Technical Bulletin

Information from Phibro Technical Services

Ken Zanzalari, Ph.D., Dipl ACAN
Phibro Animal Health Corporation, Quincy, IL

Control Milk Fever Through Prefresh Diet

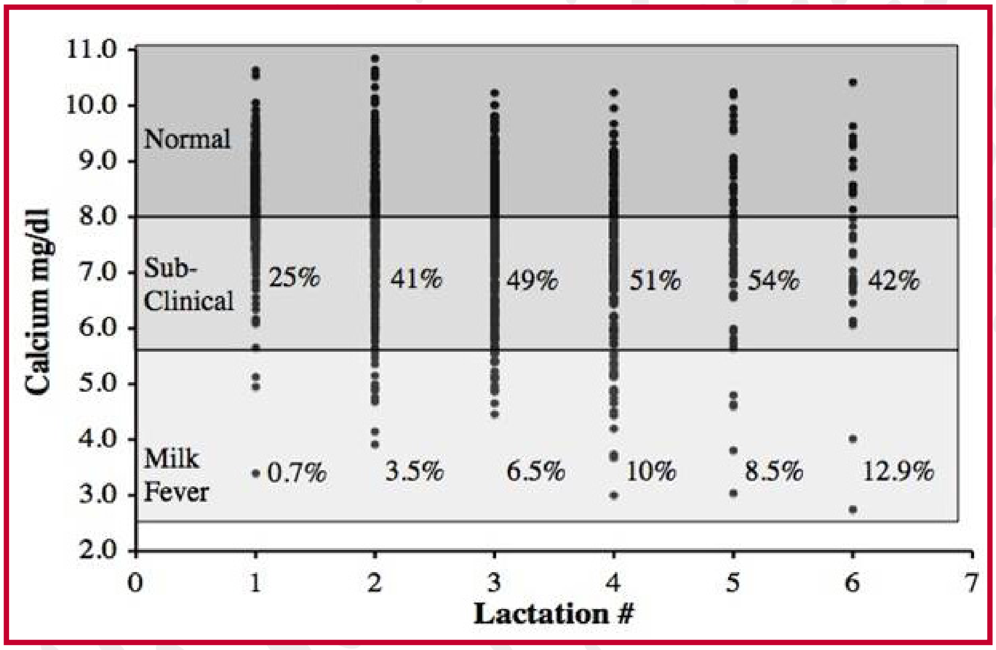
Introduction

The transition from late pregnancy to lactation requires enormous physiological adaptations by the dairy cow.

One of the most important homeorhetic changes that occurs involves the balance of calcium. Cows that are unable to maintain adequate blood calcium concentrations are susceptible to higher rates of early postpartum metabolic disorders and produce less milk.

In a 2011 publication (Reinhardt et al., 2011), the reported incidence of clinical hypocalcemia (blood calcium concentrations below 5.5 mg/dL) was found to be low, at 0.7 - 12.9%, while the incidence rate of subclinical hypocalcemia (blood calcium concentrations below 8.0 mg/dL) in the same population of cows was much higher, ranging from 25% to 54% (Figure 1).

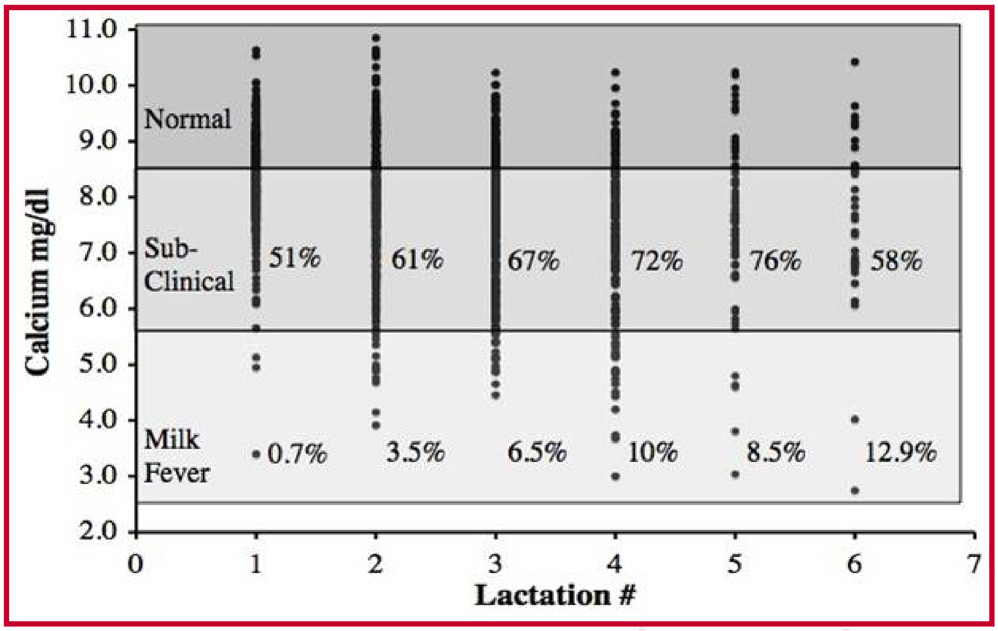
Figure 1: Incidence of hypocalcemia (8.0 mg/dL or less) in U.S. confinement herds



