selecting cases I mean we would eliminate any cow that might be diseased or might have a diseased reproductive tract, or we felt that the cow was not getting enough to eat with the proper food. In other words we treated those in which we really didn't know the cause. We have treated hundreds of cows during the last thirty years and we felt that we were getting benefit from using this type of treatment. Now, last year one of the speakers at the meeting said that there is no real good excuse for using a hormone to treat embryonic death. Well, he may be right and I'm not saying he is wrong. I'm not saying I'm wrong and when I say I, I mean there are quite a few veterinarians that I'm sure in this group who have used this treatment and found it successful. Some of you may not have found it successful, but I just wanted to bring this out because I think embryonic death is a very important situation with these valuable cattle and when you can save one, it is worth while.

Resistant Salmonellosis in Southern Alberta Feedlots

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Introduction

Reports on feedlot salmonellosis in North America over the past twenty years are scanty. During the period 1960-66, calf losses were reported in Arizona feedlots, stressors and inter-current diseases were considered to be important predisposing causes (9). Another report cites morbidity up to 30% in yearlings in a Colorado feedlot in 1961 (4). In the same feedlot in 1976, a mortality survey revealed 1.8% of total losses to be due to salmonellosis (5).

In Alberta, feedlot salmonellosis was not a problem until 1971 when a small number of deaths occurred in one feedlot. Dysentery was a prominent sign and at necropsy hemorrhagic enteritis was found. *Salmonella typhimurium,* resistant to chloramphenicol, tetracyclines, sulfonamides and streptomycin was consistently isolated. Disease continued at a low level for two years. In 1973 the use of an autogenous bacterin was reported to give good results and has been used since at this feedlot.

Losses in other feedlots continued to be very low and sporadic but in the spring of 1979, increased losses were experienced in two southern Alberta feedlots. A *Salmonella typhimurium* with a similar resistance pattern to the one mentioned previously was isolated. Antibiotic treatments which had been used with some success became relatively ineffective, losses mounted and the operators voiced much concern. An investigation was conducted to attempt to determine the causes of the outbreak and to suggest preventive measures.

History

Each of the two custom feedlots contained approximately 7,000 yearling cattle which originated either in the northern part of the province or in neighbouring Saskatchewan. Trucking distances from the various sales yards to the feedlots ranged from 320-800 km. Within two weeks of entry, animals were routinely branded and were vaccinated against infectious bovine rhinotracheitis, para-influenza-3 and clostridial diseases. The feed contained, for much of the feeding period, sufficient tetracycline to provide each animal with 75 mg. per day. The two feedlots were not particularly clean because of heavy rain the previous year. This had produced a build-up of mud or slurry in many of the pens. Furthermore, snow and rain during the spring of 1979 made the situation worse.

Death losses were normally between 1% and 2% but with the advent of salmonellosis, losses increased to between 2% and 2½%. In addition there were increases in cattle prices, in interest rates and in feed costs. Veterinary services and drugs became expensive and many treated animals developed chronic sicknesses. Finally, there was concern over the possibility of investigation by public health authorities. Losses continued at a lower level throughout the summer and fall of 1979 but rose to a second peak during the winter. Losses then gradually decreased in the spring of 1980 and finally stopped during midsummer.

Signs, Pathology and Microbiology

The common clinical signs were diarrhea and/or respiratory distress. Dysentery was less frequently noted. Necropsy studies conducted on many of the cattle with diarrhea or dysentery showed a severe fibrino-necrotic enteritis involving both large and small intestines. In these cases there was frequently a friable, yellow-grey, rope-like cast within the intestinal lumen. Necrosis and ulcers, sometimes associated with hemorrhage, were detected in the mucosa. Necrotizing or ulcerative abomasitis was often found along with the enteritis. In a small number of cases, ulcerations occurred in the omasal leaves adjacent to the omasal-abomasal junction.

