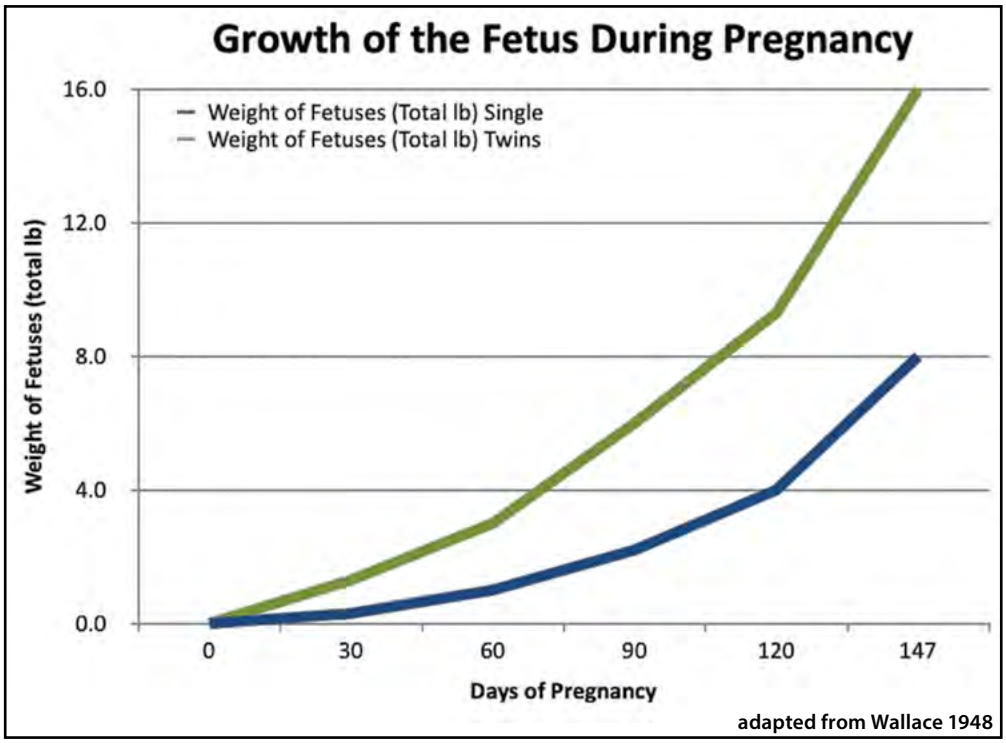
NEWS TO EWES

Is it Ketosis or Milk Fever?

by Dr. Donald G. Ely,
University of Kentucky



There are no more anticipated and exciting times in the life of a shepherd, even for a seasoned veteran, than an upcoming lambing season. Likewise, it is a much anticipated time for ewes, even for multi-parious ewes. On the other hand, the last 2 weeks of pregnancy, lambing, and subsequent lactation can be stressful times for both shepherds and ewes. How discouraging can it be when the best ewe in the flock, that is carrying twins, dies from ketosis or milk fever? The loss of three head at the same time is indeed a traumatic episode. Furthermore, did the ewe die from ketosis or milk fever? Not sure? And, why did the ewe contract ketosis or milk fever? This paper describes the causes of ketosis (pregnancy disease, pregnancy toxemia, lambing sickness, or twin lamb disease) and milk fever (hypocalcemia), as well as how to prevent them, identify symptoms, and treat them.

KETOSIS VS. MILK FEVER

Ketosis and milk fever are the two most common metabolic disorders (diseases) that affect ewes in late pregnancy and early lactation. Ketosis is the more common and can cause a high loss of ewes and lambs. These disorders initially appear similar: They can be triggered by similar factors and can occur together. Therefore, it is essential that early symptoms be identified correctly for treatment to be effective. If left untreated or not treated early enough, both can be fatal. So, prevention is far better than treatment.

