Challenging DCAD in Transition Cow Nutrition

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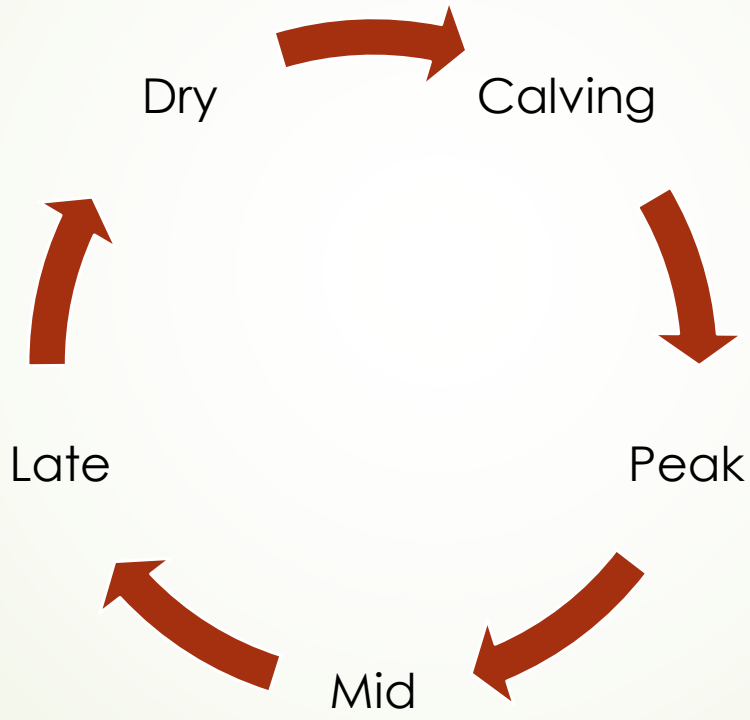
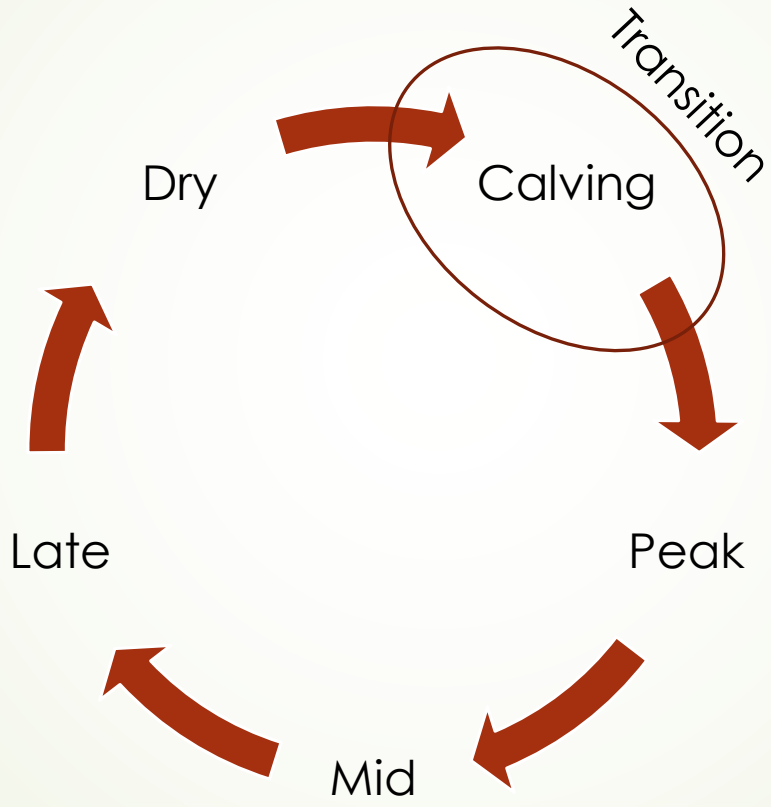
Introduction



Introduction



Metabolic disorders

Milk fever

Ketosis

Fatty liver

Displaced abomasum

Acidosis

Where did it start….?