Respiratory changes accompanied approximately half of the cases showing gastro-enteritis. A severe fibrinous pneumonia was most common while other findings included diphtheritic tracheitis (IBR) and necrotic laryngitis. Some cattle suffered a profuse thin yellow diarrhea; these did not have gastro-intestinal lesions but did have a severe fibrinous pneumonia. The feedlots were requested to submit to the laboratory dead animals or alternatively, intestine including ileo-cecal valve, liver and gallbladder, mesenteric nodes and lung from necropsied animals. These tissues were cultured and salmonellae were isolated from 46% of ileo-cecal valve tissues, from 42% of gallbladders and from 41% of mesenteric nodes. Only 5% of lungs yielded salmonellae.

Pasteurella hemolytica was the usual organism isolated from pneumonic lungs. Of a total of 248 animals necropsied over 15 months at both feedlots, resistant salmonellae were isolated from 93 (37.5%).

Gastro-intestinal tissues from cases showing abomasal and omasal ulcerations were forwarded for bovine virus diarrhea (BVD) virus isolation attempts. A variant BVD virus, the '73 Togavirus was isolated from five (1). Fecal samples were collected from a number of groups of newly introducted cattle and were cultured for salmonellae. Relatively few isolations were obtained but all were proved to be of the same multi-resistant strain.

Diagnosis

Diagnosis often proved difficult at the feedlot. There were a number of typical cases of enteric salmonellosis but problems arose in distinguishing between less typical salmonellosis and coccidiosis and possibly BVD. Further complications resulted with cattle suffering severe pasteurella pneumonia as these at times exhibited a thin, yellow diarrhea. These animals did not yield salmonellae on culture and did not contain gastro-intestinal lesions.

Laboratory diagnoses, based upon gross, histological and microbiological studies are tabulated as follows:

	Enteric Salm.	Enteric Salm. + Resp. Dis.	Resp. Dis. + Misc. Cond.	Total
Feedlot A	28	34	76	138
Feedlot B	15	16	79	110

Bovine virus diarrhea did not appear to be a significant or primary disease. The usual BVD virus was not isolated, but the variant virus, the '73 Togavirus, was recovered from five cases containing abomasal and sometimes omasal ulcers. These animals also suffered enteric salmonellosis. Coccidiosis proved to be a minor problem.

Treatment and Prevention

Until the onset of salmonellosis, therapy had given reasonably good success in each feedlot. The drugs utilized were antibiotics such as tetracyclines and chloramphenicol along with orally administered sulfonamides. After the onset of salmonellosis, many sick animals especially those showing diarrheic and respiratory signs, responded very poorly and many became chronically sick.

An autogenous bacterin, similar to the one which had been reported as beneficial in the earlier feedlot outbreak, was prepared and a single injection was given to each animal on arrival. Vaccination commenced in the late summer of 1979 and, while in one feedlot losses diminished, in the other they did not. Later on in December following severe weather fluctuations, losses increased to higher levels in both feedlots.

Treatments had been performed either in individual hospital pens or in small squeezes attached to the feeding pens. Initially, depending upon the severity of the sickness, cattle were returned to the feeding pen, maintained for a few days in the hospital pen or were turned into a recovery pen. When the presence of salmonellosis was established, the procedures were changed so that all cattle suspected of suffering from salmonellosis were segregated and did not again contact animals believed to be free of infection.

Attention was focused upon the type and the number of cattle entering the feedlots. The operators were advised to discourage entry of cattle which might have been traded at more than one auction sale. It was suggested that poorer cattle were more likely to be carriers of the infection or alternatively, would be more liable to succumb. Finally it was advocated that fewer cattle enter the feedlots in order that processing could take place more rapidly and cattle placed more quickly in permanent pens. Salmonellosis was most common in cattle 2-4 weeks after entry and it was felt that even though some were infected on entry, most became infected in the receiving pens. Thus, attention was directed towards cleaning those pens and moving cattle through them as quickly as possible. The factor over which there was no control was the weather which was deemed highly important since peaks of sickness tended to coincide with stressful climatic patterns.

