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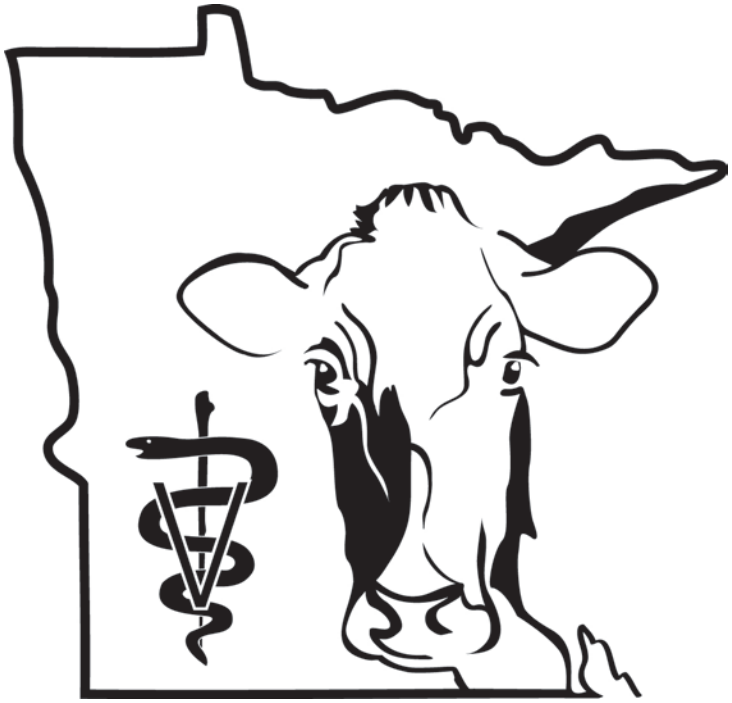

ST. PAUL, MINNESOTA
UNITED STATES OF MINNESOTA

Cows and brewmasters:
Neither one can efficiently produce desirable products from moldy ingredients

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This proceedings paper and presentation describes two dairy herd investigations. The health problems in both herds included increased late term abortions, pneumonia, marginal feed efficiency, and lower than expected milk production. The results of the herd investigations, diagnostic tests, and topics relevant to the investigations are discussed.

Herd 1: History and clinical signs

Herd 1 is a 600 cow dairy herd in Minnesota. The primary complaint was too many late term abortions and pneumonia cases during late gestation and lactation. Late term abortions (>120 DCC) approached 10% during the peak of the epizootic. Dairy cows that developed pneumonia had clinical signs that included coughing, an increased respiratory rate, and blood-tinged mucoid nasal discharges. Most pneumonia cows responded poorly to antibiotic, NAIDS, and supportive therapies. The abortion and pneumonia episodes had occurred for more than 3 years, did not have a seasonal trend, and did not correlate or respond to management changes.

During the fall and winter of 2007, the producer saved 30 aborted fetuses and froze them for periodic delivery and necropsy at the Minnesota Veterinary Diagnostic Laboratory. Necropsy results are summarized below.

14/30 Idiopathic
6/30 Enlarged thyroid glands with decreased amounts of colloid – Goiter?
4/30 Nitrates detected in the ocular fluid
3/30 *Listeria monocytogenes* abortion
3/30 Other bacterial abortions (Non-hemolytic *E. coli, A. pyogenes*)

Diagnostics to determine the potential etiologies of pneumonia were not as extensive as the abortion investigation. Infectious Bovine Rhinotracheitis Virus (IBRV) was detected by PCR in the nasal swab of one cow. Two cows that died of pneumonia were necropsies and one cow had liver abscesses with presumptive pulmonary thromboemboli. Another cow had a large blood clot in the trachea. The cause of the tracheal blood clot was not clear. The herd tested negative for bovine viral diarrhea virus (BVDV).

Herd visit #1 observations:

- Stocking density was approximately 130% (six-row barn)
- Cud chewing ranged between 42-55% in all pens
- Feces were uniform and consistent
- Lameness was minimal
- All herd dry matter intake was approximately 50 lbs./cow/day

- Milk production was approximately 70 lbs./cow/day (3X milking)
- All cow feed efficiency was 1.4 lbs of milk/lb. DM (benchmark 1.4-1.6)
- Most cows were lean and had a body condition score less than 3.0 on the 5.0 scale.

