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REVIEW ON THE TRANSITION COW MANAGEMENT SYSTEM ON COMMERCIAL DAIRY FARMS

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Abstract:
The dry period, in particular the transition period, is characterized by dramatic changes in the endocrine status. These changes prepare the cow for lactogenesis and parturition. A dry cow requires nutrients for maintenance, growth of the conception, and growth of the dam (heifers). Ration composition and nutrient content may influence prepartum dry mater intake (DMI). Etiology and nutritional management of metabolic disorders such as fatty liver, ketosis, udder edema, milk fever, grass tetany, retained placenta, metritis, and displacement of the abomasum, rumen acidosis, and laminitis will be discussed in our review.

Key words: *cow, transition period, nutrition, metabolic disorders.*

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INTRODUCTION

The transition period is a critical determinant of both productivity and profitability in a dairy herd. Nutrition and management programs during this phase directly affect the incidence of post calving disorders, milk production and reproduction in the subsequent lactation. Nutritional strategies and guidelines are outlined in other papers in this symposium. The transition period imposes a number of abrupt changes on the cow. The cessation and initiation of lactation is one example. The cow will also experience as many as 4 ration shifts during this period. She may be subjected to location and social group moves. Rapid changes in both hormonal and metabolic systems must occur. All of these tend to increase the level of stress in the cow during this period. The stress response mechanism in ruminants is a complex, multifaceted system. Nutrition and management alterations provide an opportunity to minimize the effects of stress. A key challenge for veterinarians is to educate dairy producers to devote adequate resources in terms of labour, facilities and management to implement a structured transition cow program. One approach is to build an economic basis for this approach. It is generally accepted that a good dry cow program will result in an additional 1,000 to 2,000 lbs of milk in the next lactation. At least a portion of this production response is due to a decrease in post calving disorders. Recent work from the University of Illinois [1], assists in quantifying this point. This study examined the relationships between disorders, dry matter intake and milk production. In this study, 48 cows in the university herd were monitored for the first 20 days of lactation. Average daily milk production for this 20 day period was 65 lbs. Post calving events monitored were milk fever, retained placenta, metritis, ketosis (KET), displaced abomasums (DA) and mastitis. Twenty-four cows had at least one of the listed events during this period. Dry matter intake of cows with any of the above events was significantly lower than those cows with no problems. The 305 day ME-milk production was extrapolated for each cow at 60 days of lactation. Cows which experienced DA/KET had an average milk yield of 18,901 lbs of milk. These differences in milk production represent only a fraction of the total economic cost of post calving disorders. An additional consideration is the interactions which occur between disorders. A path analysis approach was used to examine these relationships in 31 commercial herds [2]. Cows with milk fever had 4 times more risk of retained placenta and 24 times more ketosis than cows which did not have milk fever. The risk of complicated ketosis was elevated in cows which had a retained placenta, DA or milk fever.

The incidence rate of postcalving disorders in high producing herds was recently surveyed [3]. A total of 41 Holstein herds with an average production of 20,412 lbs of milk were used. Average herd size was 244 cows. The reported incidence rates were:

- Retained placenta = 7%
- Milk fever = 6.2%
- DA = 2.3%
- Ketosis = 3.7%
- Downer cows = 1.1%

These incidence levels imply that high producing herds can have relatively low rates of post calving disorders. A Field observation of high producing herds tends to confirm these survey results. How do these herd managers achieve this? The challenge is to design a management program for the transition cow to minimize post calving disorders, maximize dry matter intake and milk production. This can be done once an understanding of the changes which occur during this phase is in place. The following points need to be considered as a transition cow management program is developed.

