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METABOLIC DISEASES IN DAIRY ANIMALS: SILENT THIEVES OF MILK AND MONEY

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Abstract

Metabolic diseases such as milk fever, ketosis, fatty liver, downer cow syndrome, acidosis, grass tetany and post-parturient hemoglobinuria are among the costliest health problems of dairy animals. They occur mainly when the high-yielding animal cannot balance the sudden demand of milk synthesis with her intake of energy, minerals and other nutrients, especially during the transition period (three weeks before to three weeks after calving). Clinical cases are only the visible tip: subclinical forms reduce milk yield, fertility and immunity without obvious signs, and quietly drain farm income. Losses arise from reduced milk, treatment costs, culling, mortality, delayed conception and increased risk of mastitis, retained placenta and

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Volume 01 | Issue 05 | October, 2026

Page | 124



displaced abomasum. Most of these diseases are preventable through correct body condition at calving, balanced ration formulation, mineral supplementation, gradual feed changes, regular monitoring and timely treatment. This article explains the common metabolic diseases in simple language and gives practical prevention measures that farmers and field veterinarians can adopt to reduce economic loss.

Keywords: metabolic diseases, dairy cattle, transition period, milk fever, ketosis, prevention, economic loss

1. Introduction

Over the past decades genetic selection has raised the milk potential of dairy cows and buffaloes remarkably. A cow yielding 25-30 liters a day secretes in milk, within a few days of calving, several times the calcium, glucose and protein present in her whole blood. If feed intake does not rise quickly enough to support this, the body breaks down its own reserves, and the delicate internal balance (metabolic homeostasis) fails. The result is a metabolic disease, also called a production disease, because it is the price of high production when feeding and management do not keep pace (Radostits et al., 2007).

Unlike infectious diseases, metabolic diseases are not caused by germs and do not spread from animal to animal. They are caused by imbalance, and therefore they can largely be prevented by management. Importantly, they rarely work alone. A cow with milk fever is more likely to develop retained placenta, metritis, ketosis and mastitis, so one disorder often opens the door to several others (Goff, 2008).

2. Why the transition period matters

The three weeks before and after calving form the most critical phase in a dairy animal's life. Dry matter intake falls by 20-30 per cent just before calving, while the nutrient demand of the growing calf and the starting udder increases sharply. Calcium demand rises almost overnight at the onset of milk secretion, and energy demand outstrips intake for several weeks, creating a negative energy balance (Drackley, 1999; Horst et al., 2005). Most metabolic diseases appear in this window, so this is where management effort pays back the most.

3. Common metabolic diseases

3.1 Milk fever (parturient paresis, hypocalcaemia)

Milk fever is a drop in blood calcium soon after calving, usually within 72 hours, when large amounts of calcium are drawn into colostrum and milk faster than the body can replace them from bone and gut. Despite its name there is no fever; body temperature is normal or low. Early signs include restlessness, tremors,



cold ears and a stiff, unsteady gait. As it progresses the cow lies down, turns her head into the flank in an S-shape and finally becomes unconscious. Without treatment, death can follow within hours. Older, high-yielding cows are at greater risk, and Jersey cows and crossbreeds are particularly susceptible.

The hidden part is bigger than the visible part. Subclinical hypocalcaemia, where the cow looks normal, affects a large proportion of cows in the second and later lactations and is linked to reduced feed intake, poorer immunity, retained placenta and displaced abomasum (Reinhardt et al., 2011; Goff, 2008).

Prevention: The most reliable approach is dietary. Avoid feeding high-calcium and high-potassium forages (such as lucerne in large quantities) to dry cows in the last three weeks. Feed rations with a low or negative dietary cation-anion difference (DCAD) where feasible, keep magnesium adequate, and avoid overfeeding of calcium during the dry period, which keeps the calcium-regulating system alert. Oral calcium supplements at calving help high-risk cows. Treatment is slow intravenous calcium borogluconate given by a veterinarian; fast injection can stop the heart.