Preliminary changes after the initial farm visit:

- Increase dietary iodine to the legal limit to address the presumptive fetal goiter
- Intranasal IBRV vaccinate all cows once and all cattle prior to calving
- Routinely monitor for sub-acute rumen acidosis (SARA)
- Reduce stocking density

Results of preliminary changes:

- No noticeable improvements over 6 months.

The producer was confident that the abortions and respiratory diseases were an infectious pathogen that had not been detected or isolated. The producer wanted to detect and isolate an infectious pathogen for autogenous vaccine production. Both the nutritionist and producer were confident that the clinical problems were not a manifestation of SARA. The producer was also confident that the only thing that would decrease if the stocking density was further reduced was the total volume of milk sold and he could not afford shipping less milk.

Herd visit #2: More diagnostics, but which one(s)??

Lack of significant improvement over six months prompted a follow-up farm visit. The goal of the second visit was to focus on cows in late gestation/lactation and examine potential non-infectious causes of the herd health problems.

Ten clinically normal cows in late gestation/lactation were bled for serum chemistry analysis. The blood was collected in serum separator tubes, centrifuged, and the tubes were delivered to the U of MN clinical pathology laboratory within 4 hours. The blood was collected approximately 4 hours after morning feeding when many digestive processes and enzymes are expected to peak. A complete serum chemistry analysis was performed on all cows and included anion gab, albumin, ALP, AST, TBili, BUN, Ca, Cl, Creat., CK, GGT, Glu, Mg, Na, Osm, PO4, K, SDH, bicarb, and TP. The abnormal results are presented in Table 1. Serum chemistry results that fell within the normal reference range are not reported. All globulin values were within the normal reference range and were added to the table for Albumin/Globulin (A/G) ratio calculations.

	Clinical App	SDH	AST	GGT	Albumin	Globulin	A/G ratio	BUN
1	Normal	89 ↑	187 ↑	62 ↑	3.3	4.1	0.80 ↓	12
2	Normal	20	107	28	3.4	3.5	0.97	8 ↓
3	Normal	38 ↑	95	26	3.2	5.3	0.60 ↓	13
4	Normal	11	77	17	3.4	3.1	>1	12
5	Normal	36 ↑	96	43 ↑	3 ↓	5.1	0.60 ↓	9 ↓
6	Normal	41 ↑	77	42 ↑	3.3	3.6	0.91	9 ↓
7	Normal	41 ↑	85	42 ↑	3 ↓	4.6	0.65 ↓	10 ↓
8	Normal	26 ↑	71	23	3.3	4.3	0.77 ↓	12
9	Normal	21	81	29	3.5	3.5	1.00	13
10	Normal	38 ↑	149 ↑	39 ↑	3.6	4	0.90	9 ↓
	ref range	7-23 U/L	48-127 U/L	3-38 U/L	3.2-4.0 g/dL	3.1-5.6 g/dL	0.84-0.94	10-24 mg/dL

High ↑
Normal
Low ↓

Table 1: Serum chemistry analysis of 10 clinically normal dairy cows in late gestation.

Seven of the 10 cows had mildly elevated sorbitol dehydrogenase (SDH) values. In cattle, SDH is liver specific and considered a "leakage" enzyme. SDH has a short half-life and is a useful indicator of acute liver disease.

Five of the 10 cows had mildly elevated GGT levels. In cattle, GGT is also liver specific, but unlike SDA, has a longer half-life and is a useful indicator of chronic liver disease.

Albumin was low in 2 of the 10 cows and 9 of the 10 cows had an albumin lower that 3.6 g/dL (reference range 3.2 to 4.0 g/dL). Assuming 3.6 g/dL is "average" or "normal," then the difference between 3.6 g/dL and the 10 cows in this herd is significant ($p<0.01$). The albumin/globulin ratios were calculate and determined to be low in 5 of the 10 cows. All cows had normal globulin values which indicated that the low A/G ratio was a manifestation of low albumin and not high globulins. High serum globulins are common in cattle with chronic infection (mastitis, cellulitis, liver abscesses, etc.) and high serum globulins will lower the A/G ratio.[1] None of the cows sampled had evidence of chronic infection which would result in high serum globulins.

