milk along a groove from the oesophagus directly to the 3rd stomach compartment, by-passing C1&2. In C3, the milk is converted into curds and whey under the effects of acid and enzymes. The whey fraction (sugars and soluble proteins) is then rapidly digested, while the curds (fats and insoluble proteins) are digested slowly over the next few hours.
- Teat feeding is preferred over bucket feeding to ensure milk enters C3 and does not ferment in C1 (appear as pot-bellied, ill-thrifty crias). Teat feeding stimulates saliva secretion to enhance milk digestion.

Tips to prevent diarrhoea

- Keep concentration of milk consistent, do not fortify by adding more milk powder per litre of water as diarrhoea can develop secondary to higher sugar content of milk.
- If a cria develops diarrhoea, offer oral electrolytes by teat between milk feeds (3 hr apart) and seek vet advice.
    - Do not dilute milk by adding electrolytes to the milk as this prevents curds and whey formation and exacerbates diarrhoea.

Other hints on rearing crias

- Always have clean, cool, fresh water available to allow normal C1 development.
- Offer high quality, highly digestible creep feed and hay to develop C1 from 7-10 days of age.
- Aim to wean from milk replacer by 3-4 months provided cria eating good quality forage and weighs ≈ 25 kg.
- **Do not humanise alpacas: Do not pat cria. Bottle feed and walk away so cria bonds with alpaca herd. Crias naturally jump on their mothers and other crias, and will try to do it to you when they get bigger.**

Weaning

Weaning age

As a generalization, Australian alpaca farmers tend to wean alpaca crias from their mothers around 5-6 months after parturition. Interestingly, methods of improving fertility of domestic livestock in third world countries DO NOT include pasture improvement and artificial breeding technologies, but DO include on-farm biosecurity, seasonal management of the herd/flock, male breeding soundness evaluation and *early weaning*. Many alpaca farmers fall into the trap of leaving crias with their dams for longer if one or other or both are failing to thrive, thinking this will benefit cria growth and development. It is time to start weaning your crias sooner and here is why!

Moving to earlier weaning is based on the observations that peak milk output of female alpacas is approximately 3 weeks post-partum (Van Saun pers. comm.), 50% of milk is produced in the first 4 weeks of lactation, and that lactation is more-or-less complete by 12 weeks post-partum. Concurrently, females increase feed intake 2-3 weeks into lactation but do not reach peak intake until 8 weeks. This leads to an energy and protein mis-match (more energy and protein being lost in milk than the female is eating) and weight loss in early lactation, and weight gain (fat and muscle) later in lactation. There is a high feed cost of replacing body tissue so weight losses during pregnancy and lactation should be minimised. Weight can be gained more efficiently in females after they cease lactation.

In a good season, crias can be left with their mothers until they weigh 25-30 kg, to optimise survival and fertility rates. This target may be achieved by 12-16 weeks of age when there is plenty of good pasture and adequate gastrointestinal parasite control. E.g. cria birth weight 7.5 kg, gains 200-250 g/d for the first 30 days (6-7.5 kg), and 150-200 g/d for the next 60 days (9-12 kg) = 22.5-27 kg by 12 weeks of age, and more by 16 weeks of age.

BUT, remember that lactation is "over" by 100 days and the competition for feed between dam and cria is won by the dam! Therefore, in difficult times (over-stocking, drought, wormy pastures) it is prudent to teach crias how to eat supplements by feeding them with their mothers at least 4 times

to (a) train them onto supplements (imprint feeding) and (b) acclimatise stomach microbes to the feed which they will be fed after weaning; then drench with an effective anthelmintic and wean onto high quality feed (pasture and/or supplements) at about 12 weeks of age (no earlier than 8 weeks of age). This demonstrates how early weaning can be used as a management tool to manage nutrition and parasite burdens of both weaners and their dams. This leads to greater fleece production, healthier weaners that reach puberty sooner and allows females to regain body condition faster resulting in better conception rates during the next joining period if not already pregnant at weaning.

Weaning strategies

It is imperative to minimise stress around the time of weaning. Not only will it improve dam and weaner health after weaning, it will reduce injuries and production losses (fleece and meat).

Management strategies to help reduce the stress of weaning (taken from http://www.mla.com.au/Livestock-production/Animal-health-welfare-and-biosecurity/Husbandry/Weaning):

- Joining females to ensure a compressed birthing period to reduce age ranges at weaning.
- Maintaining females in adequate body condition through good nutritional management.
- Feeding crias at least four times with their mothers before weaning (imprint feeding).
- Using low stress stock handling.
- Selecting paddocks and yards that are securely fenced.
- Ensuring high quality pastures are available for both weaners and their mothers.
- Keeping weaners out of sight and hearing range of their dams immediately after weaning.
- Drafting and managing weaners according to sex, size and behaviour (this reduces bullying behaviour and enables allocation of small crias to the best pasture and supplementary feed if required).
- Ensuring parasite control is adequate.

After weaning (http://www.makingmorefromsheep.com.au/wean-more-lambs/procedure_10.4.htm):

- Aim to have weaners reach 50% of their mature liveweight as soon as possible after weaning. To achieve this target, weigh or assess a representative sample of crias at weaning and, when practical, draft weaners into groups for preferential management.
- Post weaning mortality is highest in the initial three months with the lightest weight weaners being most at risk.
- Most losses are in low liveweight weaners and management to increase weight gain will eliminate deaths. Formulate supplementary feed rations to account for pasture quality and availability.
- Monitor weaner liveweight. Where necessary, redraft weaners into appropriate weight groups and provide the best available pasture to the low weight group.

There is a strong association between post weaning survival bodyweight and growth rate in sheep, and it is likely true for alpacas as well. Some features of this relationship include:

- Small increases in weaning weight make a profound difference to weaner survival. For example, a 20 kg sheep weaner has a 22% lower mortality risk than an 18 kg weaner.

- Small increases in the monthly growth rates for weaners can dramatically reduce mortality rates, e.g. an increase of 0.25-0.5kg/month can reduce the mortality risk by 74%.
- Faster growing and heavier weaners accumulate more body reserves that they can use if required.
- The close association between growth rate, bodyweight and mortality risk means that supplementary feeding may be a cost-effective option in weaners.
- Apart from nutrition, special attention to weaner health is required such as drenching and flystrike prevention.