3.2 Ketosis (acetonaemia)

Ketosis develops when a cow in early lactation mobilises too much body fat to meet her energy needs. The liver cannot fully process the fatty acids that flood in and produces ketone bodies, which accumulate in blood, milk and urine. Affected animals eat less (particularly concentrate), lose weight, give less milk and may have a sweetish acetone smell in breath and milk. Some show a nervous form with licking, staggering and abnormal behaviour. Subclinical ketosis is common, with reported prevalence in the first weeks after calving of well over 20 per cent in many herds, and it is associated with a higher risk of displaced abomasum, reduced milk yield and poor reproduction (Duffield et al., 2009; McArt et al., 2012).

Prevention: Dry cows should be in a body condition score of about 3.25-3.5 (on a five-point scale) at calving; both fat and thin cows are at risk (Roche et al., 2009). Avoid excessive fattening in late lactation, provide good-quality roughage, and maintain feed intake in the close-up period. Feed a high-energy but fibre-adequate ration after calving, and minimise stress such as overcrowding, heat and sudden feed changes. Regular testing of milk or urine with ketone strips in the first 2-3 weeks identifies cases early. Treatment includes intravenous glucose and oral propylene glycol, as advised by the veterinarian.

3.3 Fatty liver (hepatic lipidosis)

Fatty liver is closely linked to ketosis. When fat reserves are mobilised faster than the liver can use them, fat accumulates inside the liver cells and reduces liver function. Overconditioned cows are the most

affected. The animal appears dull, eats poorly, has reduced milk, and recovers slowly after calving; liver damage also weakens immunity and fertility (Grummer, 1993). Prevention rests on the same principles as for ketosis: keep cows from becoming overfat in late lactation and the dry period, avoid abrupt changes in diet, and ensure steady feed intake around calving.

3.4 Downer cow syndrome

A downer cow is one that cannot rise after calving, though she remains alert and eats. The cause is often unresolved or delayed milk fever, but it may also include muscle and nerve damage from prolonged lying on hard floors, low phosphorus or potassium, or injury during calving. Prolonged recumbency leads to muscle damage that can become irreversible within 12-24 hours. Cows should be moved to a well-bedded, non-slip surface, turned frequently, and given early veterinary care. Prevention is essentially prevention of milk fever, combined with non-slip floors and good calving management.

3.5 Rumen acidosis (SARA)

When animals are fed a large amount of grain or finely chopped rations with too little fibre, the rumen pH falls, microbes are disturbed and the animal suffers from acidosis. The acute form can be fatal, but the chronic form, subacute ruminal acidosis (SARA), is more widespread and often goes unnoticed. Signs include fluctuating feed intake, loose dung with bubbles or undigested grain, reduced milk fat percentage, poor cud chewing, and later laminitis, liver abscesses and poor body condition (Plaizier et al., 2008).

Prevention: Increase concentrates gradually (over 2-3 weeks) after calving, provide adequate effective fibre such as long green or dry fodder, feed a total mixed ration or split concentrate into several small meals, and use sodium bicarbonate or other buffers where suitable. Ensure clean drinking water at all times.

3.6 Grass tetany (hypomagnesaemia)

Low blood magnesium occurs mainly in animals grazing on lush, rapidly growing, potassium-rich pasture, especially in cold, wet weather and in lactating animals. Signs are excitement, muscle twitching, staggering, convulsions and sudden death. Because the body has little magnesium reserve, the condition develops rapidly. Prevention includes magnesium supplementation (such as magnesium oxide in feed), a good mineral mixture, avoiding abrupt turnout onto lush pasture and offering some hay before grazing (Radostits et al., 2007).

