December 1999

Lifetime Effects of Respiratory and Liver Disease on Cattle

Bill Epperson
South Dakota State University

Proceedings, The Range Beef Cow Symposium XVI
December 14, 15 and 16, 1999 Greeley, Colorado

LIFETIME EFFECTS OF RESPIRATORY AND LIVER DISEASE ON CATTLE

Bill Epperson, DVM, MS
Department of Veterinary Science
South Dakota State University

Significance of Respiratory and Gastrointestinal Disease

In cattle, the respiratory and gastrointestinal tracts are the main systems affected with disease. Bovine respiratory disease (BRD) contributes the majority of illness and death loss in the feedlot segment. Historically, 15-45% of feedlot cattle have been affected with BRD, with 1-5% of total cattle placed dying of BRD (Kelly 1986). Respiratory disease, alone, accounts for 44.1% of deaths in beef feedlot cattle (Vogel 1994). Apart from death loss, the Texas Ranch to Rail Program has suggested that clinical disease (most of which is BRD), even if treated successfully, results in treatment cost ($37.90/affected), decreased average daily gain (0.21 lb/d, for a 7.2 % decrease), decreased feed efficiency, and a decrease in quality grade (27% fewer choice). Together, these contribute an economic loss of approximately $88.00/ affected animal (Anonymous 1994).

Not restricted to the feedlot, respiratory disease accounts for 16% of the known causes of death in nursing calves, second in importance only to weather, and apparently increased from estimates made 5 years earlier (USDA/APHIS/VS 1994, 1997). Though the incidence of respiratory disease in unweaned calves is generally thought to be low, (1-2%), individual herds can have up to 65% of calves affected prior to weaning (Muggli-Cockett 1992). Unweaned calves affected with respiratory disease have a 36.3 lb reduction in weaning weight (Wittum 1994). This reduction, coupled with treatment cost, is the main economic loss from BRD in the cow-calf segment.

Disease of the gastrointestinal tract is the second most common cause of death in the feedlot (Vogel 1994). Acidosis is a common condition in feedlot cattle, and liver abscesses are a possible sequella. Slaughter surveys indicate that about 12% of beef cattle livers are condemned due to liver abscesses (Smith 1998).

Recently completed work indicates that productive losses associated with BRD are greater than previously thought. Liver abscesses continue to be a significant problem. With the beef industry desiring to maximize productive potential, minimize inputs, and potentially limit the use of antimicrobials, an understanding of the biology of disease is necessary to better control these problems. Understanding of the disease process, particularly respiratory disease, implicates events in early life as risk factors for subsequent illness.

Subclinical Bovine Respiratory Disease

BRD has been regarded as a visually obvious, clinical disease with signs including depression, lack of abdominal fill, altered breathing pattern, fever, coughing, and nasal discharge. It has generally been assumed that cattle identified with clinical BRD represent the

total BRD loss. However, recent work has indicated that respiratory disease does not always present as a clinical condition, and can occur as a subclinical disease. Subclinical BRD presents with no obvious physical signs of respiratory disease, yet the lung is affected with inflammation indicative of pneumonia.

Studies examining slaughter cattle indicate that lung lesions, indicative of prior respiratory disease, are common. Many cattle with lung lesions have no history of prior respiratory disease, and these cattle have assumedly undergone subclinical bovine respiratory disease. A series of investigations using a standardized lung examination system has been completed (Bryant 1996). Lungs of 1,326 slaughtered cattle from 4 feedlots were examined. Lung lesion prevalence, recorded in commercial slaughter plants, varied from 33.6% - 76.5%, depending on feedlot and pen within feedlot. Lung lesions suggestive of previous BRD were associated with decreased average daily gain (ADG) of between 0.07-0.14 lb/day (Bryant 1997).

Another study, using more intensive lung examination, recorded a lung lesion prevalence of 72% (Wittum 1996). Clinical BRD occurred in 29% of these calves in the feedlot, and in an additional 8% from birth to weaning. Though calves with clinical BRD were more likely to have lung lesions at slaughter, 68% of calves never observed with clinical BRD had lung lesions. Lung lesions were associated with a 0.167 lb/day reduction in ADG, while clinical BRD did not effect ADG. Clearly, clinical BRD represents only a portion of respiratory disease, and subclinical BRD may be a common, costly condition associated with feedlot performance. In addition to performance, marbling or muscle tenderness may be negatively impacted by lung lesions (Gardner 1998, Whitley 1999).