Albumin is an important blood protein produced by the liver. The most notable function of this large blood protein is regulation of intravascular and extravascular hydration (oncotic pressure). Any animal with low serum albumin is at risk for generalized edema. Albumin has also been described as the "portable liver" as it scavenges free radical and transports cortisol. Albumin's other important functions include transportation of nutrients, vitamins, trace minerals, hormones, and fatty acids.

Blood urea nitrogen (BUN) was low in 4 of the 10 cows. At the cow level, there are minor differences between BUN and milk urea nitrogen (MUN) as urea will readily shuttle between serum and milk. Blood urea nitrogen in the 10 cows sampled was expected to be higher because the samples were taken approximately 4 hours after the morning feeding when soluble proteins are expected to have been degraded to urea and ammonia by the rumen microflora.[2] Unlike non-ruminants, low blood or milk urea nitrogen levels in cattle is not significant because rumen microbes use all available ammonia and urea for microbial protein production.

In summary, serum chemistries revealed 5 of 10 cows had elevated liver enzymes, 2 of 10 cows had hypoalbuminemia, 5 of 10 cows had low A/G ratios, and 4 or 10 cows had low BUNs.

Corn silage bunker:

Corn silage was the only fermented feed in the ration and comprised approximately 35% of the dry matter in the diet. The bunker was not meticulously managed and closer examination revealed a marginal packing density and large chunks of moldy crusts mixed in with the "good" feed piled at the base of the face. The face was rough because the farm did not use a mechanical "facer" to remove silage. Three walls were earthen clay and the cement base contained large pools of silage effluent. A silage inoculant was not used during harvest.

Sixteen hand-grab samples from the face and a few handfuls of moldy feed were collected, mixed, vacuum packed, and shipped frozen to NDSU for mycotoxin analysis. Zearalenone (0.6 ppm), Vomitoxin (0.8 ppm) and Aflatoxin (0.05 ppm) were detected in the corn silage by GC/MS. A brief summary of each of the mycotoxins is provided.

Zearalenone is produced by *Fusarium* molds and has an estrogen-like chemical structure. Several case reports have related Zearalenone to estrogenic responses, including abortions. *If 0.6 ppm was a representative sample, then the calculated daily consumption of Zearalenone from the corn silage was 4 mg/cow/day. Zearalenone levels above 0.3 ppm (300 ppb, 0.3 mg/kg) are not well tolerated by dairy cows.*[3] *Again, the samples collected from the silage on this farm contained 0.6 ppm Zearalenone, approximately 2 times the "tolerable" level.*

Vomitoxin (DON) is also produced by *Fusarium sp.* molds and is associated with vomiting is swine. In cattle, vomitoxin will alter rumen fermentation[4] and reduce the flow of utilizable protein to the duodenum.[5] Vomitoxin has also been described as a "marker mycotoxin" because its presence indicates the feed was exposed to a situation conducive for mold growth and the possible formation of several mycotoxins.[6]

Aflatoxins are compounds produced by *Aspergillus flavus* and *Aspergillus parasiticus.* Alfatoxins are carcinogenic and mutagenic compounds that can cause a wide variety of symptoms. Dairy cow health and production are affected with levels above 0.120 ppm (120 ppb).[7] The Aflatoxin levels in the corn silage was 0.05 ppm.

How does moldy feed cause abortions and pneumonia? Farm visit #3.

The producer was not convinced that the mold and mycotoxin-laden corn silage were contributing to the herd health problems. His justification included that the heifers were consuming the silage and were "fine" and he had been feeding a mold and mycotoxin binder for many months. Lack of evidence linking the silage to the herd health problems prompted another visit to examine three sick cows.

In August 2008, physical exams were performed on the three sick cows in late lactation. Cow 1 was acutely "off-feed" and cows 2 and 3 were diagnosed with pneumonia. Both pneumonia cows were similar to other cows previously diagnosed with pneumonia. Almost all of the cows diagnosed with pneumonia were diagnosed by farm staff and not routinely examined by veterinarians.

