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A Method for Recording Pulmonary Lesions of Beef Calves at Slaughter, and the Association of Lesions With Average Daily Gain

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Abstract

A method to record lung lesions in commercial packing plants was developed as part of this observational study. Data from calves born and raised at the Roman L. Hruska U.S. Meat Animal Research Center (MARC) were used to develop the scoring method. The method was then applied to calves from three feedlots in the private sector.

Lung lesion rates in MARC calves at slaughter ranged from 31 to 66% by sire line and feedlot pen. Lesions at slaughter were associated with a decrease of 0.057 lb in average daily gain (ADG). Lesions were grouped into categories: lesions that were sequella to cranial ventral bronchopneumonia (CVBP), other lesions, and no lesions. This grouping resulted in CVBP lesions being associated with a 0.073 lb reduction in daily gains, and the effect of other lesion types were not significant. Neither lobar location nor the amount of lesion present was associated with daily gains. Clinically diagnosed respiratory disease was not associated with increased frequencies of lesions at slaughter.

Lung lesion rates in calves from the private sector ranged from 33 to 77% at the pen level. Thirty percent of these calves had CVBP type lesions at slaughter that were associated ($P < 0.01$) with a decrease in ADG. There was a significant interaction between feedlot pen and CVBP type lesions, with an overall effect on ADG ranging from a reduction of 0.14 lb to 0.65 lb.

Introduction

Bovine respiratory disease (BRD) resulting in morbidity and mortality is common, although recent evidence suggests that non-clinical BRD is also common and results in lost production. Previous reports suggest pulmonary lesions at slaughter are associated with decreased production. Wittum et al.[12] reported 72% of 469 crossbred steer calves had pulmonary lesions at slaughter. Thirty-five percent of the population was treated for clinical respiratory disease from birth to slaughter and 78% of this sub-population had pulmonary lesions. However, 68% of the steers never showing clinical disease had similar lesions. Cattle with pulmonary lesions at slaughter had reductions of 0.17 lb in ADG for the feeding period, suggesting lesions at slaughter can reflect the occurrence of disease which was significant enough to decrease production independent of previous clinically diagnosed disease. However, the most efficient and useful methods to quantify lesions in commercial packing plant settings have not been defined. Our objectives were to develop a standardized method for measuring lesions at slaughter in a commercial packing plant environment, to identify specific types of lesions associated with reduced ADG, and apply the final method to commercially fed cattle.

Materials and Methods

Population requirements

Populations of calves from multiple feedlots were identified to address the study objectives. Calves with extensive production and health records were required for the developmental phase, and calves with unique individual identification and feedlot entry weights were required for the application phase.

MARC population

Four hundred thirty-nine steer and heifer calves born during the spring of 1995 originating in large pa-

ternal half-sib families from the Bovine Genome Mapping project at the Roman L. Hruska U.S. Meat Animal Research Center (MARC) were used for the developmental phase. Management protocol and health monitoring of this population are similar to those previously described for the MARC III composite calves,[12] except *Pasteurella haemolytica* bacterin/toxoid was not used in the calves in this study. Animals were marketed by body weight and sire line weaning groups to one commercial packing plant.

Private sector population

The application phase used calves from three different feedlots. The first group of calves (n = 265) were from Montana ranches and were fed in a central Nebraska feedlot (Feedlot A). At processing, calves were individually identified with ear tags and individual body weights were taken. The remainder of arrival processing followed the standard feedlot protocol. Following processing, calves were placed into three pens with minimal or no mixing between ranch sources.

The second group of calves (n = 111) were fed in Feedlot B, also in central Nebraska. This was one pen of single-source retained-ownership steers. At entry into the feedlot, cattle were individually identified, individually weighed and processed according to the standard feedlot protocol.

The third group of calves (n = 223) were purchased from seven livestock auction markets along the borders of Oklahoma and Texas and were fed in western Kansas (Feedlot C). The calves were individually weighed, individually identified, processed according to the feedlot protocol and backgrounded on rye pasture from March through April. When the calves were returned to the feedlot they were sorted three ways, dependent on body weight; the lower quartile, the second and third quartiles, and the upper quartile. Only animals from the upper quartile were used in this study as the others were lost to follow up (Table 1).

Table 1. Reason for loss to follow-up by source

Population		[1]Missed obs.	[2]Frame shift	[3]Lost ID	[4]Kill order shift	[5]Recorded	[6]Available
MARC		102	0	0	75	439	616
Feedlot	A	11	100	0	111	265	487
	B	21	0	8	0	111	140
	C	270	727	9	0	223	1229

[1] Lungs were observed as normal on one side and not seen on the other or the observation was missed

[2] Animals were railed off and not validated or offal observations were missed or duplicated and offal counts did not match kill order counts

[3] Animals lost their identification and trace back to feedlot entry was not possible

[4] Animals were processed at a different time than was scheduled

[5] The number of animals with valid data for analysis

[6] The number of animals available at the start of the study

Calves from Feedlots A, B, and C were not individually weighed at the end of the feeding period. Final weights were calculated by dividing individual calf hot carcass weights (HCW) by the average dressing percentage for each lot of calves.[7] All private sector calves were slaughtered during the fall of 1995 or the spring of 1996.

Data collection

The drive, lot number and head count for each group were recorded at the packing plant from the daily slaughter schedule (Table 2). The schedule was used to project the USDA sequence numbers for study calves. Calf identification and the projected corresponding USDA sequence numbers were recorded after calves were stunned and suspended from the overhead rail before tag and hide removal. These sequence numbers were validated twice, once at the hide-puller and again at the hot carcass scale. Further details on data collection in packing plants has been previously published.[2] USDA run-sequence numbers missing at the hot carcass scale represented carcasses retained for further inspection. Retained carcasses were identified with the variable "railed out".

Implants were scored as 1 if they were present with normal tissue reactions. Implants that were abscessed, missing, present but in the cartilage, bunched or crushed were scored as 0. Lung and liver pathology were evaluated at the offal table using the USDA sequence numbers to link offal to its respective carcass. Liver pathology was given a score of 1 for the presence of any large or active abscesses, or if any other abdominal structure was adhered to the liver.[6] All other liver pathology received a score of 0. Lung scoring was done on a form developed for this project (Figure 1).

Table 2. Example of a daily slaughter schedule used by packing plants

	Kill date			Kill day				Shift	
Drive no.	Lot no.	Producer	Mud	Head	Sub tot.	Kind	Type	Pen no.	Avg.
201	202			90	90	S/H		47	1166
202	225			84	174	S/H		46	1270
203	233			143	317	S		40	1162
204	204			100	417	S		12	1225

The date killed combined with lot and drive numbers uniquely identify each pen of cattle and allow recovery of group level data from the packing plant. USDA run-sequence numbers are used for individual animal identification within the plant. These numbers can be linked to kill order numbers that allows tracking in the plant at the individual animal level. Example: Drive 203 and lot 233 identifies 143 animals in the study. The (Sub Tot.) USDA run-sequence numbers for these steers start at 175 and end at 317.