The "cause" of subclinical BRD is, at present, not known. It is not known when, in the life of the calf, lung lesions form. It is likely that the risk factors for subclinical BRD are similar to those for clinical BRD, but this is unproven. The lung lesions typical of inhalation pneumonia are the only lesion type associated with decreased ADG (Bryant 1997, Gardner 1998). Inhalation pneumonia is a well-recognized form of clinical BRD, suggesting that subclinical BRD is a different manifestation of the clinical BRD disease process.

Prevention of BRD

Risk factors associated with clinical BRD include many bacterial and viral agents (Dyer 1981). These agents are all very common in the cattle population, and many are natural residents of the upper respiratory tract of cattle. It is generally regarded that clinical BRD is the result of a breakdown of natural defense systems that allow these agents to establish in the lower respiratory system (Dyer 1982).

It appears that risk factors for clinical BRD are at work very early in life. Calves that do not ingest adequate colostrum shortly after birth have a 3.2 fold increased risk of disease from birth to weaning, a 3.1 fold increased risk of clinical respiratory disease in the feedlot, and a 0.088 lb decrease in feedlot ADG (Wittum 1995). Other diseases, such as calf diarrhea, lead to increased risk of pneumonia prior to weaning, as well as decreased growth rates (Ganaba 1995). Weather stress may decrease calf resistance to disease, and cow nutrition plays a yet poorly defined role in disease resistance (Corah 1993, Odde 1988). During the grazing season, trace

mineral nutrition on pasture may be important in preventing post-weaning BRD, and preconditioning/preweaning vaccination can help increase calf resistance to disease (Larson 1995). It has been suggested that a complete preconditioning program may decrease clinical feedlot respiratory disease 23%, and feedlot death by 40% (Cole 1985). Auction market owners and cattle buyers prefer calves with a history of vaccination administration, and these calves appear to receive modest premiums (Henderson 1995, King 1997). Calf sex, age distribution of the cow herd, year, and breed of dam may influence BRD in the postweaning period (Muggli-Cockett 1992).

Events occurring near or at weaning and transport play important roles in BRD. Weather, dust level, commingled sourcing, nutrition, processing, strategic medication, and transportation factors influence subsequent feedlot disease (MacVean 1986, Martin 1982, Wilson 1985, Cole 1985, Ribble 1994, Young 1995, Schumann 1991, Harland 1991).

Seemingly, clinical and subclinical BRD are caused by numerous risk factors that act in an additive manner. Some of these, like herd demographics, may be determined before birth, others, like colostral acquisition, are determined shortly after birth, while still others are determined at later times on the ranch of origin or at the feedlot. The relative contribution of each factor to lifetime performance is unknown. There is need to attempt to prioritize these risk factors, looking at lifetime calf performance as the outcome of interest.

Liver Abscesses

Liver abscesses are the result of transfer of bacteria, mainly *Fusobacterium necrophorum* (previously called *Sphaerophorus necrophorus*) to the liver from the rumen (Jensen 1954). This transfer occurs when normal rumen defenses are impaired, usually when the internal lining (mucosa) of the rumen is damaged. When mucosal damage occurs, often secondary to rumen acidosis, bacteria can enter the bloodstream and go to the liver. *Fusobacterium necrophorum* has some unique characteristics that enable it to evade normal liver defenses and establish an infection focus (Nagaraja 1996). The bacteria stimulate an inflammatory reaction of neutrophils, and the end result is a liver abscess. Liver abscesses may resolve with time and disappear, leaving only a scar. However, large abscesses or persistent insults are likely to remain present for some time.

Liver abscesses can be observed in slaughtered cattle. The prevalence varies by feedlot and even by pen within feedlot, but about 12% of beef cattle slaughtered have severe liver abscesses. Livers affected with severe abscesses are condemned for human consumption. This loss amounts to about 11 lbs, and constitutes an economic loss of $5-6 per affected carcass (Smith 1998).

Research has indicated that cattle affected with severe liver abscesses have decreased slaughter weight, decreased carcass weight, decreased fat thickness, and decreased dressing percentage (Brink 1990, Montgomery 1985). On a carcass basis, they have decreased dry matter intakes and lower feed efficiency (Brink 1990). There has been no published work that indicates quality grade is adversely affected by liver abscesses, though some studies have suggested this possibility (Montgomery 1985).