Physical exam of cow 1 revealed an RDA. This herd had an unusually high rate of RDAs and documented approximately 9 RDAs for every 1 LDA, an upside down ratio when compared to most herds. Due to impending dry-off and having an RDA for approximately 2 days, the cow was determined to be a poor surgical candidate and was

euthanized. Other than a 2-day-old RDA, necropsy was relatively unremarkable. The cow did have a few irregular sub-capsular depressions "scars" throughout the liver. A CBC and chemistry revealed hypoalbuminemia (2.8 g/dL, reference range 3.0 to 4.0 g/dL), mild neutrophilia, and mildly elevated SDH. Histopathology of the liver revealed marked Kupffer cell hyperplasia, a finding not commonly described in cattle livers. Kupffer cells are specialized, liver-specific macrophages that are important responders to foreign antigens and toxins. Most cattle have about 20 Kupffer cells / 400X field, but this cow had more than 100 Kupffer cells / 400X field. The significance of this finding can only be described as the liver being exposed to an antigen or toxin that stimulated Kupffer cell hyperplasia. Kupffer cell hyperplasia is not specific for any one disease process or pathogen and is interpreted as chronic or persistent exposure to a foreign antigen or toxin.

Cow 2 was diagnosed with pneumonia by the producer. Physical exam revealed small amounts of blood-tinged mucoid nasal discharge and mild tachypnea. Necropsy was underwhelming as there was only a focal area of minimal lung consolidation involving the right middle lung lobe. The trachea contained a large blood clot which contained many small pieces of alfalfa. The blood clot containing alfalfa was unusual and was interpreted as an aspiration lesion. Histopathology confirmed a focal, mild purulent bronchopneumonia and *Arcanobacterium pyogenes* was isolated from the lung. There was no gross or microscopic evidence of viral pneumonia. Necropsy also revealed a focal area of chronic peritonitis involving the serosa of the abomasum. A 6-inch piece of wire was found free-floating in the abomasum, but this "hardware disease" finding was determined to be non-complicated as there was no evidence of acute peritonitis. Serum collected prior to euthanasia revealed hypoalbuminemia (2.7 g/dL, normal 3.2-4.0 g/dL) and elevated liver enzymes (SDH, GGT and AST).

Cow 3 had pneumonia and had been treated with antibiotics and NSAIDS with no effect. Physical exam revealed hypothermia (101.2 F), bilateral jugular pulses, and mild tachycardia. Auscultation of the heart did not reveal muffled heart sounds or murmurs. The cow had diffuse limb edema that gave all her legs a "stove-pipe" appearance. Vigorous tracheal manipulation did not produce a cough and auscultation of the lungs revealed raspy "fluid" sounds throughout the right lung fields. The clinical diagnosis was not obvious and preliminary rule-outs included traumatic reticuloperitonitis, vegetative valvular endocarditis, or heart failure. This cow was <u>not</u> euthanized and necropsied. This "pneumonia" cow had a normal CBC, but the serum chemistry analysis revealed hypoalbuminemia (2.5 g/dL, ref range 3.2-4.0 g/dL) and a decreased albumin/globulin ratio of 0.61 (ref range 0.84-0.94). The updated and final diagnosis for cow 3 was pulmonary and peripheral edema secondary to hypoalbuminemia. The serum chemistry results from the 10 normal cows and the 3 sick cows are summarized in Table 2.

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3	Normal	38 ↑	95	26	3.2	5.3	0.60 ↓	13
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5	Normal	36 ↑	96	43 ↑	3 ↓	5.1	0.60 ↓	9 ↓
6	Normal	41 ↑	77	42 ↑	3.3	3.6	0.91	9 ↓
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8	Normal	26 ↑	71	23	3.3	4.3	0.77 ↓	12
9	Normal	21	81	29	3.5	3.5	1.00	13
10	Normal	38 ↑	149 ↑	39 ↑	3.6	4	0.90	9 ↓
11	Pneumonia	10	220 ↑	18	**2.5** ↓	4.1	0.61 ↓	11
12	Pneumonia	50 ↑	148 ↑	38 ↑	**2.8** ↓	5.2	0.54 ↓	10 ↓
13	RDA	45 ↑	97	29	**2.7** ↓	4.1	0.66 ↓	16
	ref range	7-23 U/L	48-127 U/L	3-38 U/L	3.2-4.0 g/dL	3.1-5.6 g/dL	0.84-0.94	10-24 mg/dL

High ↑
Normal
Low ↓

Table 2: Serum chemistry analysis of all cows

Hypoalbuminemia – How low is *too* low?

