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Fundamental Research in Relation to Minimizing Adverse Effects on Metabolic Disorders

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ABSTRACT: Subclinical ketosis is a common disease in lactating dairy cows with a lactational incidence rate above 40% in many herds. This condition is associated with increased clinical disease risk, reduced milk production, and impaired reproductive performance. On a herd basis, subclinical ketosis is much more costly than clinical ketosis. Prevention is achieved largely through effective dry cow programs that encompass both good nutrition and excellent cow management. However, certain additives are helpful in reducing subclinical ketosis. These include propylene glycol, rumen-protected choline, and, where approved for dairy cattle, ionophores. Effective monitoring programs are a critical component to managing subclinical ketosis.

Key words: Fundamental, Metabolic disorders, adverse effects

INTRODUCTION

Most periparturient abnormalities have some metabolic element as a component of the sufficient cause of clinical disease. The metabolic disturbance of milk fever can be measured through low serum calcium concentrations. Negative energy balance, fat mobilization, and subsequent elevations in ketone body concentrations play a contributing role in the expression of fatty liver syndrome, clinical ketosis, and abomasal displacement. A negative energy balance may also increase the risk of retained placenta, metritis, and mastitis through impaired immune function. A third category of metabolic disease in early lactation might include rumen acidosis, which is marked by low rumen pH. Thus, calcium homeostasis, energy balance, and rumen pH are important considerations for disease prevention in transition dairy cows (Goff and Horst, 1997).In general, subclinical disease incidence is far more common than clinical disease, frequently going unnoticed. It may be associated with significant clinical disease risks, impaired production, and reduced reproductive performance. Of the three major subclinical metabolic diseases, the most information exists for subclinical ketosis. It is associated with both losses in milk production and increased risk of periparturient disease. Prevention depends on several factors, including proper transition cow nutrition and management of body condition, and may be helped through the use of certain feed additives, such as niacin, propylene glycol, rumen = protected choline, and ionophores. It is commonly accepted that subclinical hypocalcemia is an important disease, but very little information is published on the impact of this problem on subsequent risk of disease or production loss. Subclinical rumen acidosis is also thought to be a major problem on many dairy farms, but it is difficult to measure, and very little controlled research exists for this syndrome. This article will focus on the importance, prevention aspects, and monitoring strategies for subclinical ketosis.

Is Subclinical Ketosis a Disease?

Elevated levels of circulating ketone bodies occur in early lactation in response to the homeorhetic drive to sustain high levels of milk production, at a time when dry matter intake is reduced (Baird, 1982). The three major ketone bodies are acetone, acetoacetate, and beta-hydroxybutyrate (BHBA). Subclinical ketosis is simply a condition marked by increased levels of circulating ketone bodies without the presence of the clinical signs of ketosis. Subclinical ketosis has been associated with increased risk of specific periparturient diseases (ketosis, displaced abomasum, metritis, and mastitis), decreased milk production, and impaired reproductive performance. If these impacts are true, then prevention or reduction in incidence should ameliorate some or all of the negative effects of this condition. Administration of a monensin controlled-release capsule three weeks precalving has decreased the incidence of subclinical ketosis, clinical ketosis, and displaced abomasum and improved milk production (particularly in cows and herds at increased risk of subclinical ketosis).

Cows in early lactation with subclinical ketosis had an increased risk of metritis four days later (Dohoo and Martin, 1984). However, most studies have identified ketosis to be a result rather than a cause of metritis. Cows having subclinical ketosis are at increased risk of subsequently developing clinical ketosis (Dohoo and Martin, 1984). The relationship between displaced abomasum and ketosis has been identified as bi-directional (Curtis *et al.,* 1985; Grohn *et al.,* 1989). That is, ketosis may be a cause of displacement, and abomasal displacement may lead to ketosis. Correa *et al.* (1993) found that ketosis increased the risk of abomasal displacement but not the reverse.

However, ketosis, as an inciting or predisposing cause of abomasal displacement, can be further supported by some recent Guelph research. Elevated BHBA concentrations above 1000 mmol/L increased the likelihood of abomasal displacement (Geishauser *et al.,* 1997). Cows with concentrations of BHBA at or above 1400 µmol/L in the first two weeks postcalving were three times more likely to subsequently develop either clinical ketosis or abomasal displacement (Duffield, 1997).

