Involvement of microbial respiratory pathogens in acute interstitial pneumonia in feedlot cattle

Guy H. Loneragan, BVSc, MS; Daniel H. Gould, DVM, PhD; Gary L. Mason, DVM, PhD; Franklyn B. Garry, DVM, MS; Garold S. Yost, PhD; Delbert G. Miles, DVM, MS; Bruce W. Hoffman, DVM; Leon J. Mills, DVM

Objective—To evaluate viral and bacterial respiratory pathogens and Mycoplasma spp isolated from lung tissues of cattle with acute interstitial pneumonia (AIP) and cattle that had died as a result of other causes.

Sample Population—186 samples of lung tissues collected from cattle housed in 14 feedlots in the western United States.

Procedure—Lung tissues were collected during routine postmortem examination and submitted for histologic, microbiologic, and toxicologic examinations. Histologic diagnoses were categorized for AIP, bronchopneumonia (BP), control samples (no evidence of disease), and other disorders.

Results—Cattle affected with AIP had been in feedlots for a mean of 127.2 days before death, which was longer than cattle with BP and control cattle. Detection of a viral respiratory pathogen (e.g., bovine respiratory syncytial virus [BRSV], bovine viral diarrhea virus, bovine herpesvirus 1, or parainfluenza virus 3) was not associated with histologic category of lung tissues. Bovine respiratory syncytial virus was detected in 8.3% of AIP samples and 24.0% of control samples. Histologic category was associated with isolation of an aerobic bacterial agent and Mycoplasma spp. Cattle with BP were at greatest risk for isolation of an aerobic bacterial agent and Mycoplasma spp.

Conclusions and Clinical Relevance—Analysis of these results suggests that AIP in feedlot cattle is not a consequence of infection with BRSV. The increased risk of isolation of an aerobic bacterial agent from cattle with AIP, compared with control cattle, may indicate a causal role or an opportunistic infection that follows development of AIP. (Am J Vet Res 2001; 62:1519–1524)

By far the most important cause of morbidity and mortality in cattle in feedlots in the United States is bovine respiratory disease complex (BRDC).1,3 This disease complex is multifactorial and results from an interaction of stressors, susceptible cattle, and infectious respiratory pathogens that ultimately give rise to bacterial bronchopneumonia (BP).4

Quite distinct from BRDC, a second important respiratory disorder of feedlot cattle is acute interstitial pneumonia (AIP). Acute interstitial pneumonia describes a pattern of lesions that results from injury to Clara and alveolar type-I epithelial cells. Characteristic lesions of AIP include formation of hyaline membranes and proliferation of alveolar type-II epithelial cells.5 Acute interstitial pneumonia manifests with a sudden onset of respiratory distress and is frequently fatal.6

The case fatality rate of cattle with AIP may exceed 60%, and the disease appears to be refractive to available therapeutic agents. The USDA National Animal Health Monitoring System (NAHMS) reported that 97.6% of feedlots had at least 1 case of AIP in the year ending Jun 30, 1999.7 Participants in the NAHMS study reported that 3.1% of all cattle placed in feedlots during the same time period developed AIP. With the exception of BRDC, AIP affected more cattle than any other disorder. Although the criteria used by feedlot personnel to diagnose AIP were not described in the NAHMS study, it is apparent that AIP is perceived to be a substantial health concern by the feedlot industry.

More than 1 causal mechanism may lead to AIP. These include effects of bioactivated forms of 3-methylindole (3MI)8,9 or 4-ipomeanol and perilla ketones10,11 as well as infection with bovine respiratory syncytial virus (BRSV).8,12 Epidemiologic characteristics and pathogenesis of AIP in pastured cattle (fog fever, cow asthma, or acute bovine pulmonary edema and emphysema) have been described.10,11 Pasture-associated AIP results from an abrupt dietary change when cattle are moved from dry dormant pasture to lush pasture, which gives rise to large increases in the amount of ruminal 3MI that is generated.8,9,14-16 Feedlot-associated AIP, also known as dust pneumonia or allergic pneumonia, typically affects cattle that are close to market weight during a period in the feedlot when they are presumably already adapted to high-concentrate diets.17,18 Although the lesions are histologically identical to those of acute bovine pulmonary edema, the cause of feedlot-associated AIP remains obscure.