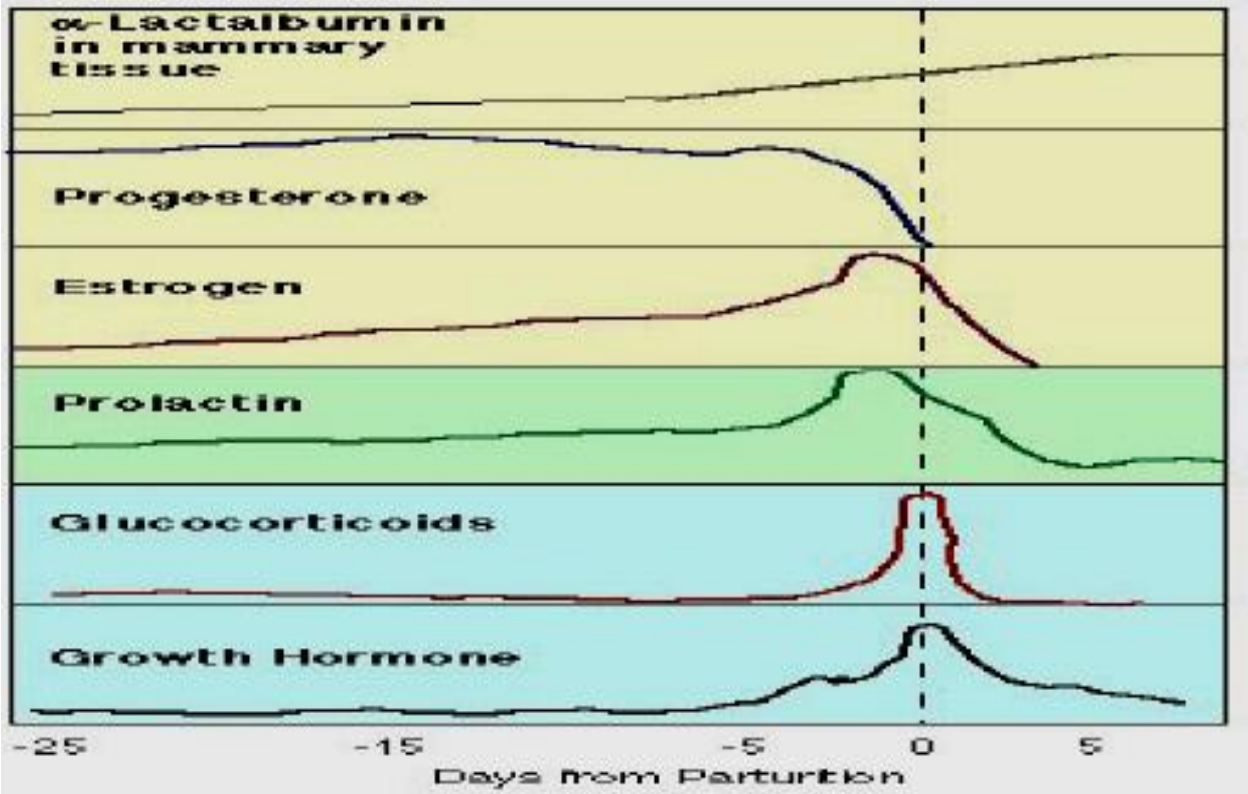

Fig 1: Physiological Hormonal Changes during Transition Cow

Fatty liver and ketosis

Fatty liver and ketosis are most likely to occur during periods when blood NEFA concentrations are elevated. At calving plasma NEFA often exceed 1000 µeq/L [4]. Liver uptake of NEFA is proportional to blood concentration. NEFA can be esterified or oxidized in liver mitochondria or peroxisomes [5]. The primary esterification product is triglyceride (TG). In ruminants export of TG from the liver, as part of a very low density lipoproteins, occurs at a very slow rate compared to other species [6], and the reason is not known. Under conditions of elevated hepatic NEFA uptake, accumulation of fatty acid esterification and triglyceride occurs. Incomplete oxidation of NEFA leads to formation of ketones - acetoacetate and beta-hydroxybutyrate. Ketonemia is common at calving during the sudden surge of NEFA, when energy requirements for milk production far exceed the energy intake, and as a secondary disorder to others that may cause DMI depression and elevated NEFA. Elevated liver triglyceride is common in cows after calving suggesting that measures to prevent fatty liver take place during the prefresh transition period [7]. Reducing severity and duration of negative energy balance (NEB) is crucial in the prevention of fatty liver and ketosis [8]. The critical time for the prevention of fatty liver is one week prior through to one week after calving [9]. This is the period when a cow is most susceptible to development of fatty liver, which is an indicator of ketosis. Maximizing DMI during that period may be achieved by avoiding over conditioned cows, rapid and frequent diet changes, unpalatable feeds, environmental stress and periparturient diseases [10]. Thin cows (BCS<3) or fat cows (BCS>4) should be avoided. Compounds to decrease fatty acid mobilization from adipose tissue or increase lipoprotein export from the liver have been suggested for prevention of fatty liver and ketosis [11]. Feeding 3-12g of niacin per day may reduce blood ketones [12] but a beneficial effect on liver triglyceride concentration has not been observed. Glucose or compounds that can be converted to glucose may decrease blood ketones following i/v administration. Propylene glycol can be given as an oral drench to reduce blood NEFA and the severity of fatty liver at calving or blood ketones post calving [13]. Salts of propionic acid may be effective in lowering blood ketones when fed.