3.7 post-parturient haemoglobinuria

Seen mostly in high-yielding buffaloes and cows in the first weeks after calving, this disease is associated with low blood phosphorus. It causes red-brown urine, weakness, anaemia, reduced milk and sometimes



death. It is common in areas where fodder and soils are phosphorus-deficient or where diets are rich in cruciferous crops such as cabbage and rape. Prevention depends on phosphorus-adequate feeding, regular use of area-specific mineral mixture and avoiding large quantities of the risk fodder. Veterinary treatment includes phosphorus injection (sodium acid phosphate) and supportive therapy (Constable et al., 2017).

4. Economic losses

The losses from metabolic diseases are both direct and indirect. Direct losses include the value of milk lost during illness, the cost of drugs and veterinary fees, discarded milk, extra labour and, in severe cases, death or premature culling. Indirect losses are often greater and include reduced milk yield for the remainder of the lactation, longer calving-to-conception interval and more services per conception, a higher incidence of retained placenta, metritis, mastitis and displaced abomasum, and the lower resale value of the animal. A single clinical case of milk fever or ketosis is generally estimated to cost many times the price of preventive minerals and feed management, and the larger share of loss comes from subclinical cases that are never treated because they are never noticed (Overton and Waldron, 2004; Duffield et al., 2009).

5. Summary of the major diseases

Disease	Key signs	Main risk period	Key prevention
Milk fever (hypocalcaemia)	Weakness, cold ears, S-shaped neck, downer cow	Within 72 h of calving	Low-calcium or low-DCAD diet before calving; calcium after calving
Ketosis	Reduced appetite, sudden drop in milk, acetone smell	2-6 weeks after calving	Correct body condition (3.25-3.5); good transition feeding
Fatty liver	Dull, off feed, slow recovery after calving	Around calving	Avoid overfat cows; avoid abrupt feed changes



Downer cow syndrome	Unable to rise despite alert state	After calving	Prevent milk fever; non-slip flooring; early treatment
Acidosis (SARA)	Loose dung, reduced fibre intake, laminitis, low milk fat	Early lactation, high-grain feeding	Adequate effective fibre; gradual grain increase; buffers
Hypomagnesaemia (grass tetany)	Excitement, muscle twitching, staggering	Lush pasture, cold weather	Magnesium supplementation; mineral mixture
Post-parturient haemoglobinuria	Red-brown urine, anaemia, weakness	Early lactation	Phosphorus-adequate diet; mineral mixture

6. Prevention strategy: the practical farmer's checklist

- **Manage body condition:** dry off cows at a BCS of about 3.25-3.5 and avoid losing or gaining condition in the dry period.
- **Feed a separate close-up ration:** for the last 2-3 weeks before calving, with controlled calcium and potassium, adequate magnesium and balanced energy and protein.
- **Use a mineral mixture daily:** an area-specific mineral mixture with vitamin A, D and E, selenium and phosphorus helps prevent many deficiency-related disorders.
- **Avoid sudden feed changes:** introduce concentrates gradually after calving and always provide adequate long fibre.
- **Reduce stress:** give comfortable, clean, non-slip housing, shade and cooling in summer, sufficient feeding space and clean water.
- **Monitor the herd:** watch feed intake, rumination, milk yield and dung consistency in fresh cows; test for ketosis in the first weeks; ask the veterinarian to run periodic metabolic profile tests (Oetzel, 2004).



- **Treat early:** call the veterinarian at the first sign of illness; early treatment is cheaper and gives a better recovery.
- **Keep records:** note calving date, body condition, disease events and milk yield so that herd problems and risk groups are easily identified.

7. Conclusion

Metabolic diseases are costly but largely preventable. The core message is simple: prepare the cow before she calves. Right body condition, a well-balanced close-up ration with proper minerals, a gradual increase in feed after calving, comfortable housing and careful observation of fresh cows can keep most animals healthy and productive. Every rupee spent on prevention and good management in the transition period returns many times over through more milk, better fertility, lower treatment costs and longer productive life of the animal. Farmers should work closely with veterinarians and animal nutritionists to design herd-specific feeding and health programmes.

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