RIVERINA LOCAL LIVESTOCK UPDATE

April

NSW GOVERNMENT | Local Land Services

www.lls.nsw.gov.au/riverina

Case study: Under Age Party in Paspalum

By Emily Stearman, District Vet

Cattle

CASE HISTORY:

The veterinary team was recently contacted about a mob of cows and calves grazing a paspalum dominant pasture.

Cases of paspalum staggers had occurred historically on the farm in yearling-mature cattle but never calves. 35 Angus cows and calves were grazing paspalum for three weeks prior to any clinical signs. Initially two calves were found dead.

The decision was made to move stock out of the paddock. When pushed, three calves were incoordinate, with muscle fasciculations and hyperesthesia (exaggerated gait); calves appeared 'drunk' while the cows were unaffected.

Clinical signs increased with further movement; calves would fall and if cast on their side, it took an extended period to right their position and move off. When rested, clinical signs regressed and the calves were able to continue the journey with the cows.

PASTURE ASSESSMENT

Seasonal conditions for summer and early autumn have been favourable for pasture growth. The area has had frequent rainfall and increased days of humidity this year. The paddock consisted of around 95 per cent paspalum in its mature stage, standing over 60cm tall. The plant material was sticky to touch. On close assessment seed heads were discoloured, coated with sticky yellow material, known to be a fungus *Calviceps paspali*.

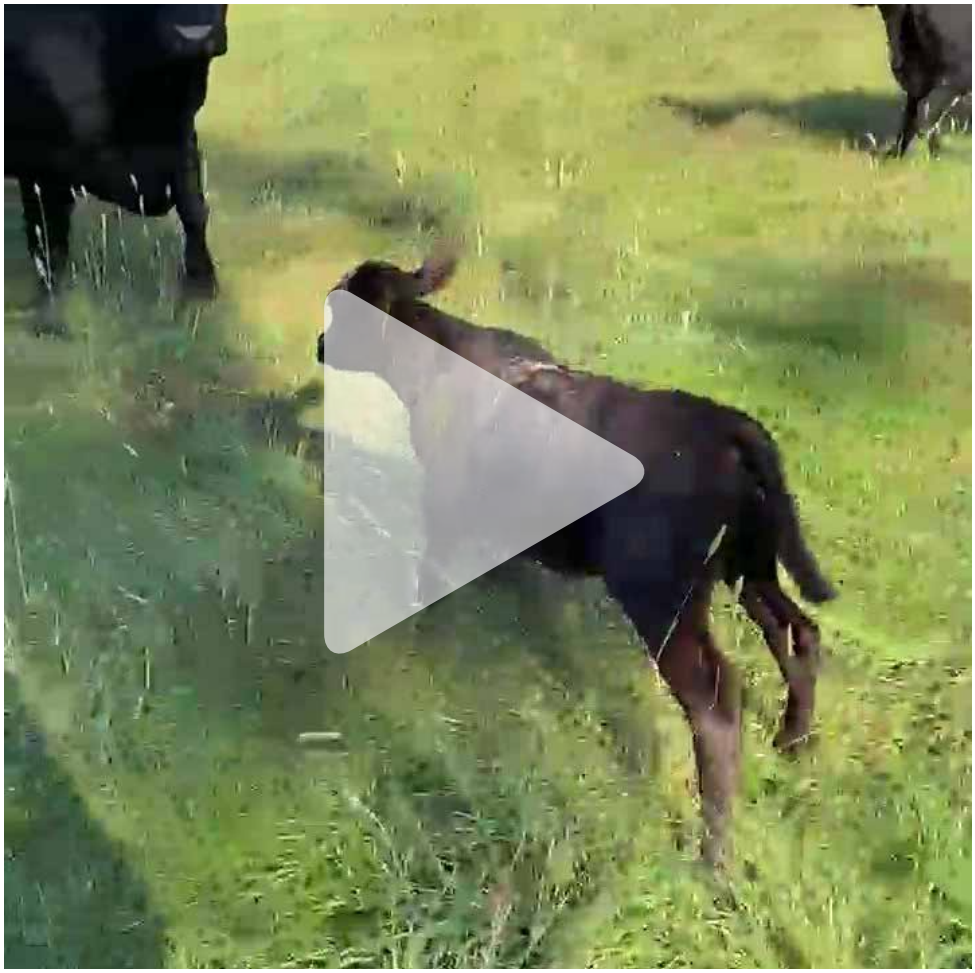
DIAGNOSIS & TREATMENT

Livestock: *Calviceps paspali* is an ergot-producing fungus, well documented to cause the neurological signs described above. Clinical signs resolve over time when livestock are removed from the infested plant material. Death is uncommon but can result through misadventure as a result of impaired mental function.

