Negative energy balance occurs in early lactation dairy cattle when the energy they need for milk production exceeds the amount available to them through feed intake. To produce more energy these cows break down fat stores to produce non-esterified fatty acids (NEFA) and ketone bodies (e.g. β-hydroxybutyrate, BHBA), which act as alternate fuel sources. An elevation of blood NEFA and BHBA is normal during this time for cows to adapt to the physiologic changes that occur from late gestation through early lactation, however cows that have a poor adaptive response to negative energy balance will produce excessive NEFA and BHBA.

This excessive production, particularly in cows with hyperketonemia (a blood BHBA concentration ≥1.2 mmol/L, i.e. subclinical and clinical ketosis), has detrimental effects on immune function, milk production and overall health. The incidence of hyperketonemia in many dairy herds during the first two weeks of lactation is reported to range from 40 to 60%. Given that 85 to 95% of hyperketonic cows do not show signs consistent with clinical ketosis, the health and production consequences of a poor transition into lactation are often unseen, as are the lost economic costs.

Current research by Dr. Jessica McArt and Dr. Daryl Nydam at Cornell University, in collaboration with Dr. Mike Overton at Elanco, describes the development of a model that estimates the economic costs behind hyperketonemia. The cost of hyperketonemia was modeled to look at the impact of this disease on milk production, treatment, and culling (referred to as the component cost), as well as the total cost of hyperketonemia, which is the sum of the component cost and additional disease-attributable costs associated with hyperketonemia (namely metritis and DA). Total cost of hyperketonemia was developed because cows with hyperketonemia are more likely to develop metritis and DA, thus those costs need to be included to fully understand the impact of hyperketonemia. Modeling was based on the incidence of disease in primiparous and multiparous animals, respectively, with the average estimated at $117. 34% of the component cost of hyperketonemia was due to future reproductive losses, 26% to death loss, 26% to future milk production losses, 8% to future culling losses, 3% to therapeutics, 2% to labor and 1% to diagnostics.

Given that future reproductive losses, future milk production losses and future culling losses are not tangible costs to producers, almost 70% of the cost of hyperketonemia is likely overlooked. The total cost per case of hyperketonemia, when including costs attributable to metritis and DA, was estimated at $375 and $256 for primiparous and multiparous animals, respectively, with the average estimated at $289. 41% of the total cost of hyperketonemia was due to the component cost of hyperketonemia, 33% to costs attributable to metritis and 26% to costs attributable to DA.

Given an average total cost of $289 per case of hyperketonemia, a herd with 1,000 calvings a year and a 30% incidence of hyperketonemia will lose approximately $90,000 due to the disease. This loss reinforces the importance of appropriate transition cow nutrition and management to decrease the incidence of hyperketonemia. For example, if the herd is able to improve management strategies and decrease the incidence of hyperketonemia in their herd to 15%, they will save almost $50,000 a year. Alternatively, if this producer makes $50,000 of changes to improve transition cow management and comfort that decrease the incidence of hyperketonemia to 15%, this cost will be recuperated in only one year.

Before determining the economic impact of hyperketonemia on a herd, it is first necessary to determine the amount of hyperketonemia in the herd. Easy, accurate and relatively inexpensive cow-side testing can be accomplished using the Precision Xtra Meter®. The meter can be purchased online for $20 to $40. BHBA test strips can be purchased through a...
Maximize insemination of cows in heat

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around 30 d after AI. More surprisingly was that only 38% of cows with a CL and that received PGF were inseminated in estrus because we carefully selected cows that should be responsive to the treatment.

We expected to detect at least 50% or more cows in estrus. It is unlikely that the method of estrus detection in the present study was responsible for the poor heat detection efficiency. The AAM system used in this study monitored cows continuously (24 h per day, 7 d per week) and used individual cow baseline data to trigger a heat event. Indeed, during the study period ~50% of the cows eligible to be inseminated before NPD were detected with increased activity by the AAM system. Although it cannot be ruled out that the AAM system failed to detect increased activity in some cows, at the moment we speculate that lack of estrus expression, rather than limitations to detect estrus resulted in poor heat detection after the PGF treatment.

Because the objective of any dairy farm is to get cows pregnant at the appropriate time in their lactation, the rate or speed at which cows became pregnant in our study was the most relevant outcome. We observed no statistical difference (P-value = 0.28) in the rate at which cows became pregnant up to 270 DIM (Figure 2). Median days to pregnancy which indicate the time when 50% of the cows became pregnant were 110 and 111 DIM for cows in the TRT and CON group, respectively.

Thus, the results of the present study support our hypothesis that more cows can be inseminated in heat after NPD, but does not support the hypothesis that the more complex TRT strategy would be superior to the simple and widely adopted one used for cows in the CON group. The relatively low proportion of cows with a CL at NPD and the poor heat expression of cows after the PGF injection contributed to the lack of difference between groups. Any potential benefit of inseminating cows in heat immediately after NPD in the TRT group was negated by the low proportion of cows that displayed estrus. Our results suggest that for a strategy aimed to maximize AI after heat detection, coupled with a delay to the beginning of the TAI protocol, the minimum proportion of cows to inseminate in heat to avoid detriment to the herd reproductive performance is ~30%. Our results also underscore the importance of immediately enrolling cows not AI in heat into a TAI program.

What this means to dairy producers is that they have the option to select a more aggressive resynchronization program that assures reinsemination of cows within 10 d of NPD but does not favor heat detection as in our control group. Or, they can adopt a strategy that maximizes insemination of cows in heat as in our treatment group.

It is imperative, however, to have in place a synchronization of ovulation protocol to submit cows to TAI immediately after the completion of the heat detection period. This is more relevant for dairy farms that due to biological limitations from the lactating dairy cow or the myriad of environmental and management factors that affect heat expression and detection cannot detect a high percentage of cows in heat after NPD. It is uncertain at the moment whether the use of a more complex, labor intensive and costly protocol such as the 5d-Ovsynch+P4 (requires two PGF injections and a P4 releasing device) protocol and presynchronization is necessary to maximize the fertility of cows not inseminated in estrus or not presenting a CL at the time of NPD.