Female alpaca weaners must have sufficient growth rates to reach the liveweight targets (> 65% of mature body weight at 12 months of age) to ensure adequate conception at first joining, regardless of the age.

Preparation of weaning paddocks

Early selection of paddocks is a critical component of managing crias immediately after weaning.

Features of ideal weaning paddocks include:

- High feed quality: mixed perennial pastures: short green feed (1,200–1,500 kg green DM/ha), preferably with improved pasture species and 20% legume. Annual clover based pastures: 2000-2500kg DM/ha feed-on-offer. In the cereal zone a special-purpose fodder crop or good quality stubble is an option. Preferentially allocate pasture to female weaners to ensure they meet growth targets.
- Low grass seed infestation: particularly barley grass or corkscrew. If crias need to graze risky paddocks either spray top or slash grass seed heads to reduce the hazard.
- Low worm risk: specially prepared paddocks with low worm burdens.
- Good quality stock water: easy access to clean drinking water and multiple watering points in larger paddocks.

Nutrition of adult alpacas

Digestion is the process by which large insoluble food molecules are broken down into smaller soluble compounds that are then able to cross the gastrointestinal lining and enter blood and lymphatic vessels and provide the animal with energy, protein, fats, vitamins and minerals, which allows them to grow, reproduce, and produce wool and milk. Adult alpacas mainly eat grasses, legumes and shrubs which contain sugars, proteins and fats along with variable amounts of fibre (provides plant structure in the form of cellulose, hemicellulose and lignin) that cannot be broken down by mammalian digestive enzymes.

The digestive tract in camelids has adapted to facilitate plant digestion by setting up a symbiotic relationship with billions of microbes in the forestomach. Therefore, when you feed an alpaca, you also feed the microbes in the forestomach. Look after the microbes and the microbes will look after your alpaca. Different rations favour different microbes so change feeds slowly to allow the microbes to adjust to the new nutrients.

1. How much to feed?

Alpacas will eat approximately 1.5 % of their body weight as dry matter to maintain body weight (i.e. not growing, pregnant or lactating). *Feed up to maintenance requirements with roughage* (pasture, hay, silage). For example, 70 kg alpaca:

70 kg x 1.5 % of body weight = 1.05 kg as dry matter (DM) ie all water removed from feed

1.1 kg DM x 100/20 = 5.3 kg lush pasture/day (grass with 20 % DM content)

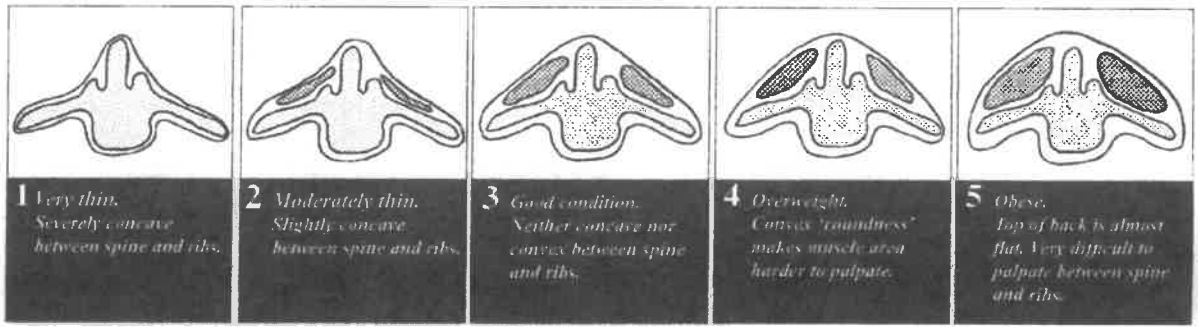
1.1 kg DM x 100/90 = 1.2 kg pasture hay/day (hay with 90 % DM content)

Feed requirements	**Maintenance**	**Growth**	**Lactation**
Dry matter intake (DMI; % BW/d)	1.5 %	1.8-2 %	2-2.5 %
Crude Protein %	8-10 %	14 % < 12mo 12 % > 12mo	14-16 %
Crude Fibre %	25 %	20-25 %	20-25 %

Growing alpacas and late-pregnant and lactating females will eat about 2-2.5 % of their body weight as dry matter. Feed up to maintenance requirements with roughage (pasture, hay, silage). Then supplement with energy/protein as required (good quality pasture hay/lucerne hay/oats/lupins/peas).

2. Body condition score to monitor if feeding too much or too little

Palpate musculature over the backbone at the level of the last ribs.



Ref: Alpaca Note 4: Body Condition Score of Alpacas. Prepared by AAA Inc. Education and Training Sub-committee.

Aim for body condition scores somewhere between BCS 2.5 and BCS 3

If alpacas are too fat, feed less. If alpacas are too thin (eg during growth, pregnancy or lactation), feed more, better quality feed. If some alpacas in the same paddock are too fat and some are too thin, then divide the group and feed accordingly.

3. Pasture and water will supply most nutrients

Alpacas require four main ingredients in their diets: water, energy, protein and fibre.

Ensure access to fresh clean water at all times. The daily requirement of water is 50-80 mL/kg body weight per day (5-8 % BW/day). So a 70 kg alpaca requires 3.5-5.6 litres water per day. The amount of water drunk is lower when grazing green pasture (20 % DM) compared with hay (90 % DM). Alpacas will drink more water in hot weather and when lactating.

Pasture will supply most energy, protein and fibre needs. The proportion of each depends on plant maturity. It will also satisfy most vitamin and mineral requirements.

Greener pastures contain more protein, therefore as pasture matures, protein decreases. Crude protein content of feed required for maintenance is 8-10 %. Higher levels are required for growth (12-15 % CP), pregnancy (12 % CP) and lactation (13-15 % CP).

4. Feed long-stemmed roughage

Alpacas need long-stemmed roughage (pasture, hay, silage greater than 4 cm in length) to keep their fore-stomachs functioning normally. A diet based on very short (or non-existent under drought conditions) pasture, chaff and grain/pellets or very lush spring pasture is not adequate to keep the stomach healthy. Ensure ad lib long-stemmed pasture/hay at all times if pasture fibre is limited.

A rough estimate of plant fibre content in pasture may be gained by manually testing the breaking strength of plant matter – more mature plants contain more fibre (thicker cell walls, more lignin) and are more difficult to break and less digestible.

