

DAIRY VETERINARY NEWSLETTER

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Heat Treating Colostrum – Should it be a Standard Practice? Is There a Place for Colostrum Replacer Products? Will the Industry Return to Group Housing of Calves, and What About Cross-suckling? Can Profitability be Increased and Methane Reduced by Avoiding Excess Crude Protein in Dairy Rations? Is Routine Ketosis Therapy Beneficial, or do we Even Know?

I recently attended the American Dairy Science Association meeting, and as usual there were some interesting presentations. I heard numerous comments, which I have to agree with, that many of the presentations were regarding research that not only had no practical application, but in addition the researchers did not seem to be considering future practical application on farms. However, research sometimes leads to tremendous practical benefit that was not clear initially. Hopefully this will be the case for some of the new information.

A couple of subjects of several ADSA presentations, with clear practical importance, were colostrum management and housing of pre-weaned dairy calves. A subject with tremendous long-term importance, methane emission reduction, was also addressed.

Colostrum heat treatment – why and how?

In recent years, the question has been posed as to whether colostrum should be heat treated to reduce bacteria counts, and whether this can be done without destroying too much of the supply of maternal antibodies in the colostrum. As stated in a story with no author citation on Dairy Herd Network on May 27, 2012, “But heat-treating colostrum is a different game than pasteurizing waste milk. Early attempts to process it using traditional - - PMO - - specifications of 62.5 to 73 °C [145 to 163 °F] resulted in a thick, denatured product that clogged pasteurization equipment and was unacceptable for feeding. - - University of Minnesota researchers led by Sandra Godden refined the process, and found that colostrum could be successfully heat-treated by heating it to 60°C [140 °F] and maintaining that temperature for 60 minutes. This process significantly decreased or eliminated bacterial organisms including *Mycoplasma bovis*, *Listeria monocytogenes*, *E. coli*, *Salmonella enteritidis*, and - - [*Mycobacterium avium* subsp. *paratuberculosis*] (the bacteria that causes Johne’s disease). At the same time, neither colostrum IgG concentration, nor viscosity, were significantly affected. These ‘60/60’ parameters have been evaluated in three other research studies with similar results.”

Two papers in Journal of Dairy Science by Donahue et al., May 2012 and Godden et al., July 2012 (same group of authors) report on the 60/60 (60 °C for 60 min) colostrum treatment. Studies of 266 colostrum batches on 6 Midwestern dairy farms housing 9,700 cows compared 60/60 treatment in commercial batch pasteurizers with no treatment; aliquots of pre- and post- heat-treated colostrum were frozen and thawed. Total bacteria and coliform counts were decreased but IgG concentration was not significantly decreased in the heat treated colostrum, remaining “high overall”. (See more details in next paragraph.)

The important question of calf health/performance was evaluated in the second paper, following 1,071 newborn calves from the same 6 farms. Calves were randomly assigned to be fed 3.8L of either fresh (FR, n=518) or heat-treated colostrum (HT, n=553) within 2 h of birth. Median total bacteria count was 515,000 cfu/ml in FR colostrum, 2,100 cfu/ml in HT (some FR samples had no bacteria, and HT samples ranged up to 240 million cfu/ml, so there was still considerable variability), coliform count medians were 51,500 cfu/ml (FR) and 90 cfu/ml (HT) (some FR samples had no coliforms, and HT samples ranged up to 9.5 million coliform cfu/ml, again high variability). Serum IgG in calves was measured once between 1 to 7 d old. Treatments and deaths were recorded until weaning. IgG concentrations were significantly higher in calves fed HT colostrum (18.0 mg/mL) compared with calves fed FR colostrum (15.4 mg/ml). There was significant decreased risk for treatment (any cause) in calves fed HT colostrum (30.9%) compared with FR colostrum (36.5%), and for scours (HT colostrum 16.5%, FR colostrum 20.7%). However, death loss in both groups was excellent, 9/518 (1.7%) for FR and 13/553 (2.4%) for HT, not significantly different.

