

Ketosis

Economics

Owners, herdsman and veterinarians are all too familiar with the fresh cow that becomes ketotic and develops a displaced abomasum. Like many diseases, clinical ketosis is just the tip of the iceberg. Sub clinical ketosis (SCK) is a far more common and frequently overlooked disorder. In a survey done on Northeast dairies, 40% of the herds had greater than 15% of their cows sick with SCK or clinical Ketosis.

Impact of Subclinical Ketosis on 1st Service Conception Rates in 781 Holstein cows from 95/96 Data

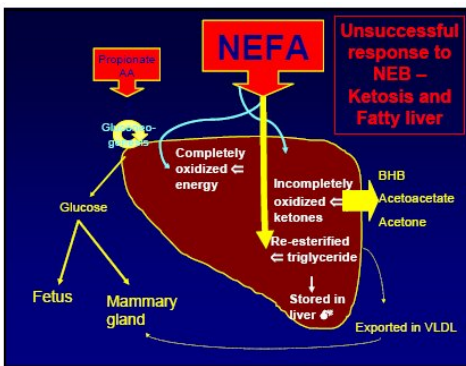
	SCK	Normal	P-value
Week 1	29%	36%	0.167
Week 2	23%	37%	0.001
All (1,2,3,6,9)	29%	38%	0.01

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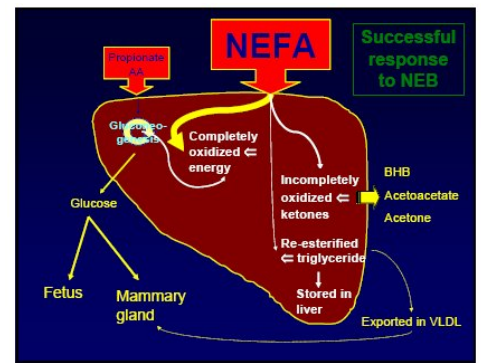
SCK has been directly linked to many post partum disorders. Retained placenta, metritis and mastitis incidence are all increased secondary to SCK. Because SCK causes an increase in periparturient disease and decreases milk production, it significantly increases the risk of culling in early lactation. SCK has also been correlated with decreased first service conception rates. A 9% reduction in 1st service CR was demonstrated in the study summarized here.

Physiology

Ketosis is defined as a condition characterized by elevated concentrations of ketone bodies – Acetoacetic acid (AcAc), Betahydroxybutyric acid (BHBA) & Acetone (Ac) – in body tissues and fluids. Clinically, this results in a gradual loss of appetite and a corresponding drop in milk production.



During the late prepartum and early postpartum periods, dry matter intake (DMI) may not meet energy demands of the cow. As a result, the cow is in negative energy balance (NEB). The physiologic response to NEB is to mobilize fat from adipose tissue stores and hydrolyze that fat to free fatty acids (FFA) and nonesterified fatty acids (NEFA). Ultimately, these substances will be used to make glucose and provide the cow with energy.



A cow that responds successfully to NEB uses the lipolysis pathway to supplement her energy demands until her DMI is adequate. The unsuccessful cow experiences SCK, then possibly clinical ketosis and is at risk to develop a fatty liver.

[READ ALL ABOUT IT! www.das.psu.edu/dairynutrition](http://www.das.psu.edu/dairynutrition)

Monitoring

There are two distinct facets of monitoring for ketosis: the individual cow and the herd. It is possible to spend too much time and energy trying to collect urine (individual cow) and not enough time examining the herd as a whole.

Herd Monitors—DMI

Precalving DMI is an excellent predictor of SCK. If DMI drops below 12kg (26.4lbs) in the prefresh group, there is a 6-fold risk of SCK. On account of the transient nature of prefresh groups – the constant changes in percent of first calf heifers, DMI's can be difficult to monitor. It does remain, however, an inexpensive and accurate predictor of a herd's SCK risk.

Herd Monitors—Pre-Partum Testing: serum NEFA's

- Currently, 0.40 meq/l is the line of demarcation for SCK.
- Herd goals – less than 15% of cows 2 weeks or less from calving have NEFA values over 0.4 meq/l.
- On farm testing not available – sent to a referral lab.
- Not accurate to predict DA's on individual cows, but useful as herd monitoring tool.

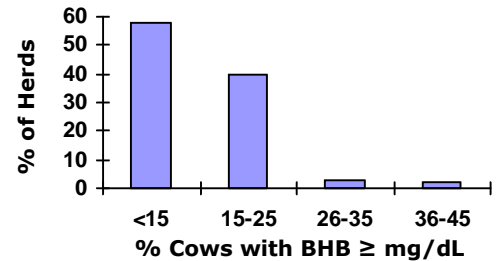
Herd Monitors—Post Partum Testing: serum BHBA

- Currently, >14 mg/dl (1400 μmol/l) is the line of demarcation for SCK.
- And, >30 mg/dl = clinical ketosis
- This test can be done on farm using a human blood ketone analyzer. The units are relatively inexpensive and the strips are approximately \$2.00 each.
- Reasonable goals are for less than 15% of the herd to be above 14 mg/l BHBA in the first 2 weeks post partum.

Herd Monitors—Protein to Butterfat Ratio on 1st Test Day

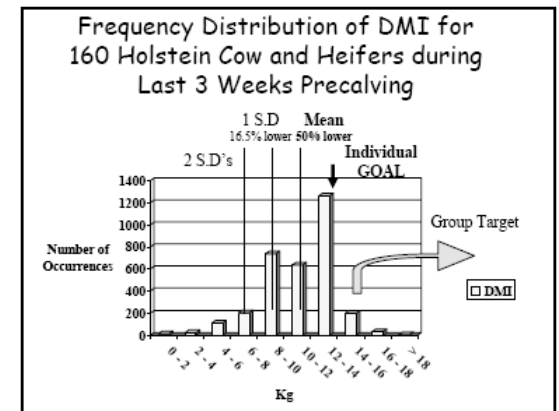
- A protein : butterfat ratio of ≤ 0.75 is indicative of SCK
- Low sensitivity 58% and specificity 69% make this a poor individual cow test
- If more than 40% of the herd has a protein to butterfat ratio ≤ 0.75 at 1st test, this is a reliable indicator of SCK.

