



FACT SHEET

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METABOLIC DISEASE IN EWES

Metabolic disease can cause a significant amount of ewe wastage. The key is prevention through adequate nutrition.

SUMMARY

Metabolic diseases can have a huge economic impact on a farming operation especially if there are large losses of capital stock.

Disease prevention is crucial. Provide adequate feed and reduce stressful situations, particularly in the critical stages of pregnancy and lactation.

The success of treating affected animals varies according to the progression or stage of the disease. Treatment should occur as early as possible to be the most effective. Farmers should consult with their veterinarian as soon as possible in order to treat the affected animals successfully and minimise further losses.

PREGNANCY TOXAEMIA

Sometimes referred to as sleepy sickness, twin lamb disease, lambing sickness or pregnancy disease. Pregnancy toxaemia usually affects multiple bearing ewes in late pregnancy.

PREDISPOSING FACTORS

These usually fall into the categories of either under nutrition or stress.

Sudden restriction of feed intake in late pregnancy due to yarding, crutching, shearing, or a snowstorm.

Falling nutrition in the last two months of pregnancy. This is when the energy demand from the growing foetus is at its peak.

Pre-lamb shearing in combination with inadequate shelter during stormy weather will decrease the feed intake of the ewe but will increase the energy demand.

Any disorder that prevents the ewe from feeding adequately may bring on pregnancy toxaemia, eg lameness, dental malformations or lack of adequate teeth, internal parasites.

In some grazing systems, where the ewes are heavily stocked, the competition for feed may be too high, and the sheep become starved or fed inadequately.

Ewes with multiples are more susceptible than single-bearing ewes.

Older ewes have been reported to be more susceptible than younger ewes.

Lack of exercise is sometimes reported to be a predisposing factor. This is thought to be because the ewe is less inclined to graze enough to meet her energy demand because she is 'unfit', especially if she is grazing on hill country.

CLINICAL FEATURES

Not all of the symptoms listed will be evident in all cases.

Early stages:

- affected ewe separates herself from the mob
- appears depressed.
- loss of appetite
- reluctant to move, often seen as the sheep who lags behind from the mob when shifted (it may take longer to notice these stages if the ewes are set stocked and not shepherded often)

Mid-stages:

- affected ewes will become more depressed
- head can be carried in an unnatural position
- ewe may appear blind
- may wander aimlessly
- show little reaction to dogs or a human
- if forced to move the ewe may stagger or crash into obstacles
- wool can be easily plucked from the fleece
- neuromuscular symptoms may also appear, such as twitching of the ears and muscles surrounding eyes and muzzle, teeth grinding, and frothing at the mouth

End stages:

- ewe may become cast
- stargazing, where the ewe will tilt her head towards the sky
- the ewe may abort the foetus; sometimes the act of lambing aids the prognosis of the ewe, although most lambs are born dead
- eventually, coma and death

TREATMENT

The treatment of pregnancy toxemia is difficult, and produces variable results.

Early treatment is the most important factor in a successful recovery. Once the ewe shows severe neuromuscular clinical symptoms or the animal is cast then renal (kidney) failure has probably occurred, and recovery is unlikely.

Maintaining appetite is also important. If the ewe starts to graze voluntarily then full recovery is more probable. It is important to leave affected ewes with normal ewes to encourage feeding, although adequate shelter is also required. Ewes must have easy access to water.

Dehydration is a key factor to manage affected ewes. As already mentioned, drinking water must be made available. Fluids can also be administered either orally or intravenously (a more expensive option). There are several solutions on the market which contain carbohydrate in the form of dextrose, and other nutrients to aid the synthesis of glucose.

Caesarean section (performed by a veterinarian) reduces the metabolic demand of the foetus. This is more successful in the early stages of the disease.

Other methods which have been used are shearing the ewe to stimulate appetite, and administering calcium borogluconate under the skin (decreased calcium levels have been shown to predispose a ewe to pregnancy toxemia).

PREVENTION

Providing adequate nutrition for pregnant ewes is the most important factor in preventing pregnancy toxemia. The main guidelines for prevention are as follows.

Prevent unnecessary fasting and stress in pregnant ewes, particular care should be taken with pre-lamb shearing or crutching.

Adequately feed pregnant ewes, including preferentially feeding ewes carrying multiples. Base feeding levels on scanning data and the use of pregnancy feed tables.

Note that inclement weather raises the energy demand of ewes, especially if they have been shorn before lambing. Provide adequate shelter. Have supplement reserves and a plan ready to feed stock as early as possible following a storm.

HYPOCALCAEMIA

Sometimes referred to as milk fever. This disorder is caused by insufficient intake and absorption of calcium into the blood.

PREDISPOSING FACTORS

A ewe's dietary requirement for calcium is 1.5-2.6g per kilogram of dry matter (kg DM). The heavily pregnant ewe will still need to mobilise some of her skeletal calcium in order to meet her calcium requirement.

This also occurs in early lactation. The amount a ewe uses from her skeletal reserves depends on the diet she is consuming. These reserves must be replaced following lambing. If they are not, the ewe may be more susceptible to hypocalcaemia in subsequent pregnancies.