Lung scoring

In preparation for this study, a lung scoring form was developed, consisting of topographical features of a

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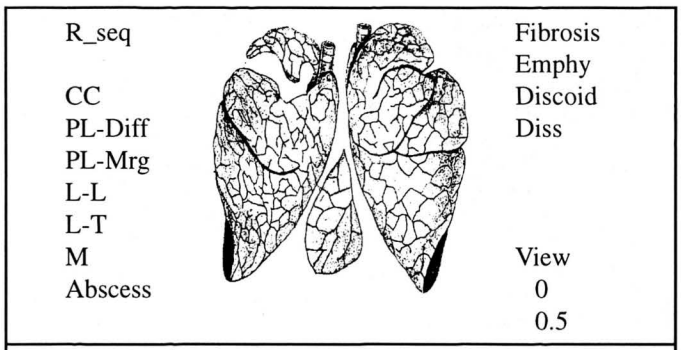

Definition of terms:

R_seq	Three digit USDA run sequence number.
CC	Consolidated and/or collasped lung parenchyma.
PL-Diff	Focal points of pleuritis in a diffuse pattern.
PL-Mrg	Diffuse pleuritis confined to the lobar margins.
L-L	Fibrous adhesion(s) extending between pleural surfaces.
L-T	Fibrous adhesion(s) extending from visceral to parietal pleura.
M	A portion of or whole lung lobe missing due to thoracic adhesions.
Abscess	Abscess(es) in the lung parenchyma.
Fibrosis	Pale sunken often yellowish-tan areas in the lung parenchyma.
Emphy	Bullae occurring interstitially or parenchymally.
Discoid	Greenish-yellow edematous interlobular and lobular areas often accompanied by hemorrhage.
Diss	Describes a lung with disseminated disease.
View 0	The lung was not observed.
View 0.5	Only half of the lung was observed.

Figure 1. Lung scoring sheet with gross descriptions of lesion classification used at slaughter.

bovine lung, for recording lesion type (gross classification) and its respective lobar location. Development was conducted in two phases. Phase one consisted of observing lungs in a commercial packing plant at chain speed (312 head per hour). From these observations a list of lesions that could be seen at chain speed was constructed. This includes the following lesions: collapse/consolidation, diffuse pleuritis, marginated pleuritis, lobe to lobe adhesions, lobe to thoracic adhesions, missing lobes, abscesses, fibrosis, emphysema, discoid and disseminated lesions. Phase two validated the system by calculating the clinical agreement between observations recorded at chain speed and those recorded on the same lungs which were tagged and retained for detailed examination in the plant. Samples from representative lesions were placed in 10% formalin, processed and stained with hematoxalin and eosin stain for histologic evaluation.

Subsequent lung scoring of MARC calves was done at chain speed using the previously developed scoring form. The presence or absence of any lesion, each specific lesion as well as the amount of parenchymal involvement was recorded. The percentage of lung affected by lesions was calculated at a later date. The denominator for calculating each lobe contribution to total lung mass was from Jericho and Langford.[4]

Analysis

MARC population

The outcome of interest was the relationship of lung lesions to ADG and specifically those associated with a reduction in ADG. This association was measured using multivariable ANOVA. Lung lesions at slaughter were investigated as present or absent, percentage of lung involved with lesions, and specific lesion type. Lesion types were also grouped into one variable representing lesions resulting from bronchopneumonia[1] occurring in the cranial ventral lobes (CVBP), other lesions (all lesions that did not occur in the cranial ventral lobes or were not sequella from bronchopneumonia) and no lesion. In order to accurately measure the relationship between lung lesions at slaughter and ADG, we recorded and tested the following variables for confounding and interaction: calf gender, calving difficulty, weaning weight, sire and dam line, feedlot pen, liver pathology, railed-out and growth implants. These variables are reported to influence ADG and could be confounded with lung lesions at slaughter, especially when carcasses are trimmed and HCW are used to estimate final live weights, and therefore ADG.

To investigate the effects of clinically diagnosed disease and the relationship of disease to lung lesions at slaughter, MARC herd records were used to classify calves as having or not having experienced clinical respiratory disease. Clinical respiratory disease prior to weaning and in the feedlot was examined as separate effects and as a single effect. Regression models were developed using multiple linear regression in a forward stepwise procedure and the association of various factors with individual calf ADG were analyzed using least squares regression.[5] Collinearity and two-way interaction between variables with plausible biological explanation were tested at each step of the modeling process followed by a test for confounding. Final least squares regression models were fit using the Statistical Analysis System (PROC GLM; SAS, 1989).[10] Cp and R^2 were used as selection criteria.[5] The assumptions of normality and homogeneity of variances were tested using standard residual analysis.

Private sector population

Independent variable screening and model building was done the same way for this population. Lung lesions were recorded at chain speed as CVBP lesions, other lesions or no lesion. Morbidity data at the individual animal level was not available in archived health files and there were no pre-feedlot health records available.

Results

Clinical agreement

During the developmental and validation phases, detailed examination of lungs in the packing plant by a pathologist, with no time constraints, was used as the gold standard for detecting the presence or absence of gross abnormalities. Substantial clinical agreement exists between this and chain speed evaluation of lungs ($k = 0.649$[9]; Table 3).

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Table 3. Measures of agreement between chain speed lesions diagnosis and detailed gross inspection

		Chain speed diagnosis	
		Lesion	**No lesion**
Gross inspection	Lesion	61	7
	No Lesion	5	17
Percent agreement		.90	
Kappa		.65	

Kappa values:[7] 0-.2 = "slight"; .2-.4 = "fair"; .4-.6 = "moderate";

Gross lesions and histology

Collapsed and consolidated lesions (slides 1, 2, and 3) appeared as slit-like alveoli, lacking normal acinar expansion, or acini filled with cellular exudate and inflammatory cells. Ten to twenty percent of these lesions also showed diffuse moderate to severe peribronchiolar and peribronchial lymphoid hyperplasia, suggesting chronic pulmonary mycoplasmosis. Lobular, raised, hemorrhagic lesions (discoid lesions; slide 4) were characterized by eosinophils, with edema and hemorrhage. This classification was observed from mid-April through mid-August. It was found more frequently in the MARC calves (23%), but was also observed in calves from the private sector (5%). Lobe to lobe (slides 2, 3, 5) and lobe to thorax adhesions (slide 6) were characterized by areas of neo-vascularization infiltrated by hemosiderin-laden macrophages and connective tissue. Marginated and diffuse pleuritis (slide 7) were similar histologically to adhesions. Fibrosis (parenchymal; slides 2, 3) typically appeared as sunken areas with increased connective tissue, smooth muscle hyperplasia, and lobular fibrosis, corresponding to resolved pneumonia. Lobes with missing portions or whole lobes missing (slides 6, 8) were classified as missing due to adhesions from previous pneumonia, based on previous work showing residual parenchyma "contains histologic evidence of previous pneumonia".[12] Emphysema (parenchymal or interstitial) was characterized by bullae and ruptured interlobular septa and was frequently accompanied by parenchymal fibrosis.

