Vitamin A Deficiency in Sheep and Cattle

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Introduction

Vitamin A deficiency occurs most commonly due to a lack of vitamin A in the diet, or inhibition of absorption from the digestive tract. It is commonly seen in cattle and sheep in feedlots, and also those being fed high concentrate diets in the absence of green pasture, as is common during droughts.

Cattle and Sheep cannot generate their own Vitamin A, they need to eat green plants containing carotenoid precursors such as β -carotene – found in the orange-yellow pigments in the green leaves of plants. The cattle and sheep then convert the β -carotene to vitamin A in the wall of the small intestine.

Vitamin A is a fat soluble vitamin and is stored in the liver, with small amounts in other tissues and organs. When adequate green feed is available, cattle and sheep meet their daily requirements, and can store excess Vitamin A in their liver. When the diet is deficient in the carotenoid precursors, Vitamin A is released from the

liver to meet daily needs. When the liver supply is exhausted, vitamin A deficiency occurs.

Signs of Deficiency

Vitamin A is important for the function of the immune system, the health of epithelial tissues, mucous membranes, the eye and the central nervous system. It is also important for reproductive performance, and normal bone growth and development of calves and lambs.

Vitamin A deficiency causes keratinisation, which breaks down the protective functions of epithelial cells in digestive, respiratory, urinary and reproductive tracts. This leads to a higher susceptibility to infections. Signs of deficiency can be varied, and are more commonly seen in young, growing animals than in adults. These signs may include

- Reduced feed intake / weight loss
- Rough, dull hair coat, sometimes with hair loss and scale or scurf on the skin

- Nasal discharge and excess tears from the eyes
- Night blindness
- Diarrhoea
- Lowered immunity, leading to increased susceptibility to infections such as pneumonia and mastitis
- Poor conception rates / infertility, due to quality of semen in males, and ability of females to conceive and maintain pregnancies
- Stillbirths
- Abortions
- Retained placentas
- Micropthalmia (small, incompletely developed eyes) in calves born to deficient mothers
- Calves and Lambs that may be malformed or born weak and unable to stand.
- Seizures in adult animals
- Seizures in lambs and calves
- Urinary infections and urinary stones
- Death

Figure 1. The neck of a cow with Vitamin A deficiency, showing a rough, dull coat with patches of hair loss. Photo: Shaun Slattery, NWLLS.



When does deficiency occur?

- Deficiency is most commonly seen in animals that are being fed stored feeds, as commonly occurs in feedlots and under drought conditions.
- Liver reserves protect cattle and sheep against short term dietary deficiencies in Vitamin A. Prolonged periods without green feed lead to clinical signs of deficiency. Calves are born with low vitamin A levels, and rely on colostrum from their mothers until their intake of green feed meets their requirements. If they are being fed by cows that are vitamin A deficient, the calves will also become deficient.
- Lambs, similar to calves, are born deficient, and rely on their mothers' colostrum until they are eating sufficient pasture to meet their requirements for vitamin A, and build their own liver stores.
- The demand for vitamin A is higher for cattle and sheep around the time of giving birth, and when they are fighting off infections such as mastitis.
- Plants that have dried off, with little to no green material have reduced levels of B-carotene. When fed these dried feeds such as under drought conditions, cattle and sheep begin to use up their liver stores to meet their daily requirements. Under drought conditions, stock are also more likely to be consuming stored feed such as hay, silage, and poorer quality feeds such as straw and crop stubble. These are generally poor sources of vitamin A. Forage feeds harvested under

drought conditions will also be low in β -carotene.

- High concentrate diets, such as grain or pelletised feeds inhibit the conversion of β-carotene to Vitamin A in the small intestine. This, combined with a lack of access to green pasture, is why deficiency is commonly seen in those being fed a diet high in processed feeds and concentrates.
- Stored fodder such as hay that has been stored for long periods, or exposed to sunlight or heat, has greatly decreased amounts of βcarotene. Similarly, mineral mixes and processed feeds including vitamin A that have been stored for long periods also have lowered levels of vitamin A, as the vitamin degrades over time.
- Diets that are deficient in protein and fat also contribute to greater levels of vitamin A deficiency, likely due to impaired vitamin metabolism.
- Feeds that are high in nitrates, such as drought stressed forage crops are also thought to cause destruction of βcarotene, and block conversion to vitamin A, further contributing to deficiency.

Diagnosis

Blood tests for Vitamin A levels can give an indication of the level of Vitamin A circulating in the bloodstream. However blood tests give little indication of liver storage levels. An animal may have normal circulating blood Vitamin A levels but have very low liver stores, which may lead to clinical deficiency in the short term future. Liver biopsies provide a more accurate indication of Vitamin A status. These must be performed by a qualified veterinarian.

Treatment

Affected sheep and cattle should be injected with Vitamin A at a rate of 400 IU/Kg. Treatment should be administered in consultation with your veterinarian. Ongoing supplementation methods are discussed below.

Response to treatment is generally rapid, however animals that are showing signs of eye damage may not recover and suffer permanent blindness.

Calves and lambs that are born with deformities due to deficiency during foetal development cannot be treated with Vitamin A. Their survival will depend on the level of damage and their ability to function.

Sources of Vitamin A

Fresh green pasture is the best source of carotenoids for conversion to Vitamin A. Preserved forages such as silage and hay also contain carotenoids, however these are destroyed over time, so feeds that have been stored for long periods will have lost much of their carotenoid content.

Cereal grains such as wheat and barley are very low in carotenoids. In addition, diets that are high in grain inhibit the conversion of carotenoids to vitamin A in the small intestine.

Supplementation

Due to the ability of ruminants to store vitamin A in the liver, they do not require

daily supplementation, and longer lasting injections are the preferred option.

Vitamin A supplement options include:

Intramuscular injection.

This is commonly sold as Vitamin A, D&E injection. Recommended doses for adult cattle range from 1 to 1.5 million IU Vitamin A per injection, with the frequency of injection depending on level of deficiency and environmental conditions.

- Calves and lambs born to deficient mothers or under conditions which lead to deficiency should be treated at birth.
- Cows and ewes should be supplemented prior to joining, to prevent deficiency during foetal development. They should also be supplemented prior to giving birth, as this is a time of high demand for vitamin A.
- The injection can cause side effects including pain around the area of injection, inappetance and lethargy, particularly in young animals.

Supplements in feed or water

Vitamin A is often added to processed feeds such as calf pellets and other feedstuffs used in full hand feeding conditions. Producers should be aware that the vitamin is readily broken down by heat, light, humidity, and when mixed with other feedstuffs.

Access to green forage, or high quality, properly cured hay or silage.

This will address the deficiency, but is not always possible under drought conditions, nor practical in feedlots.

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