At ADSA, a presentation by A. Lago et al. evaluated possible thresholds of total bacteria count (TBC) or coliform count (CC) in colostrum (not heat treated) to look for levels above which calf welfare or performance would be compromised. They studied 172 heifer calves, including death loss, but until what age was not clear. There were no differences in calf performance including average daily gain among colostrum TBC thresholds between 1,000 and 1,000,000 cfu/ml. In contrast to the study above, they found that calf mortality increased when coliform counts in colostrum were > 10,000 cfu/ml. This death loss was attributed partly to decreased GI absorption of IgG in the presence of so many bacteria, some of which antibodies attach to, but primarily to the direct pathogenic effect of the coliforms. However, only 8% of colostrum batches fed had CC > 10,000 cfu/ml, and the statistically higher mortality was based on death of only 4/11 calves (36%) fed such colostrum. This was a small group of calves compared to other categories in the study; I hope there will be more data regarding this question in the future.

I think the jury is still out regarding heat treatment of colostrum on dairy farms, but would not discourage any producers from implementing it. Some farms and calf raising experts are convinced that it is worthwhile. Unless and until more definitive evidence of consistent reduction of bacteria counts in colostrum following heat treatment is found, and more importantly, more evidence that calf mortality or performance are associated with heat treatment emerges, I question whether a different set of time and temperature procedures than those for pasteurization are practical for colostrum management. Nevertheless, maybe the best combination of time and temperature for colostrum treatment to improve calf health is yet to be found.

Colostrum replacers – is there a place?

Colostrum replacers have been of interest to me for more than 20 years. I have often found that colostrometer measurements, closely correlated with IgG concentration, improve in poor quality colostrum when a product such as Colostrx® is blended into them with a wire whisk. However, these products have frequently been essentially ridiculed, especially verbally at dairy meetings, for many years as ineffective. I have found consistently that colostrum from dairy cows calving during the summer is rarely of good quality, whether they are first calf heifers or older cows. Data continues to show that we have too much poor quality colostrum resulting in too much failure of passive transfer (FPT), associated with average calf mortality of pre-weaned calves of around 11% in the US, far too high. A recent article by Tom Quaife on Dairy Herd Network on April 10, 2012 addressed colostrum replacers.

The article mentions a related issue that is becoming almost a buzzword in dairy press, that of “calving surges”, where larger farms may have days when more than 50 calves are born. Anyone who has experienced dozens of calves born on the same day agrees with the article that when “- - upwards of 50 [calvings] a day [make it] difficult to manage the colostrum from that many individual cows on a consistent basis. And, [nutritionist Joey Ricks] does not like the idea of pooling colostrum, since it creates biosecurity concerns. ‘I believe if you keep things simple, they will get done,’ says Ricks. ‘And, (the way things were set up previously) that colostrum

program was not simple and it was not getting done.’ [Therefore], Ricks began feeding commercial colostrum replacer to the calves, and the results have been astounding. Of 780 heifer calves born this past calving season, the farm has lost just 23 of them — a death loss of 2.9%. It is a vast improvement from previous years. ‘Death loss, I feel, is costly and limits our rate of expansion through internal growth,’ Ricks says. ‘And, right now, we are in expansion mode.’”

“The fact of the matter is, many farms fail when it comes to colostrum management. ‘A significant percentage of the calves either aren’t being fed quality colostrum or they are being fed later than they should be,’ says calf-care expert Jim Quigley, [of] APC, Inc. in Ankeny, Iowa. In fact, nearly half of 890 samples tested in [a Fresno State and Iowa State] study had bacteria above - - 100,000 cfu/ml or greater, considered the upper limit for acceptable quality. - - research in California [on] more than 500 colostrum samples indicated that the degree of contamination varies by farm, level of management, season and *whether the colostrum is supplemented with commercial supplements.*”