Evolution of the ruminant

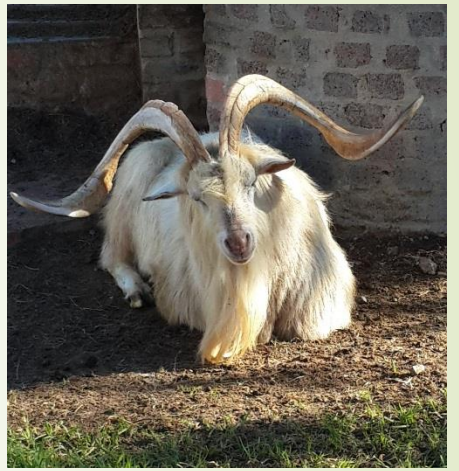
Ruminants appeared in the Eocene (38 - 54 million years ago)

Early ruminants were small animals, weighed <18 kg and were probably adapted to forest conditions. Had no horns.

Grazing ruminants evolved much later, as grasslands only appeared in the Miocene (18 - 23 million years ago)

Domestication

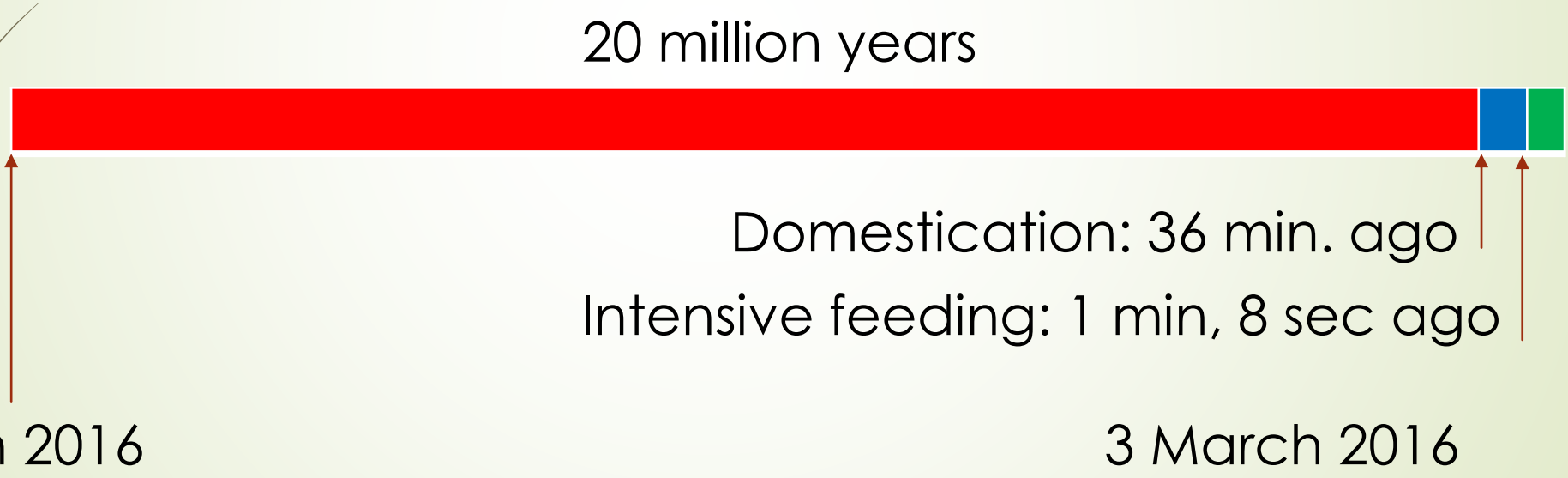
- First livestock species domesticated was the goat ~ 8 000 BC
- Most other ruminant species domesticated by 2 500 BC



The result of evolution

The rumen environment has adapted over millions of years to host a diverse and sustainable microbial population that has optimal functionality for fibre fermentation.

Perspective



So, are we surprised that cows get metabolic disorders ??

Milk fever

- Paresis puerperalis, or parturient paresis.
- Clinical manifestation of parturient hypocalcaemia.
- Blood Ca concentration < 2.0 mmol/L (< 8 mg/dL).
- Subclinical: Low blood Ca, but no clinical symptoms.

Milk fever

Hypocalcaemia occurs when Ca is extracted from the blood by the mammary gland at a faster rate than it can be replaced from the diet, skeletal reserves and renal conservation of Ca.

Result: Ca concentration drops to levels that can no longer support nerve and muscle function.

Both Ca and Mg are essential, e.g. vascular tone, nerve function and muscle contraction.

Milk fever

Normally, mammary drain of Ca causes minor decline in blood Ca, because cows respond by secreting PTH which:

- activates cells to resorb Ca from bone
- Enhances renal reabsorption of Ca from glomerular filtrate
- Begins renal production of 1,25-dihydroxyvitamin D for efficient intestinal Ca absorption
- These Ca homeostatic mechanisms do not function efficiently in hypocalcaemic cows. (Goff 2006)

History of milk fever

From Hibbs, 1950:

Can assume that milk fever did not exist 2 or 3 centuries ago (Fish 1927).