When the threshold is raised to 8.5 mg/dL (Figure 2), the incidence level of subclinical hypocalcemia increases in all parity groups.

Recent research suggests that a more appropriate threshold for blood calcium concentrations may be 8.59 mg/dL when blood calcium concentrations are monitored within the first three days of lactation (Martinez et al., 2012).

Figure 2: Incidence of hypocalcemia (8.5 mg/dL or less) in U.S. confinement herds



Source: Reinhardt, personal communication, March 25, 2013.

The researchers observed that blood calcium concentrations on the day of calving exceeded 8.0 mg/dL, irrespective of classification (low-risk or high-risk of developing uterine disease). On the day of calving, low-risk and high-risk cows had mean blood calcium concentrations of 9.02 mg and 8.86 mg/dL, respectively, well above the traditionally accepted 8.0 mg/dL threshold for subclinical hypocalcemia.

Chapinal et al. (2012) also reported that cows with low blood calcium concentrations pre and postcalving were at greater risk for displaced abomasums, lower early-lactation milk yield and decreased first-service artificial insemination (AI) pregnancy rates.

Other research has also linked clinical and subclinical hypocalcemia with health related disorders, including dystocia and ketosis (Curtis et al., 1985), a reduced rate and strength of abomasal and rumen contractions (Daniel, 1983) and impaired immune function (Kimura et al., 2006).

Lower serum calcium in fresh cows has been shown to reduce intracellular endoplasmic reticulum calcium storage, to release intracellular calcium by stimulated peripheral blood mononuclear cells (Kimura et al., 2006) and to decrease the number and killing ability of neutrophils (Martinez et al., 2012).

The impaired immune response of hypocalcemic cows may partially explain the increased risk of immune-related infectious diseases observed in this population of cows, including mastitis (Curtis et al., 1985), retained placenta (Kimura et al., 2006; Melendez et al., 2004) and metritis (Martinez et al., 2012).

Hidden Thief

Lost revenue associated with subclinical hypocalcemia far exceeds that of clinical hypocalcemia. Recently, Oetzel (2012), using data from a previous study (Oetzel, 2011) and the economic costs associated with clinical milk fever suggested by Guard (1996), estimated that the economic loss due to subclinical hypocalcemia on a typical farm can be nearly four times the loss associated with clinical milk fever.

This is primarily because the incidence rate of subclinical hypocalcemia is 15 times higher than the incidence rate of clinical hypocalcemia.

Managing Hypocalcemia

Several methods have been investigated to minimize the occurrence of clinical and subclinical hypocalcemia. These include:

Reducing dietary potassium intake - While this will primarily help reduce the number of clinical cases, it will not reduce a significant amount of subclinical cases because even if potassium levels are carefully monitored, the resulting dietary cation-anion difference (DCAD) level will not be low enough to prevent subclinical hypocalcemia.

Feeding a low-calcium diet - While restricting calcium intake prepartum has been demonstrated to reduce clinical milk fever in a research setting, this approach is very difficult to achieve in the field. Research has demonstrated that in order to successfully reduce clinical milk fever by dietary calcium means alone, the intake of calcium must be below 20 g per cow per day. Under most practical feeding situations, this is not attainable.

Vitamin D therapy - Research has demonstrated that this approach works, but it is not practical. To prevent milk fever, vitamin D injections must be given between day 1 and day 4 prior to date of calving using very high, toxic levels. If the cow calves outside of this window, additional injections cannot be given because of concerns for vitamin D toxicity.

Feeding acidogenic diets - An effective way to reduce clinical and subclinical hypocalcemia is to feed a negative DCAD diet for at least 21 days pre-partum. Proper formulation of a negative DCAD diet is important to the success of the program.