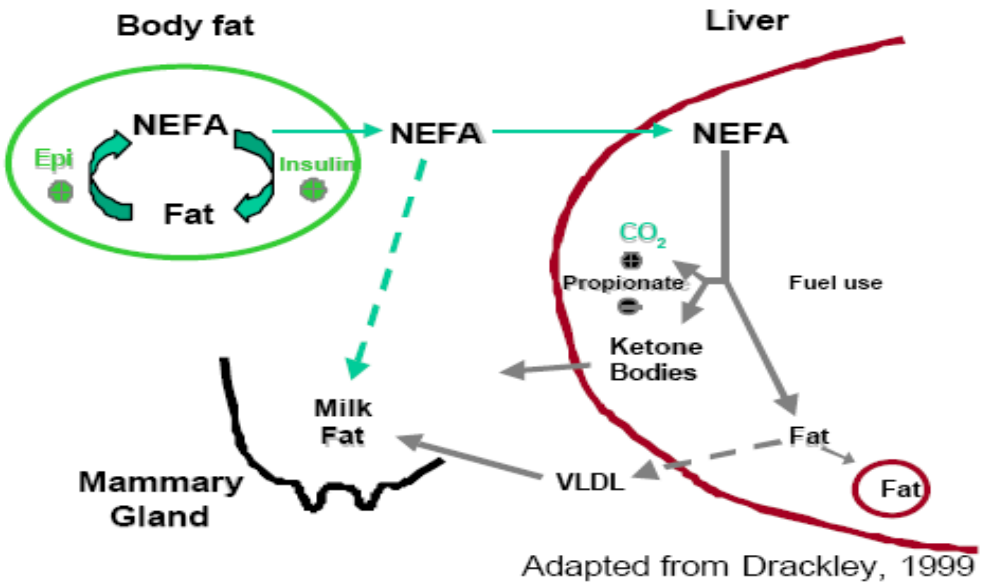

Fig 2: Oral Drench to Reduce Blood NEFA

Udder Edema

Udder edema is a periparturient disorder characterized by excessive accumulation of fluids in the intercellular tissue spaces of the mammary gland. Incidence and severity are greater in pregnant heifer than in cows [14], and tend to be more severe in older than in young heifers [15]. Udder edema can be a mayor discomfort to the cow and causes management problems such as difficulty with milking machine attachment, increased risk for teat and udder injuries and mastitis, and may also reduce milk production. The exact cause of udder edema is still unknown, most likely it is a multi-factorial condition. The remaining discussion focuses on the possible contributing nutritional factors. Emery et al. [16] found increased udder edema in pregnant heifers fed 7-8kg of concentrate per day compared with no concentrate during the last 3 weeks of gestation. Obese cows may be more predisposed to udder odema [17]. Excessive intakes of sodium and potassium were implicated as causative agents in udder edema [18] . Potassium fertilization to improve alfalfa production could be the cause of increased udder edema [19]. Tucker et al. [20] studied the effects of calcium chloride, anionic salt with diuretic properties, on incidence and severity of udder edema. The reduction od udder edema by calcium chloride was most prominent during the first week of feeding. Oxidative stress of mammary tissue resulting in reactive oxygen metabolites may play a role in udder edema [21]. A diet must supply adequate vitamin E, copper, magnesium, zinc, manganese and selenium.