Feedlot Results

Rates of clinical pneumonia, lung lesions at slaughter, and ADG are listed in Table 4 for MARC origin calves. These morbidity events are also cross tabulated with the presence of a lesion in the lung at slaughter. Similar data for the private sector cattle are listed in Table 5.

MARC

Forty-two percent of all calves had lung lesions at slaughter. Forty percent of those diagnosed with respiratory disease had lesions, whereas 42% of calves never diagnosed with clinical disease also had lesions (Table 4). Seventeen percent of the calves in this population suffered from clinically diagnosed respiratory disease between birth and slaughter. Of these, 1% occurred before weaning and 16% occurred during the feeding

Table 4. Respiratory disease, lung lesions and ADG (lb) in MARC origin calves at slaughter

No. of calves	Gender	*Treated for respiratory disease	Pulmonary lesions at slaughter	Lesions at slaughter and treated	ADG ± STD DEV
		n (%)	n (%)	n (%)	
		Sire line 1			
44	H	6 (14)	29 (66)	3 (7)	2.03 ± .29
105	S	11(11)	32 (30)	2 (2)	2.38 ± .24
		Sire line 2			
47	H	8 (8)	20 (42)	1 (2)	2.17 ± .18
96	S	17(18)	40 (42)	7 (7)	2.45 ± .24
		Sire line 3			
58	H	11(19)	31 (53)	6 (7)	2.45 ± .24
95	S	20(22)	32 (34)	10(11)	2.73 ± .12
		All calves			
	H 149	25 (17)	80 (54)	10 (7)	2.23 ± .24
	S 290	48 (17)	104 (36)	19 (7)	2.51 ± .24
	439	73 (17)	184 (42[a],19[b])	29 (7)**	2.38 ± .24

* Respiratory morbidity prior to weaning, in the feedlot or both
** Twenty-nine of 73 animals (40%) were treated for respiratory disease and had lesions at slaughter
a = Percent of lungs with any lesion at slaughter
b = Percent of lungs with CVBP type lesions

Table 5. Lung lesions, ADG (lb) and morbidity by feedlot pen in commercially fed calves

Feedlot pen	No. of calves	Lung lesions	ADG ± STD DEV	Morbidity*
		Feedlot A		
		n (%)		
3	55	38 (69)	3.31 ± .39	2
4	146	88 (60)	3.15 ± .46	2
5	64	36 (56)	3.06 ± .49	2
		Feedlot B		
2	111	86 (77)	3.42 ± .71	4
		Feedlot C		
8	223	75 (33)	3.17 ± .55	5
		All calves		
	599	323 (54[a],30[b])	3.22 ± .51	

* Pen level respiratory morbidity
a = Percent of lungs with any lesion at slaughter
b = Percent of lungs with CVBP type lesions at slaughter

