NZAGA Newsletter

NZ Arapawa Goat Association - February 2018, Issue 13

From the Editor

Hello everyone.

The first article, Shady Lady's story, part 2, written by Christine Mander, is a **must read** for **all** goat owners. I am mortified that, in my ignorance, I passed on this horrific problem to a wonderful woman who only sought to help the sweetest little kid. Several years ago I was given two beautiful old Arapawa does to care for. Both appeared healthy and settled contentedly into our family of goats. Shortly after one of the does gave birth, she grew lethargic, faded quickly and died. Within days her only kid showed symptoms of being unwell. I rushed him to the vet and he was diagnosed with, and treated for, coccidiosis. A few months later, the other older doe died with very little symptoms shown beforehand. If only I knew then what I know now. While we can't stop all the loses, I believe we can learn from them. Among the many lessons I have learnt:

1. To encourage the involvement of new breeders/owners and to establish satellite herds around the country; we must share the love for our Arapawa goats and no one owner should be allowed to monopolise the breed.
2. Become informed about everything related to goat health. Build up a relationship with your local vet and learn to **screen for worm burdens** (more about this further in the newsletter).
3. Social media is a brilliant and effective way of communicating and bringing together the 'Arapawa Goat Family'. I am not a fan of Facebook, but there are 3 groups that are on my 'priority' list: Arapawa goats, NZ Goat Talk, & Rare Breeds Conservation Society NZ.

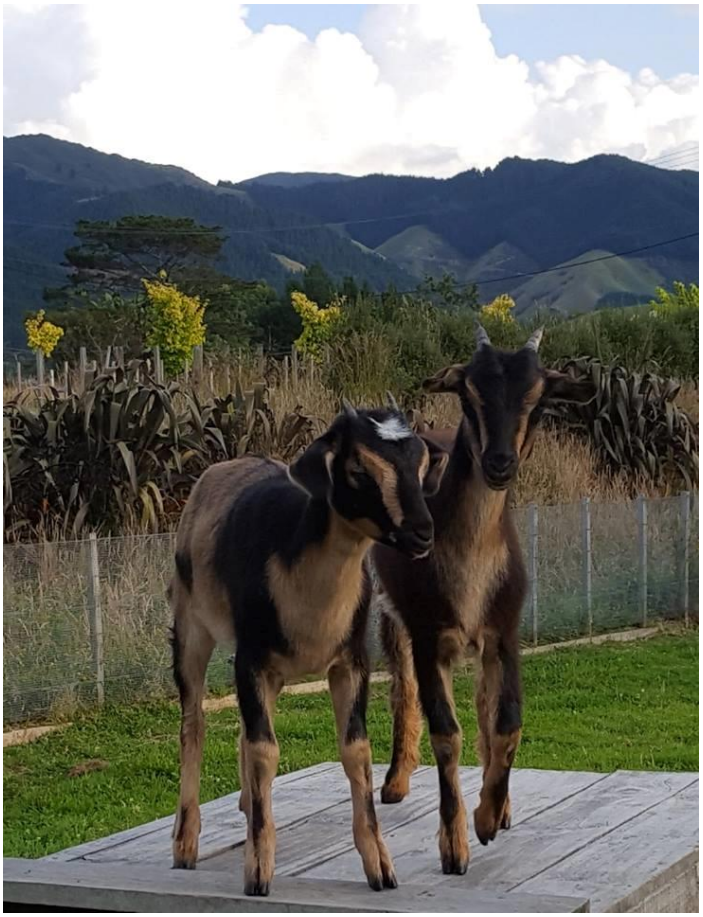
Which leads me to announce this is the last of the NZAGA Newsletters that I will be sending out quarterly. Almost everything that is in this edition comes from the Arapawa Goat and NZ Goat Talk Facebook pages. It is surpassed by what the social media platform offers: interactive and timely advice from people with a variety of skills, experiences and knowledge. Of course, **if there are any members out there who would like to continue putting out the newsletter, please put up your hand** as not everyone is comfortable on Facebook.

alison@xtra.co.nz
021 2015 007

Newsletter Contents:

The Arapawa Goat DNA samples are booked in to be run next week; results due at the end of this month. As soon as I hear, I'll send an email around.