Because BRSV may cause lesions similar to those of AIP, an infectious cause for feedlot-associated AIP often has been suggested.19 Collins et al identified BRSV in lung tissues from 11 of 15 cattle with AIP and from 3 of 18 cattle with other disorders. In that study, AIP was significantly (P = 0.01) associated with isolation of BRSV. However, other investigators have not...
Materials and Methods

Study design—A prospective case-control study was performed to evaluate associations of microbial findings for lung tissues of cattle with AIP. Feedlots were enrolled in the study by their consultant veterinarians, and sample collection was performed during routine postmortem examinations of cattle during the period of May 15 to Sep 15, 1999. At the time of sample acquisition, information pertaining to feedlot of origin, sex, days from arrival at the feedlot until death, and suspected cause of death were recorded.

Collection of samples—In accordance with typical feedlot practices, cause of death was assigned by the consulting veterinarians or trained feedlot personnel. Those who performed postmortem examinations were instructed in techniques for appropriate collection of samples. Six samples were obtained from the right lung of each animal. Tissue samples (approx 5 x 5 x 1 cm) included 2 adjacent samples from the dorsal aspect of the caudal lobe (1 for toxicologic examination and 1 for histologic examination), 3 adjacent samples from the caudal margin of the middle lobe (2 for microbiologic examination and 1 for histologic examination), and 1 sample from the apex of the middle lobe (for histologic examination). Tissues were collected in a specified order to minimize the possibility that formalin would contaminate tissue samples submitted for microbiologic and toxicologic evaluation. One sample from the caudal lung lobe and 2 samples from the base of the middle lobe were collected, and each of these samples was placed in a separate air-tight plastic bag and frozen. Then, the remaining 3 samples were collected and fixed in neutral-buffered 10% formalin. At regular intervals, 1 of the investigators (GHL) visited participating feedlots or their veterinary consultants and retrieved samples. Frozen tissue samples were sectioned at 10-mm intervals. A block for light microscopic examination was selected from each section (3 sections/animal) and embedded in paraffin, sectioned at a thickness of 5 µm, and stained with H&E. Some sections were stained with periodic-acid Schiff (to enable evaluation of protein transudate) or phosphotungstic acid hematoxylin (to enable evaluation of hyaline membranes). Two investigators (DHG and GLM) examined lung tissues, using light microscopy, and their findings were recorded. Criteria for diagnosis of AIP included multifocal or diffuse microscopic lesions of alveolar sepal edema with serofibrinous exudation into alveolar spaces or formation of hyaline membranes with or without admixed chromatoid strands or type-II epithelial hyperplasia. Cattle with this pattern of injury evident in 1 or more sections of lung tissue were categorized as AIP, even when other concurrent disease processes (such as BP) were evident. The at time of histologic examination of lung tissue, the pathologists were not aware of the microbial findings.

Histologic examination—Formalin-fixed tissue samples were sectioned at 10-mm intervals. A block for light microscopic examination was selected from each section (3 sections/animal) and embedded in paraffin, sectioned at a thickness of 5 µm, and stained with H&E. Some sections were stained with periodic-acid Schiff (to enable evaluation of protein transudate) or phosphotungstic acid hematoxylin (to enable evaluation of hyaline membranes). Two investigators (DHG and GLM) examined lung tissues, using light microscopy, and their findings were recorded. Criteria for diagnosis of AIP included multifocal or diffuse microscopic lesions of alveolar sepal edema with serofibrinous exudation into alveolar spaces or formation of hyaline membranes with or without admixed chromatoid strands or type-II epithelial hyperplasia. Cattle with this pattern of injury evident in 1 or more sections of lung tissue were categorized as AIP, even when other concurrent disease processes (such as BP) were evident. The at time of histologic examination of lung tissue, the pathologists were not aware of the microbial findings.

Virologic examination—Virus identification was performed, using fluorescent antibody detection and virus isolation techniques, at the Colorado State University Diagnostic Laboratory. Samples of frozen lung were sectioned at a thickness of 6 µm and stained, using a direct method, with fluorescent-conjugated antibodies against bovine herpesvirus 1 (BHV-1), BRSV, bovine viral diarrhea virus (BVDV), and parainfluenza virus 3 (PI-3). Sections then were illuminated with a xenon epifluorescent lamp and observed for specific viral fluorescence.