In this case, the mob was removed from the pasture and supplemented with good quality cereal hay and mineral lick block that contained calcium and other trace minerals.

The two calves found dead were a result of misadventure. Three calves were clinically affected at diagnosis, within 48 hours following removal these calves appeared normal while four days later the third affected calf had recovered.

Time of recovery will vary in animals; it can be prolonged by other underlying conditions or secondary conditions that occur as a result of misadventure.



Paspalum staggers in action (Click to play).

Pasture: When limited other pasture species are available, being able to put cattle back onto the affected paddock is important. The fungus predominates in the seed head, so slashing paddocks to remove the seed heads therefore removes the source of the fungal toxin.

Depending on the stage of plant growth, repeated treatments may be necessary. Regular monitoring of paspalum pasture in a high-risk season is recommended.

WHY WERE THE CALVES AFFECTED?

In this case, the reason calves were affected without cows is not clearly defined. It is possible that the height of the pasture provided a means for the teats to be heavily contaminated with *Calviceps paspali*, with ingestion occurring when suckling.

Alternatively, calves 3-4 weeks of age browse plant material, browsing behaviour could result in high rates of seed head ingestion, exacerbating the dose of toxin.

If you have mature paspalum dominant pastures, we recommend you assess the seed head for the presence of the fungus. Livestock should be moved off infected pasture. If clinical signs ensue, contact your local District Veterinarian for advice.

Calviceps paspali on Paspalum.

Case study: Unexpected Case of Acidosis

By Evie Duggan, District Vet

Sheep

CASE HISTORY:

1700 composite maiden ewes were introduced to a reshot barley stubble and had been grazing over the past month.

The farm employee mustered the ewes for yarding and noticed a significant tail end in the mob. Approximately 30 ewes had dropped to the back of the mob, with 20 becoming recumbent and progressing to death.

Lick blocks containing calcium and magnesium along with other minerals had been provided for the period that they had been on the stubbles. The ewes as a mob were scouring. The remaining 10 ewes that had dropped back but had not gone down were left behind in a paddock.

The District Veterinarian was contacted the next day to investigate the deaths.

CLINICAL EXAMINATION

Clinical examination: A less severely affected ewe out of the 10 that had been left behind was caught and examined – she was bright, alert & responsive (BAR), normothermic (normal temperature), had severe diarrhoea, pink mucous membranes and was sore footed (laminitic).

POST MORTEM FINDINGS

Three ewes were post mortemed. A significant volume of grain was found in the rumen of all three ewes and ruminal fluid pH was 5.

LABORATORY RESULTS

Aqueous humor (fluid from the front of the eye) from three dead ewes showed elevated D-lactate. Bloods from the less severely affected ewe (standing, BAR but scouring) also showed elevated D-lactate.

DIAGNOSIS

Ruminal acidosis.

WHAT DOES THIS MEAN?

Ruminal acidosis, commonly known as 'grain overload', occurs when large amounts of rapidly fermentable starch-containing feed is ingested that the animal is not used to.

This increase in carbohydrates causes the bacteria in rumen to produce an increased amount of lactate. Increased production of lactate causes the pH of the rumen to drop below normal pH of 6.8-7, further selecting for the bacteria that produce lactate.

When the pH of the rumen falls below 5.5, the growth of lactate utilising bacteria is inhibited, further exacerbating the issue. This change in acidity has effects throughout the body – influencing gut motility, hydration status and blood flow. In severe cases (acute acidosis) lactic acid is absorbed through the rumen wall and causes the blood stream to have a lower pH, thereby causing the circulatory system to fail and the animal to die.

In less severe cases (subacute) acidosis itself does not cause death, but the damage to the rumen wall allows the bacteria and microflora that belong in the rumen to enter the bloodstream and circulate through the body. This leads to the formation of micro-abscesses, often in the liver (due to the livers proximity and high level of blood flow).

Laminitis is also sometimes seen in cases of acidosis (both acute and subacute) and is thought to be caused by the lowering of pH in the bloodstream, stimulating substances in the blood that cause inflammation and expansion of the corium (which causes severe pain).

This complex change within the rumen can all occur within 24-48 hours of a diet change. In sheep that are not accustomed to grain, two kilograms to an adult is potentially fatal.

At first it could be assumed that the deaths were caused by the process of mustering, but the timing was coincidental, deaths would have occurred anyway. We commonly associated acidosis on stubbles with 'recent introduction'.

In this case, the fact some of the grain had reshot delayed the reaction so to speak. It wasn't until the sweet, short feed had been consumed that the feed intake reflected a sudden grain diet.

The history, grain present in the rumen and pH strip tests completed in the field showing a pH of 5 (normal is 6.8-7) in the rumen provided strong evidence for ruminal acidosis. The elevated plasma D-lactate concentration confirmed the diagnosis.