Shady's Story Part Two, by Christine Mander



It turned out that getting Shady's broken leg to heal was the easy part. From the start I knew that Shady would need to be confined on her own for at least 6 weeks. Goats prefer to hang out in groups and I was really concerned about how I could keep her healthy and happy when she would be on her own for that length of time.

On Monday 18th September I brought Shady home from Alison's. Luckily I was on annual leave for the week and so could spend plenty of time with her. Off to work I went to annoy my work mates and sedate Shady for x-rays and splinting of her fractured cannon bone. I was very grateful for the interest and help of a colleague but, even with the heavy sedation and pain relief and his help in applying traction, we were unable to reduce the fracture accurately. There would already have been some fibrosis and muscle contracture.

On recovery from her sedation I treated Shady for lice (off-label use of permethrin powder for cats and dogs), coccidia (Baycox C, again off-label) and intestinal worms (Panacur, at double the dose rate recommended for lambs) – anticipating that the stress of weaning, change of environment, pain and healing her injury would make her particularly susceptible to any diseases she might be carrying. Within hours, she developed diarrhoea and I spent days beating myself up for doing too much too soon. Two days later, her droppings were back to normal and my little princess had me wrapped around her little hoof. I found I was spending most of my spare time gathering a range of fresh browse and forage for her to choose from, along with hay, lucerne chaff and multifeed nuts, and keeping her company whenever I could. Her food preferences changed from day to day and it was difficult to know what and how much she was eating as she would regularly throw tantrums, tip over her feed bins (even when I put rocks in them) and the food would then fall through the slats of her shelter, which sits on a deck above a steeply sloping paddock. Also the local ducks, shelducks, pukekos and passerines were clearly joining the party.

On Tuesday 10th October, I sedated her again to re-xray the fractured cannon bone, remould the splint and applied fresh bandaging. A bony callus was starting to form but there would still be at least 3 more weeks before bony union could be expected. I was becoming increasingly worried over the following weeks that Shady was just not thriving. She was increasingly picky with food and not as cheeky and naughty as previously. Never chunky, she was starting to look quite gaunt and her poo production plummeted, indicating that she was eating less. I gave her a second dose of Panacur on 22nd October and took her out into the paddock on a lead to explore for a bit, thinking that might perk her up. The next day I decided a Vitamin B12 injection couldn't hurt either.

I re-xrayed her on 28th October, just under 6 weeks after first applying the splint and without sedation this time. *(See comparison x-rays on the right).*

The x-rays are of poor quality as they were taken through the splint so there would be no risk of re-fracture if she struggled and the bone was less well healed than I anticipated. Shady had not gained as much weight as she should have but I was relieved that she had at least gained some. Even though the fracture was not completely healed there was a good bony callus down the front and one side. Having not been able to re-align the fracture properly, I knew there would be a bit of distortion diagonally – but if you saw her today, you would never guess there had been a problem. I decided to remove the splint due to loss of bone density and her increasingly abnormal floppy way of placing her hoof. Because she was still confined to the shelter and seldom bothered to jump on top of her little house any more, I think this was a good decision. She was only slightly lame and this resolved rapidly with normal hoof placement returning and strength improving over the following week. That evening I spent a short time in the goat paddock with her, supervising her initial attempts at interacting with Ali and Finn, my 2 year old Arapawas. Finn just wanted to play fight and Ali acted as if Shady was the monster from the black lagoon, and so we retreated after a short time – just as well as it turned out.

Shady continued to be a little too quiet and friendly, and precious about what and how much she would eat. There was no sign of abdominal discomfort and there had been no diarrhoea since the first couple of days. I decided to do a faecal egg count on 3rd November with a view to introducing her to the goat paddock in a week or so, and collected a sample the night before. The next morning, I found she was passing quite soft faeces and so I tested some of this instead. I was horrified to find @ 7,000 coccidia oocysts per gram of faeces (and zero worm eggs) and by the time I got home from work, Shady was scouring badly. I know that coccidia are common but had never found them before when screening my sheep and goats for worm eggs. And now I had a case of full blown coccidiosis. It was just good luck that I had decided to check precisely at the onset of clinical signs.