For virus isolation, lung tissue was homogenized in minimal essential media (MEM) at a concentration of approximately 10% lung tissue (wt/vol). The homogenate was placed onto a confluent bovine turbinate (BT) cell-culture monolayer for 1 hour at 37 C and then washed. Minimal essential media containing antimicrobials and 10% fetal bovine serum (vol/vol) were added. Cells were observed for viral cytopathic effects for 7 days. Monolayers that did not have cytopathic effects were passaged onto fresh BT monolayers and observed for an additional 7 days, and any of these monolayers that did not have cytopathic effects were then passaged again onto fresh BT monolayers at 3 days, at which time they were fixed, using room-temperature acetone for 10 minutes. Monolayers then were stained by an avidin-biotin immunohistochemical technique, using antibodies against noncytopathic strains of BVDV. If cytopathic effects were observed, viruses were identified by use of fluorescent-conjugated antibodies against BHV-1, BRSV, BVDV, and PI-3. Fluorescence was detected as described previously.

Microbiologic culture—The remaining sample of lung tissue for microbiologic examination was thawed and sectioned, using alcohol-sterilized scissors. Interior samples of lung tissues were aseptically obtained. A sterile swab was placed on the sectioned surface and directly streaked onto plates containing blood, Columbia agar, and MacConkey agar. Blood and MacConkey agar plates were incubated aerobically, and Columbia agar plates were incubated microaerophobically (10% CO2) for 18 to 24 hours at 35 C. Bacterial isolates were identified in accordance with standard laboratory protocols used at the Colorado State University Veterinary Diagnostic Laboratory. Mycoplasma spp were isolated from lung tissues, using a second swab-specimen inoculated into Friis broth. The broth was incubated for 24 hours at 35 C and 5% CO2; it was then filtered (0.45 µm), swabbed on Friis agar plates, and incubated for 5 days at 35 C and 5% CO2. Mycoplasma organisms were speciated as M bovis or other species, using polymerase chain reaction techniques in accordance with protocols used at the aforementioned veterinary diagnostic laboratory.

Statistical analysis—Histologic findings were categorized into 1 of 4 groups (samples with AIP, samples with BP, control samples [congestion, edema, and atelectasis but no microscopic anomalies], and samples with other disorders). Descriptive statistics were calculated for all categories, whereas results for the last category were excluded from other statistical analyses. Viral detection methods (virus isolation and fluorescent antibody testing) were interpreted as tests performed in parallel (or positive results for either test would cause a classification of positive). Results of aerobic bacterial culture were considered positive when Mannheimia haemolytica, Pasteurella multocida, or Haemophilus somnus were isolated. Other isolates such as enterobacteria and Actinomyces pyogenes were considered postmortem contaminants and not included in the analyses. Frequency counts by virus, aerobic bacteria, and Mycoplasma spp were tabulated and validated.

A commercially available statistical program was used to generate estimates (and SEM), test statistics, confidence
limits, and \( P \) values, when applicable. Number of days from arrival at the feedlot until death was compared among histologic categories, using ANOVA techniques. Feedlot was considered a random variable, and Satterthwaite approximations for the denominator degrees of freedom were used to test estimates of least-squares means.22 Means were compared, using the Tukey method of adjustment. For count data, contingency tables were analyzed to test the compatibility of the data with the null hypothesis that there was not an association of histologic category (AIP, BP, and control) with isolation of BRSV, BVDV, BHV-1, PI-3, concomitant BRSV and BVDV, aerobic bacteria, or Mycoplasma spp. Probability values were calculated from the \( \chi^2 \) statistic or Fisher exact test, when appropriate. When the data provided evidence of an association (\( P \leq 0.05 \)), logistic regression was used to generate odds ratios (OR) for cattle with AIP and BP, compared with control cattle, and for cattle with BP, compared with cattle with AIP. The 95% confidence intervals (CI) were calculated, using partially maximized likelihood functions.22

**Results**

Samples from 186 cattle were collected during the study. Fourteen feedlots submitted samples of lung tissues from at least 1 animal during the study period. Feedlots were located in Colorado (\( n = 4 \)), Kansas (3), Nebraska (4), and Texas (3). These feedlots submitted samples of lung tissue from 101, 49, 14, and 22 cattle, respectively. Three feedlots submitted samples from only 1 animal, and 3 feedlots submitted samples from > 20 cattle. One feedlot supplied samples from 60 cattle. Histologic groupings of lung tissues were recorded (Table 1). They included AIP (\( n = 108 \)), BP (50), control samples (25), and other disorders (3). Acute interstitial pneumonia was confirmed in at least 1 animal from all participating feedlots. One feedlot provided 29 samples that were confirmed as AIP. Control samples were submitted from 7 feedlots. Nine feedlots submitted samples from at least 1 animal that was classified as BP. Two feedlots each provided samples from 1 animal with BP, and 1 feedlot provided samples from 23 cattle with BP.