CAUSES

Both are metabolic disorders (diseases) caused by inadequate nutrition. Ketosis is caused by an imbalance between energy supply and demand. It usually occurs in ewes that are 3 years or older as they approach lambing. The toxicity of ketosis is a result of insufficient carbohydrate (energy) metabolism in the last 4 to 6 weeks of pregnancy. Pregnant ewes that are too fat or too thin, carrying twins or triplets, and are fed diets deficient in energy or those containing low quality hay are the ones that usually develop ketosis. These are the ewes that are usually kept in a barn or lot during the last 4 to 6 weeks of pregnancy and, thus, have limited daily exercise. In contrast, ewes that have access to pasture and exercise in the same stage of pregnancy seldom encounter this disorder. However, they still must be supplemented with correct amounts of high energy concentrates (grains) to prevent ketosis. Do not depend on pasture forage alone to prevent this metabolic problem.

Typically, two-thirds to three-fourths of total fetal growth occurs during the last 4 to 6 weeks of pregnancy (**Figure 1**). Nutrient imbalance (primarily energy) and/or nutrient restriction (primarily energy) during this time are the instigators of ketosis. At the same time, nutrient imbalance/restriction can be the cause of light weight lambs at birth, unequal birth weights of twins and triplets, increased prenatal lamb losses, reduced mothering instincts, and subsequent lowered milk production. It is well established that ewes carrying a single fetus in late gestation (last 4 to 6 weeks) require 50% more daily energy intake than they required in early gestation (first 15 to 17 weeks). Ewes carrying twins require 75% more because of the dramatic increase in fetal growth (**Figure 1**). So, if nutrient imbalances/restrictions exist, dietary provisions can't keep up with the daily metabolic needs (requirements) of the pregnant ewes. This development may reduce the ewes' blood glucose levels, which prevent the liver from converting body fat stores to usable energy. When this occurs, ketone bodies (acetone, acetoacetic acid, and β-hydroxybutyric acid), which are normally metabolized to energy, accumulate in the blood. High levels of these ketone bodies are toxic. The adrenal glands and kidneys then respond by increasing cortisol production and reducing renal blood flow. Tissue changes in the brain, kidney, and liver are responsible for eventual death of toxic ewes.

The classic cause of the metabolic disruption of energy metabolism in the bodies of ketotic ewes is consumption of a low-quality diet of mature grass hay. Ewes that are overfat (body condition score 4+) or too thin (body condition score less than 2.0) may not consume adequate amounts of the hay because of its low palatability. Also, overfat ewes may not be able to consume enough of any roughage to meet their nutrient requirements because their uterus takes up more and more room in the body cavity as 2 or 3 fetuses grow during late gestation (**Figure 1**). Both of these scenarios prevent pregnant ewes from consuming

enough daily energy to meet their requirements. Then, they try to compensate by breaking down (metabolizing) their own body fat. When unable to do this correctly, toxic ketone bodies accumulate in the blood and kill both ewes and fetuses!

Milk fever, or hypocalcemia (low blood calcium), is a result of a calcium deficiency in the bloodstream. This metabolic disturbance usually affects the flock in outbreaks among pregnant and lactating ewes exposed to forced exercise, sudden deprivation of feed, grazing on green cereal grain crops, and/or consumption of a calcium deficient diet. Up to one-fourth to one-third of the flock may be affected at one time.

Hypocalcemia is typically caused by a sudden increased calcium demand placed on ewes by the rapid growth of fetuses (**Figure 1**) and the calcium demand for colostrum and milk production at the beginning of lactation. The milk fever syndrome, resulting from low blood calcium levels, is most often seen in 3 to 6 year-old ewes nursing twins in early lactation when both dietary calcium and energy requirements are higher than any other production period during the year. A low blood calcium level during the periods when calcium and energy requirements are highest may be a result of inadequate nutrition, even as far back as early pregnancy (during the first 15 to 17 weeks). Likewise, the shepherd may have calculated that rations to be fed would meet the ewe's daily requirement for both calcium and energy. But, there was not an anticipated sudden increase in calcium and energy demands to the level that ewes come down with milk fever.

Blood calcium regulation is controlled by the parathyroid and thyroid glands. Regulation is dependent on dietary calcium intake and absorption from the gastrointestinal tract and mobilization of calcium from bone. This mechanism makes tremendous adjustments to maintain blood calcium at the times of high demand for supply to fetal tissues, colostrum, and milk. Failure to do so, because of dietary deficiencies (calcium and energy), low calcium absorption rates or a sudden stress placed on the ewe, can result in milk fever.

