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A brief review of etiology and nutritional prevention of metabolic disorders in dairy cattle

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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 conceptus, and growth of the dam (heifers). Ration composition and nutrient content may influence prepartum dry mater intake (DMI). Etiology and nutritional prevention of metabolic disorders such as fatty liver, ketosis, udder edema, milk fever, grass tetany, retained placenta, metritis, displacement of the abomasum, rumen acidosis, laminitis and milk fat depression will be discused in our review regarding the author's USA experience.

Key words: transition period, nutrition, metabolic disorders

Nutritional and physiological status of a transition cow

A dry cow requires nutrients for maintenance, growth of the conceptus, and growth of the dam (heifers). Fetal sex does not influence the growth rates (HOUSE and BELL, 1993) Fetal tissue accounts for 45% of the uterine dry weight at day 190, and 80% at day 270 of pregnancy (BELL et al., 1995). The dry period, in particular the transition period, is characterized by dramatic changes in the endocrine status (HAYIRLI et al., 2002). These changes prepare the cow for lactogenesis and parturition. Plasma insulin decreases and growth hormone increases as the a cow progresses from the late gestation to the early lactation. Surges of both hormones in plasma concentrations are visible at the parturition (KUNZ et al., 1985). Plasma thyroxine (T4) gradually increases during late gestation,

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decreases about 50% at parturition, and then begins to increase again (KUNZ et al., 1985). Progesterone concentrations during the dry period are elevated for the sake of pregnancy maintenance, but decline rapidly two days before calving (CHEW et al., 1979). Estrogen (primarily estrone of placental origin) increases in plasma during late gestation, but decreases immediately at calving (CHEW et al., 1979). Concentrations of glucocorticoid and prolactin increase on the day of calving and return to near prepartum concentrations the day after (EDGERTON and HAFS, 1973).

Different changes in endocrine status and a decrease in dry matter intake (DMI) during late pregnancy influence metabolism and lead to fat mobilisation from the adipose tissue and glycogen from the liver (ANONYMOUS, 2001; HOLTENIUS, 2003). Plasma nonesterified fatty acids (NEFA) increase more than a two-fold between 2 to 3 weeks prepartum and 2 to 3 days prepartum, at which time concentration increases dramatically until completion of parturition (VASQUEZ-ANON et al., 1994; GRUM et al., 1996). Force feeding the cows during the prefresh transition period reduced the magnitude of NEFA increase, but did not completely eliminate it (BERTICS et al., 1992). This is an indication that at least part of the prepartum NEFA increase is hormonally induced. The rapid rise in NEFA on the calving day is due to the calving stress. Plasma NEFA concentration decrease rapidly after calving but concentration remain higher then they were before calving (BAČIĆ et al., 2006). Plasma glucose remain stable or increase slightly during the prefresh transition period, increase dramatically at calving, and then decrease immediately after calving (KUNZ et al., 1985; VASQUEZ-ANON et al., 1994). Blood calcium decreases during the last few days prior to calving due to the loss of calcium for the sintesis of colostrum (GOFF and HORST, 1997), and usually do not return to a normal level until several days postpartum.

As cows begin and terminate the dry period, there are changes in rumen dynamics. These alterations are nutritionally rather than physiologically induced. Changing from the high concentrate to a high fiber diet causes alterations in the microbial population and in the characteristics of the rumen epithelium and papillae. High concentrate diets favor starch utilizing bacteria that enhance production of lactate and propionate (BAČIĆ, 2006). High fiber diets favour cellulolytic bacteria and methane production and discriminate against bacteria that produce propionate and utilize lactate. End products of fermentation influence are papillae growth in the rumen (DIRKSEN et al., 1985). They are responsible for the absorption of volatile fatty acids. High fiber diets cause the papillae to shorten, while increasing the grain in the diet and propionate concentration in the rumen favors elongation of papillae. As much as 50%of the absorptive area in the rumen may be lost during the first 7 weeks of the dry period and elongation of papillae after reintroduction of concentrate takes several weeks (MASHEK and BEEDE, 2001). Consequently, sudden introduction of grain immediately postcalving has several negative effects. Lactate

production increases prior to the re-establishment of lactate utilizing bacteria. Lactate is very potent in reducing ruminal pH, and volatile fatty acids are absorbed at a faster rate when pH is low (GOFF and HORST, 1997). Rumen papillae will not have time enough to elongate. Therefore volatile fatty acid absorption is limited.