Acidogenic diets work by increasing bone calcium resorption, along with increasing intestinal calcium uptake (Horst et al., 1994). These coordinated processes help maintain normal blood calcium concentrations.

“Research has also demonstrated that formulating a prefresh diet to a DCAD level of approximately -15 mEq/100g (Moore et al., 2000) is most effective.

In addition to feeding the correct level of DCAD, it is also important to feed the correct dietary levels of calcium, phosphorus, magnesium, chloride and sulfur.

Practical guidelines

The benefits of maintaining adequate blood calcium concentrations in transition cows are well documented, including a reduced incidence of metabolic diseases, fewer infectious diseases, higher milk production and better reproduction.

Consider these guidelines for managing a successful prefresh DCAD diet:
Supply enough anions to the diet to result in a DCAD between -10 and -15 mEq/100g. Selection of an anionic product that is palatable can help attain a low DCAD while maintaining high dry matter intake.

Monitor urine pH values routinely, especially after significant dietary ingredient changes. Target urine pH between 5.5 and 6.0. Check water quality since hardness, pH, alkalinity and mineral content can affect acidification of the animal.

Feed for a minimum of 21 days pre-partum. Remember that some cows calve early, and it is better to be longer than shorter on days spent in the closeup pen. Properly balanced, negative-DCAD diets can be fed safely for long periods of time.

Feed proper levels of calcium (minimum of 180 g and not less than 1.5% DM), phosphorus (minimum of 45 g and not less than 0.38% DM), magnesium (54 - 60 g), chloride (96 - 120 g) and sulfur (48 - 56 g).

Periodically sample a subpopulation of fresh cows to determine total blood calcium concentrations. Taking samples at approximately 48 hours postcalving is the most informative time point.

References

Chapinal, N., M.E. Carson, S.J. LeBlanc, K.E. Leslie, S. Godden, M. Capel, J.E.P. Santos, M.W. Overton and T.F. Duffield. 2012. The association of serum metabolites in the transition period with milk production and early-lactation reproductive performance. J. Dairy Sci. 95:1301 and 95:5676.

Curtis, C.R., H.N. Erb, C.J. Sniffen, R.D. Smith and D.S. Kronfield. 1985. Path analysis of dry period nutrition, postpartum metabolic and reproductive disorders and mastitis in Holstein cows. J. Dairy Sci. 68:2347.

Daniel, R.C.W. 1983. Motility of the rumen and abomasum during hypocalcemia. Can. J. Comp. Med 47:276.

Guard, C.L. 1996. Fresh cow problems are costly; culling hurts the most. Hoard's Dairyman 141:8.

Horst, R.L., J.P. Goff and T.A. Reinhardt. 1994. Symposium: Calcium metabolism and utilization — Calcium and vitamin D metabolism in the dairy cow. J. Dairy Sci. 77:1936.

Kimura, K., T.A. Reinhardt and J.P. Goff. 2006. Parturition and hypocalcemia blunts calcium signals in immune cells of dairy cattle. J. Dairy Sci. 89:2588.

Martinez, N., C.A. Risco, F.S. Lima, R.S. Bisinotto, L.F. Greco, E.S. Ribeiro, F. Maunsell, K. Galvao and J.E.P. Santos. 2012. Evaluation of peripartal calcium status, energetic profile and neutrophil function in dairy cows at low or high risk of developing uterine disease. J. Dairy Sci. 95:7158.

Melendez, P., A. Donovan, C. Risco and J.P. Goff. 2004. Plasma mineral and energy metabolite concentrations in dairy cows fed an anionic prepartum diet that did or did not have retained fetal membranes after parturition. Am. J. Vet. Res. 65:1071.

Moore, S.J., M.J. VandeHaar, B.K. Sharma, T.E. Pilbeam, D.K. Beede, H.F. Bucholtz, J.S. Liesman, R.L. Horst and J.P. Goff. 2000. Effects of altering dietary cation-anion difference on calcium and energy metabolism in peripartum cows. J. Dairy Sci. 83:2095.

Oetzel, G.R. 2011. Diseases of Dairy Animals — Non-infectious Diseases: Milk Fever. Encyclopedia of Dairy Sciences. Vol. 2.

Oetzel, G.R. 2012. An update on hypocalcemia on dairy farms. Proceedings of Four-State Dairy Nutrition & Mgmt. Conf.

Reinhardt, T.A., J.D. Lippolis, B.J. McCluskey, J.P. Goff and R.L. Horst. 2011. Prevalence of subclinical hypocalcemia in dairy herds. Vet. J. 188:122.