Observe your alpacas to see if there is enough fibre in the diet:

- > 50 % of recumbent alpacas should be chewing their cud
- body condition score – adequate fibre in the diet is required for fat deposition
- faecal consistency – % fibre vs % DM in diet

5. Vitamins

Many of the *water-soluble vitamins* (vitamins B, C) are provided by the microbes that live in the fore-stomachs, so healthy alpacas do not require supplementation if they are healthy.

Of the *fat-soluble vitamins*, vitamins A and E are available from green grass (even from green weeds that come up after brief summer rain) so only need supplementation if pasture is completely dry for more than 8-10 weeks.

Vitamin D supplementation is required in alpacas. Inject all alpacas less than 2 years of age, and all females due to give birth in winter/early spring (to fortify colostrum) with 2000 iu vitamin D/kg body weight under the skin or into the muscle. Administer in late autumn, mid-winter (and early spring in

higher latitudes like Tasmania, New Zealand and UK). Read the label on the bottle to determine vitamin D concentration to determine what volume to administer.

For example, A 20 kg cria needs 40,000 iu vitamin D. If there is 75,000 iu per mL vitamin D in your selected source of vitamin D, then the cria would need approximately 0.5 mL of solution injected.

6. Minerals

Are minerals deficient in your area in sheep and cattle? Ask your local Department of Agriculture, local veterinarian and neighbouring farmers for information. If in doubt, you should sample soils, pasture and/or alpacas to determine if mineral levels are adequate, before supplementation.

Acid, water-logged soils (annual rainfall > 500 mm) contribute to selenium deficiency. Selenium deficiency may be treated using an annual depot injection (Deposel®) at a rate of 0.5 mL per 50 kg BW s/c once a year. Alternatively, alpacas may be supplemented by short-acting oral preparations at a rate of 0.1 mg/kg BW orally every 4-6 weeks.

7. Feeding supplements

Beware of feeding unnecessary supplements that may be costly, labour intensive to feed out and/or toxic to your animals. Do not feed out supplements designed for use in horses and pigs as they have different digestive systems and different mineral requirements.

8. Access for all

Animals should have access to long-stemmed fibre at all times (eg in drought conditions, consider placing a large round bale of pasture/oaten hay in the paddock). If supplementary feeding of concentrates (eg grain, pellets) is required, make sure all animals can access the feed at the same time. You can feed out directly onto the ground, but you may reduce wastage by feeding in long troughs (eg guttering, old conveyor belting laid out on ground, shade cloth attached to fence).

Introduce new feeds over a period of 10-14 days to allow adaptation to the new feed. Once adapted to the new feed, feeding twice as much, every second day will save you time and money, but more importantly will allow shy-feeders access to supplements. The dominant animals will fill up quickly (you have seen how they stake out the feed and spit at any animal trying to get near) and move away when full, allowing the shy feeders to eat the supplements later in the day/overnight.

9. Feed wastage

If supplements remain at the next feed out, or hay is being wasted, you are feeding too much (do their body condition scores reflect this?) Ensure that growing, pregnant and lactating animals are fed appropriately with good quality feed and feed the left-over portions to non-pregnant/non-lactating females, wethers etc.

10. Keep it simple

Complicated recipes containing scoops of this and cupsful of that do not alter cria sex or kill worms, but may waste your time, energy and money. *Feed alpacas up to maintenance requirements* (go

back to Point 1!) *with pasture/hay/silage*. Supplements for growth, pregnancy and lactation may be met by providing energy with cereal grains (eg oats) and protein with lucerne hay and lupins.

Guidelines for supplementing alpacas

The following guidelines assume that there is no pasture available. You must combine skills of pasture evaluation with body condition scoring to determine appropriate supplementation. **These are rough guidelines only**. All new feed should be introduced slowly (over a period of 10-14 days) to allow the microbes to adjust to the new substrate.

Supplement all classes of stock up to maintenance requirements with *high fibre* supplements if pasture is limiting. eg oaten hay/lucerne hay 3:1 on a DM weight basis. Ensure at least 25% of fibre is greater than 4 cm in length to optimise stomach function.

Animals requiring more than maintenance requirements (growth, lactation) but unable to obtain them from pasture can be supplemented with concentrates eg maize/oats/lupins/vitamins/minerals.

A 70 kg adult alpaca can be just **maintained** on 1.2 kg oaten hay (88% DM, 8.7 MJ ME/kg DM, 7.6% crude protein). However, a mixture of 3 parts oaten hay (0.9 kg) and 1 part lucerne hay (0.3 kg) will contain 8.5-9 MJ ME/kg DM and 10-11% crude protein. 1.2 kg of this mix will provide some safety margin for energy and protein needs.

Growing crias require 10.5-11 MJ ME/kg DM and 14-16% crude protein. An appropriate supplement could consist of 2 parts oaten hay, 2 parts lucerne hay, 3 parts oats, 3 parts lupins. This will provide enough energy and protein and calcium and phosphorus in a ratio of 1.7:1.0. Beware of excess intake when grain feeding – introduce feed slowly, do not let grain feed build up in the bottom of feeders, mix well with fibre (hay/chaff) to reduce the risk of grain poisoning and death.

Lactating hembras require 10.5-11 MJ ME/kg DM and 13-15% crude protein. A mixture of 3 parts oaten hay, 3 parts lucerne hay, 2 parts oats, 2 parts lupins would satisfy energy, protein and Ca:P needs. Again, beware of grain feeding.

Method of feeding supplements

If you need to supplement alpacas with grain or other concentrates:

- Introduce the new ration slowly over a period of 10-14 days. Begin feeding one-third of the ration, then increase over the next 2 weeks up to the full ration.
- Mix the grain with any chaff you may be feeding thoroughly to reduce the risk of some animals getting more grain than others.
- Feed out in troughs/on ground so that ALL alpacas have simultaneous access to the feed, again so that some alpacas do not get too much grain at once.
- Be aware that feeding processed grains (e.g. cracked, rolled, soaked grain) increases the risk of grain poisoning by being more rapidly available for breakdown by C1 microbes.
- Body condition score/weigh your animals to allow ration adjustments.

Rules-of-thumb for ration making

- No single supplement will suit all alpacas on all farms.
- Supplement up to maintenance requirements with forage (pasture/hay/silage).

- Grains should make up < 50% of the total feed intake.
- Read the label on all supplements carefully to avoid overdosing.
- All new feed should be introduced slowly (over a period of 10-14 days) to allow the C1-2 microbes to adjust to the new substrate.