During the transition period the immunologic status of the cow is compromised and it may also be related to nutritional and physiological status of the cow. Estrogen and glucocorticoids are immunosuppressive agents and they increase in plasma near the calving (GOFF and HORST, 1997). Intake of vitamin A and E and other nutrients essential for the immune sistem may be insuficient as DMI is reduced during the periparturient period.

Etiology and nutritional prevention of metabolic disorders

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 (GRUM et al., 1996). Liver uptake of NEFA is proportional to blood concentration. NEFA can be esterified or oxidized in liver mitochondria or peroxisomes (DRACKLEY, 1999). 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 (PULLEN et al., 1990), and the reason is not known. Under conditions of elevated hepatic NEFA uptake, accummulation 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 (GRUMMER, 1993). Reducing severity and duration of negative energy balance (NEB) is crucial in the prevention of fatty liver and ketosis (OVERTON, 2002; OVERTON and WALDRON, 2004). The critical time for the prevention of fatty liver is one week prior through to one week after calving (DUFFIELD et al., 2002). 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 overconditioned cows, rapid and frequent diet changes, unpalatable feeds, environmental stress and periparturient diseases (AGENAS et al., 2003). 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 (MANDEBVU et al.,

2003). Feeding 3-12g of niacin per day may reduce blood ketones (DUFVA et al., 1983) 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 postcalving (PICKETT et al., 2003). Salts of propionic acid may be effective in lowering blood ketones when fed.

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 (ERB and GROHN, 1988), and tend to be more severe in older than in young heifers (HAYS and ALBRIGHT, 1966). 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. (1969) 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 (VIGUE, 1963). Excesive intakes of sodium and potassium were implicated as causative agents in udder edema (JONES et al., 1984). Potassium fertilization to improve alfaalfa production could be the cause of increased udder edema (SANDERS and SANDERS, 1981). TUCKER et al. (1992) 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 (MUELLER et al., 1998). 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 USA. 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 (GOFF, 2000). This impairs muscle and nerve function so a cow is unable to rise. Intravenous calcium treatment are used to keep the cow alive long enough for bone and intestinal calcium homeostatic machanisms 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 abomasum and mastitis (especially coliform). Almost all cows experience some decrease in blood calcium (hypocalcemia) during the first days postpartum. This subclinical hypocalcemia contributes to appetence in the fresh cowm and predisposes the cow to develop other diseases. Heifers almost never develop MF. The risk increases with age (HORST et al., 1990). The Jersey, Swedish Red and White and Norwegian Red are well known to have higher incidence of MF (GOFF et al., 1995).

Nutritional considerations

Cows fed diets that are relatively high in potassium or sodium are in a relative state of metabolic alkalosis which increases the likelihood for development of MF. Hypophosphatemia and hypomagnesiemia 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 suggest 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 (GOFF et al., 1997). 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 sindrome (COX, 1998). 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 (LITTLEDIKE et al., 1983). 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 occurence of cystic ovaries, lower milk yield and greater culling; all mediated through metritis (ERB et al., 1985). 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 facors 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 (MORROW, 1976). 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 (ERB et al., 1985). Cows with RP had lower antioxidants in blood plasma during the 2 weeks before calving than cows without RP (MILLER et al., 1993). 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 (CAMERON et al., 1998). Decreasing the forage to concentrate ratio of the diet fed in late gestation and early lactation will increase the incidence of DA (COPPOCK et al., 1972). 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 (SHAVER, 1997). 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 decrease 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 (NOCEK, 1997). 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 (RADOSTITS et al., 2000). Avoiding an abrupt switch from dry-off ration to high lactation ration will be a good start for the prevention of laminitis.