All sick cows and 2 of the clinically normal cows had serum albumin at 3.0 g/dL or lower. Transient hypoalbuminemia is common during early lactation and dairy cows typically recover to have serum albumin levels above 3.5 g/dl by 6 weeks post-partum.[6] Data on the significance and potential ramifications of hypoalbuminemia in cattle is sparse, but in humans, hypoalbuminemia is an important predictor of morbidity and mortality. In a meta-analysis of cohort studies, a 1.0 g/dL decrease in serum albumin resulted in an 89% increase in morbidity and a 137% increase in mortality. Patients with a serum albumin levels of less than 3.5 g/dL at 3 months following discharge from the hospital have a 2.6 times greater 5-year mortality than those with serum albumin levels greater than 4.0 g/dL.[9]

What is causing the apparent hypoalbuminemia?

A lack of serum albumin can either result from decreased albumin *production* or increased albumin *loss*. Decreased albumin *production* can results from primary liver disease involving more than 2/3 of the liver. The liver can not sustain routine albumin turn-over and on-going losses when more than 2/3 of the liver fails to function. Malnutrition and the lack of dietary protein are common causes of decreased albumin production in many animal species. Common causes of albumin *loss* in cattle includes protein-losing enteropathies, gastrointestinal parasitism, and renal amyloidosis.

Although albumin loss through the intestine can be common in dairy cattle with enteritis or parasitism, it was unlikely in this herd because albumin loss secondary to enteritis is usually accompanied by the simultaneous loss of globlin. If the cows had enteritis, the equal loss of albumin and globlin should have resulted in normal A/G ratios. Enteric loss of albumin was also unlikely because most of the cattle in this herd tested negative for Johne's disease and there was no evidence of widespread enteric Salmonellosis or gastrointestinal parasitism. Increased loss of albumin via renal amyloidosis was unlikely because there was no clinical evidence of renal disease and animals with renal amyloidosis routinely have A/G ratios below 0.5.

In the cattle sampled, there was a mild increase of liver enzymes, but no evidence that more than 2/3 of the livers were sufficiently diseased causing decreased albumin production. After balancing all the information, it appeared that hypoalbuminemia was a result of decreased production that can we propose is explained by insufficient quantities digestible protein.

Malnutrition? Lack of dietary protein? *Really?*

The TMR on this farm was balanced to contain 17% protein. Approximately 63% of the protein was rumen degradable protein (RDP) and 36% of the protein was calculated to be rumen undegraded "bypass" protein (RUP). Soybean meal and dry alfalfa hay were the primary protein sources. Although the protein in this ration was formulated, mixed, and fed, it did not appear to be efficiently digested and utilized by the cows. The working theory is that mold and other compounds in the poorly ensiled corn silage prevented the degradation of RDP to urea and ammonia. Ammonia and urea are the essential sources of nitrogen for microbial protein production. Microbial protein is the primary protein source for cattle and is characterized as "true" protein because the digestion of microbial protein in the small intestine provides many of the essential amino acids to the cow.

Low BUN in many of the cows within this herd is consistent with a decrease in rumen degradation of soluble proteins (RDP). The 10 healthy cows were sampled 4 hours after feeding and should have had higher BUNs. The lack of protein degradation in the rumen appeared to initiate a cascade of events starting with an insufficient amount of ammonia, urea and microbial protein synthesis and ultimately resulting in a lack of essential amino acids for albumin production.

The series of events that eventually resulted in hypoalbuminemia would have taken at least one month because the half-life of albumin is about 20 days and losses are compensated by the large functional hepatic reserves. The ten normal and three sick cows sampled in this herd had consumed the forages for at least 150 days and would have had enough time to draw down albumin reserves.

What about the mycotoxins?

Elevated liver enzymes in 5 of the 10 clinically normal and 2 of the sick cows can be caused by systemic toxin exposure and the mycotoxins detected in the corn silage are the likely source. Kupffer cell hyperplasia in RDA dry-cow also indicated a chronic, active insult of the liver. However, the liver lesions in the RDA cow were not significant enough to compromise more than 2/3 of the liver. Despite having mildly elevated liver enzymes, none of the clinically normal or sick cows had clinical signs of primary liver failure (icterus, clotting disorders, etc.).