Milk fever - hypocalcemia

Milk fever (MF) affects about 6% of the dairy cows in the US A. Homeostatic mechanisms, which normally maintain blood calcium concentrations between 9-10 mg/=fail and lactational drain of calcium causes blood calcium level to fall bellow 5 mg/dL [22]. This impairs muscle and nerve function so a cow is unable to rise. Intravenous calcium treatment is used to keep the cow alive long enough for bone and intestinal calcium homeostatic mechanisms to adapt. MF is easy to treat but cows that have had MF are more susceptible to other disorders such as retained placenta, ketosis, displaced abomasums and mastitis (especially coliform). Almost all cows experience some decrease in blood calcium (hypocalcaemia) during the first days postpartum. This subclinical hypocalcaemia contributes to appetence in the fresh cow and predisposes the cow to develop other diseases. Heifers almost never develop MF. The risk increases with age[23]. The Jersey, Swedish Red and White and Norwegian Red are well known to have higher incidence of MF [24].

Nutritional considerations

Cows fed diets that are relatively high in potassium or sodium is in a relative state of metabolic alkalosis which increases the likelihood for development of

MF. Hypophosphatemia and hypomagnesaemia can also be present, and can be complicating factors in some cases. Blood magnesium level below 2.0 mg/dL within 24 h after calving suggests inadequate dietary magnesium absorption. Prevention of milk fever – hypocalcemia Reducing dietary sodium and potassium. Removing potassium can sometimes pose a problem because alfaalfa and other legumes and grasses accumulate potassium within their tissues in high concentrations. Adding anions to reduce metabolic alkaloses and induce mild metabolic acidosis. Chloride salts are more acidogenic than sulfate salts [25]. Hydrocloric acid is the most potent of anion sources available. Feeding 0.35-0.40% magnesium in prepartal rations prevents a decline in the magnesium blood level at parturition. Phosphorus requirement are met by feeding 40-50 g per cow per day. Less than 25 g may lead to a downer cow syndrome [26]. More than 80 g may induce MF. The optimal prepartal dietary calcium concentration is not well defined but very high Ca concentrations (>100 g) may reduce DMI and the animal's performance. A very low calcium diet providing less than 15 g, and fed at least 10 days before calving, will reduce the incidence of MF by placing a cow in a negative calcium balance thus stimulating parathyroid hormone before calving. This activates bone osteoclasts thereby stimulating bone calcium resorption and activates renal tubules to resorb urinary calcium and begin producing vitamin D prior to calving. Oral calcium drenches at calving reduces the incidence of MF.

Grass tetany

Hypomagnesemic tetany is most often associated with cows in early lactation grazing in pastures high in potassium and nitrogen and low in magnesium and sodium [27]. The disease will progres more rapidly and tend to be more severe if accompanied by hypocalcemia. Getting an additional 10-15 g of Mg into each pregnant cow and 30 g into each lactating cow per day usually prevents further hypomagnesemic tetany. The biggest problem is getting the extra Mg into the animal because most of the magnesium salts are unpalatable and least soluble. Magnesium is readily acceptable in grain concentrates. Including 60 g of magnesium oxide into just 0.5-1 kg of grain will be effective.

Retained placenta and metritis

Retained placenta (RP) is a failure of the fetal membranes to be expelled within 12 to 24 hours after parturition. Metritis, an inflammation or infection of the uterus, is often associated with RP. RP is indirectly associated with the higher occurrence of cystic ovaries, lower milk yield and greater culling; all mediated through metritis [28]. Multiple physiologic and nutritional factors have been associated with or implicated as causes of retained placenta and metritis. Dystocia in heifers increased the risk of RP and metritis 3-4 times. Other predisponing or associated factors include: twinning, short dry periods, various stressors, heredity, MF, exposure to toxins, mycotoxins, nitrates, abnormaly low prostaglandin F2 concentrations in placentomes, other atypical peripartum profiles of steroid, pituitary and adrenal hormones. Immunosuppression in the peripartum period has been implicated as a contributing factor.

