Feedlot Stresses

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Feedlot stresses include a number of different things. When I think of feedlot stresses, mostly I think about myself and management, although that is not what we are going to talk about today. I will discuss dealing with government intervention in business. The stress of dealing with manpower utilization and so on certainly leads to severe stress in management. Today I'd like to spend a little time with you talking about digestive stress. It follows a little in line with what we're talking about so far.

How do we start cattle on feed and how do we optimize productivity in these animals through the feeding period? Whenever I attempt to tackle a topic like this, I usually come up with more questions than answers and that holds true in this presentation today, too. I've gone back through and done a little bit of review of the literature. Coupled with this, we will see some data that we extracted in a survey from our packing house, looking at rumen wall conditions after feeding.

Predominantly, when we talk about digestive stress in cattle, I'm only going to speak of it as it occurs subclinically, so we are not going to get into the area of clinical acidosis. However, I think that as well as some of these other factors, what we are going to talk about is only the peak of the iceberg. The big problem is never detected. I think the variability in intake in cattle, feeding cattle, starting on feed is enormous. I think some eat very little and some far too much.

We spent quite a bit of time talking about intake yesterday in our seminar and generally you can define it somewhere between 2-3% of body weight on a dry matter basis and I think that is just exactly a mean value. I think it might extend considerably on both sides of that range with individual animals. I think what we see manifest in the form of rumenitis, parakeratosis and the liver abscess complex are usually not detected clinically unless we get into liver abscess rupture and peritonitis. The same with acidosis. I think many of these cattle go through acidosis and never manifest it in any type of a clinical picture.

By rumenitis we are simply speaking of the inflammation of the rumen wall by parakeratosis, the degenerative changes in the mucosa; specifically, hardening, enlargement, darkening, occasionally clumping, and we see ulceration on the tips of the papillae. These clumps can vary from an inch to several inches in diameter. They are very hard and you cannot separate the papillae.

It is a real problem processing the tripe in a packing operation. The question that I am interested in is what effect does that have on performance? The only literature of value that I could find was the work done by Jensen back in the early '50's, 1954 specifically. He found it occurring in 8.8% of fed cattle. Well, I think certainly feeding conditions have changed enormously since then and we are going to see some updated figures on the incidence of these conditions.

The etiology of rumenitis I think most of you are familiar with. I will briefly mention them again–trauma, of course, can be one factor, rapid changes from high roughage to high concentrate diet, and feeding cattle on very high concentrate diets over very extended periods of time can also result in rumenitis. There is some hint of viral infections, vesicle-forming viruses, as being potential causes. With parakeratosis, the major factor appears to be processing the feed, specifically, fine grinding or pelleting.

As you go back through the literature and try to dig out what is available, there is not a lot that deals with the topic. Much of it is pretty much dated, say 20 years ago or even some past that. Hinders and Owens at Nebraska did some work with parakeratosis. They determined, at least in their trial, that it will inhibit the absorption of volatile fatty acids which could supposedly, at least theoretically, interfere with the energy utilization in an animal. So you might think about that a bit. Garrett and co-workers at California did some work with coarsely ground and then finely ground feed and they found that a coarsely ground feed certainly would give you a less affected rumen wall or a wall, say, that was in better shape than with very finely ground feed. Long oat hay would completely obliterate or at least improve the sores from parakeratosis. A research worker in Georgia, working on concentrate rations, improved parakeratosis by the adding of roughage, specifically long oat hay. A USDA researcher observed darkening and clumping in the rumen epithelia of steer fed soybean meal when compared with steers fed urea. That is the only report of that particular type of finding.

Haskins and his co-workers found more variation between individuals than they did between treatment groups dealing with this particular syndrome and I think this maybe gets us back to perhaps the variability in feed intake between cattle. Feeding sodium bicarbonate seemed to alleviate the severity of some of these signs.

I would like to discuss some of the survey work we did in our packing house. Dr. Bob Pearson from

Colorado State did the scoring of the lesions. I would like to just basically take you through the operation quickly and then show you the results of our survey.

Dr. Pearson recorded lesions on the dorsal curvature. We have some data there. We also saw some anterior dorsal lesions, but these were very minimal. The majority of the lesions are in this area. Occasionally we saw some on the pillars in the rumen wall. We did notice some color variation between rumens and we don't know if this is breed-related. There is some hint to it in the literature that it might be associated with some type of an iron metabolism factor but it is not well understood.

The classification system was basically based on severity of the lesions.

The anterior ventral pouch had the least severe lesions. In what we call an AV-2, there is a little larger area of involvement, little more severe pathology and larger areas of ulceration. In AV-3, there is very massive involvement over larger areas and lots of chronic granulation tissue and fibrosis.

Now we are going to talk a little about parakeratosis. The rating system was basically the same. AV-3 shows thickened and slightly more severe than the previous. I want to talk just a minute too about clumbing. This is the situation that I described to you where you get ulceration on the borders of the papillae, serum accumulating and then coalescing and causing the papillae to clump. These areas are all matted together. It would be very difficult for you to separate these areas with your fingers.