Became known at the time when it became customary to feed cows more generously to increase milk production (Hutyra & Marek, 1926).

First documented in Germany by Eberhardt in 1793 (Hutyra et al., 1938).

History of milk fever

Early treatments included sweating (hot packs and blankets) and bleeding.

Prevention before turning to pasture: 1 oz. each of alum, nitre and cream of tarter, mixed and added to 1 qt. of boiling ale and beer.

In 1879, Tellor reported that veterinarians started to question whether or not MF fever and mastitis were synonymous.

History of milk fever

Navin (1872): Attributed MF to inflammatory disease of the womb and recommended prompt and copious bleeding as key treatment. Also recommended sedatives and rubbing the cold legs with cayenne pepper and alcohol.

Williams (1884) Attributed MF to congestion of the brain and apoplexy associated with heavy milk production and parturition. Recommended bleeding to prevent congestion of the brain, plus physics, stimulants, etc.

History of milk fever

Hibbs (1950) listed 30 early milk fever theories.

- General inflammation
- Derangements of the nervous system
- Cerebral anaemia
- Cerebral congestion
- Apoplexy (cerebral haemorrhage)
- Trombosis
- Fat embolism
- Aeremia (too much air in the blood)
- Anaphylaxis
- Faulty protein metabolism
- Alkalosis (1947)

Jerseys are more susceptible to milk fever than Holsteins

Milk fever

- Does not only affect dairy cow welfare, but impacts negatively on economy of milk production.
- Treatment usually effective, but hypocalcaemic cows more susceptible to other metabolic conditions and infectious diseases:
  - Poor rumen motility (leading to reduced feed intake)
  - Increased risk of ketosis and displaced abomasum
  - Dystocia due to poor uterine motility (increased risk of retained placenta and metritis)

Aetiology

Milk fever only metabolic disease associated with increased milk production.

Cows secrete ~ 20 – 30 g of Ca/d into colostrum and milk.

Blood Ca declines from 8.5 – 10 mg/dL to < 7.5 mg/dL.

Results in hyperexcitability of nervous system and reduced strength of muscle contractions, resulting in tetany and paresis.

Aetiology

Aetiology relates primarily to macro-mineral composition of transition cow diets.

Evidence that mobilization of Ca from bone by PTH and calcitriol might be impaired under insulin resistance.

Biochemical and Physiological Aspects

Prevailing theory since the 1960s: Milk fever caused by high Ca in dry period, causing a shutdown of parathyroid gland activity during dry period (Goff, 2006).

At calving, PT gland too sluggish to stimulate Ca mobilization from the bone.

With low pre-partum Ca diets, PT gland is stimulated to release PTH several weeks before calving.

Biochemical and Physiological Aspects

Reported that hypocalcaemia reduces the release of acetylcholine (ACH). (Horst & Reinhardt, 1983)

With hypocalcaemia, endogenous opioid peptides (EOP) are increased during parturition (Petraglia et al., 1985).

One of many cellular responses is a blockage of voltage-gated Ca^{++} channels and opening of K^{+} channels, inhibiting release of ACH (Kim et al., 2005).

Rizzio et al. (2008) suggested that ACH release may occur in milk fever, but that it cannot bind adequately to its specific receptor.

Biochemical and Physiological Aspects

Reactivation of Ca homeostatic mechanisms takes time:

- Increase in Ca abs. after 1,25$(OH)_2$D stimulation: 24 h.
- Increase in bone resorption after PTH stimulation: 48 h.
- In milk fever cows, even longer (Reinhardt et al., 1988).

Low Ca dry cow diets remained strategy until 1984 when the DCAD principle was developed.

Low Ca diets

Milk fever risk highest with prepartum dietary [Ca] of 1.35%.

If Ca is too low (< 0.4% of diet DM), risk also increases.

Dietary Ca can be reduced by:

- Feedstuff selection.
- Ca binding with a commercial Ca binder, such as aluminium silicate, or with rumen bypass phytic acid from rice bran.
- (Too much Ca binding can have opposite effect)

Biochemical and Physiological Aspects

A 1997 USDA study suggested that metabolic alkalosis, induced by high K or Na diets, reduced PTH responsiveness of bone and kidney tissues and that high Ca diets *per se* did not cause milk fever.

