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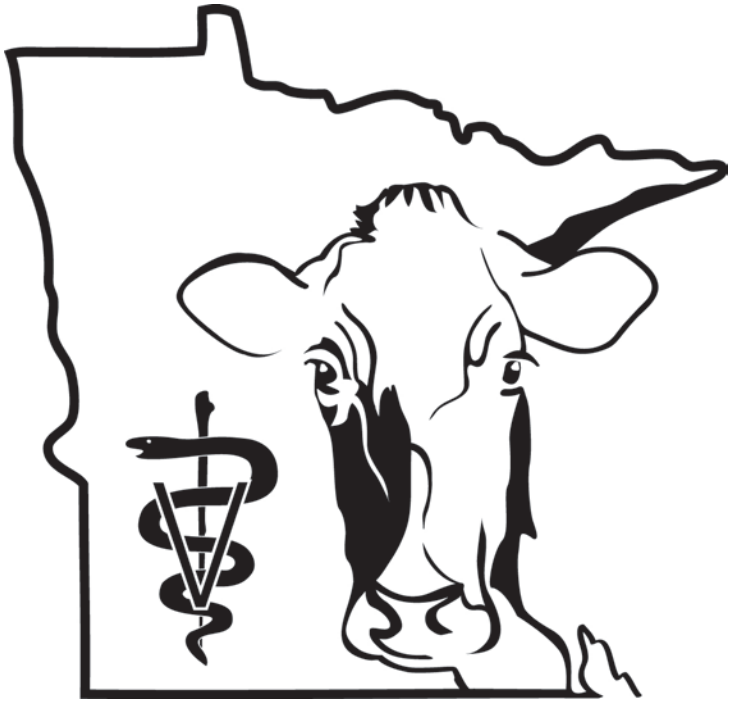

ST. PAUL, MINNESOTA
UNITED STATES OF MINNESOTA

IMPACT, PREVENTION, AND MONITORING OF SUBCLINICAL KETOSIS IN TRANSITION DAIRY COWS

Todd Duffield, DVM, DVSc, Assistant Professor,
Department of Population Medicine,
Ontario Veterinary College,
University of Guelph
tduffiel@uoguelph.ca

Introduction:

Subclinical ketosis is an important and common condition of early lactation dairy cattle. 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, management of body condition, and the use of certain feed additives such as niacin, propylene glycol, and ionophores.

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 (BHB). Ketone bodies represent an integral part of ruminant intermediary metabolism and they provide an important form of energy to peripheral tissues when carbohydrate levels are reduced. A baseline level of circulating ketone bodies is therefore normal in the ruminant. 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, mastitis), decreased milk production, and impaired reproductive performance. If this is 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 3 weeks precalving, has decreased the incidence of subclinical ketosis, decreased the incidence of clinical ketosis, and displaced abomasums, and improved milk production (particularly in cows and herds at increased risk of subclinical ketosis).

Association with periparturient disease

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 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 BHB concentrations above 1000 μmol/L increased the likelihood of abomasal displacement (Geishauser et al, 1997). Cows with

concentrations of BHB at or above 1400 μmol/L in the first two weeks post calving 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 et al, 1984; Syvajarvi et al, 1986). Mastitis increased the risk of ketosis in Finnish Ayrshires (Grohn et al, 1989). Hyperketonemic cows with BHB 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.

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 of milk per day for a lactation (Dohoo and Martin, 1984). Test day milk production was negatively correlated with milk acetone levels in several Scandinavian projects (Andersson et al, 1985; Gustafsson et al, 1993; Steen et al, 1996). Kauppinen (1984) reported that subclinically ketotic cows had significantly higher annual milk yields than nonketotic cows. Herdt et al (1981) found higher levels of BHB in higher producing cows; but individual milk tests preceded blood measurement for BHB. 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. Studies measuring both ketone levels and milk production on the same day should identify any potential negative impacts of hyperketonemia on milk output. Trials where milk measurements precede ketone evaluation could identify high producing cows that later become subclinically ketotic. Therefore, due to the difference in timing of ketone measurement and milk yield determination, a positive correlation between milk production and hyperketonemia would be observed.

Effect on milk components

Milk fat and milk protein are significantly altered in hyperketonemia. Milk fat percentage was increased in subclinically ketotic cows (Miettenen, 1994; Miettenen and Setala, 1993). The association between milk fat and hyperketonemia is, presumably, because of increased availability of BHB and fatty acids for milk fat synthesis. It is unclear whether increased levels of circulating ketones cause increased milk fat, or if cows that are prone to higher milk fat yields are more susceptible to subclinical ketosis. Milk protein percent 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 percent is positively associated with net energy balance.