We actually surveyed approximately 13,000 cattle; 5,000 of these came from one feedlot, 8,000 came from another. The feeding programs between the feedlots did vary slightly.

I was very interested in the work of Jensen, and his initial work where he studied the effects of concentrate level and its effect on the development of rumenitis and the liver abscess complex where he was looking at a wide spread in the concentrate-roughage ratio. Say, from very little to a ration that contained a great deal. He saw very good correlation between the incidence of rumenitis and the development of liver abscesses with the type of ration and the higher concentrate ration.

We looked at a much narrower range. In two feedlots the difference in concentrate, our roughage ratio, was feedlot 1 had approximately 10% roughage in the ration and feedlot 2 about 7%. So a very narrow range. We are looking specifically to see if there would be any difference in rumen pathology. Of this total number of cattle that were slaughtered and examined, we looked at over 3300 rumens from the lot 1 cattle and 4500 rumens from lot 2 cattle for a total of about 7800 cattle. This is approximately 60%.

We did an analysis to determine if there was a difference in the incidence of lesions between the two lots. The only place that we saw statistical significance in difference was in the mild form of rumenitis, the AV-1 cattle from feedlot 1 containing the slightly higher, significantly higher level of rumenitis than did those from lot 2. Keep in mind that those are the cattle that received slightly higher roughage. In the area of parakeratosis in the dorsal curvature, we saw slightly higher, and it was significantly higher, incidence of parakeratosis in the dorsal curvature of those cattle from lot 1 also.

I think this maybe shows us a little bit about the fact that there might be more variation from animal to animal than there is between treatment. I find this very inconsistent with previous research findings. Overall, from the survey we found about 23% of the rumens from cattle slaughtered having the mild form of rumenitis, about 26% having the moderate degree of rumenitis and about 5% real severe rumenitis, for a total incidence of about 52-53% in those cattle, which is up considerably from Dr. Jensen's 8%. The incidence of parakeratosis is about 47% for the mild form, 22% for the moderate form and 1.5% for the severe form in the anterior ventral sac. For the dorsal column or dorsal curvature, about 16%, 8% and 3%, respectively; clumping about 10%. So certainly it appears that the incidence of these conditions is greatly increased over what it was in 1954 and I suspect a lot of that follows along with our current feeding practices.

The next thing we did with the data was try to establish how well it correlated with gain, feed efficiency, and incidence of liver abscesses. We performed a simple linear regression between rate of gain, feed efficiency and liver abscess percent and each of the levels of incidence of this particular, either rumenitis or parakeratosis. The measurement that we looked at is what we call a correlation coefficient. The very best correlation we have in any of the data was a value of 0.66 which is not very good. We'd like to see it over 0.9 to really say it is meaningful.

Just briefly, we did this on all of our various combinations for liver abscess, gain, and feed efficiency and found in general very poor correlation. I can't explain it. It is something that I would have aniticipated a very good degree of correlation. We did get good correlation between gain and feed efficiency, since feed efficiency is calculated from gain. It's just a difficult thing to say and, again, Dr. Jensen in his article didn't specify exactly how good his correlation was with the incidence of rumenitis and high grain rations. I suspect a lot of our problem is the fact that we had too narrow a set of test animals. They were too much alike so that we are just looking at only individual variation.

I think one thing that really comes to mind, going through this type of data, is that we see very definitely the effects of digestive stress in these rumens. There is no question about it. The animals have been insulted. I think some of it is the result of superior performance. You have to stress that animal a little bit to get maximum gain. I think a lot of the milder forms of rumenitis parakeratosis perhaps are the result of this animal's ability to take in vast quantities of feed and process it, and the severe cases, I think, on the other hand, have to be creating some

type of a performance inhibition in these animals. The incidence, of course, is low. We saw less than 5% generally and I imagine that this is just washed out in close-outs where you've got 5% of the cattle as poor doers. I think we all can walk out into a pen of fat cattle and see 5% that are poor doers.

In summarizing then, this is an area that I think we need to do more work in. I think that the literature is certainly limited in relationship to the wealth of information dealing with this particular area and how it affects production. I think it gives us some hints, some areas that we need to work in. But, I think that up in the kinds of rations we feed routinely in feedlot cattle, say, 80% in concentrate or better, we don't know very much about specifically which will get a little bit better performance, stress the digestive system a little less. If we stay off the 90-95% concentrate level a little bit, could we enhance performance by feeding 15% roughage instead of 10% or 5%?

We did a trial a couple of years ago where we looked at this on silage rations. We did it unfortunately in the summer months where we did feed a 5% silage ration and unfortunately, well, fortunately, we had the best gain on that particular ration–a significantly better gain in those cattle. We did go through and look at the rumens in those lots of cattle that we slaughtered, again to see how severely the rumens were stressed. We could find no real correlation in those cattle either. I think that we are looking at a problem that is somewhat of an aftermath but also in the severe forms can certainly interfere with performance.