Nearly all dietary K^+ and Na^+ are absorbed by cows, making them powerful alkalinizing cations (NRC, 2001).

Ca^{++} and Mg^{++} poorly absorbed; not strong alkalinizing agents.

Low Ca diets are still a strategy to "trick" the PT gland into secreting PTH, but low Ca diets sometimes difficult to formulate.

Biochemical and Physiological Aspects

The DCAD principle

Primary aim is to reduce metabolic alkalosis and induce mild metabolic acidosis.

Cows are usually metabolically alkalotic (urine pH in lactating cows > 8 mg/dL).

- Reduced efficiency of PTH to bind to its receptor.
- Reduces activity of Vit D_3 (function to increase Ca release from bone and Ca absorption from gut).
- Induces a state of pseudohypoparathyroidism.

Biochemical and Physiological Aspects

The DCAD principle

The DCAD concept is an empirical hypothesis, not a physiological mechanism (Ramberg et al., 1996).

Criticism towards the current model to explain the mode of action pertains primarily to the statement that PTH is more efficient to bind to its receptors under acidic blood conditions.

The argument is that in early lactation, when blood pH is high, PTH responsiveness is adequate (Martin-Tereso and Verstegen, 2011).

Biochemical and Physiological Aspects

The DCAD principle

An alternative model involves two transient receptor potential vanilloid channels.

The main Ca entry channel in the intestine is TRPV6.

The only entry channel in the kidney is TRPV5.

Under acidic conditions, TRPV5 is inactivated and TRPV 6 stimulated.

Biochemical and Physiological Aspects

Various DCAD formulae have been published.

Most widely used and accepted:

DCAD (mEq) = $(Na^{+} + K^{+}) – (Cl^{-} + S^{=})$

This relates to mEq/100 g of DM =

[(%Na in diet/.023) + (%K/.039)] - [(%Cl/.0355) + (%S/.016)]

Biochemical and Physiological Aspects

Five equations evaluated by Charbonneau et al. (2006)

The following one showed highest correlation with milk fever incidence and urinary pH:

DCAD (mEq) = $(Na^{+} + K^{+}) - (Cl^{-} + 0.6\ S^{=})$

Biochemical and Physiological Aspects

A DCAD value of -10 to -15 mEq/100 g DM is accepted to be sufficient to prevent milk fever.

Various anionic salts are available to manipulate DCAD.

These include sulphates and chlorides of ammonium, calcium and magnesium.

Reducing DCAD with anionic salts should only be considered if DCAD of diet is below 25 mEq/100 g DM.

Prevention via DCAD approach

Select feedstuffs that are low in Ca and K.

Ammonium chloride and calcium chloride application to lucerne at the beginning of growing season can elevate Cl^- content and lower DCAD value of lucerne (Goff et al., 2007.)

$MgSO_4$ and $MgCl_2$ more palatable than NH_4Cl, and also supply useful supplementary Mg which will generally be beneficial to the pre-calving cow.

Prevention via DCAD approach

First inclusion should be the sulphates of Mg, Ca or ammonium, or combination.

Supplement until S-content of diet reaches 0.4%.

$MgSO_4$ has additional benefit of increasing Mg^{++}, but limit to 0.4% Mg in diet DM.

If max Mg is reached before max S content, use $CaSO_4$ or $(NH_4)_2SO_4$ to reach 0.4% S.

Then use chloride salts to reach required DCAD.

Ca can then be included at 1.5 or 1.8%.

Partial DCAD

Phased-in approach to decrease K^{++} and Na^{+} and/or to increase Cl^{-} and $S^{=}$.

Select low K forages.

Or use high Cl^{-} and $S^{=}$ feeds, e.g. feedstuffs treated with H_2SO_4 or HCl (SoyChlor), or lucerne fertilised with NH_4Cl or $CaCl_2$.

Supplement with $MgCl_2$ 100g/d, up to 150-170 g/d, depending on DCAD of total diet. Most popular anionic salt in combination with low K forages.

Or merely use less anionic salts if intakes become problematic.