Nutritional factors

Nutritional factors of RP are primarily due to the diet fed the last 6-8 weeks before calving. Extreme deficiency of dietary energy, protein or both can result in RP. Cows fed diets low in dietary crude protein (8%) for the entire dry period had a higher incidence (50%) of RP compared with cows fed 15% crude protein (20% incidence). Fat cow syndrome is also frequently associated with increased incidence of RP and metritis [29] . The rate of RP was associated with imbalances in Ca and P metabolism. Path analyses showed that multiparous cows having MF were two times more likely to have RP and metritis [28]. Cows with RP had lower antioxidants in blood plasma during the 2 weeks before calving than cows without RP [30]. Supplementation of diets with antioxidants (vit E and Selenium) to meet the requirements is crucial during the periparturient period. When the diet contained at least 0.12 mg/kg of Se and 1000 IU of dietary vitamin E per cow/day, the incidence of RP is lower. Avitaminosis of vitamin A and β-Carotene was shown to increase the incidence of RP.

Displacement of the abomasum (DA)

The transition period from 3 weeks before calving until 4 weeks postpartum is the major risk period for development of DA. About 85% of cases involve left displacement of abomasum. Cows dried off with high BCS are at increased risk of DA as a result of poor DMI around parturition [31]. Decreasing the forage to concentrate ratio of the diet fed in late gestation and early lactation will increase the incidence of DA [32]. TMR that are easiliy sorted by cows may affect the ratio of forage to concentrate of total feed consumed by an individual cow and will contribute to DA [33]. When a TMR is not fed, grain intake after calving should be increased slowly (0.25 kg/day) until peak grain intake is achieved. Grain fed to cows should be divided into at least 3 meals per day. A decline in the calcium concentration around parturition linearly decreases abomasal contractility,

which is suspected to lead to distension and abomasal atony.

Rumen acidosis and laminitis

Rumen acidosis is associated with the feeding of diets with higher grain amounts and acidosis commonly occurs in the first month of lactation [34]. Following the dry-off a cow is fed a high forage ration that is less energy dense and higher in neutral detergent fiber than the lactation ration. This affects rumen function in two ways. The bacterial population shifts away from the lactate producers as a result of the decrease in readily fermentable starches in the diet. Another effect is a reduction in the papillae length and VFA apsorptive capacity of the ruminal mucosa. If the fresh cow is now abruptly switched to a high energy lactation diet, she is at risk of developing rumen acidosis because the lactate producers will respond rapidly to the higher starch diets and produce high amounts of lactate. The lactate converting bacterial population responds slowly to a change in diet, requiring 3-4 weeks to reach levels that will effectively prevent lactate from building up in rumen. The lactic acid, and the endotoxins and histamine released as the rumen flora die, are absorbed systemically and affect the microvasculature of the groowing hoof wall, which then result in clinical laminitis [35]. Avoiding an abrupt switch from dry-off ration to high lactation ration will be a good start for the prevention of laminitis.