SYMPTOMS

Symptoms of ketosis and milk fever in heavily pregnant ewes can be similar. Ketosis most often occurs during pregnancy whereas milk fever can occur during both pregnancy and lactation. Even though post-lambing symptoms will most likely be those of milk fever, it is important to be able to distinguish between the two disorders. The following is a progression of the typical symptoms of these metabolic disorders.

Typical Symptoms of Ketosis and Milk Fever

Disorder Progression	Ketosis	Milk Fever
Early and late symptoms	<u>Earliest signs:</u> 1. Separation from flock. 2. Apparent blindness but still alert. 3. Standing still when approached. 4. Running into objects when forced to move. 5. Pressing head against objects. 6. Lapping water. <u>Later stages:</u> 1. Marked drowsiness. 2. Tremors/spasms of head, face, and neck muscles. 3. Head pulled back or sideways. 4. Abnormal postures, elevation of chin (star-gazing). 5. Leg muscle tremors, incoordination, falling, convulsions. 6. May have a thick, yellowish, candle-wax like discharge from nose.	<u>Early and late symptoms:</u> 1. Stilted, proppy gait. 2. Muscle tremors, especially shoulder muscles. 3. Alert and struggles when approached. 4. Weak, staggering, goes down. 5. Once down, tends to stay down unless treatment is effective.
Recumbency (lying down)	1. Slow progression to recumbency 2 to 3 days after initial signs. 2. Profound depression or coma until death 2 to 6 days after onset of signs.	1. Rapid progression to recumbency over 3 to 4 hours. 2. Sternum recumbency, chin on ground or floor, head stretched out. 3. Legs stretched out behind is usual. 4. Watery nose discharge maybe. 5. Vaginal prolapse maybe. 6. Severe depression/coma. 7. Death within 6 to 24 hours without treatment. 8. Some cases linger up to 3 days.
Response to treatment	1. No response to milk fever treatment. 2. Usually poor even when given while ewes are still alert.	1. Rapid and good recovery after injection of treatment doses of calcium solutions, even in later stages. 2. Signs of recovery seen within a few minutes to half an hour after injection.

TREATMENT

Blood glucose levels must be increased if ewes with ketosis are to survive. Miracle recoveries cannot be expected, but intravenous infusion of glucose and oral administration of glycerin or propylene glycol (PG) at 4 to 8 ounces daily may save a ketotic ewe. The problem here is that PG reduces appetite even more than it may already be reduced, thus compounding the problem of low blood sugar levels. Taking lambs from the uterus by Caesarean section early in the course of the disorder may assist in recovery. But, the lambs are usually too immature to survive if they are taken 3 to 7 days before term. Another treatment, if the ewe lives long enough, is the oral administration of a half cup of molasses, with a quart of water, given 4 times every 24 hours. If the affected ewe will eat, provide her with the grain she was previously receiving plus alfalfa hay. Remember, she needs energy to increase her blood glucose levels. Any other treatment is simply "a shot in the dark" that may save a ewe once in a while. **Preventative nutritional management is more successful than treatment.**

In contrast to ketosis, response to treatment of milk fever can be rapid. Treatment is usually by intravenous infusion of 50 to 100 cc of calcium borogluconate solution (usually those prepared for milk fever in cows). Subcutaneous injection may be effective, but absorption is slower so it takes longer before the effects can be seen. Having on hand the equipment needed to infuse, maintaining a supply of calcium borogluconate solution, and having the proficiency to infuse are required if shepherds are to administer their own treatment. Otherwise, a veterinarian will be needed. **Like ketosis, preventative nutritional management will render more success than treatment.**

