PGC FEEDS

# PETTISVILLE GRAIN CO.

*Your Success. Our Future.*

## Monthly Newsletter – May 2021

Grain:

### Get the Latest Updates on Your Phone with Our Bid Texts

Pettisville Grain Co. is proud to offer our customers the most relevant marketing information, right at your fingertips! One of the ways that we can keep everyone up to date is through our grain bid texts. We send out a text with our grain bids at 10:30 a.m. and at 3:30 p.m. every day that the grain markets are open. Don't miss out on this opportunity to get a daily glance at the markets right from your phone!

### What's Happening with The Markets?


Since the start of this price rally in September 2020, farmers have began forward contracting their grain because of the profitable prices. Those who sold early in the rally may feel like they made a mistake and missed out on even better prices. Now that we have reached that time of year when farmers would typically start forward contracting for 2021, we want to encourage growers to take another look at their marketing plans and be proactive. PGC promotes calculating the farm's 5-year yield average and using that to find out how much you should forward contract for any given year. We like to see farmers contract up to 60% of their 5-year average yield to hedge against a poor harvest.

June is typically the best time for forward contracting corn and soybeans because the prices are highest in June and July on average. However, 2021 seems to be a fluke because of the unpredicted rally early in the year. It now seems that prices have downside potential due to deals with China and weather conditions. Most of the U.S. has gotten the crops planted and there are rains across the corn belt and other key parts of the country. This has given the U.S. a great start to the 2021-22 crop year and seems to have led the corn and soybean prices downward for the last week of May.

We say it often here at Pettisville Grain, but it is important to emphasize that farmers should continue to take an in-depth look at their costs of production and compare that with their anticipated revenue based on yield. What price per bushel would result in a profit for the farm? This question is an important one because profitability is what keeps a farm in operation, not price. Reaching the high point of the markets brings high risk. Now is a great time to take another look at profitability because much of the seed is in the ground and we are closer to buying inputs, if they haven't already been priced. Input prices have shifted recently to follow the commodity prices, so it may be necessary to take another look at target prices.

In summary, the grain markets have been volatile lately, so to manage risk, we encourage farm marketing managers to take a look at the updated input costs along with yield expectations and put in realistic target orders for remaining forward contracting.

Feed:

## Ruminal Acidosis (Grain Overload)

Dr. Richard Bowen, Professor, Department of Biomedical Sciences, Colorado State University

The rumen encases a complex ecosystem containing numerous species of bacteria and protozoa that collectively provide the capacity for efficient fermentation of carbohydrates. Among the major products of such fermentation are volatile fatty acids and lactic acid. Wild ruminants and those raised on pasture consume a diet rich in grasses that consist mostly of cellulose. Cellulose is a molecule that might be called a "slowly fermentable carbohydrate". In contrast, grains such as wheat, barley, and corn are considered "highly fermentable carbohydrates", meaning that they can be very rapidly fermented to generate large quantities of volatile fatty acids and lactic acid. **Ruminal acidosis results from consumption of an unaccustomed quantity of highly fermentable carbohydrate, described as grain overload.**

Ruminal acidosis is most commonly a disease of dairy and feedlot cattle, and occasionally sheep in feedlots. All of these animals are typically fed large quantities of grain to promote production of milk and enhance growth. The key point is that animals and their ruminal microbes must be adapted *over time* to a high grain diet, otherwise acidosis commonly ensues. In some cases, animals develop acute acidosis "accidentally", when, for example, they escape from their pen and get into a store of grain.

**Pathogenesis**

The fundamental pathogenesis of ruminal acidosis is straightforward. Ingestion of large quantities of a highly fermentable carbohydrate (grain) by an animal not adapted to such a diet results in rapid production of massive quantities of acids in the rumen. These acids have several deleterious effects:

- Acid damages the epithelium of the rumen, resulting in ruminitis and allowing leakage of bacteria from the rumen into the systemic circulation.
- The drop in pH associated with acid production has profound effects on microbial populations in the rumen, resulting in proliferation of lactic acid-producing bacteria and death of others populations, including protozoa. This constitutes a vicious cycle in which more acid is added to ruminal contents, with further reduction in pH.
- Absorption of lactic acid produced in the rumen into blood results in a profound metabolic acidosis.

There are several common sequelae to ruminal acidosis:

- Liver abscesses: Molecules such as volatile fatty acids produced in the rumen are absorbed across the epithelium into ruminal veins, then into the hepatic portal vein and through the sinusoids of the liver. When the ruminal epithelium is damaged, as it is with ruminal acidosis, ruminal bacteria often leak across the epithelium, travel the liver and establish abscesses. *Fusobacterium necrophorum* is the most common bacterium to cause such lesions.

- Inflammation of the hoof wall (laminitis) and subsequent hoof wall deformities have been associated with ruminal acidosis, but is difficult to substantiate in terms of cause and effect.
- Damage to the ruminal epithelium associated with ruminal acidosis frequently is followed by colonization by fungi, resulting in mycotic rumenitis.

**Clinical Signs and Diagnosis**

Although there is a spectrum of clinical pictures associated with ruminal acidosis, two predominant forms are recognized, dependent on how rapidly and extensively excessive acid production in the rumen occurs. To a large extent, severity also relates to the quantity of grain ingested relative to the animal's adaptation to a high grain diet.

*Acute ruminal acidosis:* Animals have a rapid onset of profound depression, with recumbency, staggering or standing immobile. The animals are inevitably off feed and frequently show profuse diarrhea. They become dehydrated and body temperature is normal or low. This form of the disease has quite a high mortality rate and even those that survive often suffer from serious sequelae such as liver abscesses.

The pH of rumen fluid is normally 6 – 7 for animals on a diet of roughage and 5.5 – 6 for healthy animals on a high grain diet. Animals with acute ruminal acidosis display a lack of rumen motility and the pH of rumen fluid obtained with a stomach tube is less than 5.5, getting as low as 4 – 4.5 in lethal cases.

*Subacute and chronic ruminal acidosis:* This condition results from repeated episodes of decreased ruminal pH (5.2 – 5.6) due to elevated levels of volatile fatty acid production in the rumen. Affected animals show reduced feed intake and milk production, poor body condition with weight loss, and sometimes diarrhea. As with the acute form of disease, these repeated episodes result in rumenitis, with damage to the epithelium, which is often associated with development of liver abscesses. The repeated low pH environment also alters the normal flora within the rumen.

**Pathology**

Post-mortem diagnosis of acute rumenitis can be difficult and is best accomplished by microscopic examination of the ruminal epithelium. Mycotic ruminitis presents rather spectacular patches of darkened epithelium at post-mortem examination.

**Treatment and Control**

Acute ruminal acidosis is a disease with high mortality and it is often recommended that affected animals be slaughtered prior to their death. If treatment is desired, it must be very aggressive and consist of removing ruminal contents via rumenotomy, instituting intensive fluid therapy to combat dehydration and acidosis.

Less severe, subacute cases of ruminal acidosis are managed by oral administration of bases to neutralize acid, reduction of grain intact, and coincidentally placing the animal on long stemmed hay.

**The key practice required to minimize development of ruminal acidosis is to adapt animals slowly to a diet containing large amounts of grain.** Some authorities suggest feeding animals a buffer such as sodium or potassium bicarbonate, although this practice is unlikely to totally prevent acidosis.