So what had gone wrong? I had given her a treatment of Baycox C (Toltrazuril) when she arrived and there was very little opportunity for her to have picked up a new infection on my property as she had been living on a wooden deck that had never seen a goat. I had been removing any faeces once to three times daily, before they could have become infective (most take at least a couple of days to sporulate). Grass had been picked for her from places that my goats had never been.

Had I under-dosed her? Possibly. I had given her the label dose (for calves; the product is not licenced for use in goats) of 15 mg per kg (i.e. 3 ml as she was just under 10 kg at the time). I later consulted Doctor Google and found that there is some evidence that goats require twice the calf dose (i.e. 30 mg per kg or 6 ml per 10 kg) although others have had excellent results with a dose similar to or lower than the label dose for calves. A Turkish study found that 25 mg per kg for 2 consecutive days was safe and effective and so I decided to follow that protocol on 3rd and 4th November.

Was the treatment failure due to poor hygiene? I had kept the floor and food bins meticulously clean, removed any soiled bedding whenever I saw it, and replaced all straw and water weekly. But perhaps she was re-infecting herself because I wasn't scrubbing her water trough and replacing her straw bedding every day. Following the 2nd treatment course of Baycox, I would pursue this obsessive cleaning regime, also sponge bathing Shady whenever I saw any faecal soiling – oocysts can be so numerous that a goat can potentially superinfect itself through grooming.

Shady's scour stopped after the first dose of her November Baycox treatment and her demeanour, appetite and body condition improved immediately, suggesting that coccidia infection and not social isolation was most likely the main cause of her stunted growth. Two days after the 2nd dose, her faecal sample was completely clear of oocysts. I continued to repeat weekly faecal tests until I was confident that Shady was thriving and it was safe for her to join the herd. I wanted her to be part of my herd as soon as possible but did not want to put the most vulnerable younger kids at risk if she was still shedding large numbers of coccidia oocysts.

And just when I thought it was over – embarrassingly the day before Onyx the buckling went to his new home on New Year's Eve – I did another routine faecal screen and found that Shady was again excreting oocysts in quantity. Onyx's sample was clear but I had to inform the new owners that he had been exposed and may well start shedding over the next days or weeks. I gave up waiting for Opal to provide a sample that day but she started scouring the day after her brother Onyx left the property – his mother didn't seem to care but Opal was initially distraught by his absence. Unsurprisingly, she too was excreting large numbers of coccidia oocysts in her faeces (along with too many nematode eggs for my liking; no more Panacur for my goats as it is no longer effective enough). She too was treated with Baycox and recovered rapidly. Clearly my plan of "eradicating" Shady's coccidia and isolating her until she was "safe" to join the herd had failed. I now accept that coccidia will be endemic in my herd – much like most people's I suspect - and will have to manage accordingly. Because young kids are the most vulnerable to coccidiosis, they will be fed **calf pellets containing a coccidiostat that is safe for goats to prevent the onset of clinical signs and massive shedding.** I plan to introduce the food from 8 weeks and continue until beyond weaning. It goes against the grain but I see no alternative. I can muck out and fill damp spots with sand as often as I can but the goats will still poo in their shelters and eat their straw bedding the minute my back is turned. These behaviours make it easy to understand why coccidiosis can be such a huge problem for goat farmers. It will also be interesting to monitor whether and which does excrete large numbers of coccidia around kidding time and consider selective breeding from the ones that don't – or at least see if mothers and daughters tend to show the same tendencies.

A one-off dose of Baycox can potentially cure an animal of coccidiosis – i.e. put a stop to the scouring and disease. But it turned out that I was very naïve to hope that I could completely eradicate Shady's coccidia infection. Any resistance will be specific to the type(s) of coccidia that the animal has previously been exposed to. When goats are moved between flocks, they bring with them a whole ecosystem of microorganisms and parasites. Goats of all ages will be susceptible to new infections with coccidia that they have not previously been exposed to but kids will be especially vulnerable to developing clinical disease. Previously exposed (and likely chronically infected?) individuals that have recovered from disease are best thought of as having built up some tolerance rather than being immune to coccidia and this tolerance is likely to break down at times of stress, with certain individuals thought to be prone to bouts of heavy shedding of oocysts.