Two studies have found a relationship between the diagnosis of ketosis prior to identifying mastitis (Dohoo and Martin, 1984; Syvajarvi *et al.,* 1986). Mastitis increased the risk of ketosis in Finnish Ayrshires (Grohn *et al.,* 1989). Hyperketonemic cows with BHBA blood levels above 1400 µmol/L were found to suffer a more severe experimental mastitis than normal cows (Kremer *et al.,* 1993). There may be some important immune function implications associated with decreased energy balance and subclinical ketosis. Recently, two separate studies have identified subclinical ketosis as a risk factor for the subsequent occurrence of clinical mastitis (Leslie *et al.,* 2000).

Impact on milk production

In general, there is consensus that a negative association between hyperketonemia and milk production exists. In one study, the loss of production associated with a positive milk ketone test was 1.0 to 1.4 kg/day of milk for a lactation (Dohoo and Martin, 1984). Test day milk production was negatively correlated with milk acetone levels in several Scandinavian projects (Andersson and Emanuelson, 1985; Gustafsson *et al.,* 1993; Steen *et al.,* 1996). Kauppinen (1984) reported that subclinicallyketotic cows had significantly higher annual milk yields than nonketotic cows. Herdt *et al.* (1981) found higher levels of BHBA in higher-producing cows, but individual milk tests were not collected on the same day but preceded blood measurement for BHBA. It is possible that higher milk yields put cows at increased risk of developing subclinical ketosis. Increased levels of milk production may be associated with increased fat mobilization and a greater risk of hyperketonemia.

Effect on milk components

Milk fat and milk protein are significantly altered in hyperketonemia. Milk fat percentage was increased in subclinicallyketotic cows (Miettenen, 1994; Miettenen and Setala, 1993). The association between milk fat and hyperketonemia is, presumably, because of increased availability of BHBA and fatty acids for milk fat synthesis. It is unclear whether increased levels of circulating ketones cause increased milk fat or whether cows that are prone to higher milk fat yields are more susceptible to subclinical ketosis. Milk protein percentage has been reported to be lower in cows with subclinical ketosis (Miettenen, 1994; Miettenen and Setala, 1993). This may be the result of a reduced energy supply since milk protein percentage is positively associated with net energy balance. Recently, we have identified an impact of subclinical ketosis on milk fat percentage at first DHI test postcalving only but a reduction in milk protein percentage for the first three DHI tests.

Impact on reproductive performance

Increasing the degree of negative energy balance in early lactation has been shown to increase the interval from calving to first ovulation (Butler and Smith, 1989). Butler and Smith (1989) suggested that cows with a longer interval from calving to first ovulation experience a decrease in pregnancy at first service because conception risk is related to the number of ovulatory cycles that occur prior to insemination (Stevenson and Call, 1983; Whitmore et al., 1974). Sincehyperketonemia is a symptom of a disturbed energy metabolism, many authors have investigated the relationship between subclinical ketosis and reproductive performance. No effect of either subclinical or clinical ketosis on individual cow fertility was found in two studies (Andersson and Emanuelson, 1985; Kaupinnen, 1984). However, significant correlations between the herd prevalence of hyperketonemia and herd mean intervals from both calving to first service and calving to last service have been noted (Andersson and Emanuelson, 1985). A link between subclinical ketosis and the increased incidence of cystic ovaries has also been reported (Andersson and Emanuelson, 1985; Dohoo and Martin, 1984). Miettenen and Setala (1993) found an increased interval from calving to conception in cows with high milk and fat yields. The associations between fertility and increased fat and milk yields do not necessarily imply a relationship between impaired fertility and hyperketonemia. The duration of either clinical or subclinical ketosis may be too short to exert a negative effect on calving interval.

However, Whitaker *et al.* (1993) found cows with a better energy status at 14 days postpartum had a reduced interval from calving to the onset of cyclicity and fewer services per conception. No effect was observed when energy status was evaluated at 21 days postpartum or at first service. This study was only conducted on 24 cows within one herd. In a much larger dataset, Cook *et al.* (2001) reported significantly longer calving to conception intervals and higher culling rates in cows that had high milk acetone concentrations in early lactation. Initial screening of our 1,010 cow dataset from 1995 indicates a significant reduction in first service conception rate for cows identified to be subclinicallyketotic in the second week postpartum.