Least-squares means ± SEM for number of days from arrival at a feedlot until time of death were 127.2 ± 8.3, 98.6 ± 9.8, and 84.0 ± 11.8 for AIP, BP, and control samples, respectively. Values for this variable ranged from 23 to 249 (AIP), 5 to 236 (BP), and 3 to 145 (control samples). Cattle that died as a result of AIP were in feedlots for a significantly (\( P = 0.01 \)) longer period than cattle that died as a result of BP or control cattle. Number of days from arrival at a feedlot until death did not differ significantly (\( P = 0.40 \)) between control cattle and cattle with BP.

Bovine respiratory syncytial virus, BVDV, or BHV-1 was identified in 56 (30.1%) samples of lung tissue. Parainfluenza virus 3 was not identified in any cattle. Bovine herpesvirus 1 was identified in lung tissue from 2 cattle with AIP and 3 cattle with BP. We did not detect evidence to support an association of histologic category with detection of BHV-1 (\( P = 0.33 \)).

Twenty-one (11.3%) cattle had positive results for BRSV. Similar percentages of cattle had positive results for BRSV when tested by use of fluorescent antibody detection, virus isolation, or both techniques (Table 2). Bovine respiratory syncytial virus was detected in 24.0% of control samples, 8.3% of AIP samples, and 12.0% of BP samples. There was weak evidence for an association of histologic category with isolation of BRSV (\( P = 0.09 \); Table 3).

Bovine viral diarrhea virus was identified in 38 (20.4%) samples. Similar percentages of cattle had positive results for BVDV when tested by use of fluorescent antibody detection, virus isolation, or both techniques (Table 2). Seven (28.0%) control samples, 19 (17.6%) AIP samples, and 11 (22.0%) BP samples had positive results when tested for BVDV. The data did not support a hypothesis that detection of BVDV varied with histologic category (\( P = 0.47 \); Table 3).

Seven cattle had positive results when tested for both BRSV and BVDV. These included 1.9% of cattle with AIP, 6.0% of cattle with BP, and 8.0% of control cattle. We did not detect evidence for an association of concurrent BRSV and BVDV infection with histologic category (\( P = 0.18 \)). None of the cattle with other disorders had positive results when tested for BRSV and BVDV.
BVVD. One animal that had BP had positive results for BHV-1 and BVVD.

Overall, 37 aerobic bacterial isolates were identified as *M. haemolytica* (n = 9), *P. multocida* (26), or *H. somnus* (2; Table 4). These isolates were cultured from 34 (18.28%) cattle. There were 3 cattle from which >1 isolate was cultured (*P. multocida* and *H. somnus* were isolated from 1 animal with AIP, and *M. haemolytica* and *P. multocida* were isolated from 2 cattle with BP). Isolation of aerobic bacteria varied significantly (P = 0.01) with histologic category (Table 3). One (4.0%) control sample, 15 (13.9%) AIP samples, and 18 (36.0%) BP samples had positive results for microbial culture.

The odds of an aerobic bacterial isolate being cultured from an AIP sample did not differ significantly (P = 0.20) from the odds of a control sample having an aerobic isolate (OR, 3.9; 95% CI, 0.73 to 71.7). Samples from cattle with BP were at significantly (P = 0.01) greater risk for isolation of aerobic bacteria than samples from cattle with AIP (OR, 3.5; 95% CI, 1.6 to 7.8). *Mycoplasma* spp were cultured from 63/186 (34.4%) cattle. Isolation of *Mycoplasma* spp was significantly (P = 0.01) associated with histologic category (Table 3). The proportions of control, AIP, and BP samples that had positive results when cultured for *Mycoplasma* spp were 16.0, 30.6, and 50.2%, respectively. We did not detect evidence that samples from cattle with AIP were at increased risk for isolation of *Mycoplasma* spp, compared with control samples (OR, 2.3; 95% CI, 0.8 to 8.4; P = 0.15), whereas samples from cattle with BP were at increased risk, compared with control samples (OR, 5.7; 95% CI, 1.8 to 21.7; P = 0.01). Risk of isolation of *Mycoplasma* spp was significantly (P = 0.01) greater for BP samples than AIP samples (OR, 2.6; 95% CI, 1.2 to 4.9).