Ruminants are more resistant to mycotoxins than monogastrics. Ciliates and other protozoa in the rumen are effective "detoxifiers" of many mycotoxins. Slower rates of passage allow rumen microbes more time to "detoxify" mycotoxins and the extent of detoxification depends on rumen turnover rates. Beef cows appear more resistant to mycotoxins because the average rumen turnover rates in a beef cow is about 8 times longer than lactating dairy cow.[10]

In vitro experiments demonstrate that rumen protozoa are more efficient than rumen bacteria at detoxifying various mycotoxins.[11] Research studies indicate that moldy feed negatively affects rumen protozoal populations. In one study, microscopic examination of rumen content in steers fed moldy hay indicated a reduction in protozoa concentrations. Digestibility of proteins and ruminal ammonia levels in the steers fed moldy alfalfa hay were also significantly lower.[12] Mycotoxin detoxification appears inefficient in mold-laden rumens that have reduced numbers of ciliates and protozoa.

Herd #2:

Herd #2 was far less complicated, but did provided another perspective of the significance of moldy forages in dairy cow diets. The information gathered from this herd suggests that a "harmless" mold that does not contain a known detectable mycotoxin can have a significant impact on cow health and milk production.

The dairy consists of 120 lactating dairy cows. Clinical signs included poor milk production (~60 lbs.), brisket edema, chronic health problems and an unacceptably high cull rate. Serum chemistry analysis of five affected cows is summarized in table 3. Values which fell outside the published reference range are presented. Unlike herd #1, all liver enzymes were normal.

Cow ID	Clinical Signs	Albumin	Globulin	A/G ratio	BUN
767	Brisket and peripheral edema	3.0 ↓	5	0.60 ↓	14
757	Poor performance	2.4 ↓	5.7 ↑	0.42 ↓	15
807	DA cow	2.2 ↓	6.4 ↑	0.34 ↓	9 ↓
204	Fresh cow	2.7 ↓	4.5	0.60 ↓	10 ↓
737	Fresh cow	2.8 ↓	4.7	0.60 ↓	10 ↓
	ref range	3.2 - 4 g/dL	3.1 -5.6 g/dL	0.84 - 0.94	10-24 mg/dL

Low ↓
Normal
High ↑

Table 3: Herd 2 serum chemistry analysis.

Like herd #1, corn silage was the primary fermented forage in the TMR of herd #2. The corn silage bunker was 30' x 150' and the dairy fed more than 6 inches per day for the first 6 months. In March, approximately 2/3 of the bunker was gone and the dairy realized they would run out of corn silage before fall harvest. After the discovery, the feed-out rate was reduced to 1-2 inches per day. The nutritionist sent the visibly moldly corn silage to Dairyland Laboratories for mold and yeast counts. The results with guidelines are below.

Mold count	20 million colonies/gm
Yeast count	25 million colonies/gm
Mold Identification	100% *Rhizopus sp.*
Mycotoxins (ELISA)	None detected.

Mold Count Guideline:

10-10,000	Relatively safe
10,000 – 100,000	Transition zone
100,000 – 1 million	Caution advised
Over 10 million	Feeding not recommended

High levels of *Rhizopus sp*. and yeasts were detected in the corn silage. The warm summer weather and an insufficient removal rate of 1-2 inches per day amplified yeast and mold growth at the bunker face. *Rhizopus sp.* has been described as a common "bread mold" that is produces no known mycotoxins. The high level of molds, even in the absence of a detectable mycotoxin, appeared to have a negative impact on rumen function and protein digestion/metabolism. Hypoalbuminemia in many of the cows was striking and a few cows had obvious brisket edema. The cows with peripheral edema did not have clinical diarrhea and there were no gastrointestinal parasites detected on fecal parasite exams.

Infectious pathogens were ruled-out with ten non-vaccinated sentinel heifers ranging in age from 6 to 14 months that all tested negative for BVDV, IBRV, and Leptospirosis antibodies. Hypoabuminemia was likely a result of excess mold consumption and a lack of microbial protein production and digestion as described in herd #1.