Summary

- Ensure crias receive 10% of their body weight as colostrum by 6 hours of age.
- Aim to wean crias at 12-16 weeks, weighing 25-30 kg.
- Ensure unlimited access by all stock to clean, fresh *water*.
- Maximise *pasture* intake. Alpacas have evolved on high fibre diets and will eat about 1.5 % of their body weight as dry matter daily to maintain body weight.
- Supplement all classes of stock up to maintenance requirements with *high fibre* supplements if pasture is limiting. E.g. good quality pasture hay or oaten hay. Ensure at least 25% of fibre is greater than 4 cm in length to optimise stomach function.
- Animals needing more than maintenance requirements (growth, late pregnancy, lactation) but unable to obtain them from pasture can be supplemented with better quality hay or chaff (e.g. clover or lucerne/alfalfa) and/or concentrates (e.g. oats, lupins).
- *Vitamin D* is likely to be limiting in alpacas in winter. Supplement all alpacas less than 2 years of age with 2000 IU vitamin D/kg body weight injected subcutaneously in mid-late autumn and in mid-winter (and in early spring if in higher latitudes such as Tasmania, New Zealand, northern Europe, UK, Canada, northern USA). Supplement heavily pregnant alpacas due to give birth in late winter 4-6 weeks prior to birth to ensure an adequate level of vitamin D3 is achieved in the colostrum.
- Determine *mineral requirements* by animal, pasture, hay, grain and soil testing. Contact your veterinarian and Department of Agriculture regarding minerals most likely to be limiting locally.
- *BODY CONDITION SCORE* ANIMALS REGULARLY TO MONITOR FEEDING PRACTICES. Farmers with scales are encouraged to weigh young alpacas regularly to ensure they reach targets weights and ages for weaning and first mating.

MATING MANAGEMENT IN YOUR ALPACA HERD

Understanding normal reproductive physiology in alpacas is essential when alpaca breeders want their alpaca herd pregnant efficiently and economically or when a veterinarian is undertaking infertility investigations in alpacas. The emphasis in this paper is on learning what is normal in alpacas and developing a simple mating protocol based on the unique reproductive physiology of South American camelids. This information may then be used to ensure alpacas conceive efficiently or combined with general veterinary principles to diagnose and treat causes of infertility in alpacas.

FEMALE REPRODUCTIVE PHYSIOLOGY

Puberty

Camelids are induced ovulators. They do not exhibit clear signs of oestrus and it is therefore difficult to assess when they attain an age at which conception may occur. Evidence of oestrus is based on submission to a macho for mating. Information on ovarian activity in llamas has been attained by measuring urinary hormones, and suggests that ovarian follicular growth may start as early as 5-6

months of age (Bravo 1997). As in ruminants, nutritional status plays a major role in when ovarian activity begins. Camelids need to attain approximately 60-66 % of their mature weight to ensure a good chance of conception (Smith et al. 1994, Johnson 1989), which is at approximately 12 months of age under reasonable climatic and nutritional conditions. Good nutrition after weaning together with monitoring of live weight and body condition score are essential for continued reproductive success.

Anatomy

Camelids have a bicornuate uterus with a single cervix. In alpacas, the body of the uterus is approximately 3 cm long and 3 cm diameter and the two uterine horns are about 8 cm long. The uterus resembles the letter "Y" and the tips of the uterine horns are blunt and rounded. The endometrium is lined with uterine glands. There are 2 long, tortuous oviducts, which join each uterine horn to the ovarian bursa. The ovaries are approximately 1 cm x 1 cm x 1.5 cm (peanut-sized). The cervix is usually 2 cm long and the vagina is 15 cm long. The vulva is 1-2 cm long, with vertical orientation.

1. The unmated, non-pregnant female (unstimulated, non-pregnant)

Camelids exhibit new waves of ovarian follicular growth every 12-20 days (Vaughan et al. 2004). Recruitment of a cohort of follicles is likely under the control of FSH and occurs before follicles in the cohort are visible in the ovary. Follicular wave growth involves synchronous emergence of several (3-6) follicles (2-3 mm diameter). These follicles develop to 4-5 mm diameter. By an unknown mechanism, one follicle in the ovary becomes dominant at an average diameter of 5-6 mm and continues to grow, the others in the cohort regress. The dominant follicle takes about 4-6 days to reach its mature size, remains at its mature size of 7-12 mm for 4-8 days, it then regresses over a period of 4-6 days. Regression is associated with emergence of the next follicular wave (Bravo et al. 1989, Sumar 1983, Adams et al. 1990, Vaughan et al. 2004)

Growth characteristics of follicular waves are consistent for the first 10 days after emergence (Vaughan et al. 2004). There is variation in interwave interval within and between animals, a longer interwave interval is associated with a larger maximum follicle diameter and there is no correlation between live weight and interwave interval.

Consecutive follicular waves do not necessarily alternate between ovaries. Large follicles are equally distributed between ovaries (Vaughan et al. 2004). A follicular wave emerges as soon as the dominant follicles starts to regress which means that both the regressing and emerging dominant follicles can be visualised on the ovaries at the same time (Bravo et al. 1990). The significance of follicular dynamics is that repeated ultrasonography is required to establish whether a relatively large follicle is in the growing phase, static phase, or regressing phase. This has important implications if ovarian ultrasonography is used in the management of mating.

Behavioural oestrus appears to be associated with a lack of progesterone rather than a presence of oestrogen (Adam et al. 1989, Sumar et al. 1988). Behavioural studies have shown that most non-pregnant females are receptive all the time whether they have an ovulatable size follicle present in one of their ovaries, or not, with intermittent periods of non-receptivity lasting 1-2 days as a new follicle wave develops (Johnson 1989, Sumar et al. 1993).

Camelids are non-seasonal breeders so ovarian follicular activity occurs all year. Follicle waves continue during lactation and early pregnancy (up to 6 months gestation; Bravo et al. 1989).

2. The mated female that fails to conceive (ovulatory but non-pregnant)

Copulation takes place in a sternal sitting position and usually lasts 20-25 minutes. During copulation, the macho penetrates the cervix with his penis, and places semen into both uterine horns.

A mating that occurs in the absence of an ovulatable-size follicle will not induce ovulation, and conception will not occur.

