



Transition Cow

Part 1

Without any qualifications, the transition from dry cow to fresh cow is the most influential time on farm profit. No other investment or activity on farm can match transition management in terms of return on investment in either labour or cost.

We will briefly run through the 9 common metabolic disorders at calving and discuss transition management's (not just lead feeding) role in, at best, eliminating, or at least, minimising these problems.

Milk Fever: is simply inadequate blood calcium to meet both muscle function and demand for milk secretion. This is resolved by creating a mild blood acidosis 14 to 21 days prior to calving to activate calcium absorption in the rumen and to begin drawing from the skeleton, or bones of the cow.

We achieve a mild blood acidosis through feeding a transition ration with a negative DCAD (Dietary Cation Anion Difference). Usually in Australian dairying this requires the addition of anionic salts to this ration. The major cations (+ charge) in a diet are potassium and sodium (K + Na). The most common anions (- charge) in anionic salts are chlorine and sulphur (Cl + S). The equation then is $DCAD = (K + Na) - (Cl + S)$ resulting in a negative or minus figure. Bear in mind, pasture, pasture hay/silage are very high in K, which is why I usually recommend oaten hay as the forage in transition rations, this year, cereal straw due to cost, quality and availability.

Ketosis: related to energy shortage and most common 3 weeks post-calving when energy demand for high milk production is well in excess of ration energy. This produces excessive body fat mobilization (negative energy balance) followed by the liver, in trying to deal with this fat, unloading high numbers of toxic ketones into the blood stream. Clinical ketosis, especially severe cases are quite difficult to reverse; prevention is far cheaper.

Ketosis can start right back at the 14 – 21 days prior to calving, the transition period, due to falling dry matter intake associated with late gestation. The transition ration needs to allow for this decline by increasing both energy and protein density to prevent body fat being mobilised to meet the cow and calf's energy/protein needs.

Fatty Liver: is a complication of ketosis in which the liver becomes clogged with mobilised body fat due to negative energy balance – the consequences are fairly obvious.

Retained Placenta: has 2 common causes. 1) Interference with the process that disconnects fetal placenta from the cow's caruncles or uterine attachment. 2) The uterus lacks strength for good contractions to push the placenta out. In heifers it is frequently associated with difficult calving. We have all experienced the consequences of retained membrane in infections and fertility. Again, transition nutrition plays a major role in reducing this problem, especially in mineral nutrition.

Displaced Abomasum: The dairy farmer's dilemma – surgery or the knackery? During pregnancy the abomasum is forced below the rumen, and with either inadequate rumen fill or poor muscle tension can slip either up the left or right side of the rumen effectively cutting off the digestive system's 'plumbing'. Poor muscle tension to pull the abomasum to its correct position is due to low blood calcium level as with milk fever. But, blood calcium can be sufficient to not cause milk fever but not high enough to have adequate muscle strength to pull the abomasum back in place!

Udder Edema: although science is unclear on the exact processes which cause edema, sodium (salt) and potassium levels are certainly contributing factors. We've all tried milking, especially heifers, with 'rock-hard' udders and swollen teats with frustrating results. Treatment is more in time consuming therapies than quick jabs in the rump. Another transition nutrition issue aimed at prevention rather than cure.

Mastitis: needs no explanation, nor me to describe sound dry cow therapy. However, good mineral supplementation, especially selenium and vitamin E have proven their worth in helping prevent mastitis infection around calving; part of transition nutrition.

Uterine Infection: we have touched on under retained placenta. Prevention begins with good pre-calving nutrition, both in energy/protein/fibre balances and mineral nutrition so any metabolic causes are eliminated.

Rumen Acidosis: Last but by far not least. Get all the above right, but cause acidosis in the rumen and you could end up with all the above. Nothing reduces (even annihilates) a cow's immune system like acidosis. Acidosis is the number one, undisputed, profit robber in nutritionally induced disease in Australian dairy farming!
This disease can be caused through management at any stage of lactation, but none so easily as at calving – back to transition management again.

All these diseases are interrelated, in other words, one can be the primary issue, but trips off the rest as secondary problems. The space available for this article restricts me from further detail, but hopefully highlights the massive ramifications in terms of avoiding costly productivity losses through poor transition management.

I cannot overemphasise the greatest financial losses in these metabolic diseases around calving are from sub-clinical status – not seen, not treated, and can take month's of milk robbing energy for the cow to self-correct the problem. It is well worth getting sound advice on transition management. Next month we will look at a good transition management program, but I need to add now, the first issue is dry off body condition score which I highlighted with concern in February's article.