References

AGENAS, S. E., E. BURSTEDT, K. HOLTENIUS (2003): Effects of feeding intensity during the dry period. 1. Feed intake, body weight and milk production. J. Dairy Sci. 86, 870-882.

ANONYMOUS (2001): National research council (2001): Nutrient Requirement of Dairy Cattle. 7th revised ed., National Academy Press, Washington D.C. pp. 184-186.

BAČIĆ, G. (2006): Što sve treba znati o estrusu. Mljekarski list 12, 26-27.

BAČIĆ, G., T. KARADJOLE, N. MAČEŠIĆ, M. KARADJOLE (2006): Special aspects of dairy cattle nutrition etiology and metabolic disease prevention. 7th Midle European Buiatric Congres, Radenci, Slovenia, March 2006., Slov. Vet. Res. Vol. 43 (Supl. 10), pp. 169-173.

BELL, A. W., G. SLEPETIS, R. A. ERHARDT (1995): Growth and accretion of energy and protein in the gravid uterus during the late pregnancy in Holstein cows. J. Dairy Sci.78, 1954-1961.

BERTICS, S. J., R. R. GRUMMER, C. CADORNIGA-VALINO, E. E. STODDARD (1992): Effect of prepartum dry matter intake on liver triglyceride concentration and early lactation. J. Dairy Sci. 75, 1914-1922.

CAMERON, R. E., P. B. DYK, T. H. HERDT, J. B. KANEENE, R. MILLER, H. F. BUCHOLTZ, J. S. LIESMAN, M. J. VANDEHAAR, R. S. EMERY (1998): Dry cow diet, management and energy balance as risk factors for displaced abomasum in high prosucing dairy herds. J. Dairy Sci. 81, 132-139.

CHEW, B. P., R. E. ERB, J. F. FESSLER, C. J. CALLAHAN, P. V. MALVEN (1979): Effects of ovariectomy during pregnancy and of prematurely induced parturition on progesterone, estrogens and calving traits. J. Dairy Sci. 62, 557-566.

COPPOCK, C. E., C. H. NOLLER, S. A. WOLFE, C. J. CALLAHAN, J. S. BAKER (1972): Effect of forage-concentrate ratio in complete feeds fed ad libitum on feed intake prepartum and the occurrence of abomasal displacement in dairy cows. J. Dairy Sci. 55, 783-789.

COX, V. S. (1998): Downer cow sindrome. In: Current Veterinary Therapy 4: Food animal practice, (Howard, J. L., R. A. Smith, Eds.), Philadelphia W. B. Saunders Co. pp. 215-218.

DIRKSEN, G. U., H. G. LIEBICH, E. MAYER (1985): Adaptive changes in ruminal mucosa and their functional and clinical significance. Bovine Pract. 20, 116-120.

DRACKLEY, J. K. (1999): Biology of dairy cow during the transition period: the final frontier? J. Dairy Sci. 82, 2259-2273.

DUFFIELD, T., R. BAGG, L. DESCOTEAUX, E. BOUCHARD, M. BRODEUR, D. DUTREMBLAY, G. KEEFE, S. LEBLANC, P. DICK (2002): Prepartum Monensin for the reduction of energy associated disease in postpartum dairy cows. J. Dairy Sci. 85, 397-405.

DUFVA, G. S., E. E. BARTLEY, A. D. DAYTON, D. O. RIDDELL (1983): Effect of niacin supplementation on milk production and ketosis of dairy cattle. J. Dairy Sci. 66, 2329-2336.

EDGERTON, L. A., H. D. HAFS (1973): Serum luteinizing hormone, prolactin, glucocorticoid and progestin in dairy cowsfrom calving to gestation. J. Dairy Sci. 56, 451-458.

EMERY, R. S., H. D. HAFS, D. ARMSTRONG, W. W. SNYDER (1969): Prepartum grain feeding effects on milk production, mammary edema and incidence of diseases. J. Dairy Sci. 52, 345-351.