Cost of subclinical ketosis

When negative impacts of milk production losses, increased risk of disease, and reduced reproductive performance are considered, the cost of one cow with subclinical ketosis is estimated to be $78 U.S. (Geishauser *et al.,* 2001). This number will vary depending on several variables, including the value assigned for milk, impaired reproduction, and metabolic disease. Regardless, the individual disease value for subclinical ketosis is less than clinical disease. However, because the subclinical form is more prevalent, the cost at the herd level is much higher. For example, if an average dairy herd has an incidence rate for clinical ketosis of 5% and the disease costs $145, a 100-cow dairy herd would have a cost of clinical ketosis of $725 in a year, whereas an average 100-cow dairy would have a subclinical ketosis incidence of 41%, with an annual cost of $3,198.

How common is subclinical ketosis?

Before a program is instituted, the veterinarian and farm manager need to know what the average incidence of subclinical ketosis is for the herd so that a reasonable and achievable target can be set. In a recent trial conducted at Guelph, the median incidence of subclinical ketosis (BHBA > 1400 µmol/L) in untreated cows was 41% for the first nine weeks of lactation (Duffield *et al.,* 1998).

This was roughly equivalent to two cows identified as subclinicallyketotic per10 cows examined in each of the first and the second weeks postcalving. The range across 25 herds for the total nine weeks was 8 to 80%. The four highest herds had incidence rates above 65% and also had the largest milk production response to prophylactic treatment.

PREVENTION

General guidelines

Since ketosis occurs in early lactation, recommendations for prevention have focused on the nutritional management of the dry and transition cow. Detailed recommendations for nutrition during the dry period can be found elsewhere (Oetzel, 1998). It is a common recommendation to divide the dry period into two feeding groups: far-off and close-up (Radostits *et al*, 1994). Typically, far-off diets follow NRC (2001) guidelines for dry cows. The close-up diet is usually balanced according to recommendations that are halfway between those for the dry cow and those for the early lactation cow and should be fed starting at least three weeks before expected calving (Oetzel, 1998). The goals of the transition diet (specifically designed to prevent subclinical ketosis) are to maximize dry matter intake (DMI) to provide adequate energy density (Oetzel, 1998). Avoidance of ketogenic feedstuffs (Tveit *et al.,* 1992) and increased frequency of feeding concentrates (Andersson, 1988; Gustafsson *et al.,* 1993) have been advocated as preventive measures against subclinical ketosis. The reduction of overconditioning cows in late lactation and the early dry period, as well as lead feeding with concentrates about three weeks prior to calving, have also been suggested as aids in prophylaxis (Andersson, 1988; Lean *et al,* 1991). Maximizing DMI and maintenance of a consistent intake through the last three weeks prior to calving is likely the hallmark of a successful transition cow program. Recent work at Guelph (Tera Osborne, MS candidate, personal communication) indicates that a DMI of less than 12 kg/day per cow in the last three weeks prior to calving substantially increases the subsequent risk of subclinical ketosis (Odds Ratio 5.7, $P < 0.05$). Achieving group DMI targets above an average of 12 kg/day per cow is possible, and based on the above finding, should be a goal for the close-up group. More important than ration formulation and ration ingredients, close attention should be paid to cow comfort and environmental issues. These factors include, but are not limited to, adequate pen space or stall space per cow, adequate feed bunk space, sufficient and comfortable bedding, adequate water supply, and minimization of heat stress.

Feed additives

In addition to good nutrition, certain feed additives have been found beneficial in reducing subclinical ketosis, when administered prophylactically.

Niacin fed prior to calving at the rate of 3 to 6 g/day may be helpful in reducing blood levels of BHBA (Dufva *et al.,* 1983; Fronk and Schultz, 1979). Propylene glycol has been used successfully for the prevention of subclinical ketosis (Emery *et al.,* 1964; Sauer *et al.,* 1973). Treatment of cows for eight weeks starting at calving with either 3 or 6% propylene glycol in a concentrate mixture significantly reduced the incidence of positive milk ketone tests (Fisher *et al.,* 1973). Precalving oral treatment with 300 g/day of propylene glycol for 10 days lowered serum non-esterified fatty acids (NEFA) concentrations and improved some measures of reproductive performance in one study (Formigoni *et al.,* 1996). A dose of propylene glycol of 1 L/day as an oral drench for nine days prior to calving decreased BHBA and NEFA and increased glucose concentrations (Struder *et al.,* 1993). It appears that a bolus of propylene glycol is necessary for maximum effect since mixing in a total mixed ration is not as efficacious as either an oral drench or when mixed with a small quantity of grain (Christenson *et al.,* 1995). Schultz (1958) reported that sodium propionate could be given to prevent clinical ketosis in dairy cattle. Propylene glycol requires repeated daily oral administration, and sodium propionate may reduce feed intake (Sauer *et al.,* 1989). Ionophores have been proposed as potential prophylactic agents for reducing hyperketonemia (Lean e*t al.,* 1991; Tyler *et al.,* 1992). In contrast to propylene glycol and sodium propionate, ionophores are relatively inexpensive and much easier to administer. Rumen-protected choline reduced liver triglycerides and increased liver glycogen (Piepenbrink and Overton, 2000). More recently, preliminary Guelph research shows reductions in NEFA and BHBA concentrations postcalving in cows receiving rumen-protected choline (Reashure™; Balchem Encapsulates, Slate Hill, NY) during transition compared to control cows. The cost benefit of this prevention tool needs to be investigated.