**Discussion**

Acute interstitial pneumonia describes a distinct pattern of histologic lesions that includes edema of the alveolar wall and interlobular spaces, accumulation of protein-rich fluid within the alveolar spaces, formation of hyaline membranes, and proliferation of alveolar type-II epithelial cells. Hemorrhage from pulmonary capillaries also may be evident and probably reflects the severity of tissue injury. Grossly, lesions may be diffuse, but the caudodorsal aspect of the lungs is typically the most severely affected. During postmortem examination, lungs fail to collapse, have a rubbery texture, and exude clear edema fluid from cut surfaces. Interlobular edema often is marked, and emphysematous bullae may be evident. Lobules vary in the extent to which they are affected, and an apparently normal lobule often may be adjacent to an affected lobule. When variation is evident within the lungs of an animal, it results in a distinctive checkerboard appearance.

Although there has been considerable interest in infectious organisms and feedlot-associated AIP, we did not find any evidence to support an association between infection attributable to commonly identified respiratory pathogens and AIP of feedlot cattle. Hjerpe reported that BP was detectable in a greater proportion of cattle with AIP than in cattle that died as a result of other causes, and cattle with AIP were more likely to have been administered therapeutic agents. In the study reported here, gross evidence of concurrent BP was not recorded. Although not significantly (P = 0.20) different, the only aerobe cultured from the 25 control samples was a single isolate of *M. haemolytica*, whereas 13.9% AIP samples had positive results for at least 1 aerobic bacteria. Cattle suspected of being affected by AIP in the participating feedlots were treated with antimicrobials in accordance with their treatment protocols. Therefore, aerobic culture may have been unsuccessful in some AIP cattle with bacterial infections of the respiratory tract because of residual antimicrobial effects. Most of the isolates were *P. multocida*, although *M. haemolytica* (n = 2) and *H. somnus* (2) were isolated. It is unclear from this study whether bacterial infection of the respiratory tract occurred prior to the development of AIP. Because proliferation of alveolar type-II epithelial cells was evident in some cattle, these cattle must have survived an initial AIP episode for at least 2 to 3 days. Bacterial colonization of the respiratory tract would presumably be more likely in cattle with severe pulmonary injury than in healthy cattle. The susceptibility to bacterial colonization would also likely increase as time-at-risk increased (eg, duration of respiratory distress attributable to induced pulmonary injury increased). Although an association was not detected in the study reported here, it is still possible that aerobic bacterial pathogens may have a causal role in development of feedlot-associated AIP; however, they may simply be opportunistic invaders. An opportunistic infection could have contributed to the death of some of the cattle without involvement in the pathogenesis of AIP.
Etiologic factors responsible for feedlot-associated AIP are not clearly defined. This disease reportedly affects cattle during the later portions of the finishing period. The data reported here support this finding, with the mean interval from arrival at a feedlot until death as a result of AIP being 127.2 days. At this stage, cattle presumably are adapted to their finishing diet, and, in contrast to pasture-associated AIP, sudden dietary changes are unlikely. Because root crops are not commonly fed to feedlot cattle, it is doubtful that feedlot-associated AIP results from contamination of the diet with 4-ipomeanol. Furthermore, root crops were not included in the diets of cattle in this study. If perilla ketones were involved, then those cattle with the greatest exposure to purple mint would have been at greatest risk for AIP. Purple mint could potentially contaminate the roughage component of the diet. The proportion of roughage fed to new arrivals at feedlots is greater than that fed to established cattle, but AIP typically affects cattle on low-roughage diets (eg, those at lowest risk of exposure to purple mint). Perilla ketones are not likely to be involved in feedlot-associated AIP. Although sudden increases in dietary ketones are not likely to be involved in feedlot-associated AIP, the greatest exposure to purple mint would have been at greatest risk for AIP.

The potential role of a subclinical bacterial infection of the lungs or liver in BRDC has been suggested. It has been proposed that a bacterial nidus may result in increased concentrations of proinflammatory cytokines, particularly tumor necrosis factor (TNF-α) and interleukin (IL)-1β, thereby priming the lungs for AIP. This hypothesis has been supported by data in reports of humans with acute respiratory distress syndrome (ARDS), which shares many histologic lesions characteristic of AIP in cattle. Patients at risk of ARDS who subsequently develop ARDS have higher concentrations of TNF-α and IL-1β in bronchoalveolar lavage fluid than those who do not develop ARDS. Concentrations of TNF-α and IL-1β are increased in lung tissues and lavage fluid obtained from calves experimentally infected with *M hemolytica*. It may be possible that TNF-α and IL-1β prime the lungs for an AIP trigger such as an increased production of 3-methyleneindolenine. More research is required to evaluate the association between bacterial infections of the lungs or other organs, particularly the liver, with feedlot-associated AIP.