“Sam Leadley, calf/heifer management specialist at Attica Veterinary Associates in upstate New York, [sometimes finds that] colostrum replacer is short-term solution until the farm can get a better handle on things. ‘The most obvious example is disease control, and the most obvious disease is Johne’s,’ he says. With Johne’s, until you can establish which cows are high-risk, you have to assume that all cows can pose a risk to the calves, he adds. In that case, it may be prudent to switch to a colostrum replacer. - - And, while there are emergency situations for feeding colostrum replacers, there are other situations where colostrum replacers are fed because they are convenient. Perhaps a farm will want to feed maternal colostrum to its calves during the day, when the herdsman and others are normally there. But, it may be more convenient for the night crew to feed colostrum replacer if it is busy with other chores. ‘I think it’s a staffing question, I think it’s a convenience question,’ Leadley says. ‘A lot of the time, the night crew has very little time to take care of newborn calves.’”

The article continues with more interesting discussion of colostrum replacers or use as supplements. If dairy veterinarians have not recently discussed colostrometer measurements of colostrum quality, at least some measurements of calf serum IgG, and colostrum management with some of their clients, these would be good subjects to revisit. Colostrum supplement use may be a worthwhile practice to try on many farms.

Will the industry return to group housing of calves?

I still remember when calf hutch housing was new, and then when small fences instead of chains and collars to contain calves to individual hutches gained favor. In my experience, and from what I recall, according to research during the 1980’s, this resulted in great improvement in calf health. Compared to many forms of group housing, putting calves in equipment sheds or old barns as farms became larger, and “calf castles”, hutches were a major improvement. Many calves were noticeably cleaner and calf performance was increased when farms had a mix of group pens or enclosed housing for some calves compared with others in hutches at the same time. However, more farms are now returning to the adoption of group housing of pre-weaned dairy calves.

Two ADSA presentations by C. Cobb et al. addressed group housing. One study was of *indoor* calf housing; 72 Holstein calves were “randomly assigned” to individually housed (IH, n = 29), 2 calves/pen (G2, n = 22), or 3 calves/pen (G3, n = 21). Why the IH group was so much larger was not explained; calf mortality was not presented leaving the possibility that more calves survived in IH, but this was not addressed. Space was 2.5 m²/calf. Weaning was from approximately 49 to 53 d old. Calves’ blood was sampled 9 times between 3 d and 99 d old for neutrophil activation measures and TNF α secretion. Dry matter intake (DMI) and average daily gain (ADG) were measured. While all calves varied in “immunological measurement” and DMI at different ages, and during some weeks of age the G2 and G3 calves had higher DMI, the authors’ overall conclusion was that, “Although group [indoor] housing increased post-weaning DM intake, it did not influence ADG or any biochemical or immunological measurement throughout neonatal, weaning, or commingling periods.”

The second presentation by the same group was of *outdoor* calf housing; 49 Holstein calves were “randomly assigned” to individually housed (IH, n = 22) or 3 calves/pen (G3, n = 27). Again, the difference in reported group sizes was not explained. Space was 4.75 and 7.0 m²/calf, respectively. The reason for more space per calf in the G3 housing may have been facilities that could not readily be modified, but this was not addressed. The rest of the study design was the same as above. The G3 calves had higher DMI from 56 d to 84 d old, and ADG was either nearly or significantly higher from 21 d to 52 d old compared with IH calves. From 3 d to 47 d old, G3 calves had higher measures of neutrophil activation, and again before comingling at 94 d and 99 d. No raw numbers were presented, but this time the authors’ conclusion was, “Outdoor group housed calves had improved performance and heightened innate immune responses when compared with individually housed calves, which may be due to increased immunogenic stimulation.”

Cobb also mentioned cross-suckling as a concern of group housing, and had some photos of it. I had also wondered about this problem, which can result in *Streptococcus agalactiae*, *Staphylococcus aureus*, or *Mycoplasma* spp. infecting udders of baby calves, resulting in their calving with contagious mastitis; I associate it more with calves being group housed while still on milk. I have seen this problem in literally hundreds of herds in the past, including heifers culture-positive for any of the above 3 types of contagious mastitis when they first calve and begin to milk. During the question and answer period, I asked Cobb whether based on his experience and what he had read, he was more in favor of group or individual housing for calves. He said that he did not really see a clear advantage or disadvantage for either method. However, he speculated that eventually, as has happened in some parts of the European Union, the public will demand US regulations specifying that calves must be group housed as a perceived improvement in animal welfare.