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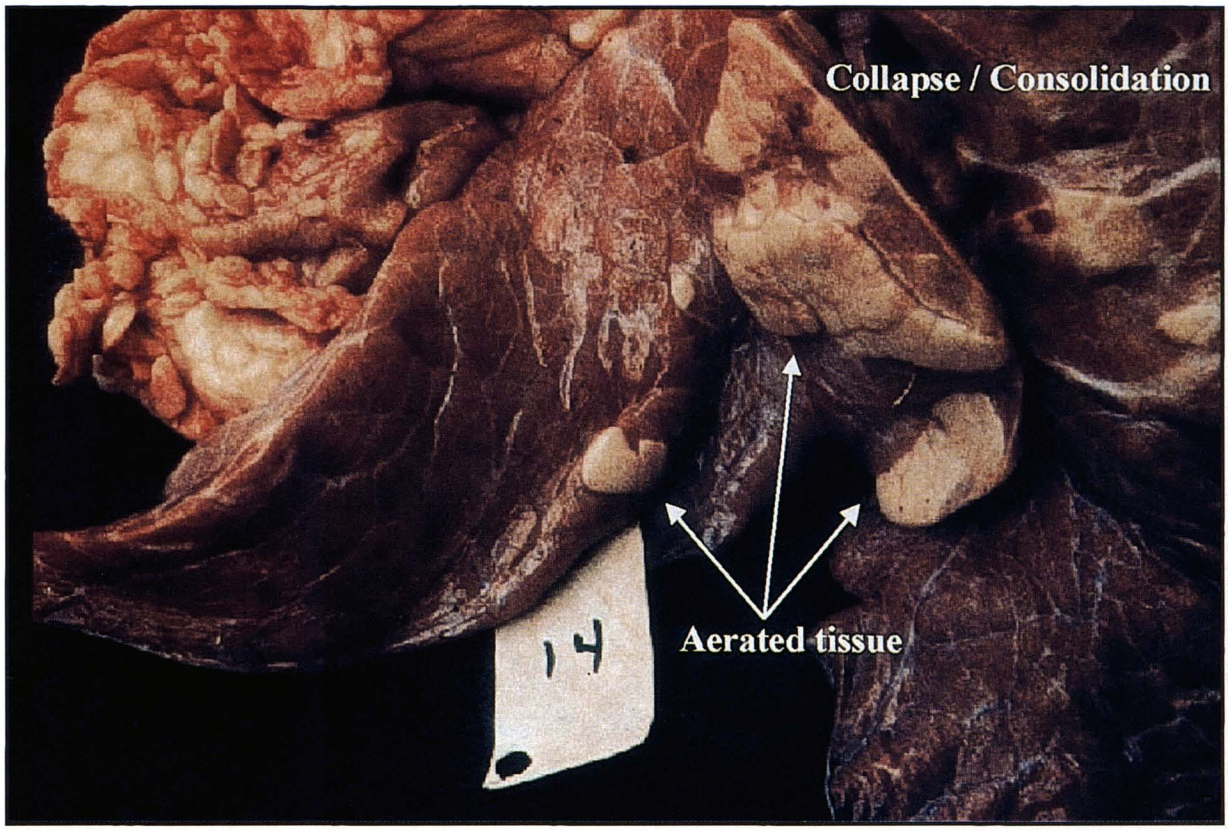
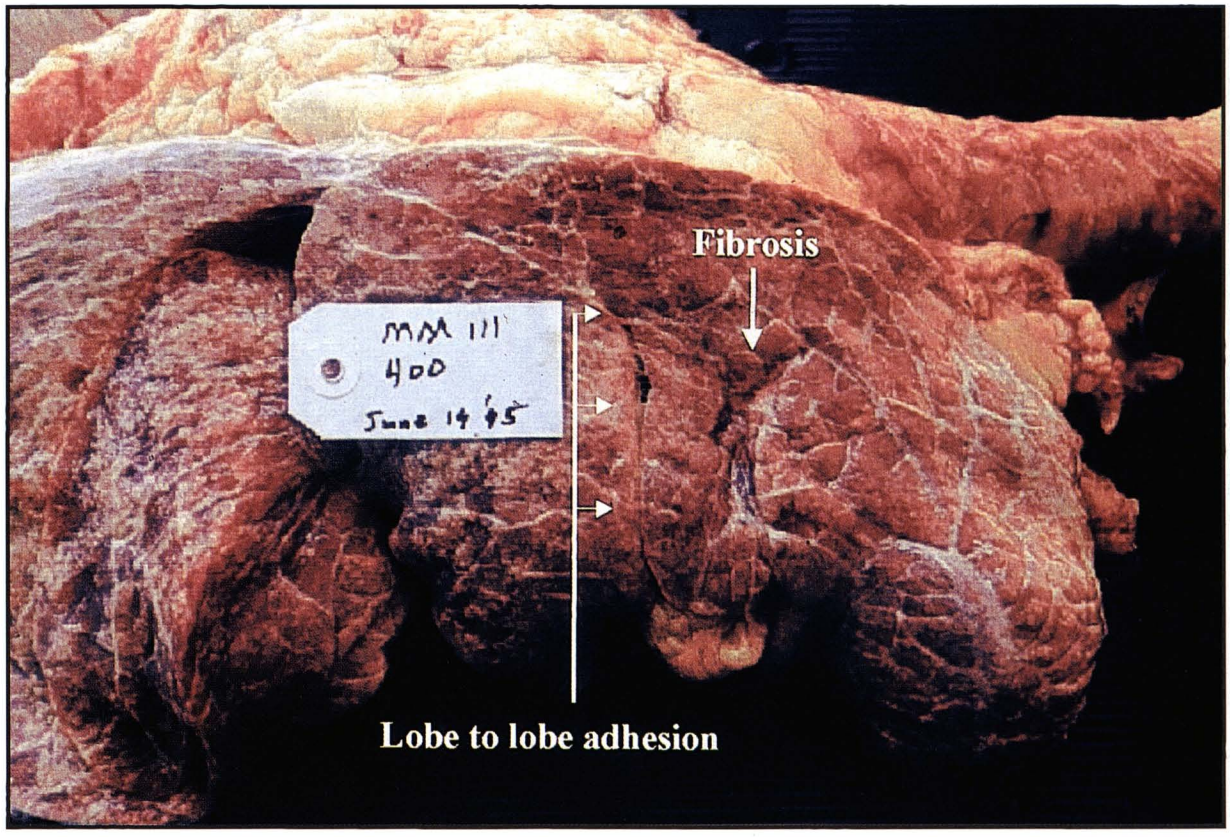