Ionophores

The gluconeogenic potential of monensin has attracted researchers to investigate its possible role as an antiketogenic agent in dairy cattle. Rogers and Hope-Cawdery (1980) first described the beneficial effects of monensin for reducing the incidence of ketosis in a herd with a clinical ketosis problem. The antiketogenic properties of monensin were later investigated in a Canadian trial involving two levels of monensin and three groups of 12 Holstein cows (Sauer *et al,* 1989). Monensin included at 30 g/ton of total ration (high group) decreased the incidence of subclinical ketosis and significantly reduced blood BHBA concentrations in the first three weeks postpartum (Sauer et al., 1989). The incidence of subclinical ketosis, defined as total blood ketones > 9 mg/100 ml (900 µmol/L), was reduced by 50%, and blood BHBA concentrations were reduced by 40% for the high monensin group. Based on the average feed intakes observed in this trial, the low monensin group received approximately 208 mg/day of monensin and the high group 399 mg/day. The monensin treatment commencing at two to four weeks prior to calving reduced serum BHBA and NEFA in lactating dairy cows during the first 28 days postpartum when monensin was fed at 300 or 450 mg/day, but not by a daily dose of 150 mg/day of monensin (Thomas *et al.,* 1993). Serum glucose was not influenced by monensin feeding. Australian cows treated with a monensin controlled-release capsule (CRC) during the first week postcalving had significantly lower plasma BHBA concentrations and tended to have higher glucose concentrations than controls (Abe *et al.,* 1994). A CRC that delivers 335 mg/day of monensin sodium for 95 days reduced the incidence of subclinical ketosis by 50% and also decreased the duration of the condition when it was administered three weeks prior to expected calving (Duffield *et al.,* 1998).

The most closely linked diseases occurring subsequent to subclinical ketosis are displaced abomasum and clinical ketosis. Estimates of milk production loss range from 300 to 450 kg (660 to 990 lb) for a lactation (Dohoo and Martin, 1984; Gustafson *et al.,* 1993). These losses must be weighed against the cost of any prophylactic measure.

Administration of a monensin CRC precalving reduced the incidence of clinical ketosis by 50%, abomasal displacement by 40%, and multiple illness by 40% (more than one disease) (Duffield et al., 1999b). The milk production response depended on body condition and was 0.85 kg/ day (1.9 lb/day) at peak lactation in cows with a precalving body condition score (BCS) of 3.25 to 3.75 and was 1.2 kg/day (2.6 lb/day) for the first 90 days of lactation in fat cows (BCS³4.0)(Duffield et al., 1999a). No milk production response was noted in thin cows, presumably because they had the lowest BHBA concentrations and were at decreased risk of subclinical ketosis. A subsequent Canadian study conducted in 45 dairy herds confirmed that monensin CRC reduces the incidence of displaced abomasum (Duffield *et al.,* 2002). A pooled summary of the two Canadian projects showed that monensin CRC reduced the incidence of displaced abomasum and clinical ketosis by 40% each. In addition to the impact of monensin on displaced abomasum and clinical ketosis, pooled anlaysis of the two Canadian CRC studies showed that the incidence of retained placenta tended to be lowered by25% in monensin-treated cows (P = 0.09). Monensin is currently not approved in the United States for use ketosis prevention or treatment in lactating dairy cows.

MONITORING SUBCLINICAL KETOSIS

When do I test cows?

By most definitions, the theoretical testing period for transition cows would extend from three weeks prior to calving until three weeks after calving. Practically, however, the most important time periods are during the last week prior to calving and within the first two weeks after calving.