ERB, H. N., R. D. SMITH, P. A. OLTENAW, C. L. GUARD, R. B. HILLMAN, P. A. POWERS, M. C. SMITH, M. E. WHITE (1985): Path model of reproductive disorders and performance, milk fever, mastitis, milk yield and culling in Holstein cows. J. Dairy Sci. 68, 3337-3349.

ERB, H. N., Y. T. GROHN (1988): Epidemiology of metabolic disorders in periparturient dairy cow. J. Dairy Sci. 71, 2557-2571.

GOFF, J. P. (2000): Pathophysiology of calcium and phosphorus disorders. Vet. Clin. N. Am. Food Anim. Pract. 16, 319-337.

GOFF, J. P., R. RUIZ, R. L. HORST (1997): Relative acidogenic activity of commonly used anionic salts-re-thinking the dietary cation-anion difference equations. J. Dairy Sci. 80 (Suppl. 1) 169-173.

GOFF, J. P., R. L. HORST (1997): Physiological changes at parturition and the relationship to metabolic disorders. J. Dairy Sci. 80, 1260-1268.

GOFF, J. P., T. A. REIHARDT, D. C. BEITZ, R. L. HORST (1995): Breed affects tissue vitamin D receptor concentration in periparturient dairy cows: A milk fever risk factor? J. Dairy Sci. 78 (Suppl. 1) 184-187.

GRUM, D. E., J. K. DRACKLEY, R. S. YOUNKER, D. W. LACOUNT, J. J. VEENHUIZEN (1996): Nutrition during the dry period and hepatic lipid metabolism of periparturient cows. J. Dairy Sci. 79, 1850-1864.

GRUMMER, R. R. (1993): Etiology of lipid-related metabolic disorders in periparturient cows. J. Dairy Sci. 76, 3882-3896.

HAYIRLI, A., R. R. GRUMMER, E. V. NORDHEIM, P. M. CRUMP (2002): Animal and dietary factors affecting feed intake during the prefresh transition periods in holsteins. J. Dairy Sci. 85, 3430-3443.

HAYS, R. L., J. L. ALBRIGHT (1966): Udder edema: Its incidence and severity as affected by certain management practices. Illinois Res. 8, 6-8.

HOLTENIUS, K., S. AGENAS, C. DELAVAUD, Y. CHILIARD (2003): Effects of feeding intensity during the dry period. 2. Metabolic and hormonal responses. J. Dairy Sci. 86, 883-891.

HORST, R. L., J. P. GOFF, T. A. REINHARDT (1990): Advancing age results in reduction of intestinal and bone 1,25 dihydroxyvitamin D receptor. Endocrinol. 126, 1053-1057.

HOUSE, W. A., A. W. BELL (1993): Mineral accretion in the fetus and adnexa during late gestation in Holstein cows. J. Dairy Sci. 76, 2999-3010.

JONES, T. O., R. KNIGHT, R. K. EVANS (1984): Chronic udder edema in milking cows and heifers. Vet. Rec. 115, 218-219.

KUNZ, P. L., J. W. BLUM, I. C. HART, H. BICKEL, J. LANDIS (1985): Effects of different energy intakes before and after calving on food intake, performance, and blood hormones and metabolites in dairy cows. Anim. Prod. 40, 219-231.

LITTLEDIKE, E. T., J. A. STUEDEMAN, S. R. WILKINSON, R. L. HORST (1983): Grass tetany syndrome. In: Role of Magnesium in Animal Nutrition, (Fontencot, J. P., G. E. Bunce, K. E. Webb, V. G. Allen, Eds.) Blacksburg, VA, Virginia Polytechnic Inst. and State University. pp. 173-195.

MANDEBVU, P., C. S. BALLARD, C. J. SNIFFEN, D. S. TSANG, F. VALDEZ, S. MIYOSHI, L. SCHLATTER (2003): Effect of feeding an energy supplement prepartum and postpartum on milk yield and composition, and incidence of ketosis in dairy cows. Anim. Feed Sci. Tech. 105, 81-93.