Slide 1



Slide 2

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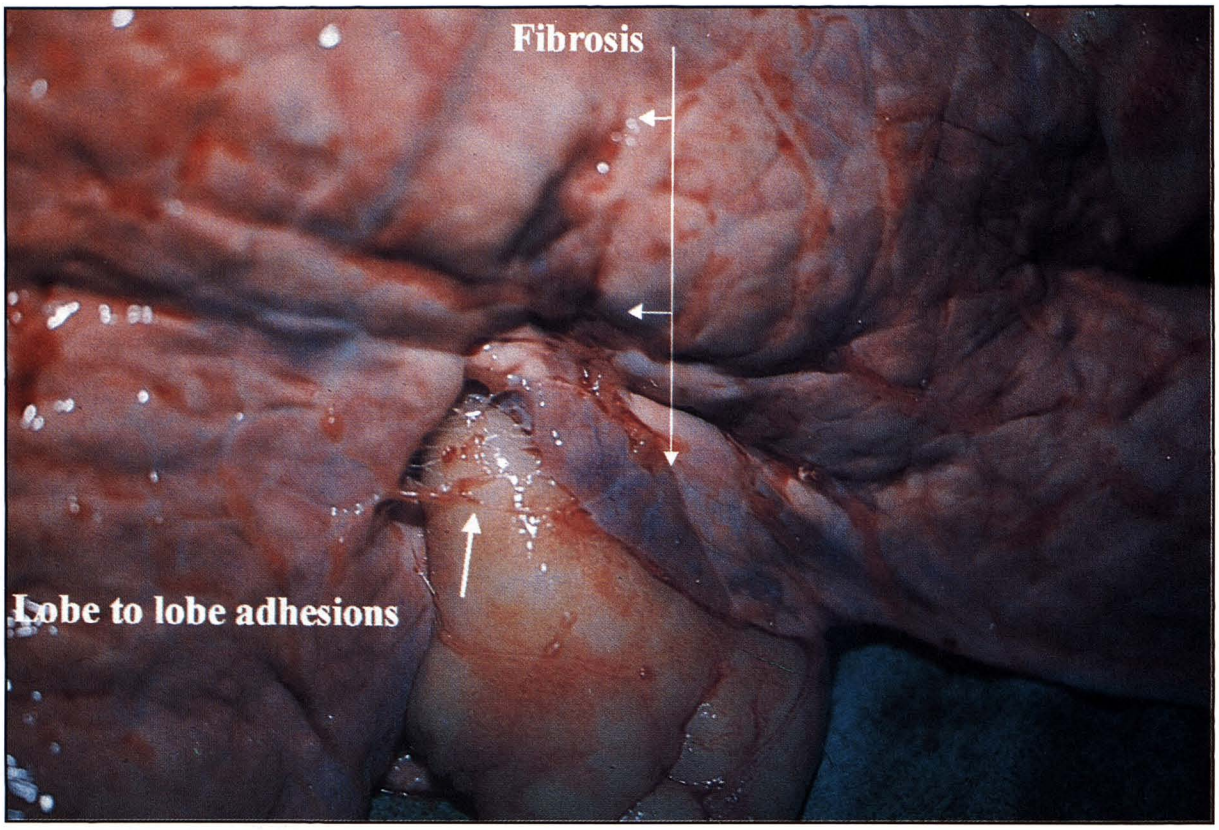
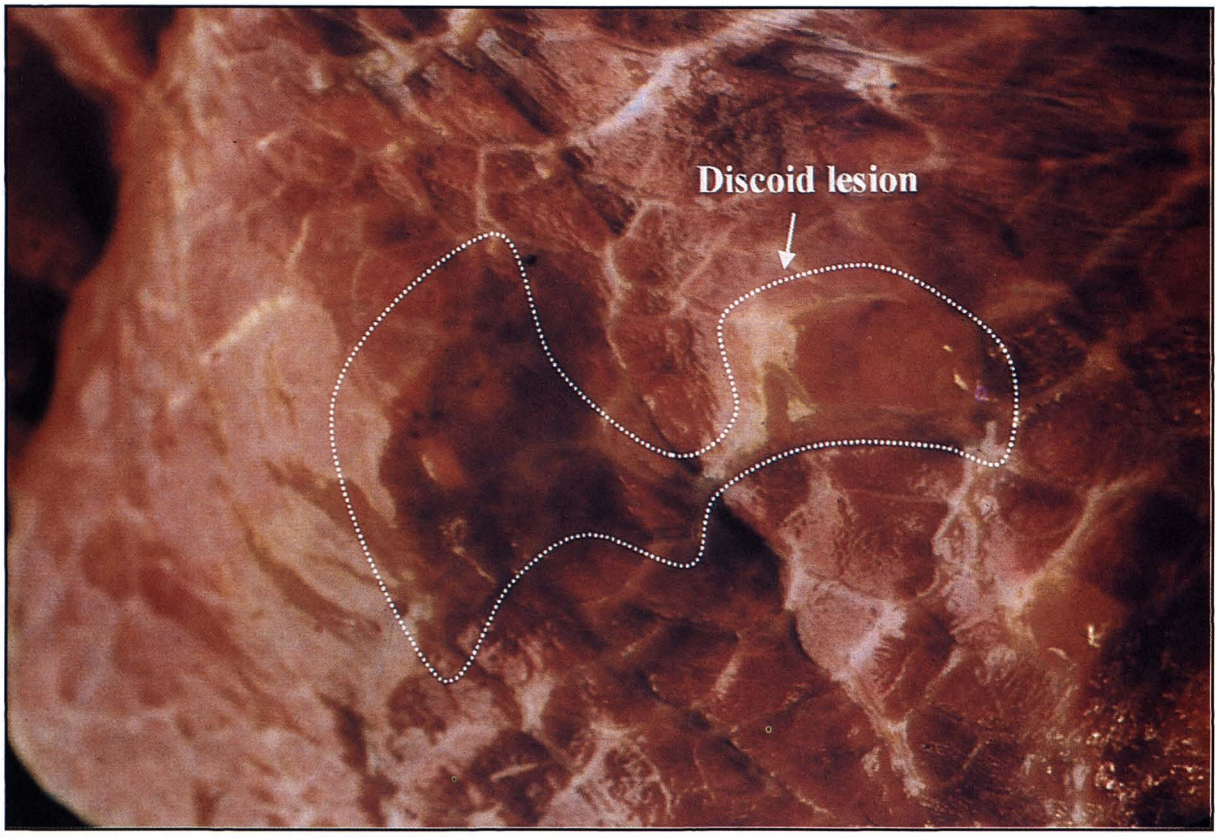