Summary:

Common sense dictates that moldy feeds and mycotoxins will have a negative impact on health and production. Many research publications have highlighted the negative effects of molds and mycotoxins, but few research projects have followed experimentally challenged cattle for more than one month. If moldy feed negatively affect microbial protein synthesis and albumin production, cattle will need at least 1 month to draw down hepatic reserves before hypoalbuminemia is detected. Cows will always be exposed to mold and yeast, yet the effect of prolonged exposures to high levels (>100,000 colonies/gram) is not known. In the two herds described, long term consumption of moldy feeds appeared to cause hypoalbuminemia, a phenomenon that we propose is explained by a chronic deficit of rumen microbial protein production.

As the dairy industry consolidates and more forages are stored in large open-faced bunkers, the risk of feeding poorly preserved and spoiled forages will increase. Producers are reluctant to discard marginal or slightly spoiled feed when quantities may be low and feed prices are high. The problem of feeding spoiled feed is further complicated when TMR-mixed feeds mask spoiled feeds and inclusion of spoiled feeds does not *appear* to reduce dry matter intake.

Quantifying and estimating forage dry matter losses at the bunker level during ensiling and storage (shrink) can be easily calculated. Losses at the cow level are much more difficult to determine and cow-level losses have not been described or quantified. Quantifying losses at both the bunker and cow level will give nutritionist, veterinarians, and producers a reference when allocating resources to improve feed quality, reduce yeast and mold growth, and minimize nutrient losses.

Brewmasters???

So what's with the goofy title? High quality cereal grains (wheat, barley, etc.) are essential for brewing high quality beer. Beer can only be as good as the malted grains, hops, yeast and water. Molds, mycotoxins, insects and other foreign material (weed seeds, etc.) will not make tasty beer. Moldy flavor would result in the loss of the batch. It doesn't matter if the fermentation vat is a cow's rumen or stainless steel vessel, neither cows nor brewmasters can efficiently produce desirable products from moldy ingredients.

References:

1. Russell K., Roussel A., 2007. Evaluation of the Ruminant Serum Chemistry Profile. Vet Clin Food Anim 23:403-426.
2. Gustafsson A., Palmquest D., 1993. Diurnal variation of rumen ammonia, serum urea, and milk urea in dairy cows at high and low yields. J Dairy Science 76:475-484
3. Mycotoxins: Economic and Health Risks. 1989. Council for Agricultural Science and Technology. Task Force Report No. 116, Ames, Iowa.
4. Seeling K., Lebzien P., Danicke S., *et al.* 2006. Effects of level of feed intake and Fusarium toxin-contaminated wheat on rumen fermentation as well as on blood and milk parameters in cows. J. An. Physiol. and Nutr. 90:103-115.
5. Danicke S., Matthaus K., Lebzien P., *et al.* 2005. Effects of Fusarium toxin-contaminated wheat grain on nutrient turnover, microbial protein synthesis and metabolism of deoxnivalenol and zearalenone in the rumen of dairy cows. J An. Physiol. and An. Nutr. 89:303-315.
6. Whitlow L., and Hagler Jr. W., 2008. Mold and Mycotoxin Issues in Dairy Cattle: Effects, Prevention and Treatment, North Carolina State University @ http://www.extension.org/pages/Mold_and_Mycotoxin_Issues_in_Dairy_Cattle:_Effects,_Prevention_and_Treatment
7. Guthrie, L. 1979. Effects of Aflatoxin in corn on production and reproduction in dairy cattle. J Dairy Science. 62 (abstr.):134.
8. Jordan E., Swanson L., 1979. Effect of Crude Protein on Reproductive Efficiency, Serum Total Protein, and Albumin in the High-Producting Dairy Cow. J Dairy Science 62:58-63
9. Peralta R., Rubery B., Guzofski S., 2009. Hypoalbuminemia @ http://emedicine.medscape.com
10. Ontario Ministry of Agriculture Food and Rural Affairs. 2006. Moulds and Mycotoxins – Effect of Mouldy feed and Mycotoxins on Cattle.
11. Kiessling K., Pettersson H., Sandholm K., *et al.,* 1984. Metabolism of Aflatoxin, Ochratoxin, Zearalenone, and three Trichothecenes by Intact Rumen Fluid, Rumen Protozoa, and Rumen Bacteria. Appl and Environ Microbiol, p. 1070-1073
12. Mohanty G., Jorgensen N., Luther R., Voelker H., 1968 Effect of Molded Alfalfa Hay on Rumen Activity, Performance, and Digestibility in Dairy Steers. South Dakota Agriculture Experiment Station Publication no. 832.