Looking for answers to

Atypical Interstitial Pneumonia

Pieces of the puzzle likely include feed intake, feed composition, individual animal physiology and possible environmental triggers.

*Story & photo by **Troy Smith,** field editor*

Not everyone agrees that atypical interstitial pneumonia is the second leading cause of feedlot mortality. While it's generally agreed that bovine respiratory disease is easily the No. 1 cattle killer, atypical interstitial pneumonia, or AIP, is seldom seen in some feedlots. For others, its occurrence is more common. It's a malady of the perplexing kind because the reason or reasons why AIP occurs are uncertain, and a remedy remains elusive.

During the tour in conjunction with last fall's National Angus Conference, participants heard Pratt Feeders General Manager Jerry Bohn explain how, over the past few years, feedlot AIP has become a more significant animal health challenge at the Pratt, Kan., yard and at sister yards located in Ashland and Hays, Kan., and Buffalo, Okla.

In the experience of the Pratt Feeders group, the disease tends to become more prevalent during hot, dry weather and dusty conditions. The disease often comes on suddenly, therapy often is unrewarding, and most affected animals die. And most AIP victims rank among the best-performing animals, including Angus cattle.

High performers

"It appears to show up in the high-quality, high-performing, big-eating cattle," states Bohn. Cattle with the best [performance] genetics appear to be most susceptible to the disease."

Consulting veterinarian Scott MacGregor says the incidence of AIP is no higher at the Pratt feedlots than at others having what he calls "superlative conversion rates," and similar observations are reported by other clients with feeding operations in the Great Plains region. Affiliated with Feedlot Health Management Services USA, MacGregor says clients find more cases of AIP among cattle fed a high-megacalorie ration based on steam-flaked corn, as opposed to a lower-megacalorie ration based on ground corn. It appears that AIP is also more common among cattle fed finishing rations containing a beta-agonist to enhance performance.

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Most AIP cases occur late in the finishing period, or at least after cattle have been on feed for a couple of months. MacGregor's data also suggest incidence is higher among heifers than steers.

"It's discouraging to see a big, beautiful heifer die suddenly, just 35 to 45 days away from market. It often happens like that, but death from AIP is pretty easy to confirm with a postmortem exam," says MacGregor, explaining how the lung tissue has a rubbery texture and dark lesions creating sort of a mottled or checkerboard appearance.

Asked if Angus cattle are more susceptible, MacGregor explains that AIP is not breed-specific. Many AIP cases in feedlots he serves do involve high-performing Angus cattle, but it strikes cattle of other breeds, too.

"Some Angus cattle may be more susceptible. Genetics may be a factor. We're looking at that," MacGregor states.

A production disease

It seems likely that some combination of factors may trigger AIP, but it's important to understand that AIP is not an infectious disease caused by a bacterial agent. It isn't caused by some "bug" that spreads from animal to animal, and cattle cannot be immunized through vaccination. It can't be effectively treated with antibiotics. Like acidosis and bloat, AIP is a production disease, so AIP might be "managed" if all contributing factors and their respective roles were better understood.

Evidence suggests previous respiratory infection may influence susceptibility. Tim McAllister, a research scientist at Agriculture and AgriFood Canada in Lethbridge, Alberta, says animals that die

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from AIP often show evidence of prior lung insult that might be attributed to bacterial pneumonia experienced earlier in life. But signs of previous respiratory disease are not consistent with all AIP victims.

McAllister's rumen nutrition and microbiology research suggests diet and rumen metabolism are pieces of the AIP puzzle. They are with a kind of AIP that can affect grazing cattle when they are moved from dry, mature pasture to lush, green forage. With the sudden change to a diet rich in the amino acid tryptophan, rumen metabolism of tryptophan produces 3-metholindole (3-MI), which is rapidly absorbed into the circulatory system. After reaching the lungs, 3-MI is converted to several toxic substances. Most toxic is 3-MEIN, which is believed to cause cellular damage resulting in the edema, hemorrhage and emphysema associated with pasture AIP.

McAllister says studies in Alberta showed that 3-MEIN might also be associated with feedlot AIP. Plasma levels were significantly higher in feedlot animals with AIP, as compared to animals that died from other respiratory disease.

"There is strong evidence that the rumen metabolism is involved with feedlot AIP, but there probably are other factors," offers McAllister. "In our experience, feedlot AIP is at least three times more likely to affect heifers than steers. There's some evidence that feeding MGA (melengestrol acetate) to heifers may increase the likelihood of AIP, but it isn't conclusive. There is growing anecdotal evidence that feeding beta-agonists might be linked to increased incidence of AIP. There is no doubt that these additives improve feed efficiency, and AIP is most often associated with high-performing cattle near finish. Feeding (higher levels of) monensin may help prevent it. There's not enough data to say for sure."

Scott MacGregor and his colleagues are convinced that AIP occurs as a result of multiple factors, but they strongly suspect genetics is one of them. Could cattle that are genetically programmed to excel in the feedlot be more at risk? If so, feeding management strategies may help lower the incidence of AIP.

"In the cattle-feeding business, feed efficiency is important. Conversion rates drive the bus. We have good, cost-effective tools, including potent feed additives, that enhance feed conversion, but their use can increase the risk of digestive disorders, like acidosis, and probably AIP. We can put up with it, and suffer the morbidity and mortality, or try to be proactive," says MacGregor.

"Our approach is to see if susceptibility goes along genetic lines, and see if we can designate risk for AIP. Maybe we can tell that pushing higher-risk cattle toward a 5.5 conversion rate (for example) is likely to be accompanied by a higher incidence of AIP. Could we control that by feeding for a little lower conversion rate? Where is the sweet spot? And can we reduce AIP enough to offset the cost of lower efficiency?"

MacGregor admits he has no answers yet. It's a work in progress. If it succeeds, it will further emphasize the importance of diligent feeding management.

"There's always room to improve," he adds. "You have to keep trying to do the common things uncommonly well."

Editor's Note: *Troy Smith is a freelancer and rancher from Sargent, Neb.*