Precalving

It is unusual for cows to develop subclinical ketosis precalving because the etiology of the condition depends on the homeorhetic drive for milk production. However, cows in an energy deficit precalving will start mobilizing energy reserves in the final week before parturition. This can be measured via serum or plasma NEFA. The challenge for this precalving sample is predicting when the animal is going to calve. In most cases, a serum bank needs to be established and then samples are submitted retrospectively once the actual calving date is known.

Post calving

A ketone testing program should commence after calving. The primary risk period for subclinical ketosis is the first month of calving. Our work at Guelph has indicated that the first two weeks postcalving is the time of peak incidence. In addition, the median days to diagnosis of clinical ketosis and displaced abomasum was 11 days. Thus, in order to try to prevent subclinical disease from becoming clinical disease (if that is possible), cows must be identified early. For these reasons, a subclinical ketosis monitoring program should focus on the first two to three weeks of lactation.

What Test Do I Use?

NEFA. This test should only be used precalving on samples obtained within one week of parturition. Unfortunately, these restrictions make the utility of this test limited. However, it may be useful in certain situations, such as a herd investigation or an intervention follow-up. The data for this variable are frequently right skewed, and thus averages can be very misleading. One suggested threshold is 0.5 units/L. In recent work, cows within one week of calving with serum NEFA above this threshold were at a 3.5 times greater risk of subsequently developing a displaced abomasum. Whole herd interpretation is best made by calculating a proportion of cows above a threshold value; however, at this point, there are not a lot of good data on an appropriate goal for this parameter. In a multi-herd 1,060 cow study near Guelph, 30% of cows were above 0.5 U/L during the last week prior to calving.

Serum BHBA. In contrast to NEFA, serum BHBA should only be used postcalving. The first two weeks are the primary risk period for subclinical ketosis, defined by a serum concentration of 1400 umol/L BHBA or greater. Although BHBA is the most stable of the ketones, it is the most subject to variation associated with feed intake; thus, all samples on a given farm should always be taken at the same time of day. In addition, hemolysis is known to artificially elevate values; therefore, hemolyzed samples should be avoided. Other disadvantages of serum BHBA are the cost (approximately $5.00 per sample) and the laboratory turnaround time (minimum 24 hours). However, all things considered, serum BHBA analysis is the gold standard from which to compare cowside tests. A reasonable goal is to have less than two cows per 10 with BHBA above 1400 umol/L in the first two weeks postcalving.

Milk ketone tests. Most milk ketone tests measure acetone and acetoacetate through a chemical reaction with nitroprusside which causes a color change from white to either pink or purple. These tests in general are poorly sensitive in milk (< 40%) but highly specific (> 90%). One exception is the milk ketone test that measures BHBA. It is marketed in Europe as "Ketolac BHBA," in Japan as "Sanketopaper," and in Canada as "Keto-Test." This test has a much higher sensitivity in milk (>60%) and reasonably good specificity (> 70%, up to 90%). This is a semi-quantitative test that allows choosing a lower threshold for screening to increase sensitivity and a higher threshold for diagnosis to increase specificity.

Urine ketone tests. The urine ketone tests are based on the same nitroprusside reaction as the milk powder ketone tests. These tests are highly sensitive (approaching 100%) but are poorly specific. Thus, they are great tests for ruling out subclinical ketosis with a negative test result. However, their use overestimates a subclinical ketosis problem because of a high probability of false positive reactions. If the urine test was used to evaluate the goal of less than two cows per 10 with BHBA above1400 umol/L in the first two weeks postcalving, an adjustment of the goal to less than 5 cows per 10 with positive urineketone tests would be required (Table 1). More work needs to be done to fully assess the utility of urine ketone tests.

Table 1. Use of cowside ketone tests in screening programs for identifying subclinical ketosis.

Test	20% Prevalence			40% Prevalence			60% Prevalence		
	PV1 +ve	PV2 -ve	AP3	PV +ve	PV -ve	AP	PV +ve	PV -ve	AP
Keto-Test® using 100 µmmol/L	62%	93%	23%	81%	83%	35%	91%	68%	48%
Ketocheck™ (milk)	90%	86%	8%	96%	70%	16%	98%	51%	23%
Urine	38%	100%	53%	62%	100%	65%	78%	100%	76%

1PV +ve: Predictive value of a positive test result.
2PV –ve: Predictive value of a negative test result.