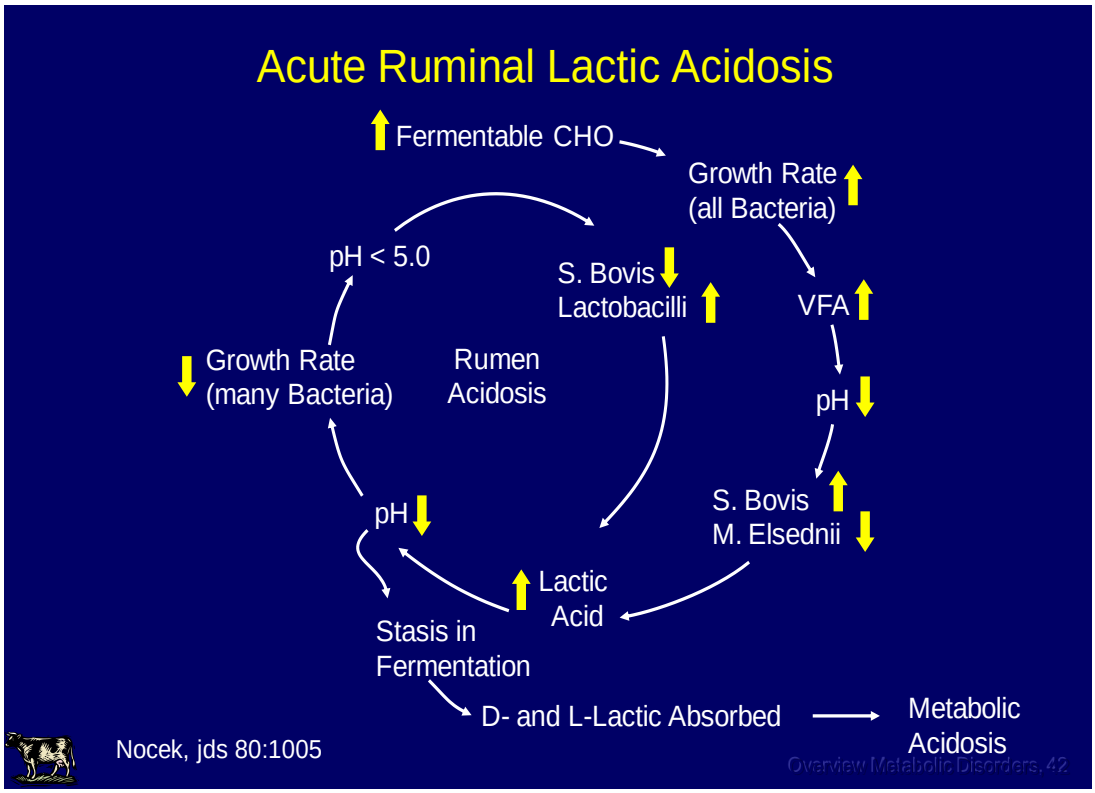

Fig 3: Acute Ruminal Lactic Acidosis

Management of transition cow

1. Dry-off
- Abrupt changes may be needed here to enhance the cessation of milk synthesis.
- Most recommendations suggest discontinuing milking abruptly.
- Dry cow treatment should be done at this time.
- Observation of the cow and udder for the first few days is critical.
- The feeding program will normally shift to high forage.
- Restrict water only if necessary.
- A change of physical facilities or location may be beneficial.

2. Early dry period
- Provide a balanced ration.
- The goal is to maintain body condition in the dry period.
- The cow should gain weight due to fetal growth.
- Feed a bulky forage to keep the rumen expanded and working.
- Avoid high K forages (> 2.5% K).
- Limit corn silage to about 1/2 of the forage dry matter.
- Provide adequate feedbunk space, feed availability and water.
- Some exercise may be helpful to maintain muscle tone.
- Clean, dry environment.

3. Close-up period (last 3 weeks)
- Adjust ration density for lower DMI.
- It may be good to bring in some of the postcalving forages or feeds to minimize palatability problems.
- Limit grain to 0.5 - 1% of bodyweight.
- Vitamin E - selenium injection if needed.
- Avoid poorly fermented silages.
- Clean, dry environment.

4. Calving
- Clean, dry environment.
- Be there to observe the calving process.
- Assist if needed but don't get overanxious.
- Don't use excessive force when assisting.
- Avoid smooth or slippery floors.
- Offer the cow water and a highly palatable feed (hay, calf starter).
- The key is to get the cow eating, drinking and ruminating.

5. Fresh cows
- Observe chewing and rumination activity.
- Some producers are taking body temperatures once or twice a day on these cows.
- Make sure fresh, palatable feeds are available.
- Water should always be available.
- A separate area to house these cows is preferable. The primary reason for this is the ability to frequently observe these cows.
- Avoid crowded, competitive environments.
- Clean, dry environment.
- The first 1 - 2 weeks postcalving set the stage for the entire lactation. Observation and reaction are the management keys during this time.
- Don't increase concentrate feeding too rapidly. The daily increase in DMI in the first week is about .6 lbs/day for older cows (11).

CONCLUSION

Dairy cows undergo a tremendous set of metabolic adaptations as they go from late pregnancy to early lactation. These changes normally are exquisitely coordinated by hormonal changes to support the new physiological state of lactation, the concept known as homeorhesis. However, these adaptive processes fail or are overcome by environmental influences in too many cows, resulting in peri parturient illness. Research to continue to increase our understanding of the adaptive processes and how they are affected by pre calving nutrition and environmental influences will undoubtedly improve our management capabilities for cows during the transition period. We are currently studying the impact of nutritional management during the long-ignored far-off dry period as a key in these adaptations.

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