Liver abscesses are mainly associated with grain feeding, probably because grain feeding results in large increases in rumen *Fusobacterium necrophorum* populations (Tan 1994). Animals affected with liver abscesses exhibit no direct clinical signs of illness. Liver abscess control has traditionally relied on in-feed antimicrobials. The 1999 feed additive compendium lists 5 feed additives labeled for liver abscess control. Tylosin (Tylan®) at 60-90 mg/head/day has been popular, and reduces liver abscesses by 73% (Vogel 1994). Feed additive antimicrobials appear to work by simply decreasing rumen *Fusobacterium necrophorum* populations, thereby decreasing exposure to high bacterial levels (Nagaraja 1999). There has been recent concern about antibiotic use that has prompted the European Union to ban most feed additives, including tylosin. It is unknown whether this ban will be permanent in the EU, or whether some form of restricted use of feed additives may appear in the US. There is little scientific evidence to suggest that *Fusobacterium necrophorum* has acquired resistance to the common feed additive antibiotics (Lechtenberg 1998). However, this does not eliminate the risk of selecting for antibiotic resistance among other bacteria (Prescott 1993). Therefore, other methods of liver abscess control are needed.

Experimental vaccines have been used in attempt to decrease liver abscesses, with some past success (Garcia 1974). Recent reports have again indicated that vaccination can decrease liver abscess prevalence at slaughter (Terhaar 1996). Currently, a vaccine product is licensed for liver abscess control in the US. The product is reported to decrease liver abscesses at slaughter by 48-61%. In addition to liver abscess control, effective immunization against *Fusobacterium necrophorum* may also provide control for footrot, since footrot is also associated with *Fusobacterium necrophorum* infection.

Better control of liver abscesses is needed since they continue to be a prevalent condition of slaughter cattle and are associated with reduced performance and economic loss. Control through reliance on antimicrobials appears to be a politically dangerous position. Other methods of control, including immunologic, need to be examined. Such methods will likely require cooperation in the cow-calf and stocker sectors for maximum benefit.

References

Kelly AP, Janzen ED. A review of morbidity and mortality rates and disease occurrence in North American feedlot cattle. *Can Vet J* 1986; 27:496-500.

Vogel GJ, Parrott C. Mortality survey in feedyards: the incidence of death from digestive, respiratory, and other causes in feedyards on the Great Plains. *Comp Cont Ed Prac Vet* 1994; Feb: 227-234.

USDA/APHIS/VS. National Animal Health Monitoring System. Part III: Beef Cow/Calf Health & Health Management. 1994.

USDA/APHIS/VS. National Animal Health Monitoring System. Part II: Reference of 1997 Beef Cow-Calf Health & Health Management Practices. 1997.

Muggli-Cockett NE, Cundiff LV, Gregory KE. Genetic analysis of bovine respiratory disease in beef calves during the first year of life. *J Anim Sci* 1992; 70:2013-2019.

Wittum TE, Salman MD, King ME, et al. The influence of neonatal health on weaning weight of Colorado, USA beef calves. *Prev Vet Med* 1994;19:15-25.

Smith RA. Impact of disease on feedlot performance: A review. *J Anim Sci 1998;* 76:272-274.

Anonymous. 1993-94 Texas A & M Ranch to Rail North/South Summary Report. *Texas Agricultural Extension Service* 1994.

Bryant LK, Perino LJ, Griffin DD, et al. Lung lesions in feeder cattle at slaughter: a proposed method for lesion recording, and lesion effects on calf growth and carcass traits. *Proc Am Assoc Bovine Pract* 1996; 29:147-151.

Wittum TE, Woolen NE, Perino LJ, et al. Relationships among treatment for respiratory tract disease, pulmonary lesions evident at slaughter, and rate of weight gain in feedlot cattle. *J Am Vet Med Assoc* 1996; 209:814-818.

Gardner BA, Dolezal HG, Bryant LK, et al. Health of finishing steers: effects on performance, carcass traits and meat tenderness. *Animal Science Research Report*, Oklahoma Ag. Exp. Station 1998; P-965:37-45.

Whitley EM, McCollum FT, Montgomery DL, et al. Performance measures and pulmonary lesions at slaughter in feedlot cattle. *J An Sci Supplement* 1999; 232.

Bryant LK. Lung lesions in feedlot aged beef calves at slaughter. Masters Thesis, University Nebraska-Lincoln 1997.

Dyer RM. The bovine respiratory disease complex: infectious agents. *Comp Cont Ed Pract Vet* 1981; 3 (10): 43-51.

Dyer, RM. The bovine respiratory disease complex: a complex interaction of host, environmental, and infectious factors. *Comp Cont Ed Pract Vet* 1982; 4 (7):52-61.

Wittum TE, Perino LJ. Passive immune status at postpartum hour 24 and long-term health and performance of calves. *Am J Vet Res* 1995; 56 (9) 1149-1154.