Selection and Interpretation of Cowside Tests

It is most likely that in screening a group of fresh cows, there would be two possible actions resulting from a test. One action might be to treat positive animals with the goal to prevent subsequent development of clinical disease. In this case, a high predictive value of a positive test is desired so that normal animals are not unnecessarily treated. The second action might be to compare the percentage of positive reactors to a goal for determining the effectiveness of either the transition ration or some prophylactic measure in reducing the incidence of subclinical ketosis. In this situation, the apparent prevalence is the parameter that actually would be used. Note from Table 1 that the urine ketone test would substantially overestimate the prevalence of subclinical ketosis, while the Ketocheck™ test would grossly underestimate the prevalence. This does not preclude these tests from being used. However, the impact of the inherent sensitivity and specificity of the test must be remembered when establishing goals and intervention thresholds.

Herd Disease Records

Herd records (computerized or paper) are important tools for monitoring the incidence of periparturient disease. Producers should set goals for minimizing the incidence of metabolic disease. Herd consultants should periodically review herd performance relative to the goals. In addition, intervention levels should also be considered. Several diseases are associated with increasing age, and this must be taken into account when assessing herd performance. For example, in monitoring and comparing herd incidence of milk fever and clinical ketosis, it is important to stratify this by parity. A high proportion of first lactation animals will give a herd a much lower incidence of milk fever and clinical ketosis since risk increases with age.

Can herd incidence of certain diseases be used to decide whether a herd has a problem with subclinical ketosis? Herd level analysis of our 1995/1996 dataset involving 25 dairy herds indicates that the herd incidence of displaced abomasum is positively associated with the probability of a herd having a high incidence (> 20% in the first two weeks of lactation) of subclinical ketosis. The only other predictive variable was precalving body condition score (higher average increased risk). If greater than 10% of the herd had a BCS > 4.0 at three weeks precalving, that herd was extremely likely to have a problem with subclinical ketosis.

DHI Test Day Data

Since milk fat and milk protein percentages are altered in sublinical ketosis, these parameters have been investigated for their utility in defining subclinical ketosis. Among all protein and fat parameters, a protein-to-fat ratio of < 0.75 was the best test for diagnosing subclinical ketosis, at the cow level, in a Canadian study (Duffield *et al.*, 1997). However, the protein-to-fat ratio was not a good test overall, having a sensitivity of 58% and a specificity of 69%. Using data from a 25-herd study conducted in Guelph in 1995, the median cumulative herd incidence of subclinical ketosis was 41% in the first two months postcalving. Summary data for each herd from each cow's first DHI test postcalving were used to assess the protein-to-fat ratio as a test at the herd level for classifying a herd as a high or low incidence herd for subclinical ketosis. A herd mean protein-to-fat ratio of < 0.78 yielded a sensitivity of 69% and a specificity of 75% for identifying herds with subclinical ketosis problems. Further, if more than 40% of cows in the herd at first DHI test had a protein-to-fat ratio of less than or equal to 0.75, those herds were likely to be problem herds. This test had a sensitivity of 69% and a specificity of 83%. Although more work needs to be done on herd-level indicators of subclinical ketosis, herd-level protein-to-fat ratios appear to be better indicators of herd-level issues than individual cow protein-to-fat ratios are of identifying cows with subclinical ketosis problems.

A recent study was conducted through the Ontario DHI testing facility to evaluate the utility of milk acetone measurements for diagnosing subclinical ketosis. At the cow level, test day milk acetone values were not found to be useful in identifying cows at risk for developing clinical metabolic disease (displaced abomasum or ketosis) or clinical lameness. This is not an issue with the acetone test methodology but more likely a problem with the timing of the milk sample collection relative to the occurrence of the disease event.

The highest prevalence of subclinical ketosis occurs within two weeks of calving. Since most of our DHI testing programs have an interval of 30 to 45 days and cows less than five days in milk on test day are not sampled, the probability of testing all cows within two weeks of calving is low.

Therefore, from an implementation standpoint, the testing of routine DHI samples for the purpose of identifying cows at risk of subclinical or clinical disease is inefficient.

CONCLUSIONS

Subclinical ketosis is an important and common disease in lactating dairy cows. Prevention depends largely on effective dry cow nutrition and management. However, certain feed additives, such as ionophores and rumen-protected choline, may be beneficial. Given the cost of subclinical ketosis, the fact that it is a common problem in early lactation, and the strong association with clinical disease, monitoring programs for subclinical ketosis during the first few weeks of lactation may be warranted. There are several cowside tests for subclinical ketosis available; however, all of the current tests have their strengths and weaknesses. The design and frequency of a subclinical ketosis monitoring program will depend on the purpose of the program and the frequency of disease within the herd.

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