Figure 2. Frequency distribution for post-calving BHB results for Northeast dairy herds.



Percentage of tested herds (n=35) with different proportions of sampled cows having BHB values equal to or above a critical "production" decision limit of 14 mg/dL.

Because SCK causes an increase in periparturient disease and decreases milk production, it significantly increases the risk of culling in early lactation. SCK has also been correlated with decreased first service conception rates. A 9% reduction in 1st service CR was demonstrated in the study summarized here.



Precalving DATA

1050 Cows: NEFA
 ≥ 0.5 U/L
 - 35% Prevalence
 - 9.2% DA (High) vs 2.8% DA (Normal)
High NEFA vs DA
 Sensitivity: 64%
 Specificity: 66%

Individual Monitoring—Cow-side Tests

The two most reliable tests are the milk keto test, by Elanco, and the urine keto stix, Diascreen.

Frequency of testing individual fresh cows should depend on a number of factors:

1. overall incidence of SCK based on herd testing
2. facilities
3. herdsman's time and ability to detect ADR (ain't doing right) cow

Test	Medium	Substance Detected	Reliability
Smell	Breath/milk	Ac	Poor (false negatives)
Acetest	Urine	AcAc	Poor (false positives)
Ketostix	Urine	AcAc	Good
Ketochek Powder	Milk	Ac, AcAc	Poor (false negatives)
Keto-Test	Milk	BHBA	Good

Ketosis - So What Test to Use?

Best Tests


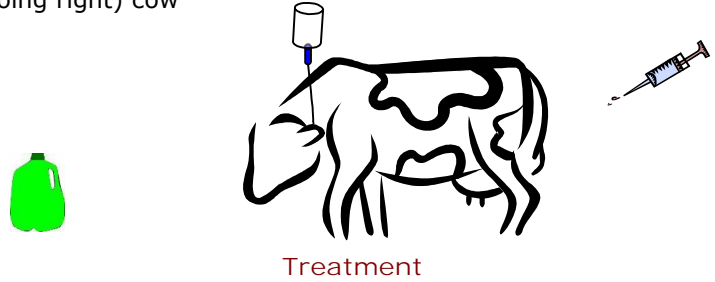
Keto-Test in milk

- ≥ 100 umol/L
 - SENSITIVITY - 83%
 - SPECIFICITY - 82%
- ≥ 200 umol/L
 - SENSITIVITY - 54%
 - SPECIFICITY - 94%

OR

Ketostix in Urine (interpret in 5 to 10 seconds)

- ≥ Small (15 umol/L)
 - SENSITIVITY - 79%
 - SPECIFICITY - 96%
- ≥ Moderate (40 umol/L)
 - SENSITIVITY - 50%
 - SPECIFICITY - 99%

Treatment

The goal is to supply glucose to the affected animal or to stimulate those metabolic pathways which promote gluconeogenesis. Appropriate treatments raise insulin levels, which drive more glucose into cells.

1. Dextrose
 - a. causes large elevations in serum glucose and stimulates insulin production
 - b. removed from the body within 2 hours of administration
 - c. should be used in combination with other treatment modalities
2. Corticosteroids (Dexamethasone, Predef)
 - a. gluconeogenic - increases glucose production for 2-4 days
 - b. works best when used with dextrose
 - c. may cause hypokalemia, low potassium levels, especially with repeated use
 - d. may cause immunosuppression
 - e. causes decrease milk production in healthy cattle
3. Propylene Glycol
 - a. absorbed directly through the rumen wall
 - b. metabolized by the liver to propionate (energy source to cow)
 - c. serum glucose peaks 75 minutes after drenching
 - d. 300 - 500 mL dose is adequate
 - e. should be given for 3 days

Steroids and Clinical Efficacy

2. Israel study (Shpigel et al, 1996)

- No difference between steroids and steroid + dextrose on changes in Ketones or Glucose.
- Steroids + Dextrose more efficacious in clinical recovery vs Steroids alone:

Parameter	Dexamethasone + Dextrose	Dexamethasone	Flumethasone	Flumethasone + Dextrose
Ketosis - Simple	85%	64%	63%	91%
Ketosis- Comp.	69%	41%	40%	79%

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Prevention

1. Optimize DMI in dry, prefresh and post fresh pens
 - a. adequate bunk space
 - b. stall comfort
 - c. limit overcrowding
 - d. best feed available should be fed to these groups
 - e. pay close attention to particle length— as more straw has been added to those diets, sorting has become an important concern
2. Control Body Condition Score (BCS)
 - a. if greater than 10% of herd has BCS greater than or equal to 4.0, then significantly increased risk for SCK
 - b. most common cause of fat dry cows is poor reproduction, get them bred early - avoid long lactations
3. Nutritional Management
 - a. proper balance dry, prefresh, post fresh rations
 - b. consider use of ionophores (rumensin) at correct level
 - c. consider use of rumen protected choline (reassure)

Conclusion

Clinical ketosis is just the tip of the iceberg. Sub Clinical Ketosis is a costly condition. Monitoring the herd with the most appropriate testing method is important for the well being of your business, as well as, that of your cattle. Talk with your veterinarian for specific ideas about detecting and treating these conditions on your farm.

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