Slide 3



Slide 4

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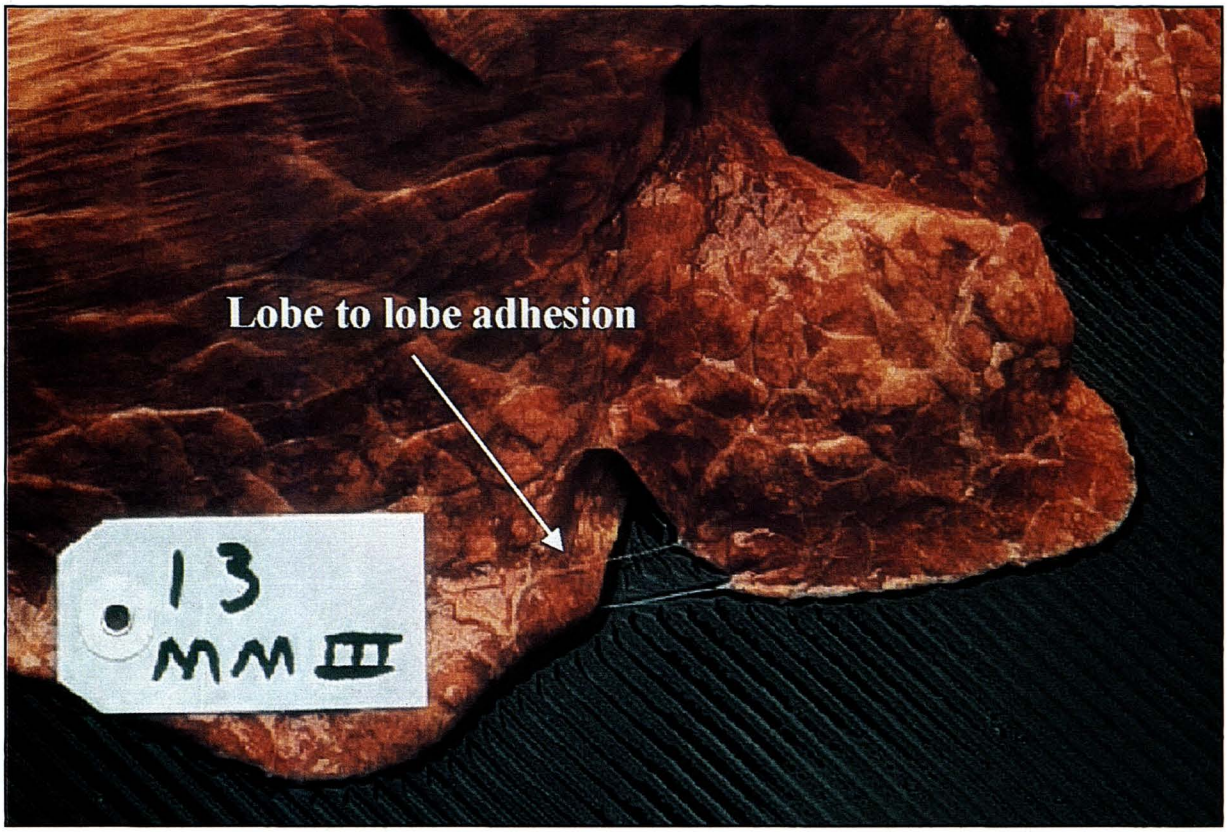
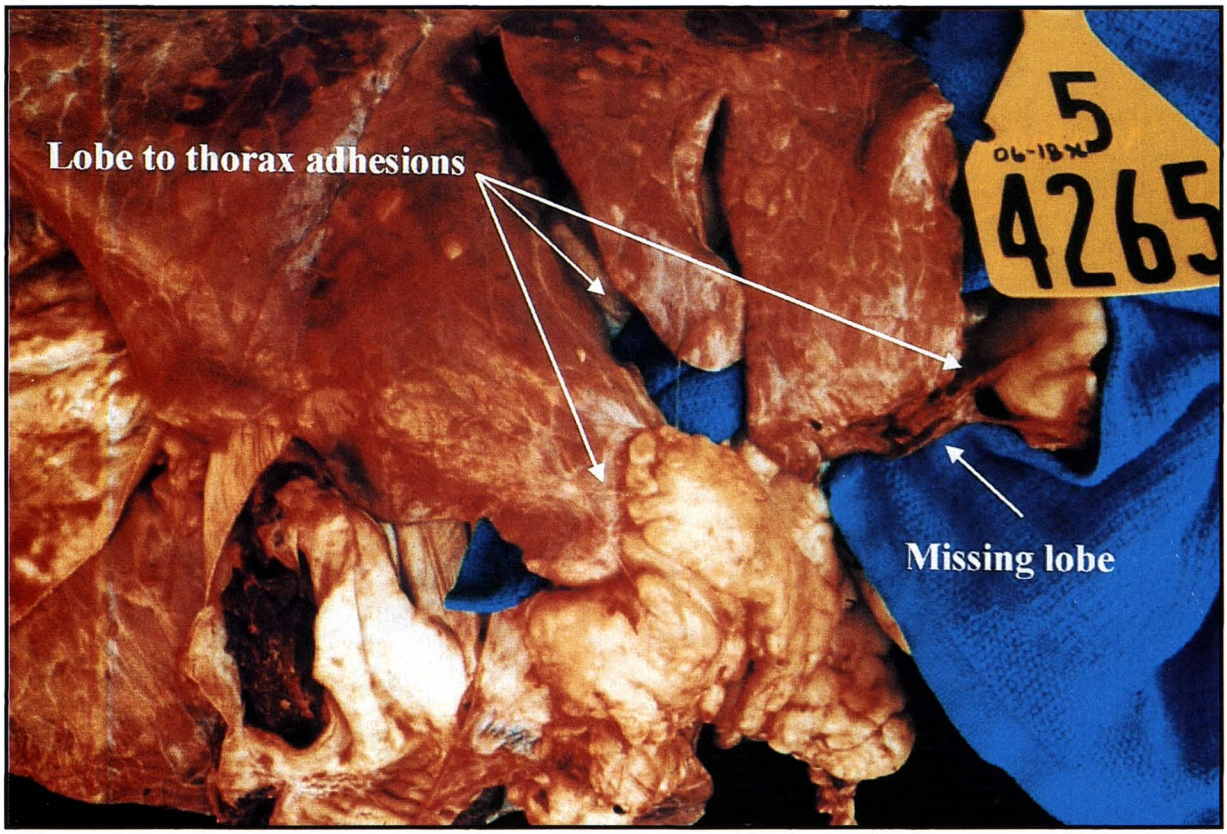