Methane reduction and crude protein levels in dairy rations, including financial aspects

There were several ADSA talks regarding methane (CH₄) or ammonia (NH₃) emission from dairy cow manure. Some interesting data was presented that addressed something I have noticed in recent years; the dairy industry often seems to take the public position that we are not a major source of methane in the atmosphere, and that vehicle traffic is a much bigger source. It often comes across as a very defensive response, which I think just reflects our perception, often accurate, within agriculture that the small percentage of the population engaged in farming gets blamed and/or regulated too much. However, there is no denying when the data is seen that one major source of methane in the atmosphere is emission from dairy cattle GI tracts, and all other steps of dairy production and distribution are dwarfed by cow emissions as a source of methane. Opinion polls also show that in addition to more than 99% of scientists who study the question, the majority of the population now agrees that human (and human-raised livestock) activities are associated with atmospheric change that is associated with more extreme weather, including droughts and high temperatures. Ultimately this issue may become of paramount and life-threatening or lifesaving consequence to our children and grandchildren. Even a very small proportional change in methane emission per cow will result in a major reduction in total methane emission into the atmosphere each year. In addition, what if dairy farm profitability can also be increased?

A presentation by A. Hristov et al. was from a 2-year study of 12 Pennsylvania dairies (169 lactating cow average herd size) with scrape, gravity flow, or flush manure systems. Background barn floor emission data was collected at 4 samplings during Year1 for NH₃, N₂O, CH₄, and CO₂ on each farm, and after ration changes, 4 samplings during Year2. The farms were selected for dietary crude protein (CP) in the ration of approximately 16% (CP16); the mean was 16.5% CP during Year1. During Year2 the goal was to reduce CP to 15% (CP15); mean was indeed 15.4% CP during Year2; rations were sampled regularly to verify CP. Adjusting for ambient temperature, barn floor NH₃ emissions from manure were 23% lower (P < 0.001) with CP15 vs. CP16 (292 vs. 378 mg/m²/h). Emissions of CH₄ and CO₂ were not affected by dietary CP and emissions of N₂O were negligible. It was noted that all emissions were drastically reduced with flush manure systems, *but only within barns*; atmospheric emissions outside the barn were not expected to be affected. Milk yield actually slightly increased (not significantly) during Year2 with CP15 vs. CP16, 32.5 vs. 32.2 kg/cow/d, and milk composition did not change. It was also noted that despite a strong national trend in the opposite direction, income over feed cost increased

during Year2 with 15% CP fed increased. The authors' conclusion was, "- - manure NH₃ emissions can be significantly reduced by moderately decreasing dietary CP content without affecting milk yield and composition in dairy cows." Some other attempts to reduce methane per cow will be covered in future newsletters.

Routine ketosis therapy - is it beneficial?

I thought we knew that the answer to this question was yes, and had for many years. However, several presentations at the American Association of Bovine Practitioners conference in September 2011 cast some doubt on the practice of routine treatment of ketotic dairy cows.

A presentation by J. Gordon et al., AABP 2011 Proceedings pg 153, stated that in most dairy herds, the percentage of lactations affected by ketosis ranges between 30% and 61%. They performed an extensive literature search and found "1,056 unique articles, theses, and conference abstracts - - [evaluating] over 35 distinct treatment[s] used alone or in combinations" for ketosis in cows. They found only 42 (4%) of the articles appropriate for analysis according to their criteria, well designed studies including a control (controls could be treated with baseline accepted treatment, or untreated) group and looking at naturally occurring ketosis in lactating dairy cows.