SAMPLE			01773 EWE.3	01773 1.AH	01773 2.AH	01773 EWE.4.AH
GGT	0-55	U/L	53			
GLDH	0-30	U/L	16			
AST	0-130	U/L	165 H			
BIL	0.0-6.8	umol/L	8.6 H			
CK	0-300	U/L	89			
CREAT	0-265	umol/L	667 H			
PHOS	1.13-2.58	mmol/L	1.74			
URE/CREA	0.00-0.07		0.05			
PROTEIN	55.0-80.0	g/L	69.1			
ALBUMIN	26.0-36.0	g/L	27.0			
GLOB	30.0-57.0	g/L	42.1			
ALB/GLOB	0.5-1.1		0.6			
CA	2.12-2.87	mmol/L	2.17			
MG	0.74-1.44	mmol/L	1.10			
HAPTO	0.00-0.30	g/L	2.88 H			
SERUM HB	0.00-0.20	g/dL	0.19			
UREA	2.9-7.1	mmol/L	32.5 H	13.6 H	19.1 H	25.1 H
BHB	0.00-0.80	mmol/L	0.80	0.12	0.17	0.16
D-LACT	0.0-0.5	mmol/L	1.6 H	15.7 H	5.7 H	12.9 H
AQ.HU CA	1.13-2.03	mmol/L		2.03	2.30 H	1.25
AQ.HU MG	0.65-1.55	mmol/L		1.45	1.98 H	1.82 H
GLUCOSE	<4.4	mmol/L		0.0	0.8	0.1
NITRATE	<10	mg/L		<10	<10	<10
NITRITE	<1	mg/L		<1	<1	<1

TREATMENT?

If identified at an earlier stage, recumbent/ obviously-affected sheep should be administered one gram per kg live weight of magnesium oxide, or 0.5 g per kilogram of live weight of sodium bicarbonate (baking soda), mixed in with one litre of water.

The ewes that were subacute were treated with oxytetracycline long-acting injection to prevent the formation of micro-abscesses in both the liver and lungs.

The mob was put onto improved pasture and offered hay.

FOR FURTHER INFORMATION

Grain overload, acidosis, or grain poisoning in stock | Agriculture and Food

Grain poisoning of cattle and sheep (dpi.nsw.gov.au)

Rumenal acidosis (flockandherd.net.au)

Announcements and additional warnings

MANAGING LAMENESS IN THE UPCOMING AUTUMN

It looks like it's shaping up to be another wet autumn, which will bring with it some challenges for managing lameness and body condition. Read on to see the most common causes of lameness to be aware of, and how lameness and pregnancy toxaemia can go hand-in-hand.

Lameness (what's causing it?)

1. Foot abscess

Appearing as a severe lameness, typically in one foot. When undisturbed the sheep will often hold the foot up. Foot abscess is commonly seen in heavier sheep (heavily pregnant ewes, rams, etc).

It is associated with damage to the sole of the foot caused by muddy conditions in rocky terrain, or poor foot conformation.

Antibiotics as a treatment for abscess should only be after consultation with a veterinarian and in combination with foot paring, this allows the abscess to drain.

2. Ovine interdigital dermatitis (scald)

In conditions where the foot remain moist for most of the day (when grazing crop or lush pasture), the skin between the toes becomes damaged and loses natural resilience against environmental bacteria.

With the skin damaged, *Fusobacterium necrophorum* (*F. necrophorum*) and other bacteria that are normally found in the environment establish and infect the tissue. The disease often resolves without intervention as seasonal conditions dry, facilitating the feet to dry and skin damage to heal.

Management for scald should focus on changing the environment of the foot - if the skin is no longer moist and damaged, the bacteria are not able to survive.

Foot bathing with 10% zinc sulphate can be effective in drying the foot, but is ineffective if sheep return to moist pastures.

3. Footrot

Footrot is caused by *Dichelobacter nodosus (D. nodosus)* and can be a debilitating disease of sheep.

For footrot to occur, first the foot must be infected with *F. necrophorum* (which occurs with scald). Once *F. necrophorum* is established, if exposed to *D. nodosus*, it may then also establish itself.

D. nodosus damages the cells of the hoof that generate new horn, which is why it is capable of creating 'under-run'.

Lesions associated with footrot are scored. A Score 0 is a normal/healthy foot, through to a Score 5 which is a severe lesion with separation of the walls of the hoof.

There is no other disease in the sheep feet that will create this characteristic underrunning (a score 3 lesion or greater).

Transmission and expression of footrot is completely dependent on the environment. For descriptive and regulatory purposes, there are two distinct forms; benign and virulent.

Benign footrot is a common disease and may require management, particularly during good seasons (due to good conditions for expression and spread). Virulent footrot is a notifiable disease in NSW.