Slide 5



Slide 6

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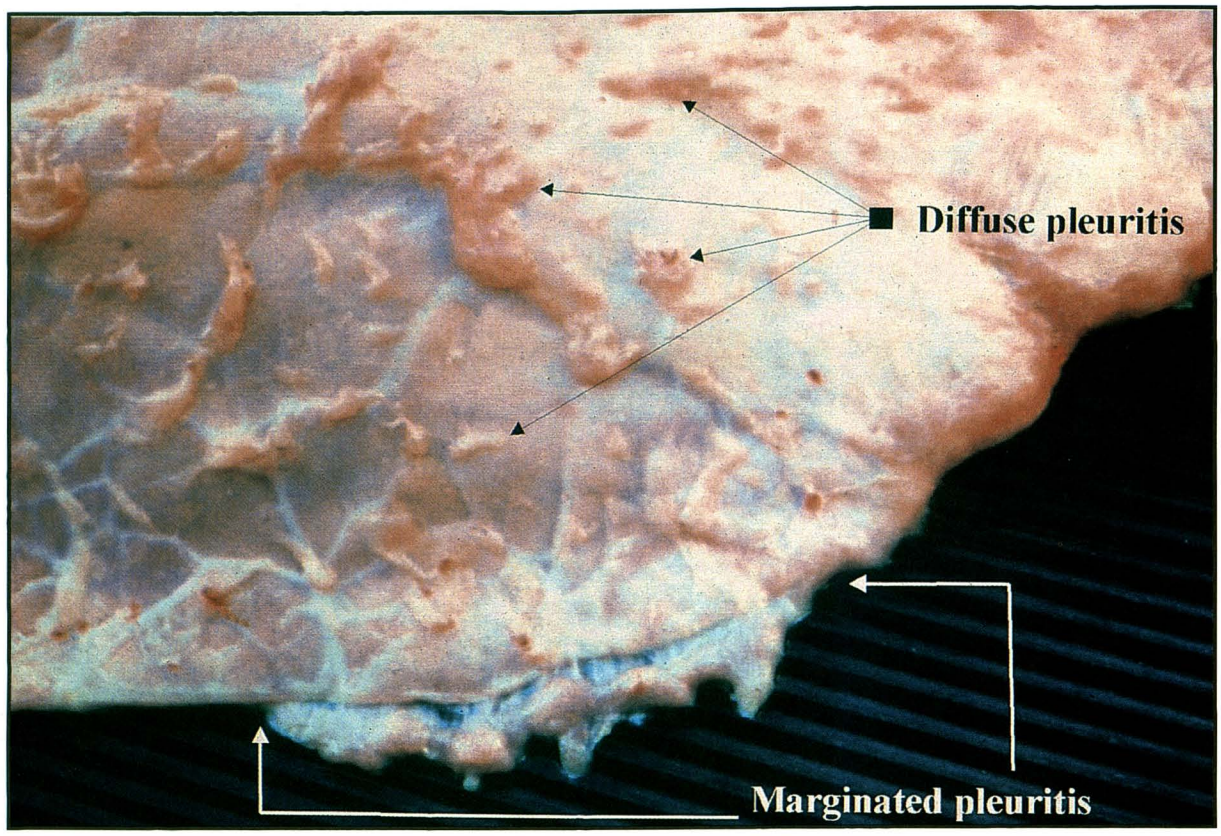
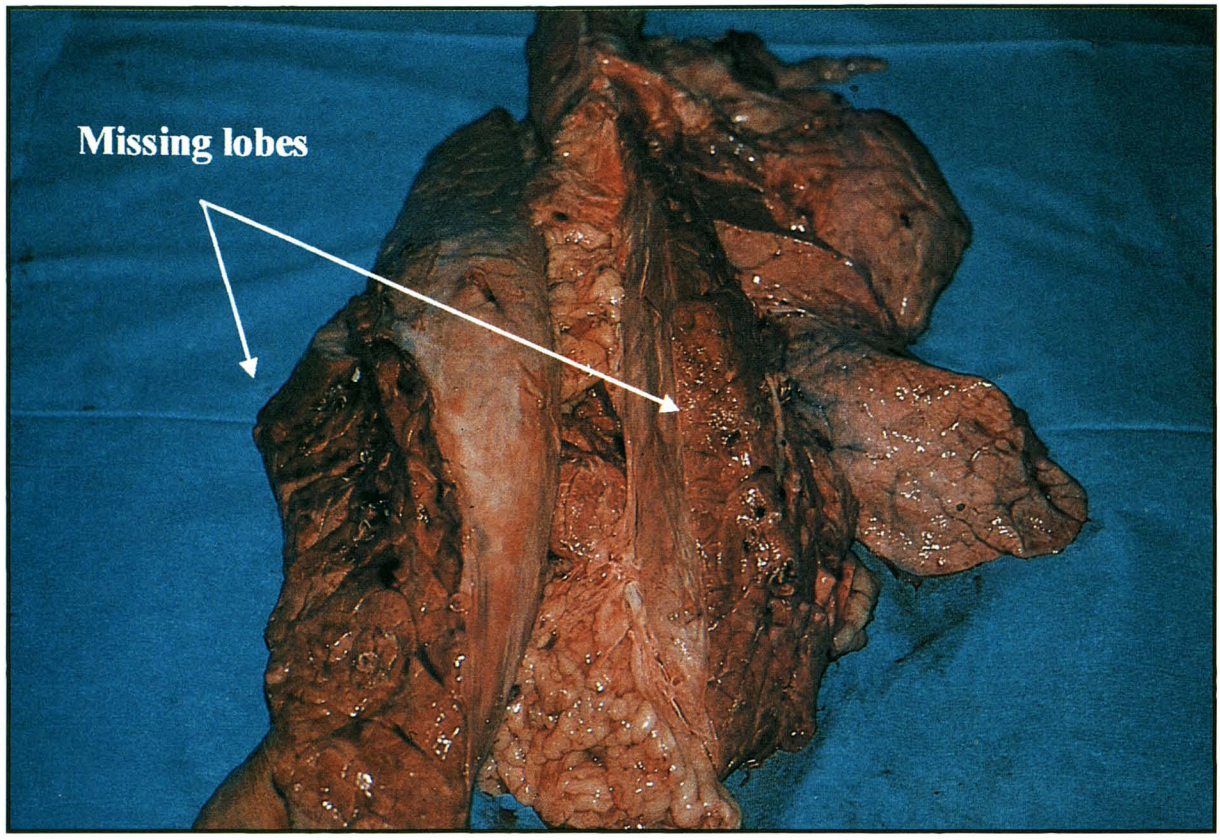


Slide 7



Slide 8

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Table 6. Final ANOVA model for estimating effects of pulmonary lesion at slaughter on ADG (lb) in the feedlot among 149 heifers and 290 steers in the MARC population

Variable	df mean	F	β	SE of β	Least-squares
Lung lesion[1]	1	*6.72	-0.057[b]	0.035	2.40[a] 2.34[b]
CVBP lesion[1]	1	*6.89	-0.073[d]	0.012	2.40[c] 2.31[d]
Calf gender	1	*96	- 0.27[e] ref[f]	0.011 ref	2.45[e] 2.49[f]
Weaning weight	1	*6.21	0.00053	0.0001	Na
Sire line	2	*35	(-.22, -.35)	0.013	Na

a = lung lesion absent at slaughter
b = lung lesion present at slaughter
c = CVBP lesion absent at slaughter
d = CVBP lesion present at slaughter
e = heifers
f = steers
* P < 0.01
ref = referent cell used by the model

[1] Model effects are calculated with one variable represented at a time, variables are reported simultaneously for comparison only.

period. One animal had clinical disease during both periods. Clinical disease between heifers and steers was not different (P = 0.95), however a larger portion of the heifers, 54% vs. 36% (P < 0.01), had lung lesions. Gender effect is totally confounded with, and controlled for in the model by, feedlot pen.

The final model estimating independent variables and covariant effects on ADG is shown in Table 6. The presence of any lung lesion observed at slaughter was associated (P < 0.01) with a decrease of 0.057 lb in ADG for the feeding period. Lesion types were grouped into one of two variables: lesions produced as a result of cranial ventral bronchopneumonia (CVBP) or other lesions representing separate effects. The variable representing CVBP was the only sub-classification significantly associated with decreased ADG. The presence of CVBP lesions was associated with 0.073 lb less ADG with no change in other calculated estimates. The percentage of parenchymal involvement was not associated with ADG as separate effects.

Private sector population

The highest frequency of pulmonary lesions at slaughter existed in calves from Feedlot B. This was not different from calves in Feedlot A, but both were different from calves in Feedlot C (P < 0 .001; Table 5). Data from the private sector calves were pooled for regression analysis.

Table 7. Final ANOVA model for estimating effects of CVBP lesion at slaughter on feedlot ADG (lb) among 599 steers from commercial feedlots

Variable	df	F	β	SE of β	Least-squares mean	
CVBP lesions		*49.9	-0.14	0.044	3.02[a]	2.64[b]
FP x CVBP lesions		**2.97	...	...	...	
A 2	—	—	-0.051	0.062	3.11[a]	2.91[b]
A 3	—	—	-0.33	0.077	3.04[a]	2.58[b]
A 4	—	—	-0.22	0.060	2.98[a]	2.62[b]
B 5	—	—	-0.51	0.078	2.84[a]	2.18[b]
C 8	—	—	Ref	ref	3.06[a]	2.93[b]
Entry weight	1	*16.65	- 0.0015	-0.0002	Na	
Railed out		*8.36	-0.35	0.056	3.00[c]	2.65[d]
Feedlot pen (FP)	4	*9.51	Na	Na	Na	Na
Implant palpation		**3.73	-0.10	0.024	2.76[e]	2.89[f]
Liver pathology		*6.14	-0.21	0.039	2.93[g]	2.71[h]

F = test statistic
* P < 0.01
** P < 0.05
— = values contained in parent variable
... = values contained in subsequent variables
ref = referent cell used by the model
a = CVBP lesions absent
b = CVBP lesions present
c = carcass not railed
d = carcass railed
e = implant abnormal or missing
f = implant present
g = liver pathology absent
h = liver pathology present

A final model estimating the effect of CVBP lung lesions at slaughter on ADG was developed (Table 7). CVBP lesions observed at slaughter and feedlot pen had a significant (P < 0.05) interaction effect. The main effect, CVBP lesions, combined with the interaction effect were associated with (P < 0.01) a decrease in ADG for the feeding period ranging from 0.14 to 0.65 lb.