Camelids are induced-ovulators: A mating that occurs in the presence of a growing or mature follicle (≥ 6 mm diameter in alpacas) will result in ovulation (Vaughan et al. 2003). An LH surge begins 30-60 minutes after mating and subsides 5-6 hours later (Aba et al. 1995). This surge results primarily in response to an ovulation-inducing factor in the semen identified as ß-nerve growth factor (Kershaw-Young et al. 2012) and from a neuro-hormonal pathway (physical contact of male with female, stimulation of female reproductive tract, orgling). In South American camelids, ovulation occurs approximately 26 hours after mating (Stuart, unpublished). 10-25% of females with an ovulatable size follicle present on the ovary may fail to ovulate. Ovulations occur with equal frequency from both ovaries (Fernandez-Baca et al. 1970a). There is no significant relationship between follicle size and time of ovulation post-mating.

About 3 days after mating, (2 days after ovulation), a corpus luteum (CL) develops on the ovary at the site of ovulation. The CL reaches a maximum size of 10-15 mm and maximum progesterone output 8-9 days after mating (Ratto et al. 2006). When conception fails to occur, prostaglandin is released from the uterus and luteolysis occurs. The progesterone output of the CL starts decreasing around 9-11 days after mating. Females will be receptive again approximately 12-14 days after mating if conception fails.

Regression of the CL in non-pregnant camelids is under the influence of the uterine horns as partial hysterectomy prolongs lifespan and secretory activity of the CL on the same side as the missing horn (Fernandez-Baca et al. 1979). Difference in luteolytic effects of left and right uterine horns has been observed and is probably explained by asymmetrical blood supply to the uterine horns and ovaries. Luteolytic activity of the right uterine horn acts locally on the CL in the right ovary while the left uterine horn has both a local and a systemic effect, thus affecting a CL on the right or left ovary.

When circulating progesterone levels are greater than 2 ng/ml, a CL is present on the ovary. Female South American camelids placed in a pen with a macho will strongly reject the male by spitting, kicking and running away (a process known as "spitting-off").

Females not completely deprived of visual, auditory and olfactory stimuli from a macho may ovulate too. Spontaneous ovulation, where ovulation occurs without coital stimulation or male influence, may occur in 3-15% of females but many recorded cases included some form of male contact. Multiple ovulations occur in up to 10% of natural matings (Fernandez-Baca 1993, Vaughan et al. 2004).

3. The pregnant female (ovulatory and pregnant)

Oocyte maturation occurs following copulation and LH release. Fertilisation occurs in the oviduct and a hatched blastocyst enters the uterine horn about 6.5 days after mating. The embryo starts elongation by Day 10-12 post-breeding and attaches to the uterine wall around Day 18-20. Placentation is epitheliochorial. An extra foetal membrane, the epidermal membrane, consists of a layer of epidermal cells that covers the foetus and attaches at muco-cutaneous junctions (lips, nose, eyes, coronary bands, vulva/prepuce, anus). This membrane is thought to play a role in lubricating the foetus during parturition.

The CL is the major source of progesterone throughout pregnancy and its presence is required to maintain pregnancy (Sumar 1988). There is a temporary decline in blood progesterone from Day 8 to 12 after mating during the period of maternal recognition of pregnancy. The embryonic signal for maternal recognition of pregnancy must be transmitted as early as Day 8-9 after mating in order to rescue the CL of pregnancy (Aba et al. 1997). Thereafter, progesterone levels increase and the diameter of the CL reaches a maximum of 10-19 mm on about Day 21.

98% of pregnancies occur in the left uterine horn, even though the CL of pregnancy is found equally in the left or right ovary (Fernandez-Baca et al. 1973, 1979).

Ovarian follicular activity continues in a wave-like fashion during pregnancy (Bravo et al 1993b). Periodic emergence of dominant non-novulatory follicles 6-12 mm diameter have been shown until 6 months of gestation in South American camelids. Follicles greater than 6 mm were attained irrespective of which ovary contained the CL. Thereafter, 3-4 mm diameter follicles are seen on the ovaries.

Early embryonic death is common in camelids. 10% (and perhaps up to 50% in adverse climatic and/or nutritional conditions) of embryos may be lost in the first 60 days of pregnancy (Fernandez-Baca et al 1970b, Adams 1997). Factors responsible for this high attrition rate are unknown but nutritional constraints, hormonal imbalance, low grade infection or chromosomal aberrations may be principle causes. There are approximately 5% abortions between 60 days gestation and full term.

Twin births are rare. Camelids probably have a mechanism to reduce twin conceptions to singletons in early gestation as multiple ovulations are common (up to 10%) and live singleton pregnancies have been observed in females with multiple CLs on the ovaries.

The average gestation period of alpacas is 342 days (range 300-380 days) – longer in spring-mated females (348 days average) than autumn-mated females (340 days average; Vaughan et al. 2004).

Pregnancy diagnosis

Indirect methods of pregnancy diagnosis involve detection of the CL/elevated plasma progesterone:

- Sexual behaviour – 'spit-off' test. Female South American camelids placed in a pen with a male will usually strongly reject the male by spitting, kicking, squealing and/or running away if they have a CL and elevated plasma progesterone.
- Elevated blood/milk progesterone. When plasma progesterone levels are greater than 2 ng/mL, a CL is likely to be present on the ovary.

Direct methods of pregnancy diagnosis involve detection of the foetus:

- Transrectal or transabdominal ultrasound. A foetal heart beat is detectable from 25 days of gestation.
  A useful rule of thumb (up to 220 days gestation) is:

  Approximate days gestation = [Bi-parietal diameter (mm) x 4] + 20

- Manual rectal palpation is possible from 35 days of gestation. Good restraint, lots of lubrication and a small hand are needed.
- Transabdominal ballottement is possible from 8 months of gestation.

Parturition (unpacking)

The full-term alpaca or llama female will separate from the herd, hum, urinate frequently, and lie down and stand up frequently. Stage I (preparatory phase) lasts 1.5-2 hours, Stage II (delivery of foetus) 30-45 minutes and Stage III (delivery of foetal membranes) 1-4 hours but all stages may be longer in maidens. The dam will often remain standing throughout Stage II. She does not lick the newborn cria nor eat the placenta. The cria usually sits up in 10 minutes. Investigate retained placenta if > 6 hours and treat with oxytocin. Do not pull membranes. Lochia is passed for 7-10 days post-partum and will appear and white-pink mucoid discharge on the communal dung pile.