Dietary risk factors

Adams, Ishler & Moore, 1996:

Excessive Ca intake (0.7 – 1.0% TRDM)

Low Ca intake (< 0.4% in TRDM)

Excessive P intake (> 0.4% TRDM)

Low P intake (< 0.28% TRDM)

Low Mg intake (< 0.2% in TRDM)

High K intake (> 1.2% TRDM)

Control principles

Urinary pH (Brown, 2015):

8.0+	**Too High:** This is "normal" for lactating cow urine,
7.0 – 8.0	**Still a Little High:** Some milk fever will be prevented.
6.0 – 7.0	**Ideal Range for Partial DCAD:** Many noticeable benefits will be realized.
5.5 – 6.0	**This Range Indicates Full DCAD:** Maximum benefits will be derived, but monitor closely to ensure cows don't become overly-acidified (pH less than 5.5).

Control principles

Oral Ca drenching around calving:

Recommend 3-4 evenly spread doses from 12-24 h before calving up to 24 h after calving.

Drawbacks:

Requires single cow handling.

Risk of aspiration pneumonia.

$CaCl_2$ and Ca formate products may result in GIT irritation and systemic acidosis.

Control principles

Acidifying rations (DCAD):

DCAD of -10 to -15 mEq/100g DM, at least 10 d prepartum, preferably 14 d.

Drawbacks:

Low palatability resulting in reduced DMI.

Only attempt if diet DCAD < 25 mEq/100 g DM.

Control principles

Low Ca intake:

Highly effective, provided daily Ca intake is below 20 g/d.

Exposure period at least 20 d before calving.

Drawbacks:

With commonly used feeds, Ca intake < 20 g/d is difficult to obtain.

Consider Ca binder, but keep min [Ca] in mind.

Control principles

Prepartum administration of vit D:

Controversial.

Timing for treatment critical. Injection 2-8 days optimal.

Drawbacks:

Efficient doses close to toxic dose.

Control principles

Peripartum Mg control:

Check Mg status as part of MF control programme.

Blood [Mg] < 2.0 mg/dL within 24h p.p. suggest inadequate dietary Mg absorption (Goff,1998).

Mg content of dry cow diet should be 0.4% of DM.

Mg involved in production, release and binding of PTH to its receptors.

Drawback:

Mg uptake is severely decreased by high [K^+]

Control principles

Antioxidants:

Methionine (from which glutathione peroxidase can be derived), natural tocopherols, coated vitamin C, flavonoids, etc., would reduce oxidative stress and calving stress.

Antioxidants would also increase immune status and liver function of the cow.

Control principles

Body condition control:

Over-conditioned cows have higher risk of MF and other diseases.

Ensure BCS of ≤ 3.75.

Control principles

Controlling CHO intake peripartum:

Information conflicting.

Large amounts of CHO may result in "overfed" cows that might loose appetite around calving.

Control principles

Shortening the dry period:

Cows with too long dry period may become too fat. Depend on feeding program.

Less profound drop in blood Ca of cows with dry period of 4 days vs. 8 weeks.

Dry period of 4 wks also reduced MF.

Drawback:

Reduction in milk production next lactation.

Control principles

Prepartum milking:

Conflicting results.

Not regarded as an option in herd control programmes to prevent MF.

Control principles

Reduced milking in early lactation:

Not enough research results to make meaningful conclusion.

Buffering diets

If prepartum diets have to be buffered:

$NaHCO_3$ would add Na.

At 0.8% inclusion, $NaHCO_3$ would add 9 mEq of Na/100 g DM.

Acid Buf would add Ca^{++}

A daily intake of 90 g of Acid Buf would relate to 27 g of Ca^{++}

Post partum

Positive DCAD required. ($Na^+ + K^+ + Cl^-$)

Increasing DCAD with K^+ rather than Na+ increased milk fat concentration.

At DCAD of 25 – 40 mEq/100 g DM, K had no effect on DMI, but DMI increased at higher DCAD. (Iwaniuk et al., 2015)

High DCAD (50 mEq/100g DM) increased milk production and improved protein utilisation. (Wildman, West & Bernard, 2007)

Milk fat and DMI increased with DCAD ranging from 23-88 mEq/100 g DM in pasture cows. (Roche, Petch & Kay, 2005)

Conclusions

Milk fever has far reaching economic implications.

World wide, 3.5-10% of cows suffer from clinical MF.

Subclinical MF results in loss of milk production.

The prevalence is 23-39% and in some herds it can be as high as 50%.

Conclusions

The most successful preventative measures include:

Feeding a low Ca prepartum diet.

Using Ca binders.

Selecting feedstuffs, especially forages, with low K content.

Acidifying diets (DCAD principle); partial or full DCAD.

Ensuring sufficient Mg in prepartum diet.

Oral drenching around calving.

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and

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Thank mooo!