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 rate at first service because conception rate is related to the number of ovulatory cycles that occur prior to insemination (Stevenson and Call, 1983; Whitmore et al, 1974). Since hyperketonemia 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 yield and high fat yield. The associations between fertility and increased fat and milk yield 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. 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. It is not clear if hyperketonemia truly has a negative effect on reproduction, or whether hyperketonemia and impaired reproductive performance are both simply a consequence of a prolonged negative energy balance associated with increased milk production.

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 frequent, 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 $145.00, a 100-cow dairy 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 $3198.

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 (BHB $\geq$ 1400 µmol/L) in untreated cows was 41% for the first 9 weeks of lactation (Duffield et al, 1998). This was roughly equivalent to 2 cows identified as subclinically ketotic per 10 cows examined in each of the first and the second week post-calving. The range across 25 herds for the total 9 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. An appropriate monitoring program would assess cows in the first 2 weeks of lactation, since this is the time peak incidence occurs. Also, identification of positive cows earlier in lactation might allow time for prevention of subsequent clinical disease.

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 guidelines for dry cows. The close-up diet is usually balanced according to recommendations that are halfway between the dry cow and 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 and 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).

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 grams per day may be helpful in reducing blood levels of beta-hydroxybutyrate (BHB) (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 8 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 of propylene glycol per day 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 1L per day as an oral drench for 9 days prior to calving decreased BHB, 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 et al, 1991; Tyler et al, 1992). In contrast to propylene glycol and sodium propionate, ionophores are relatively inexpensive and much easier to administer.

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 grams per ton of total ration (high group), decreased the incidence of subclinical ketosis and significantly reduced blood BHB levels 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 BHB levels 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 of monensin per day and the high group 399 mg per day. Monensin treatment commencing at two to four weeks prior to calving reduced serum BHB 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 during the first week postcalving had significantly lower plasma BHB levels and tended to have higher glucose concentrations than controls (Abe et al, 1994). A controlled release capsule that delivers 335 mg of monensin sodium per day for 95 days reduced the incidence of subclinical ketosis by 50% and also decreased the duration of the condition when it was administered 3 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 for a lactation (Dohoo and Martin, 1984; Gustafsson et al, 1993). These losses must be weighed against the cost of any prophylactic measure. Administration of a monensin controlled release capsule 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 at peak lactation in cows with a precalving BCS of 3.25 to 3.75, and was 1.2 kg/day for the first 90 days of lactation in fat cows (BCS ≥ 4.0)(Duffield et al, 1999a). No milk production response was noted in thin cows presumably because they had the lowest BHB 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 DA and clinical ketosis, pooled anlaysis of the two Canadian CRC studies showed that the incidence of retained placenta tended to be lowered by 25% in monensin treated cows (P=0.09). Monensin is currently not approved in the United States for use in lactating dairy cows.

Economics of Prevention

An economic assessment of the health improvement imparted by the monensin CRC will depend on several variables including the disease incidence for the herd, estimated cost of disease, and cost of the capsule. From the untreated groups in both studies combined, the mean incidence of abomasal displacement, clinical ketosis, and RP were 5.9%, 2.5%, and 10.3% respectively. Using estimates of disease costs of $340 for DA, $145 for clinical ketosis and $285 for RP (Guard, 1994), the approximate health savings for 100 cows per year using the monensin CRC precalving would be $1681. The estimated cost of the capsule would be $10 per cow, thus the benefit cost ratio for health improvement is about 1.7:1. This does not include any additional benefit of monensin CRC for improving milk production, which is expected to range from no benefit in thin cows, to 0.85 kg per day at 2nd DHI test in cows in good body condition, up to

over 1 kg per day for the first 90 days of lactation in fat cows (Duffield et al, 1999). Two farm scenarios estimating the total impact of the monensin CRC in herds with low and average periparturient disease incidences yielded 1.5:1 and 3:1 benefit cost ratios (Duffield, 2000).

Monitoring subclinical ketosis

Given the strong association between DA and subclinical ketosis, monitoring programs designed for subclinical ketosis detection could be structured to predict the occurrence of DA.

When do I test cows?

By most definitions, the theoretical testing period for transition cows would extend from 3 weeks prior to calving until 3 weeks after calving. Practically however, the most important time periods are: during the last week prior to calving, at calving itself, and within the first 2 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 non esterified fatty acids (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.

At Calving

Cows at calving are an attractive group for monitoring because they must be handled for the collection of colostrum. Preliminary work at Guelph has suggested that glucose monitoring on the day of calving could be useful. With a small dataset, it appeared that those cows that went on to develop displaced abomasums post calving failed to have a normal stress-induced glucose spike on the day of calving. By contrast, those cows that went on to develop mastitis within the next month after calving appeared to have an exaggerated glucose spike. Considerably more work is required on this before any "at calving" program can be recommended.