PREVENTION

If ewes become over-conditioned during the 15 to 17 weeks of early pregnancy, the high level of nutrition cannot be maintained during the last 4 to 6 weeks because the capacity of the rumen is reduced by the distended uterus. A shortage of critical nutrients (energy and calcium) associated with ketosis and milk fever can then occur. Under-conditioned ewes that enter the last 4 to 6 weeks of pregnancy may not consume enough feed to increase their body fat reserve, especially if the diet contains a large amount of low-quality roughage. If a concentrated energy source, like shelled corn, is fed, the energy requirement of ewes may be met. But, concentrates have limited amounts of calcium, which may make the ration deficient in calcium and result in low blood calcium levels causing milk fever. Therefore, performance of ewes in the last 2 weeks of pregnancy and early lactation depends on the nutritional management the shepherd imposes during the other 50 weeks of the production year. **Figure 2** illustrates how body condition scores (BCS) of ewes change during a production year. In reality, the BCS of ewes in the last 4 to 6 weeks of gestation are a function of how they were managed during maintenance, nutritional flushing, breeding, and early gestation. The situation that most producers face is that ewes coming out of lactation and entering the maintenance phase of production are relatively thin (BCS = 1.5 to 2.0; **Figure 2**). Generally, ewes gain weight and body condition after weaning lambs regardless of the nutritive value of the diet. The shepherd's responsibility is to keep ewes from becoming obese during this maintenance period by maintaining them on a low-quality hay diet or pasture until the next nutritional flushing/breeding phase. Other ewes may remain thin (1.5 to 2.0 BCS) during maintenance because they are "hard-doers", infested with internal parasites, or have responded negatively to management in other ways. Consequently, some ewes may be too fat (BCS = 3.0 to 3.5 or too thin (BCS = 1.5 to 2.0) as they leave flushing/breeding and enter early gestation (first 15 to 17 weeks). Ideally, ewes should have a BCS of 2.0 to 2.5 as they enter early gestation (**Figure 2**) and gain only enough weight in early gestation so they have a BCS of 3.0 as they enter the last 4 to 6 weeks of gestation. Unfortunately, the "easy-keeping", fatter ewes will likely continue to gain weight as fat, whereas the "hard-doers" will not change very much. The end result may be excessively fat (BCS = 4.0 to 4.5) or excessively thin (BCS = 1.5 to 2.0) ewes that are carrying twins or triplets when they are within 2 weeks of lambing. Irregular feeding caused by inclement weather, excessively cold weather, lack of exercise, or improper late gestation diets can trigger ketosis and/or milk fever. So, what can shepherds do to keep this from happening?

The fact that few cases of ketosis and milk fever occur when ewes are on pasture speaks to their ability to handle inclement weather, the exercise they get, and the

