



The Dry Cow Phase

Despite considerable research work done overseas, the standard eight week dry period remains the most economic both financially and cow health-wise; and the two are never separate. Shorter dry periods have been successful in mature cows, but require micromanagement of nutrition, achievable only with total mixed rations and close attention (monitored) to DCAD.

An eight week dry period allows for some error margin in calving date, reducing the risk of inadequate dry spell and antibiotic residue from dry cow therapy. Six weeks is believed to be necessary for udder tissue regeneration and rumen wall restoration. Beyond eight weeks there is a risk of fat deposition in udders reducing capacity. Heifers can benefit from slightly longer dry periods as growth will utilise extra time and nutrient, but fatty udders are a risk here too.

The goal of the first five weeks is stable weight. Variations in weight through this period can predispose the cow to metabolic disorders of ketosis, milk fever, retained membrane, metritis and mastitis (and they are rarely separate, if not clinical, certainly sub-clinical). Through this time we need an energy intake of 90 MJME for Holstein (70 MJME Jersey), and crude protein around 13%.

At this time of year, 10 kgs of hay plus 2 kgs of grain (dairy mix) will provide this. Dry cows need mineral nutrition so grain from the dairy silo is ideal. A diet of 10 kgs DM hay alone will only provide 70 MJME (Holstein – 8 kgs Jersey = 56 MJME). Such a ration will cause weight loss, fat mobilization and head the cow toward Fatty Liver Syndrome and ketosis at calving.

Although the transition period includes several weeks post-calving, we will focus on the pre-calving three weeks in this article. Lead feeding has many benefits, but our goal is to prepare the cow for best possible lactation performance (genetic potential is almost never achieved in Australian dairy farming). This is never separate from best possible profit performance either!

There are four areas we need to focus on to achieve the preparation of our cows to enable them their best chance of performing to their genetic potential. Obviously post-calving nutrition will determine whether that does happen, but we'll look at those areas throughout the season, but if she has not been prepared, post-calving nutrition will not compensate.

Firstly we are adjusting the rumen environment (bacteria populations) for a post-calving ration. The early dry period (first five weeks) is essentially a “pit stop”. A diet wind-down to repair rumen and udder tissue. A time of rest and, ‘re – creation’. Refuel and new tyres. During this time bacteria numbers decrease and change in type densities due to high fibre/low energy/protein diet. Rumen papillae (absorption sites) decrease in size and capacity.

We need to re-introduce grain, some pasture (if available) to encourage feed-specific bacteria populations to multiply in preparation for post-calving rations. Without this bacteria population changes rumen upset (acidosis usually being the result) is inevitable. To spin-out on the first corner has already demolished her chances of ‘winning’. The increase, or re-introduction, of grain at this time also stimulates rumen papillae to expand increasing nutrient uptake potential.

Dry matter intake (feed intake) declines during these three weeks heading the cow to negative energy balance = ketosis/Fatty Liver Syndrome. Low dry matter intake will also predispose the cow to displaced abomasum. This is one of the causes of RDA/LDA’s, but a greater one we’ll look at shortly. To counter this, we need to increase energy/protein density of the springer ration. Rumensin will improve energy utilisation by increasing glucose levels reducing ketosis.

The increase in grain intake to 3 kgs will improve energy density, but the lead feed needs a high crude protein content to meet both energy density and drive intake. I run Lead Feeds at 30% canola meal for this purpose, and canola’s high by-pass protein is very effective in increasing intake. Another major consideration in regard to protein is that colostrum quality (passive immune transfer) is directly related to protein level of the pre-calving ration.

The final area that lead feeding should address is ration DCAD (dietary cation anion difference). DCAD is responsible for calcium/magnesium control. A high DCAD (+ charge) will produce low blood calcium and reduce calcium absorption in the rumen. A low DCAD (- charge) will cause calcium digestion to increase, but also start re-absorption of calcium and magnesium from skeletal reserves to neutralise the mild blood acid condition we have deliberately created via anionic (- charged) salts in the Lead Feed. Every dairyperson understands Milk Fever!

Why do we need anionic salts in Lead Feed? Simply, all Australian grazing/silage/hay rations are very high in potassium. Potassium is a very strong cation (+ charge). I even use a small amount of anionic salts in lactating rations to counter this problem which manifests itself as mid-lactation milk fever and grass staggers (both clinical and sub-clinical). I always observe lactating herds for ‘flightiness’, a good indicator of high DCAD. Your Lead Feed must supply a DCAD of -2500 msq over 3 kgs of grain. Ensure your supplier’s Lead Feed meets this level. DA’s are often caused by low blood calcium reducing ligament elasticity and tension.

In summary, none of these four aspects of Lead Feeding are independent; on the contrary, they are highly interdependent. There is a synergy between them. Miss one, ‘you miss the start’.