Postcalving

A ketone testing program would begin after calving. The primary risk period for subclinical ketosis is the first month of calving. Our work at Guelph has indicated that the first 2 weeks postcalving is the time of peak incidence. In addition, the median days to diagnosis of clinical ketosis and displaced abomasum were 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 2 to 3 weeks of lactation.

What test do I use?
NEFA

This test should only be used precalving on samples obtained within 1 week of parturition. Unfortunately these restrictions make the utility of this test limited. However, it may serve useful in certain situations. The data for this variable is frequently right skewed and thus means can be very misleading. One suggested threshold is 0.5 units/L. In recent work, cows within 1 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 is not a lot of good data on an appropriate goal for this parameter.

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 is the cost (approx $5.00 per sample) and the laboratory turn around time (minimum 24 hours). However, all things considered, serum BHBA analysis is considered the gold standard from which to compare cowside tests. A reasonable goal is to have less than 2 cows per 10 with BHBA above 1400 umol/L in the first 2 weeks post-calving.

Milk Ketone Tests

Most milk ketone tests measure acetone and acetoacetate through a chemical reaction with nitroprusside which causes a colour 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 BHB. It is marketed in Europe as "Ketolac BHB", in Japan as "Sanketopaper", and hopefully soon in North America 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 2 cows per 10 with BHBA above 1400 umol/L in the first 2 weeks post-calving, an adjustment of the goal to less than 5 cows per 10 with positive urine ketone tests would be required (see table 1). Recent work suggests that at least some urine test strips may have higher specificity performance relative to

urine tablets (Geishauser, personal communication). More work needs to be done to fully assess the utility of urine ketone tests.

Selection and Interpretation of Cowside Tests

The poor specificity and relative inconvenience in sample retrieval has limited the utility of urine ketone evaluations in field studies. Milk by contrast, is relatively easy to obtain and the high correlation between milk and blood ketones, as well as the inexpensive cost of nitroprusside powder has allowed these tests to gain popularity. Choice of an appropriate test is difficult. Ketolac® has been shown to be a very useful test; however, it is presently unavailable in North America and in Europe it is considerably more expensive than the currently available ketone nitroprusside tests. The milk ketone nitroprusside tests have excellent specificity, so there is little danger of false positives. However, these tests lack sensitivity limiting their utility as screening tests. The nitroprusside tests in urine provide almost a zero probability of a false negative (ideal for screening) but there is close to a 50% chance of a false positive. The prevalence of disease will also alter the predictive values of the various tests. Predictive values for three cowside tests used within hypothetical herds having 20%, 40%, and 60% prevalence of subclinical ketosis in the first 2 weeks of lactation are presented in Table 1. The Ketolac® test performs well across all prevalence levels. The Ketocheck™ test tends to give better predictive values at lower prevalences and the urine ketone test yields higher predictive values at higher prevalences. The predictive value should be compared to the prior prevalence of disease (true prevalence) and the difference between the two numbers is the information gained from the test. For example, at 20% prevalence, there is a 20% chance that any animal selected prior to testing would have subclinical ketosis. Using the urine ketone test, a positive result would gain only 18% (PV +ve: 38%), whereas the Ketocheck™ test would gain 70%.

It is most likely that in screening a group of fresh cows, there would be two possible actions resulting from the 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 percent 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.

Table 1. Use of Cowside Ketone Tests in Screening Programs for Identifying Subclinical Ketosis.

Test	20% Prevalence			40% Prevalence			60% Prevalence		
	PV +ve	PV -ve	Apparent	PV +ve	PV -ve	Apparent	PV +ve	PV -ve	Apparent
Ketolac® using 100 μmol/L	62%	93%	23%	81%	83%	35%	91%	68%	48%
Ketocheck™ 1400 μmol/L BHB	90%	86%	8%	96%	70%	16%	98%	51%	23%
Utrecht Urine Ketone 1400 μmol/L BHB	38%	100%	53%	62%	100%	65%	78%	100%	76%

PV +ve: Predictive Value of a positive test result.
PV –ve: Predictive Value of a negative test result.

Herd Disease Records

Herd records (computerized or paper) are important tools for monitoring the incidence of periparturient disease. Producers should set goals for the 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 2 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 $\geq$ 4.0 at 3 wks 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 $\leq$ 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 cows first DHI test postcalving was 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 1st 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 (DA 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 5 DIM 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

Given the cost of subclinical ketosis, the fact it is a common problem in early lactation, and the strong association with DA, 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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