As with worm drenches, there is concern about coccidia rapidly developing resistance to many of the available treatments. This is one of the reasons that you need to do everything you can with management

to minimise the goats' level of exposure to coccidia oocysts (and worm larvae) in the first place. Strict hygiene and avoidance of wet muddy areas around shelters and feeding stations etc. will help to reduce the number of oocysts stock are exposed to. On properties with a history of coccidiosis, where you know that kids will be exposed it may be preferable to introduce a feed containing a coccidiostat that is safe for goats before they reach an age when you might expect to see clinical signs. This avoids stunted growth and gut damage in the treated individuals and also reduces the level of contamination for future generations that can occur when kids with coccidiosis start excreting gazillions of oocysts.

I learnt from this experience how persistent and insidious coccidia can be. In future I will screen regularly for coccidia, especially in the kids from a young age. This will not provide total assurance as disease can develop before there is evidence of infection in the form of oocysts in faeces. Although you cannot generally avoid low level infection with coccidia, you want to prevent clinical disease if at all possible by avoiding overcrowding and wet conditions, and practicing good hygiene etc. But the reality is that many of us with multiple goats on smallish areas of land will come up against cases of clinical coccidiosis, with acute disease especially common in kids under 6 months old. Coccidia are ubiquitous, and the stress of kidding means that does may pass increased numbers of oocysts, pretty much guaranteeing that the kids will have some exposure. If you are unlucky enough to have kids infected with significant burdens of the more pathogenic coccidia species, you may have very little time to treat affected and exposed animals. There may be a lot of damage to the gut before the diagnosis can be confirmed through finding oocysts in the faeces. This is because the parasite goes through several cycles of asexual reproduction before starting to produce oocysts. It therefore may be necessary to treat without delay on the basis of a presumptive diagnosis. If there is a history of coccidiosis in the herd, it may be better to start preventive treatment in the form of coccidiostat (there are several that are safe to use in goats) in the kids' feed well before the age when you would expect to see clinical disease. This allows for earlier suppression of the shedding of oocysts, less ill thrift, and reduces the level of contamination of pasture and shelters etc. and thus a lower level of challenge to susceptible animals in future. Once the oocysts are sporulated (the infective stage), they are very hard to kill except through freezing. If you are using a feed containing coccidiostat, please remember to be very careful to prevent other species, particularly horses and dogs, from eating it as it is extremely toxic to them.

In a situation where treatment with Toltrazuril (Baycox) is necessary, goats may or may not require higher doses than do other livestock species. The same applies to some of the worm drenches – for example, Panacur on its own has a wide safety margin but there could be a real risk of toxicity if you increased the dose rate of a drench that also contained selenium or other mineral additives. Very few worm drenches are licenced for use in goats and many appear to be metabolised faster by goats compared with sheep. Increased dose rates or dosing twice on consecutive days are recommended for some drenches – e.g. Panacur, Cydectin, Startect, Zolvix. Many of the commonly used multi-agent drenches on the market do contain added minerals, are not licenced for goats and it is difficult to find even anecdotal information on suitable dose rates. I would be very careful with these and would strongly recommend people get faecal egg counts done to find out whether their drenches are actually effective.

For anyone who feels they want to better understand the principles of controlling intestinal worms in goats, I would strongly recommend the following information source:

wormwise.co.nz/wp-content/uploads/2014/06/Wormwise-for-Goats.pdf

---

Christine Mander BVSc (NOT a livestock vet so please listen to your regular vet rather than taking any of my long rant as gospel)

Screening for faecal eggs in goats and sheep

Goats are susceptible to worms and will die if they are not caught in time

If only I had known this years ago! Aware that regular trips to the vet is just too expensive for most lifestylers, I asked Christine if there was a way to do our own faecal egg counts. This is her rely …

"Faecal egg counts are really easy (though time consuming) to do but the initial investment in a microscope would be daunting for many people. Other than that, all you need is a McMaster egg counting slide, scales for weighing out 2g of poo (ideally more accurate than the kitchen scales I use), a sieve, pipettes, sugar and/or salt to make up a supersaturated flotation solution and something for measuring this."