MASHEK, D. G., D. K. BEEDE (2001): Peripartum responses of dairy cows fed energy-dense diets for 3 or 6 weeks prepartum. J. Dairy Sci. 84, 115-125.

MILLER, J. K., E. BRZEZINSKA-SLEBODZINSKA, F. C. MADSEN (1993): Oxidative stress, antioxidants and animal function. J. Dairy Sci. 76, 2812-2823.

MORROW, D. A. (1976): Fat cow syndrome. J. Dairy Sci. 59, 1625-1629.

MÜELLER, F. J., J. K. MILLER, M. H. CAMPBELL, F. C. MADSEN (1998): Prevention of udder edema in dairy cows. Proc. Tri-State Dairy Nutr. Conf. Fort Wayne, IN. pp. 79-95.

NOCEK, J. E. (1997): Bovine acidosis: Implications of laminitis. J. Dairy Sci. 80, 1005-1028.

OVERTON, T. R. (2002): Could feeding CLA improve energy balance, decrease metabolic disorders and improve reproductive performance. Pro Dairy, Northeast Dairy Business, June 2002., pp. 22.

OVERTON, T. R., M. R. WALDRON (2004): Nutritional management of transition dairy cows: Strategies to optimize metabolic health. J. Dairy Sci. 87 (E Suppl), 105-119.

PICKETT, M. M., M. S. PIEPERBRINK, T. R. OVERTON (2003): Effects of propylene glycol or fat drench on plasma metabolites, liver composition, and production of dairy cows during the periparturient period. J. Dairy Sci. 86, 2113-2121.

PULLEN, D. L., J. S. LIESMAN, R. S. EMERY (1990): A species comparison of liver slice synthesis and secretion of triacylglycerol from nonesterified fatty acid in the media. J. Anim. Sci. 68, 1395-1399.

RADOSTITS, O. M., C. C. GAY, D. C. BLOOD, K. W. HINCHCLIFF (2000): Veterinary Medicine, W. B. Saunders LTD. London, New York, Philadelphia, San Francisko, St. Louis, Sydney, pp. 1805-1806

SANDERS, D. E., J. A. SANDERS (1981): Chronic udder edema in dairy cows. J. Am. Vet. Med. Assoc. 178, 1273-1274.

SHAVER, R. D. (1997): Nutritional risk factors in the etiology of left displaced abomasum in dairy cows: A review. J. Dairy Sci. 80, 2449-2453.

TUCKER, W. B., J. F. HOGUE, G. D. ADAMS, M. ASLAM, I. S. SHIN, G. MORGAN (1992): Influence of dietary cation-anion balance during the dry period on the occurrence of parturient paresis in cows fed excess calcium. J. Anim. Sci. 70, 1238-1250.

VASQUEZ-ANON, M., S. BERTICS, M. LUCK, R. R. GRUMMER, J PINHEIRO (1994): Peripartum liver triglyceride and plasma metabolites in dairy cows. J. Dairy Sci. 77, 1521-1528.

VIGUE, R. F. (1963): Management of udder edema in cattle. Can. Vet. J. 4, 236-241.

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SAŽETAK

Za suhostaj, a naročito prijelazni period, karakteristične su velike promjene u endokrinom statusu krave. Ove promjene pripremaju kravu za porođaj i laktaciju. Krave u suhostaju metaboliziraju hranjive tvari za održanje bazalnog metabolizma, za rast i razvoj ploda, te za vlastiti rast i razvoj. Mješavina hrane i sadržaj hranjivih tvari utječu na količinu unosa suhe tvari u organizam u danima prije porođaja. Etiologija i hranidbena preventiva metaboličnih poremećaja kao što su zamašćena jetra, ketoza, edem vimena, mliječna groznica (hipokalcemija), pašna tetanija, zaostala posteljica, upala maternice, dislokacija sirišta, acidoza buraga, laminitis i smanjenje količine mliječne masti u mlijeku ukratko su obrađene u ovom članku.

Ključne riječi: tranzicijski period, hranidba, metabolični poremećaji