Bovine respiratory syncytial virus has been implicated in AIP, because BRSV-induced disease can cause clinical signs and pathologic changes similar to those induced by AIP. Calves experimentally challenged with BRSV developed acute respiratory distress and had gross and microscopic lesions similar to those of AIP. In 1 study, BRSV was identified in 11 of 15 (73.3%) cattle with feedlot-associated AIP, whereas it was only identified in 5 of 18 (27.7%) cattle that died as a result of other causes. Data collected by others have not supported an association of AIP with infection attributable to BRSV. In the study reported here, we identified a weak association (P = 0.09) between histologic category and infection with BRSV. However, control samples had the greatest proportion of positive results for BRSV (24.0%), whereas AIP samples had the lowest proportion of positive results (8.3%). Analysis of these data indicates that BRSV commonly infects feedlot cattle without inducing lesions identifiable by use of standard light microscopy techniques. Although only 25 cattle served as control animals, the prevalence estimate of BRSV infections in cattle without observed pulmonary pathologic changes can be adequately estimated. The prevalence of BVDV infection in similar cattle probably cannot be estimated accurately, because infection with BVDV may be associated with increased risk of death as a result of nonrespiratory disorders such as enteric disease. Infection with BRSV would not likely lead to increased risk of death as a result of nonrespiratory disorders. The SE of the estimate of BRSV infection was 8.5%. On the basis of the data reported here, the prevalence estimate and 95% CI for BRSV infection in cattle without pulmonary disease were 24.0% and 7.3 to 40.7%, respectively.

Substantial differences exist in clinical signs and signalment of BRSV and feedlot-associated AIP. These include calves that develop severe pyrexia and are obtunded when BRSV-associated respiratory tract disease is induced experimentally, whereas cattle with AIP are typically not febrile and are alert. In fact, feedlot personnel have often noticed that AIP-affected cattle can be unusually aggressive. Acute interstitial pneumonia affects feedlot cattle with a substantial number of days in a feedlot (127.2 days in the study reported here). Infectious respiratory tract disease such as that caused by BRSV typically causes greatest morbidity in newly arrived cattle. Furthermore, cattle with AIP in our study had the smallest proportion of samples with positive results when tested for BRSV. Therefore, most cases of AIP seen in feedlot cattle are not likely to be induced by BRSV.

In situ coinfection of alveolar macrophages with BRSV and BVDV results in a greater inhibition of specific macrophage function than infection with BRSV or BVDV alone. Bovine viral diarrhea virus and BRSV coinfection was identified in 4.0% of cattle in the study reported here. We did not detect evidence that coinfection was associated with histologic grouping; however, the number of cattle with positive results was limited. The greatest proportion of BRSV-BVDV coinfection was identified in control samples (8.0%). It is possible that there is synergy between these 2 viruses in vivo, but it was not detected in this study.

The role of *Mycoplasma* spp in feedlot-associated BRDC is unclear. Samples from cattle with BP were at significantly (P = 0.01) greater risk to have a positive result for *Mycoplasma* culture, compared with samples from cattle with AIP or control samples. The OR were 2.6 and 5.7, respectively. Fifty-two percent of BP samples were positive when cultured for *Mycoplasma* spp. It is unclear whether these organisms serve a primary role in the pathogenesis of BP or are merely opportunistic invaders. Sixteen of 50 cattle with BP had been at feedlots for ≥120 days. Therefore, some of the cattle that developed fatal BP were presumably close to marketing. Commonly used antimicrobials in cattle close

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to marketing include those with a short withdrawal period prior to slaughter such as sodium ceftiofur. This cell-wall inhibitor would not be effective against mycoplasmas, but this may be inconsequential if mycoplasmas are not involved in the pathogenesis of BP, do not propagate disease as an opportunistic pathogen, or when effective treatment of aerobic bacteria leads to resolution of infection attributable to Mycoplasma spp. Additional research is required to better understand the ecologic ramifications of mycoplasmas in feedlots and the role Mycoplasma spp play in BP.

SAS System for Windows, release 8.00, SAS Institute Inc, Cary, NC.

References