Discussion

The multi-resistant *Salmonella typhimurium* involved in these outbreaks is similar to the organism recovered in recent years from dairy calves in Britain. These calves, born on widely scattered dairy farms are shipped, within a few weeks of birth, to collecting points or sales yards and they then go to intensively managed fattening farms. Excepting age differences, the stocking system is similar to that practiced in Canada and other countries with modern beef industries. Bacteriological and epidemiological studies in Britain lead to accusations that the veterinary profession had misused antibiotics and that selective pressures had given rise to dominance by resistant salmonellae (10, 11). The argument was also put forth that shipping and stressful husbandry practices might be as much or more to blame since the resulting debilitating effects predisposed animals to salmonellosis and to other diseases (8). In Alberta feedlots it is common practice to add antibiotics to feed and to treat sick cattle with chloramphenicol and other antibacterials however salmonellosis has not become a frequent problem.

Stressors, inherent with stocking practices may be augmented by delays, overcrowding and inadequate facilities or manpower etc. . . Irregular feeding is conducive to the proliferation of rumen salmonellae and to increased shedding (2). Such insults are multiplied if cattle are resold as might happen on upward markets. Finally, if there are too many, or if there is lack of regulation on cattle entering

feedlots, processing delays might be expected. Stressors such as prolonged confinement, handling and certain infections such as BVD may depress the immune response of the animals (3, 6).

Vaccinations may be detrimental. Human study has hown a decrease in respiratory disease which corresponded o a decrease in the numbers of vaccinations performed on naval recruits (7). Thus it is important to ensure that the vaccines and bacterins administered to cattle are all really required.

The acquisition of salmonellosis in most animals is related to the infective dose and so attention should be focused on the cleanliness of pens at auction sales and of cattle liners. At the feedlot attention should be directed towards cleanliness of receiving pens, holding pens and hospital pens.

Feedlot veterinarians should be aware of the existence of multi-resistant salmonella species and of the conditions under which they cause disease. It is obviously very difficult to predict the future but in Europe, disease caused by these organisms has been increasing. Veterinarians should play a greater role in feedlot animal health. The feedlot manager should be advised on the number and the type of cattle to be admitted. Feedlot construction should be examined, particularly the number and type of receiving and hospital pens. Only the necessary vaccinations should take place. Finally, there will be much public health concern and enquiry if resistant feedlot salmonellae are found to cause human disease. In such a situation intervention similar to that applied to the poultry industry might be expected.

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BVD Vaccination Trial

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Introduction

BVD is an infection of cattle that occurs around the world. Evidence of BVD infection is found in 70-90% of the cattle population.[1] This level of infection is determined by random serological surveys and you can appreciate that if 70-90% of the cattle population is seropositive, then BVD virus is quite prevalent. BVD may be a source of economic loss to the cattle industry by causing a clinical or subclinical infection.

Clinical Infection

Clinical cases of BVD are either enteric or reproductive problems. The enteric form causes economic loss in animals that die of acute or chronic diarrhea. The reproductive form may contribute to losses by causing abortions, repeat breeders or the birth of malformed calves usually following a BVD infection in non-immune heifers.[2]

The magnitude of the economic losses due to the reproductive form has not been accurately determined and this should be done so the cost of the disease can be compared to the cost of control.

Subclinical Infection

Reports, which have not been substantiated by controlled studies, suggest that subclinical BVD infections may lead to economic losses.

THis type of loss may be due to the fact that BVD is an immunosuppressive virus. BVD field virus is capable of decreasing the numbers of T & B lymphocytes in a calf, as well as impairing the functional capabilities of T & B cells and macrophages.[3]

This immune suppression following a mild or subclinical infection may occur and subsequently lead to secondary infections that impair growth rates in calves. This project was concerned with investigating the enteric and subclinical form of BVD infections. When considering the enteric form there are 2 epidemiological patterns involved; sporadic and enqootic.

Sporadic

These outbreaks occur from time to time in a herd and following the resolution of the disease, the herd remains free from clinical BVD for several years due to herd immunity.

After the total number of susceptible cattle increases due to the addition of susceptible replacements, i.e. calves, an outbreak may occur again.

Enzootic

This epidemiological pattern is much less common but it is being recognized more and more, especially in cow-calf operations.