Discussion

Our results indicate that lesions resulting from bronchopneumonia in the cranial ventral lung lobes are the most useful indicators for determining the effect of respiratory disease on rate of gain in feedlot cattle. These data suggest that CVBP lesions present at slaughter have an important, but highly variable effect on ADG. The effect of CVBP on ADG ranged from 0.073 lb in the single-source MARC population to as high as 0.65 lb in the commercial feedlots. This variability in effect may be due to differences in exposure to infectious respira-

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tory pathogens, differences in management and animal characteristics, or other factors. However, little is currently known about the epidemiology of pulmonary lesions in feedlot calves. Investigation of the factors which influence the incidence of CVBP lesions in feedlot cattle may lead to effective strategies to prevent significant production losses attributable to unrecognized respiratory disease.

Biologically it is reasonable to represent lesions resulting from bronchopneumonia[1] by one variable, CVBP. To do this, a dummy variable was created that returned a value of "1" if any of the following lesions occurred in the cranial ventral lung fields: collapse/consolidation, adhesion lobe-lobe, adhesion lobe-thorax, missing lobe, abscesses, parenchymal fibrosis or emphysema. This simplifies the scoring method allowing one person to successfully observe and record lung lesions at the individual animal level on slaughter groups containing up to 90 head. This is dependent upon familiarity with the plant and the number of animals between the blood pit and the offal table.

Wittum et al.[12] suggested grouping all pulmonary lesions at slaughter into one category. However, the current study identified several lesions not associated with ADG. Discoid lesions (slide 4) have been described as eosinophilic pneumonia[8] representing inflammatory reactions stimulated by first-instar migration of *Hypoderma lineatum*. These lesions can be differentiated from cranial ventral bronchopneumonia by their gross appearance and pattern of distribution. The gross appearance is lobular and the pattern resembles a metastatic/embolic distribution compared to a lobar appearance with a cranial ventral distribution for bronchopneumonia. The discoid lesions can appear in the cranial ventral fields but most often appear in the diaphragmatic lung lobes. Panceria et al.[8] also described this lesion as occurring in a seasonal pattern and accompanied by fibrous tags on the pleural surface and along the lobar margins. These latter lesions are identified as diffuse and marginated pleuritis (slide 7) in the current study. However, these were not confined to a seasonal occurrence and at times occurred independently of each other. They frequently occurred independently of discoid lesions, reducing the possibility they are related. We are not unaware of other descriptions of marginated and diffuse pleuritis. Speculation on their cause would be theoretical.

Other subclasses of lesions deserve further investigation, such as Mycoplasma-like lesions and active lesions. Mycoplasma-like lesions were frequent (≤ 37%) in some pens of cattle. At times, they were observed concurrently with other lesions, and at times they occurred alone. In this study, they were included in the variable collapse/consolidation. The current dogma suggests *Mycoplasma sp*. do not cause clinical respiratory disease but are frequent isolates from pneumonic bovine lungs. Information gained from this study should stimulate further investigation into Mycoplasma-like lesions, allowing differentiation of performance parameters associated with this lesion and perhaps redefine our understanding of *Mycoplasma* organisms and their role in respiratory disease. In addition, a small portion of lungs (≤ 6%) had active lesions, represented by lymphadenopathy in the hilar and/or mediastinal lymph nodes. These active lesions were not recorded in a formal manner so their effect on ADG is included with non-active lesions.

Examination of the individual morbidity in MARC calves suggests that there is little relationship between clinical BRD diagnosis and lung lesion presence at slaughter. This is consistent with previous reports.[3,12] Lack of individual morbidity records precludes evaluation of the agreement between clinical BRD diagnosis during the feeding period and lung lesion presence at slaughter for the commercial calves. However, the low rate of group morbidity and the high rate of lung lesions in the commercial calves supports earlier findings that there is little relationship between clinical BRD diagnosis during the feeding period and lung lesion presence at slaughter.

CVBP type lesions were significantly associated with ADG but the amount of parenchymal involvement was not associated with ADG. One possible explanation of this is that the extent of parenchymal damage is not relevant to calf growth. Alternately, differences in fibrin contraction of damaged tissue, coupled with possible differences in the initial inflammatory response and healing rates, result in the final scar not being representative of the initial magnitude of infection.

Acutely, lesions in feedlot calves with clinical disease are more extensive than in calves with asymptomatic disease.[11] However, the effect of lesions on ADG during the feeding period are similar between cattle that are diagnosed with clinical infections and not treated with antibiotics and calves with asymptomatic disease.[3] This suggests that disease occurrence extensive enough to produce lesions is the key to causing production losses, regardless of whether it manifests as a clinical or non-clinical event.

Conclusions

Only lung lesions resulting from cranial ventral bronchopneumonia were significantly associated with growth performance. Therefore, all lesions occurring in the cranial ventral lung field that result from bronchopneumonia should be used as a measure of pulmonary disease.

The purposes of this study were to were develop a standardized method for measuring lung lesions at slaughter in a commercial packing plant

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environment, to identify specific types of lesions associated with reduced ADG, and apply the final method to commercially fed cattle. The populations of cattle used to accomplish these goals were not all managed identically. The study was not designed or executed in a manner that allows evaluation of the effects of the different management factors on lung lesion prevalence. Additionally, varying amounts of loss to follow-up in sub-populations may have introduced bias into the group-level data. It may be tempting to evaluate the differences between lesion rates from different groups of cattle and make inferences about these lesion rates. However, this is outside the limits of the usefulness of the data and is not appropriate.

Future investigations should utilize the method reported here combined with health records to measure the true incidence of respiratory disease. Our current methods of disease diagnosis are not adequate for evaluating management changes or product efficacy if we are interested in understanding the cost effectiveness of our decisions. The methods proposed here should be used to identify risk factors that are associated with the frequencies of lung lesions at slaughter. Once these have been identified management strategies can be changed accordingly.

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