She also told me instructions and photographs to help identify nematode eggs and coccidia oocysts are readily accessible on the internet. So, online I went and found the following link that could be useful:
http://blog.microscopeworld.com/2012/10/parasites-in-sheep-goats.html

Probably the most useful information there, was that the microscope can be a simple high school model. I was now on a mission. One supplier recommended what he described as a cost-effective monocular microscope that would do the job. So at $259 I bought it – and he was wrong. A good learning tool but I would soon learn my new toy wasn't good enough. Reading and/or advice from people with a marketing agenda is fine, but learning is best done through hands-on tuition by experts. So I reached out to our local Keinsley AgVet, and wow! Amazing. I felt absolutely empowered.

First we established that this is about **screening for faecal eggs, not diagnosing**. The more information we give them just helps them do their job. It is about rare breed enthusiasts and vets working together for the benefit of our animals (limited to goats and sheep as other species have different problems). The rationale is we screen rather than drench routinely. When screening suggests a worm burden, take a sample to the vet for diagnosis and treatment advice. Catch it early before it kills. Following treatment, check to determine effectiveness.

Off to the farm to collect goat poo samples and, armed with my new microscope, back to the vet I went to learn the procedure. Lesson one: to do a proper job we need a microscope with a built in light, an eyepiece with a 45^{o} angle and a mechanical stage. My one will do the job (with its little mirror to reflect light) but we can do better for the same price (via importation). An ordinary glass slide and cover will do the job, but again, we can do better by purchasing a McMaster slide ($65 but you only need 1; again cheaper if we import).

So **where to from here**? Well, I now feel confident that I can screen for faecal eggs and even identify some of the common variety parasites. But I have no goats, or sheep for that matter. But I do have my teaching skills and so I have created a step by step lesson plan on how to screen for faecal eggs. If anyone is interested, maybe we could meet up in small groups and share the knowledge. I would be happy to make up kits (with or without a suitable microscope) that learners can reimburse me for at cost.

Alison

1 Coccidia

3 Hook worm

2 small intestinal worm

First Aid kit for goats, Lex McKay

1) A good torch
2) Thermometer
3) Vaseline and Lanoline
4) Dettol or similar
5) Hydrogen peroxide
6) Iodine (liquid) and antiseptic powder with puffer
7) Terramycin cream (for eyes or open wounds) Comes in a tube
8) Baby's bottle and teats. Plastic drink bottles and lamb teats
9) Eyedropper and plastic syringe with tip cut off
10) Brandy (*for the goat or the owner? Editor*)
11) Roll of elastoplast and adhesive bandage
12) Roll of wadding
13) Plaster of paris bandages or similar
14) Clean pieces of wood or halved pvc for splints
15) Cod liver oil (a good source of Vitamin A & D)
16) Pentavite
17) Ketol (for Pregnancy Toxaemia)
18) Electrolytes (for dehydration)
19) Poison plant book
20) Clean soft rags, paper towels and newspaper
21) Strong plastic spoon
22) Small narrow necked bottle (to administer tonics)
23) Probotic capsules (from chemist)
24) Old woollen jerseys that can be made into rugs, etc
25) Frozen colostrum

I would add to that list: Stock Brew, scissors and wooden spatulas (great splints for smaller animals). Having read Christine's article, I would now strongly recommend those who live away from town, talking to your vet about having both penicillin and Baycox on hand.

A goat's vitals:

Average pulse rate **90** (variation 70 to 135) beats/minute

Average temperature (daytime) **102.8** (100.0 to 105.0)

Average respiration **19 breaths** per minute

Show quality animals

Exciting to see members discussing the characteristics of the Arapawa Goats and looking towards quality animals rather than pets. No doubt we'll be seeing them at livestock shows soon. Heaven forbid we'll start hearing about champions! Based on the expertise of the first people to rescue and advocate for the Arapawa goats, a breed standard was drawn up in 2014 and can be accessed via the arapawagoat.com website link: http://arapawagoats.com/standard.html.

4 *Jo with Baccus at Canterbury A&P show*

History in brief:

The New Zealand Arapawa goat breed can be traced back to six goats left on Arapawa Island during Captain Cook's journeys to the southern ocean. It is believed that one or more of these goats were of the Old English Milch goat breed. Because of a 'goat control programme' operated by the Department of Conservation, and which has been ongoing since the 1970s, at Betty Rowe's initiative a number of Arapawa goats were taken off Arapawa Island and given sanctuary by people determined to protect the integrity of the breed. All goats determined to be purebred Arapawa goats, and registered as such, can be linked back to Arapawa Island.