Virulent footrot is a severe, debilitating disease with significant economic loss from reduced wool growth, poor ewe fertility, flystrike, and costs associated with control of the disease.

If you suspect that you have virulent footrot it is your biosecurity duty to notify your District Veterinarian.

Lame sheep will have a lower feed intake. For pregnant ewes, a reduced feed intake increased the likelihood of developing metabolic diseases, such as pregnancy toxaemia. If you are experiencing issues with lameness, please call your local district veterinarian to discuss a management plan tailored to your property conditions and flock.

PREGNANCY TOXAEMIA

What is it?

Pregnancy toxaemia is a disease of ewes in late pregnancy characterised by dullness, inappetence, isolation from the mob with a classic 'hollowed' out appearance, they progress to recumbency. Unless treated in the early stages death will occur within a few days of clinical signs first appearing.

Anything that impacts feed intake in late gestation (e.g. a lame ewe in heavy body condition) predisposes the ewe to pregnancy toxaemia. The ewe will make up for the short fall in nutrition by mobilising her own stores of body fat, but too large of a short fall and the rapid mobilisation of fat causes a significant disruption to the metabolic system and results in death. Without early intervention, irreversible metabolic collapse occurs.

Treatment

The more severe the clinical signs a ewe is showing, the poorer the prognosis. If treatment is able to be administered to a ewe that is still standing, her prospect of recovery is much better compared to a recumbent ewe.

Down ewes or ewes standing on their own showing clinical signs should be treated with:

- 4-in-1 Flopack under the skin twice daily.
- Ketol or Vytrate (propylene glycol) orally twice daily.
- Shelter, thick bedding and energy dense feed.

Prevention

Prevention is key when talking about pregnancy toxaemia. Best practice is through management of body condition score (easier said than done!) and provision of high quality, energy dense feed during late gestation and lactation.

Upcoming events

PNEUMONIA IN LAMBS IS ON THE RISE, ARE YOU PREPARED?!

22 April 2021, 1 pm, Webinar

Joan Lloyd will discuss the rise in incidence of pneumonia in lambs, which is being seen across the state.

To register please follow: Sheep Connect NSW - WEBINAR- Pneumonia in lambs is on the rise, are you prepared?!

AUSTRALIAN MERINO SIRE EVALUATION IN AUSTRALIA

6 May 2021, 1 pm, Webinar

Merino Superior Sires (MSS) is a sire evaluation program that compares the breeding performance of a sire by evaluating his progeny and their expressed traits relative to the progeny of other elite MSS sires.

Progeny are evaluated to assess a sire's breeding performance for a large number of traits which are important to breeders and commercial producers. The results assist in the selection of sires suitable for a large range of breeding objectives. Join this webinar to learn all about the program.

To register please follow: Sheep Connect NSW - WEBINAR- Australian Merino Sire Evaluation in Australia

LIVESTOCK PRODUCTION IN THE RANGELANDS

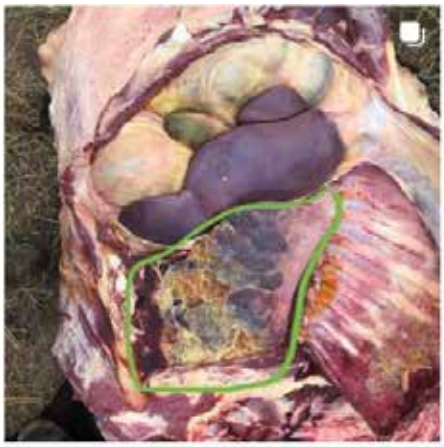
5 May 2021, 8.30 am, Workshop

All are welcome to attend an interactive free workshop on livestock production in the rangelands. This is an opportunity to hear about the latest research and paddock-based experience.

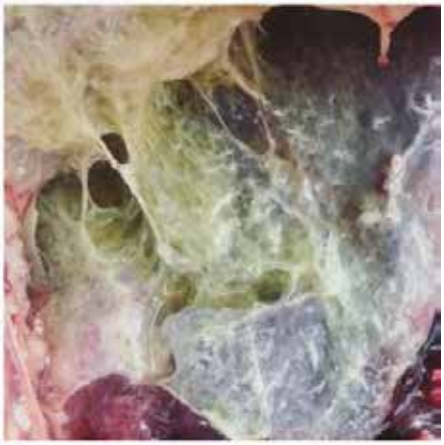
Speakers will be presenting from Meat and Livestock Australia, Melbourne University, Productive Livestock Systems, WoolProducers, Australian Wool Innovation and other organisations.

RSVP by Friday 30 April to assist with catering Sally Ware (Riverina LLS Hay) - sally.ware@lls.nsw.gov.au or 0429 307 627.

Follow us at **@locallivestockvets** on Instagram to see photos and videos direct from the paddock!





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