Ganaba R, Bigras-Poulin M, Belanger D, et al. Description of cow-calf productivity in northwestern Quebec and path models for calf mortality and growth. *Prev Vet Med* 1995; 24:31-42.

Corah L. How pre-calving nutrition affects calf survival. *Large Animal Veterinarian* 1993. October; 8-10.

Odde KG. Survival of the neonatal calf. *Vet Clin of N Am Food An Prac* 1988; 4 (3):501-508.

Larson BL, Arthington J, Corah LR. 1995. Recognizing and treating copper imbalances in cattle. *Vet Med 1995;* June: 613-619.

Cole NA. Preconditioning calves for the feedlot. *Vet Clin of N Am Food An Prac* 1985;1 (2): 401-411.

Henderson G. Now is the time to add value to your calves. *Drovers Journal* 1995*;* August: 6.

King ME, Wittum TE, Odde KG. The effect of value added health programs on the price of beef calves sold through nine superior livestock video auctions in 1996. Report prepared for Pfizer Animal Health 1997;159-165.

MacVean DW, Franzen DK, Keefe TJ, et al. Airborne particle concentration and meteorologic conditions associated with pneumonia incidence in feedlot cattle. *Am J Vet Res* 1986;47 (12): 2676-2682.

Martin SW, Meek AH, Davis DG, et al. Factors associated with mortality and treatment costs in feedlot calves: the Bruce County Beef Project, years 1978, 1979, 1980. *Can J Comp Med* 1982;46:341-349.

Wilson SH, Church TL, Acres SD. The influence of feedlot management on an outbreak of bovine respiratory disease. *Can Vet J* 1985; 26:335-341.

Ribble CS, Shoukri MM, Meek AH, et al. Clustering of fatal fibrinous pneumonia (shipping fever) in feedlot calves within transport truck and feedlot pen groups. *Prev Vet Med* 1994; 21:251-261.

Young, C. Antimicrobial metaphylaxis for undifferentiated bovine respiratory disease. *Vet Clin of N Am Food An Prac* 1995; January:133-142.

Schumann FJ, Janzen ED, McKinnon JJ. Prophylactic medication of feedlot calves with tilmicosin. *The Veterinary Record* 1991;128:278-280.

Harland RJ, Jim KG, Guichon PT, et al. Efficacy of parenteral antibiotics for disease prophylaxis in feedlot calves. *Can Vet J* 1991;32:163-168.

Jensen R, Flint JC, Griner LA. Experimental hepatic necrobacillosis in beef cattle. *Am J Vet Res* 1954;January:5-14.

Nagaraja TG, Laudert SB, Parrott JC. Liver abscesses in feedlot cattle. Part I. Causes, pathogenesis, pathology, and diagnosis. *Comp Cont Ed Pract Vet* 1996; Sept: S230 - S256.

Brink DR, Lowry SR, Stock RA, et al. Severity of liver abscesses and efficiency of feed utilization of feedlot cattle. *J Anim Sci* 1990;68:1201-1207.

Montgomery TH. The influence of liver abscesses on beef carcass yields, in Special Technical Bulletin 1985. Canyon, TX, West Texas State University.

Tan ZL, Nagaraja TG, Chengappa MM. Selective enumeration of Fusobacterium necrophorum from the bovine rumen. Appl Environ Micro 1995; 60:1387-1389.

Vogel GJ, Laudert SB. The influence of Tylan® on liver abscess control and animal performance - a 40 trial summary. *J Anim Sci* 1994;72, Suppl. 1:293.

Nagaraja T, Sun Y, Wallace N, et al. Effects of tylosin on concentrations of *Fusobacterium necrophorum* and fermentation products in the rumen of cattle fed a high-concentrate diet. *Am J Vet Res* 1999;60 (9):1061-1065.

Lechtenberg KF, Nagaraja TG, Chengappa MM. Antimicrobial susceptibility of *Fusobacterium necrophorum* isolated from bovine hepatic abscesses. *Am J Vet Res* 1998;59 (1):44-47.

Prescott JF, Baggot JD. *Antimicrobial Therapy in Veterinary Medicine, 2nd edition,* 1993. ISU Press, Ames, IA.

Garcia MM, Darward WJ, Alexander DC, et al. Results of a preliminary trial with *Sphaerophorus necrophorus* toxoids to control liver abscesses in feedlot cattle. *Can J Comp Med* 1974; 38:222-226.

Terhaar BL, Lechtenberg KF, Hale RL. *Fusobacterium necrophorum* Bacterin/Leukotoxoid efficacy in the control of naturally occurring hepatic abscesses in cattle. *Agri-Practice* 1996;17(7):15-17.