In camelids, parturition is under photoperiodic control so the majority of births occur between 7 am and 2 pm. Never let the sun set on a camelid trying to give birth!

Receptivity After Unpacking

The interval from parturition to resumption of ovarian follicular activity is about 5-7 days in South American camelids and females can be ready to ovulate by 10 days post-partum. The uterus only takes about 20 days to involute, probably because of the diffuse (microcotyledonary) nature of placentation.

Some owners have described difficulties in getting some females pregnant again if mated later than 3 or 4 weeks post-partum. These females generally conceive easily once their cria has been weaned. Explanations for this observation include:

- Females of other species of domestic livestock produce certain hormones during lactation that may reduce/inhibit ovarian function. This may also occur in camelids.
- Females reach peak lactation approximately 3 weeks post-partum. Lactation is the most metabolically demanding time for alpacas, and nutrients are diverted preferentially to the udder, possibly to the detriment of ovarian function.

Reproductive efficiency

Alpacas are as fertile as other domestic livestock. Conception and birth rates in alpacas average more than 50% from a single mating when mating system, fertility and body weight of sire and dam, post-partum interval, environmental conditions and nutrition are taken into consideration (Vaughan and Tibary 2006). This translates into 90% of females conceiving in the first three mating attempts after reaching puberty or post-partum. Sexual receptivity, ovulation, fertilisation and embryonic

survival rates in alpacas are similar throughout the year (Fernandez-Baca et al. 1972; Knight et al. 1992; Vaughan et al. 2003).

The ideal mating time appears to be during the late growing and early mature phases of follicular growth when follicles are most likely to ovulate, approximately 8 days after new wave emergence (Vaughan et al. 2004). Unfortunately, there is no association between sexual receptivity and follicular status or ability to conceive (Vaughan et al. 2003) and therefore farmers have no simple measure of when a female may be more likely to conceive. Paddock mating may allow the male to determine an optimal time for mating, but when males and females are together in the same paddock continuously, male libido may decline within a week (Sumar 1999). Where supervised yard-matings occur, the first two matings by a male on any day are equally as likely to result in pregnancy (Vaughan et al. 2003). While more matings per day by a male are possible, conception rates decline and depend on the fertility of individual males (Bravo et al. 1997).

Breeding recommendations for supervised yard matings

- First breeding at 12+ months of age (maiden) when reached 65 % of estimated mature body weight OR 15-20 days after unpacking if the delivery was straightforward and unassisted.
- Breed once when receptive. Multiple matings may lead to metritis as male penetrates cervix with penis.
- Spit-off at 7 days to check for ovulation. If receptive, ovulation did not occur and female should be mated again. If non-receptive at 7 days ....
- Spit-off at 14 days to check for pregnancy. If receptive, conception did not occur and female should be mated again. Non-receptivity indicates presence of elevated plasma progesterone and a corpus luteum .... And probably pregnancy.
- Spit-off regularly (every 2-4 weeks) until ultrasound pregnancy test at (30 and) 60 days post-joining to observe foetus and therefore confirm pregnancy status.
- Spit-off intermittently throughout gestation as up to 5% foetal loss occurs after 60-days gestation.

A female should be given three rounds of this management before seeking veterinary advice for failure to conceive. 90% of females will conceive in the first 3 mating attempts after reaching puberty or post-partum. Ensure that dates and findings are recorded at each mating to assist with further reproductive investigations.

FERTILITY AND INFERTILITY IN YOUR ALPACA HERD

Steps to minimise infertility in your alpaca herd

Prevention, prevention, prevention

- Cover all bases with a good, all-round husbandry plan
- A farm management plan helps you make sure your day-to-day actions are working toward your goal of better reproductive performance
- Avoid in-breeding to minimise congenital defects that are hereditary

Rear good crias

- Ensure colostrum intake adequate

10% of liveweight in first 6 hours of life

- Ensure feeding, hygiene and disease control allow high growth rates and minimise disease occurrence

Prevent JD, coccidiosis, bacterial/viral diarrhoeas etc

Monitor GIT worms

- Schedule vaccination and disease control programs
- Imprint feed: train crias to feed supplements by feeding crias 4 times with their mothers before weaning
- Wean crias at 12-16 weeks onto high quality, low parasite pastures

When reached target weight 25-30 kg

AND regularly eating their weaning ration (pasture +/- supps)

Maidens

- Set target liveweights and feed appropriately so > 65% of mature body weight by joining

65% of 70 kg = 45 kg

Continue supplementary feed as necessary

Schedule vaccination, vitamin D and parasite control programs

Prevent JD

- BCS regularly, weigh every 4-6 weeks
- Look after underweight maidens

Consider differentially feeding groups of young females according to their size

First mating

- Decide what age/liveweight
- Weigh before mating, ensure BCS 3 or more
- Feed so maidens gain weight throughout mating period
- Choose an unpacking time to match pasture quality/supplement availability
- Select males that
    - Reduce birthing difficulties
    - Are experienced
- Prepare males so healthy 2 months before joining starts
- Monitor male fertility throughout joining

First pregnancy

- Feed maidens to ensure achieve target liveweights

Management of female nutrition during pregnancy increases cria birth weight and survival and lifetime wool production of offspring

- Pregnancy test to confirm pregnancy status
- Place pregnant females in a paddock next to mating yards every 2-3 months

Females that have aborted will come up and sit next to fence

Investigate females that have lost a pregnancy

Unpacking time: all females

- Weigh maidens before full term
- Minimise health problems of all late pregnant females

Make sure females shorn > 4-6 weeks prior to unpacking date so recovered from stress and long fleece does not interfere with joining

Consider vitamin D, Se supplementation, 5-in-1 vaccination 4-6 weeks before due date

Monitor worm burdens and treat accordingly

- Minimise health problems of males

Ensure fit and healthy 8 weeks before joining starts so ready for action soon after females unpack – shearing/vaccination/toenails/parasite control/lupins/vitamin D & Se supplementation

- Record every unpacking

Female ID, date, cria ID

Note whether difficult or assisted birth/RFM/vaginal discharge/twins

- BCS every female at unpacking

Should be BCS 3 or more

Early lactation = pre-mating

- Minimise BCS loss in early lactation & monitor BCS before mating

Keep an eye on females with first cria to ensure both are growing and in good BCS

- Vet check females that had difficult deliveries/RFMs/discharges/lameness/severe weightloss

Mating time

- Record date and sire for all matings
- Check interval between unpacking and first mating – can interval be reduced to keep females in annual birthing pattern?
- Check male fertility on regular basis

Do you have enough males and are they healthy?