Figure 2

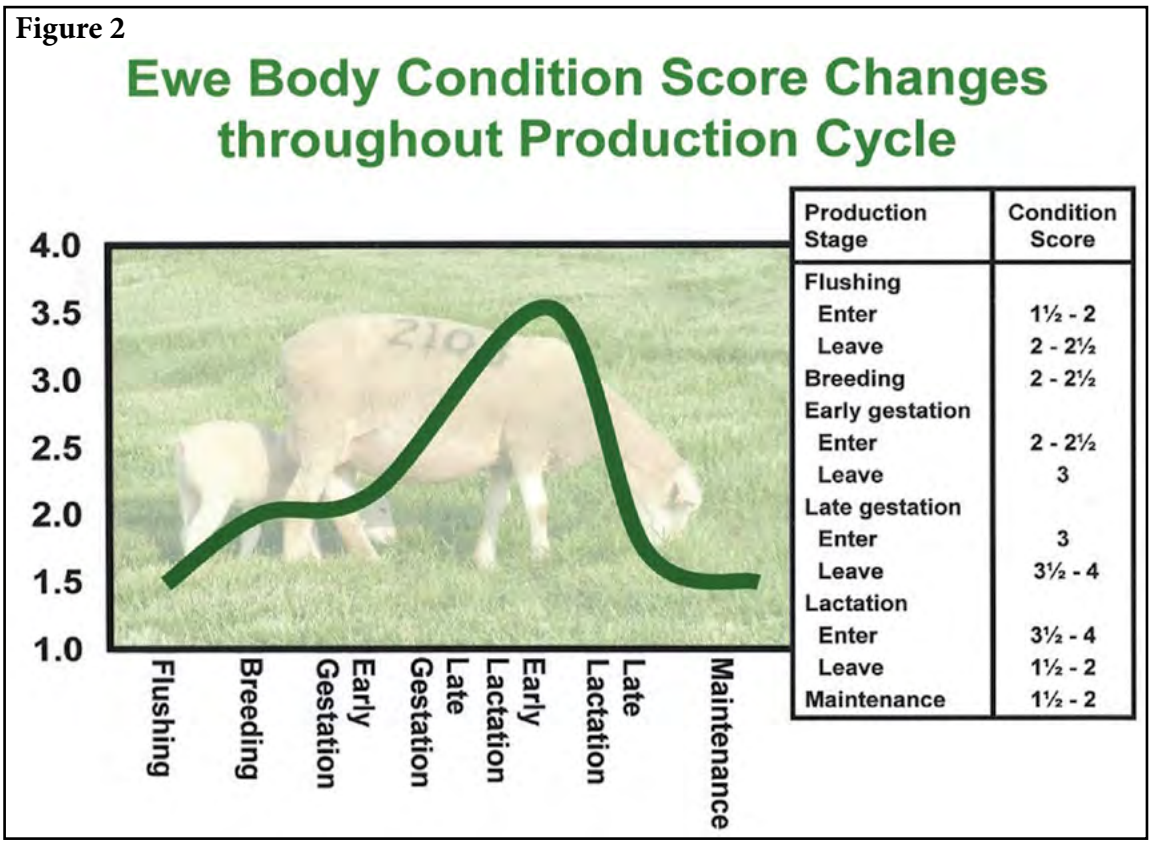


Table 1. Daily Early and Late Gestation Rations (lb/hd/d) for Ewes of Different Weights

Ewe wt., lb.	154		176		198	
Production phase[a]	EG	LG	EG	LG	EG	LG
Ration ingredient:						
Low quality hay[b]	3.4	-	3.7	-	3.9	-
Medium-quality hay[c]	-	4.0	-	4.0	-	5.0
Shelled corn	-	0.5	-	1.0	-	1.5
Cost, ¢/ewe/d[d]	10.2	29.0	11.1	35.0	11.7	46.0

[a] EG = first 15 to 17 weeks of gestation; LG = last 4 to 6 weeks of gestation.
[b] Late bloom to mature grass.
[c] Boot stage for grass, mid-bloom for legume.
[d] Low quality hay at $60/ton; medium quality hay at $120/ton; shelled corn at $6.00/bu

correct late gestation diet they consume. In reality, however, these ewes usually don't carry as many twin and triplet fetuses or become as fat as those maintained in more confined areas during early and late gestation. An analysis of the scenarios when ketosis or milk fever might occur points to the 15 to 17 weeks of early gestation management as the key to prevention. Ewes must not become over-conditioned or under-nourished during this 15 to 17 week period. **Table 1** shows some daily early and late gestation rations that, theoretically, should prevent both disorders if ewes are in recommended BCS (**Figure 2**) when they enter each production stage. The amounts of hay and corn that make up each ration will meet the daily requirements for protein, energy, minerals, and vitamins for ewes of each weight class. However, a complete mineral mix that contains vitamins A, D, and E should be available ad libitum, as a safety factor, every day of the year. The low-quality hay fed in early gestation should be replaced with pasture, if available. Pasture can also replace the medium-quality hay for late gestation, although this may be the time of the year when pasture forage becomes unavailable (December/January). Of particular importance in this table is the principle of feeding low-quality hay (or pasture) in early gestation and medium-quality hay (or pasture) in late gestation. There is no reason to feed medium-or high-quality hay (or pasture) if ewes are in correct BCS (**Figure 2**) as they come out of maintenance, nutritional flushing, and breeding phases. Note that low-quality hay (or pasture) intakes increase as ewe weights increase. Ewes in late gestation require more medium-quality hay (or pasture) than low-quality hay in early gestation because of increased fetal growth (**Figure 1**). Ewes weighing 154 and 176 lb require the same amount of hay, but different amounts of shelled corn, in late gestation. The corn supplementation reduces the chances of encountering ketosis and/or milk fever. Feeding the rations in **Table 1** to ewes in correct BCS should also produce a feed cost savings because the highest quality hay (not shown in table) is saved for the lactating ewes.

SUMMARY

Planning ahead is the key to the prevention of ketosis or milk fever. Centered in this planning is the knowledge of body condition scores of ewes in relation to their annual production phases. Once these scores are established, quality and quantity of feed required to maintain them in each phase can be determined. Nutritional management in maintenance, flushing, breeding and early gestation has a profound effect on late gestation performance. Following these guidelines established for body condition scores and feedstuff quality and quantity for production efficiency will concurrently provide preventative nutrition for ketosis and milk fever.

Dr. Donald G. Ely, *Professor in the Department of Animal and Food Sciences at the University of Kentucky*