The review found that 300-500 g of propylene glycol daily for 3 to 5 d "was the only commonly used product to show consistent improvement in cure rates for subclinical and clinical ketosis, decreased risk of clinical ketosis, and increased milk production." I.V. dextrose or insulin (probably S.Q. but not stated) treatment for ketosis varied in association with outcomes from beneficial to detrimental. Steroids including dexamethasone "showed the poorest performance and often increased the duration of ketosis [and] increased the risk of - - clinical ketosis [and] other disease, such as pneumonia." The authors commented on the relatively small number of "appropriately designed ketosis treatment trials currently available in the literature", and said that more well-designed studies of ketosis treatment are needed. Their conclusion was that based on this data, propylene glycol drench appeared to be the most rational ketosis therapy for cows.

A presentation by J. McArt et al., AABP 2011 Proceedings pg 155, also supported propylene glycol (PG) treatment. On 2 New York and 2 Wisconsin dairy farms, 1,823 cows tested 6 times between 3 to 16 DIM for beta-hydroxybutyrate (BHBA) resulted in 741 cows (40.6%) having subclinical ketosis defined by BHBA levels between 1.2 and 2.9 mM/L. 372 cows were assigned (whether randomly, randomized block, or how was not stated) to the PG treatment group and 369 cows to the control group ("no PG", presumably no treatment). Oral PG 300 ml (300 g) was given daily from when cows tested 1.2 to 2.9 mM/L BHBA until when they tested < 1.2 mM/L (or > 2.9 mM/L which was defined as clinical ketosis and they apparently were treated differently but otherwise left the trial). It seems that cows were studied for the first 30 DIM; at any rate some never were "cured" of ketosis. However, whether BHBA was tested only until 16 DIM for cows still defined as ketotic, or until 30 DIM was not clear (abstracts have word limits that make it difficult to fit in everything authors would usually like to say).

PG treated cows were 1.5 times more likely to "resolve" ketosis and 0.5 times as likely to develop clinical ketosis (both P < 0.009); raw numbers in each category were not shown. The 3 herds varied significantly in milk production difference/cow per d from 0.04 lb to 3.50 lb per d over the first 30 DIM in favor of PG treated cows. The authors concluded that PG treated cows had better outcomes in terms of subclinical or clinical ketosis, and would produce more milk than untreated cows in some herds.

A third presentation by J. Carrier et al., AABP 2011 Proceedings pg 157, was from a study of 4 Holstein herds, "mostly" cows 1 to 15 DIM screened for urinary acetoacetate > 15 mg/dl with no concurrent DA, defined as ketotic. Of 3,969 cows 561 (14%) were ketotic and randomly assigned to treated (279 cows) or

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untreated control (282 cows; an undefined number of control cows were treated for humane reasons “as a last resort”). Treatment was I.V. 500 ml 50% dextrose, 20 mg dexamethasone, 5 mg Vit B12, oral 500 ml (500 g) PG, with PG repeated on days 2 and 3. Cows in treated group “could be retreated” but details were not stated.

Mean number of ketosis “courses of therapy” per cow were 1.35 for treated, 0.15 for control group. Ketosis treatment had no significant effect on DA within 60 DIM (0.75 odds ratio), milk production (-2.2 lb per d), or time until pregnant (0.84 hazard ratio). However, with all cows studied until next calving or culled, 161 cows (58%) were culled from treated group and 127 (45%) culled from control group (1.4 hazard ratio, P = 0.005). The authors concluded, “standard ketosis therapy failed to prevent (and even increased) the negative impacts of ketosis in fresh cows”. This appears to support the concept that I.V. therapy may be detrimental vs. only oral PG therapy (300-500 g of propylene glycol daily for at least 3 d) for ketosis. More studies of long-term outcomes in cows treated or not treated for ketosis appear to be needed. This is especially true with larger herds needing standard treatment protocols.

Please let us know your comments and also suggestions for future topics. I can be reached at (435) 760-3731 (Cell), (435) 797-1899 M-W, (435) 797-7120 Th-F or David.Wilson@usu.edu.



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