General Appearance:

Smaller than modern milking goats, the general appearance of the Arapawa is a small, light-framed goat with all parts of the body in balanced proportion relative to its size. The buck presents as heavier in the head, neck and forequarters. All Arapawa goats have distinctly patterned faces, which are long and narrow. Dark brown or black-striped facial markings are distinctive features of the Arapawa breed. The ears are placed at the upper part of the skull and are small and expressive. Horns should be symmetrical, with the does' sweeping up towards the back and the bucks' sweeping up, back and curling outwards. The overall picture is that of an alert, good-natured, attractive animal. Arapawa goats do not have tassels.

Size:

Females and males are considered mature at 24 months. As a general guideline, the height at withers of a mature animal should be 24 to 28 inches (61 to 71 centimetres) for females and 26 to 30 inches (66 to 76 centimetres) for males.

Desirable Features:

Distinguishing male from female: Sexual differences between doe and buck should be immediately obvious.

Eyes: Full of expression, alert and bright, the Arapawa goats' eyes have an amber iris and a black pupil.

Head and Neck: Strong and in proportion to the length of the body. The head is wide at the eyes and tapers to form a distinct bridge at the nose. Some (male and female) sport elegant goatee beards.

Back: Must be strong, broad and relatively straight with the presence of a dorsal stripe or shading along the centre of the spine.

Loins: Should give the appearance of strength. These attach to a wide, generous croup.

Croup: Continues the topline, and should have a gently sloping appearance, to allow for easy kidding. Too steep a slope is undesirable.

Tail: Short in relation to the body, carried gaily, pointing upwards.

Ears: The ears are pixie type that when folded reach just below the eye level.

Limbs: The front legs should be in proportion to the depth of the body. The legs should be straight, strong and well placed, with strong pastern joints and well-formed, dark hooves.

Colour: Arapawa goats vary in colour but are predominantly tan, brown, white and black in varying combinations with dark brown or black badger stripes on the face.

Coat: Lustrous and in good condition, free of foreign debris and parasites, the coat can be long on the hind quarters (petticoat), or all over, with some fringing along the back. In winter they have fine, matted underwool coats.

Doe (Nanny): A strong emphasis on femininity. The round-bellied look of the Olde English goat, slender and fine-boned. The horns of the does are round, shorter than the bucks and curve backwards over the head. The doe should appear finer than the buck. Both her fore and hind legs should appear strong, but correspondingly finer and more delicate than the male's.

Buck (Billy): A strong emphasis on masculinity. The round-bellied look of the Olde English goat, but should be solid and stocky. Flattened, wide-sweeping horns. The buck's body structure should show more massive muscling than that of the doe.

Alison Sutherland

I have learnt

When you suspect a goat has eaten too much grain, never, NEVER give them water!

Unwanted bucks

A question from the Facebook page: "Just wondering if you could ask in the newsletter if anyone in the South Island might be thinking about establishing a buck paddock, like Kitt is up his way? (north of Auckland)

My response:

We certainly do need a buck paddock, but rarely does anyone put their hand up for this. Personally, I rarely sold my mature bucks, instead asking the new owner to cover the cost of transport. This is one of the reasons why I wether the majority of my bucklings and sell them as pets (or free as a companion for a doeling). In the real world of farming, males that are surplus to requirements go into the freezer. As our numbers grow, this is likely to become the outcome for unwanted bucks. Alison

More questions on the Arapawa goat Facebook page:

"Miss Bunny and Blaze free range on my section which is full of trees, bushes and grass. They are free to eat anything as we have fenced off plants/trees that we want to keep. They have pumpkin seeds daily to help with any parasites and Stock Brew everyday to help with their rumen. They also get a daily dose of Megimmune Cu which contains all the minerals a goat should need and free access to hay 24/7. Please advise me if I need to get them anything else and if I should keep in medicines for just in case. We are a long way from the nearest vet."
This question led to Lex McKay's reply with the First Aid kit. Excellent advice.

"Wethered boys – how big do they grow?"

Once neutered, they grow to a similar size to your average doe, although sometimes a little leggier. Their horns resemble those of a doe and they often have a gentler nature than both genders.

So that is me, signing off for now. Warm cheers everyone, Alison