How many supervised yard matings can each perform per day?

How many spit-offs/positive pregnancies does he achieve per mating?

Can he be used for paddock matings?

Observe him working and get females ultrasounded to positively identify pregnancies

Handle males carefully

- Avoid inbreeding
- Get females examined by vet if have had 3 matings and failed to conceive

Pregnancy

- Wean at 12-16 weeks (lactation over by 100 d/14 wk) to allow:

Skinny females to conceive when start gaining weight after weaning

Pregnant females to regain weight and feed the foetus (foetal ovaries, wool follicles etc)

- BCS pregnant females and monitor

Consider options to maintain or increase BCS during gestation

Focus on females that have just had first cria

- Get females pregnancy tested

Know the reproductive status of your females

Assess male performance

- Place pregnant females in a paddock next to mating yards every 2-3 months

Females that have aborted will come up and sit next to fence

Investigate females that have lost a pregnancy

Monitor abortion rate and seek help if excessive

Culling

- Prepare a culling list
- Does culling affect future fertility?
- Get females suspected to have aborted pregnancy tested by a skilled pregnancy tester before culling
- Will you carry over non-pregnant females?

COMMON REPRODUCTIVE PROBLEMS IN FEMALES

Female alpacas are usually presented for infertility examination because they are unable to conceive following multiple matings, they have aborted, they have visible abnormalities of the external genitalia or they are continuously rejecting the male (Vaughan and Tibary 2006). Diagnosing the cause of infertility requires a full reproductive history, a general physical examination and thorough evaluation of the reproductive tract using transrectal ultrasonography, vaginal examination, uterine cytology/culture/biopsy, laparoscopy and chromosomal testing. The congenital and acquired conditions of infertility described in the female alpaca are listed in Table 1.

Table 1. Disorders of the female reproductive tract in alpacas

Ovary/bursa/oviduct	Uterus	Cervix/vagina/vulva
ovarian hypoplasia*	segmental aplasia*	double cervix*
paraovarian cysts*	uterus unicornis*	segmental aplasia*
hydrosalpinx*	uterine hypoplasia*	persistent hymen*
teratoma*	mucometra*	vulvar aplasia*
ovulation failure	endometritis#	infantile vulva*
persistent corpus luteum	endometrial fibrosis#	intersex*
anovulatory large follicles	pyometra#	adhesions post-dystocia#
oophoritis#	peri-uterine adhesions#	cervicitis#
granulosa cell tumour	uterine cysts/abscesses#	
salpingitis#	uterine adenocarcinoma	
pyosalpinx#		

*congenital condition and should not be used for breeding

#infection/inflammation may result from abortion and/or poor management

The most common cause of infertility in a study of approximately 150 infertile females was ovarian hypoplasia (Sumar 1983). The condition may be partial or complete, unilateral or bilateral. Animals with complete bilateral hypoplasia will have no primordial follicles and are therefore sterile, but females with partial hypoplasia should not be used for breeding as the condition is likely to be heritable. Segmental aplasia of the uterus, cervix or vagina is usually accompanied by mucometra as there is no outlet for glandular secretions produced by uterine glands.

Common causes of acquired infertility include endometritis, persistent corpora lutea and dystocia-related trauma resulting in secondary fibrosis of the uterus, cervix and/or vagina. Thorough examination of the reproductive tract will allow diagnosis and treatment of these conditions.

It is important for owners to understand reproductive physiology of both males and females to manage their breeding program correctly. Try to keep clear, concise notes on animal

identity/matings/spit-offs/ultrasounds to assist with infertility investigations. Learn what is normal so you can recognise the abnormal!

Risk factors for uterine infections

Reduced general health

- Maidens
- Thin females
- Poor nutrition
- Metabolic diseases
- General stress such as adverse weather, transport, overcrowding, other diseases
- Selenium/vitamin E deficiency which may suppress immunity

Reduced uterine health

- Difficult births
- Uterine tears
- Dead crias (stillbirths)
- Assisted delivery of crias
- Uterine prolapses
- Retained foetal membranes (placenta)
- Twins

Inexperienced owners should not attempt to correct a mal-positioned foetus as they risk damaging both the dam and the cria. Rupture of the uterus, trauma to the cervix and/or vagina can lead to infertility and at worst to death. Owners attempting to correct a mal-positioned foetus MUST do the following:

1. Ensure the dam is in a large, clean area (dry, grassy yard ideal) and well restrained. Do not attempt to pull a foetus in muddy yards or to chase a female around a paddock while pulling on the cria.
2. Scrub your hands and arms with warm soapy water before attempting any traction/correction. Every time you introduce your hands, you carry bacteria into the reproductive tract, which will contribute to uterine infections and vaginal fibrosis.
3. Wash the perineum of the dam with warm dilute disinfectant to remove faeces/hair/foetal fluids so you do not carry them inside when you put your hand through the vulva.
4. Use plenty of clean, water-based lubrication. Do not use lubrication that has been sitting around in the shed for months (e.g. used for ultrasound examinations) that is contaminated with dirt/insects.
5. If in doubt, seek veterinary assistance sooner rather than later. The longer you intervene without success, the more likely the cria will die and the dam will suffer complications. If you cannot work out the dystocia, it is likely your neighbour/visiting alpaca farmers will not either. Get on the phone to your veterinarian.
6. 'Tears and spares'. If a female still looks in discomfort after you have successfully extracted a cria, then she may have a tear in her uterus/vagina or there may be another cria still inside. Call your veterinarian to investigate.
7. Do not pull on foetal membranes after delivery of the cria as it may result in tearing of foetal membranes or uterine prolapse. In consultation with your veterinarian, consider use of oxytocin to assist with expulsion of membranes and contraction of uterus, and use of systemic antibiotics to reduce uterine infection.

Reproductive efficiency of male alpacas

Male fertility plays a key role in reproductive efficiency in alpacas. Sperm production is correlated with testicular weight and testicular length in alpacas and Table 2 indicates how mean testicular length may be used to estimate the likelihood of sperm production (Galloway 2000). Selection of early maturing males with large testicles should ensure optimal conception rates in any alpaca herd. Both genetics and environmental influences govern age at puberty and testicular size.