Lush, actively grown pasture, dry mature summer pastures, wheat or concentrate feeds containing low calcium (less than 1g/kg DM) and high phosphorus may predispose the ewe to hypocalcaemia.

Hypocalcaemia usually occurs in mature ewes in late pregnancy, although has been reported in ewes post-lambing and in dry ewes.

The predisposing factors for hypocalcaemia are very similar to those of pregnancy toxemia, such as:

- sudden changes in feed type or grazing regime
- sudden increases in green feed
- mustering and holding for crutching or shearing

as well as access to sorrel or other oxalate-containing plants.

CLINICAL SIGNS

The diagnosis is usually based on a history of a sudden change of feed or method, clinical signs and a rapid response to treatment. Blood samples can also be taken to assess calcium levels (these should be taken from at least five ewes).

- initially ewes will stagger and seem hyperactive
- this rapidly progresses to the ewe becoming cast and then comatose. Their body position is usually one of sitting up with their head bent around to their flank
- there may be no corneal reflex
- you will often see bloating and regurgitation of stomach contents
- bearings (prolapse) can also occur in affected ewes
- untreated animals will usually fall into a deep coma and die within 24 hours

TREATMENT

Low blood magnesium and glucose is also common in ewes affected with hypocalcaemia, therefore treatment with calcium borogluconate, magnesium sulphate, and glucose may be necessary.

Solution of calcium borogluconate is available in several concentrations. It is usually administered by injection under the skin.

The response of a ewe following treatment is usually quick; the ewe will get up within 15-30 minutes, urinate, walk away and start feeding (muscle tremors may also be evident).

If the ewe does not respond quickly to treatment it may be necessary to treat for pregnancy toxemia (see section above) as this is a common effect of hypocalcaemia.

PREVENTION

Avoid stressful situations in late pregnancy and early lactation such as unnecessary mustering or yarding for long periods without feed.

Introduce sheep gradually to any green feed crops.

Do not transport heavily pregnant ewes.

Give calcium supplements to grain fed animals, especially during drought conditions where pasture is not available.

HYPOMAGNEAEMIA

Also termed grass tetany or staggers (not to be confused with ryegrass staggers). It is most commonly seen in mature lactating ewes, although it has also been seen occasionally in dry ewes.

PREDISPOSING FACTORS

Magnesium absorption is influenced by many factors including decreased saliva production and/or increased potassium levels and decreased sodium in the rumen.

Changing sheep from hay to lush pasture may bring on hypomagnesaemia, simply because the sheep may reduce the amount of chewing and salivating needed to digest the feed.

There are virtually no body reserves of magnesium readily available, therefore animals are reliant on dietary intake of magnesium. The ewe's highest demand for magnesium is during lactation.

Hypomagnesaemia may be caused by the use of high rates of potassic fertiliser.

The most common cause is inadequate feeding relative to energy demand, either due to inclement weather or under nutrition over a period of time.

Ewes may be suffering from other disease such as footrot, pink eye or a high internal parasite burden.

CLINICAL SIGNS

Ewes affected with hypomagnesaemia are usually found dead in the paddock.

Initially sheep will appear dull and stop eating. If disturbed these sheep may display muscle tremors and nervous signs (this is because magnesium is necessary for the central nervous system to function properly).

Other clinical signs include:

- ewe collapsed on side with her head thrown back
- severe convulsions while paddling legs
- frothing at the mouth
- nystagmus (rapid rhythmic repetitive and involuntary eye movements, either horizontal, vertical or rotary)
- rapid heartbeat and breathing

Death usually occurs in four to six hours.

TREATMENT

As with all metabolic diseases, early detection and therapy greatly increase the success of treatment and recovery.

The usual treatment advised by veterinarians is to inject magnesium sulphate and calcium borogluconate under the skin. Ewes that respond positively to this treatment may also be drenched with magnesium oxide to boost their magnesium intake.

Other sheep within in the flock may also be suffering sub-clinically from low magnesium levels. It is therefore advisable to administer magnesium oxide at a rate of 10 grams per sheep per day.

Increasing the amount of hay the flock consumes may also help to treat the rest of the flock by increasing salivation (this increases the sodium and potassium ratio in the rumen).

PREVENTION

To prevent further cases in subsequent seasons, pay attention to increasing the intake of dietary magnesium, especially in mature ewes that may be carrying multiples.

Magnesium can be administered to sheep as either calcined magnesite (in powder form) or Causmag (magnesium oxide) which can be sprayed onto hay. The sheep must be used to eating hay, because the application of the magnesium may make the hay relatively unpalatable. Pasture samples can be taken and analysed for their magnesium content. It may be necessary to increase magnesium fertiliser application.

You should also:

- minimise stressful procedures such as yarding, trucking or mustering
- introduce sheep gradually to lush feed and incorporate hay into their ration
- provide adequate shelter
- quickly attend to animals suffering from other ailments such as foot problems or internal parasites

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West, DM, Bruère, AN, Ridler, AL (2002) The sheep - health, disease and production (2nd edition) Foundation for Continuing Education of the New Zealand Veterinary Association, Massey University.

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