Table 2. Development of testicular function in alpacas with testicles of different length (Galloway 2000).

Mean testicular length (cm)	Proportion of males (%)	Percentage of testicular tissue producing elongated spermatids*
<3	100	0
3-4	68	<10
	31	30-60
>4	36	<10
	31	10-60
	31	>60

* Elongated spermatids are the last step in the cellular production line before spermatozoa are produced in the seminiferous tubules of the testes.

Disorders of the male reproductive tract

It is essential to establish whether a male is sexually mature during an infertility investigation, as age at onset of puberty in alpacas ranges from less than 12 months of age to greater than 4 years of age. Breeding soundness examination of infertile male alpacas includes collection of a thorough breeding history, a general physical examination and palpation and ultrasonography of the scrotum, testes and prepuce (Tibary and Vaughan 2006). Testicular length is a very important indicator of sperm production ability and fertility (Table 2). Testicular biopsy, mating ability and semen evaluation will complement the examination. The congenital and acquired conditions of infertility described in the male alpaca are listed in Table 3.

Testicular hypoplasia was the most common cause of congenital abnormality in the male reproductive tract in a study of more than 3000 alpaca males (Sumar 1983). The condition is usually bilateral, but may be unilateral, total or partial. Histologically, there is a reduced number/absence of seminiferous tubules and no spermiogenesis. The condition is suspected to be inherited in alpacas and affected males should not be used for breeding.

Testicular degeneration is probably the most common cause of acquired infertility and results from heat stress, trauma or chronic inflammation of the testes secondary to severe systemic disease (Tibary and Vaughan 2006). The testes are usually smaller than normal and may be soft for very firm and fibrous. The testes may recover following degeneration if the cause is removed but long-standing degeneration may further deteriorate into testicular atrophy.

Table 3. Disorders of the male reproductive tract in alpacas

Scrotum/Testis	Penis/prepuce	Epididymis
testicular hypoplasia*	urolithiasis	epididymal cysts
cryptorchidism (intra-abdominal)*	balanoposthitis	
ectopic testes (extra-abdominal		
and extra-scrotal)*	paraphimosis	
monorchidism*	preputial prolapse	
testicular cysts*	preputial swelling	
testicular degeneration		
orchitis		
hydrocoele		
seminoma		
scrotal trauma		

*congenital condition and should not be used for breeding

Male management to maximise fertility in the herd

- Check scrotum for testes at birth
- Measure testicular length every 6 months and record findings
- Check for scrotal oedema during hot/humid conditions
- Complete breeding soundness examination, vaccinations, drenches, shearing 2 months before breeding season
- Adopt early pregnancy diagnosis to observe early infertility problems

REMEMBER THAT NO DRUGS ARE REGISTERED FOR USE IN CAMELIDS!

References available upon request

Thomas Westermann

Veterinary Student – Adelaide University Vet School

I grew up in Kuitpo forest, in the Adelaide hills, on our family hobby farm with cows, sheep, alpacas, rabbits, mice, cats, dogs, frogs and fish. I've been fascinated by animals my whole life, and have a special bond with rabbits and alpacas, both of which I have bred for many years, and my cats and dogs. I have a keen interest in exotics and native fauna, such as the joey pictured, right. I particularly like raptors, which I've been lucky to often observe on our property's bush land, and bordering forest.

I am a committed and hardworking student, and have thoroughly enjoyed my veterinary studies so far, and look forward to what the future will bring.

THE PREVALENCE OF DERMATOLOGICAL LESIONS IN A SUBPOPULATION OF ALPACAS (*Vicugna pacos*) IN THREE REGIONS OF SOUTH AUSTRALIA

Authors: Thomas Westermann and Dr Mandi Carr

Objective

To determine if the prevalence, lesion characteristics and bodily distribution of dermatological disease in a subpopulation of South Australian alpacas is comparable to international findings.

Design/Procedure

Three hundred and thirty eight alpacas were surveyed during the 2013 shearing season, from the Fleurieu Peninsula, Adelaide Hills and Mid-North regions of South Australia, using a comprehensive skin scoring chart. Local shearers assisted with data collection following an induction program. The results were then tabulated in a spreadsheet (Excel; Microsoft), and statistically analysed.

Results

The prevalence of skin disease in South Australian alpacas was shown to be 60.36% ±5.21%, with the majority having one lesion (11.24% ±3.37). Body areas most often affected include the pelvic limbs (31.07% ±4.93), axillae (24.56% ±4.59) and thoracic limbs (19.23% ±4.20). The most common lesion characteristics include scale and thickening (41.42% ±5.25), crusts (36.69% ±5.14), alopecia (31.07% ±4.93) and erythema (30.18% ±4.89). Significant relationships were established between

geographical region and lesion bodily distribution (P = 0.0001), geographical region and lesion characteristics (P = 0.0001), and lesion bodily distribution and characteristics (P = 0.00).

Conclusions

Skin disease was seen in the majority of the sample population, although lesions were often few in number and present in body areas of little significance to fleece production. Despite this dermatological disease still has the potential to decrease stock value, cause increased management and treatment costs, and impact negatively on animal welfare. Further research into dermatological disease in Australian alpacas is required.

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Chris Williams

Chris Williams diverted his professional attention to alpacas in 1992, excited by the opportunities and business potential of the industry. With an interest in the vertical integration of his alpaca business, the desire to expand his alpaca expertise and participate in the development of the Australian and International Alpaca Industry, Chris has developed a diverse alpaca portfolio over the last 20 years.

He is a passionate alpaca breeder, owning around 1,100 alpacas within his herd *Ambersun Alpacas*. Chris has also exported over 200 alpacas to Europe, and runs these herds with the assistance of his European network, in five different countries.

Chris has also been President of South Australian Region of the Alpaca Association, a member of National Committee of the Australian Alpaca Association and a member of Australian Wool Development Board, representing the alpaca industry. Chris is also a qualified alpaca judge.

Chris is an experienced alpaca shearer, shearing up to 4,000 alpacas each year in Australia and 10 different countries in Europe.

Chris has also participated in the manufacture of alpaca clothing under the label of *Alpaca Classica*, retails alpaca products through his Alpaca Shop in Hahndorf, and is now also involved with the promotion and sales of alpaca meat, in one of his newest ventures of *Fleurieu Prime Alpaca*, working with Prados Alpacas.

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