LUNG LESIONS IN LAMBS

J. A. Daniel[1], J. Held[1], C. S. Schauer[2], W. Epperson[3*]

[1]Department of Animal & Range Sciences,
South Dakota State University, Brookings, SD 57007
[2]Hettinger Research Extension Center, Hettinger, ND 58639
[3]Department of Veterinary Science, South Dakota State University, Brookings, SD 57007
[*]Current address: College of Veterinary Medicine, Ohio State University,
Columbus, OH 43210

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Introduction:

Maintaining lamb respiratory health is critical for efficient lamb production. Clinical respiratory disease has long been acknowledged to have negative effects on lamb production, but subclinical respiratory disease has only recently become a concern. Subclinical respiratory disease in lambs can be identified by moderate (5-50% of any lobe) or severe (>50% of any lobe) consolidation of the lungs at slaughter. In previous studies, we have observed that although only 8% of spring born lambs at South Dakota State University (SDSU) required individual treatment for respiratory disease, 38% (34 of 89) of the lambs had severe lung lesions, and 64% (57 of 89) of the lambs had either moderate or severe lung lesions (Daniel et al., 2003). Post weaning to finish growth performance was 0.07 pounds per day lower for those lambs diagnosed with severe lesions than moderate lesion or normal lungs (Daniel et al., 2003). Goodwin et al. also observed reduced daily weight gain during the month prior to slaughter in New Zealand lambs with greater than 20% of the lung surface area affected by lesions (2001). However, in fall born lambs at SDSU only 7% (7 of 108) of the lambs had severe lung lesions and 29 % (31 of 108) had either moderate or severe lung lesions (unpublished observations). The different incidence in lung lesions between spring and fall born lambs suggests that lung lesions can be prevented. Therefore, we designed a series of experiments to determine the timing of the onset of lung lesions, identify some risk factors associated with lung lesions, and test possible means of reducing the incidence of lung lesions.

Research Procedures:

Experiment 1: Timing of onset of lung lesions in lambs.

In spring of 2003, white-faced composite (Dorper sired) wethers born at the SDSU Sheep Unit were divided into three slaughter groups prior to weaning. Lambs were born in a 32 day lambing period (February 4 - March 8, 2003). Lambs were weaned at 78 ± 0.13 days of age and either slaughtered the following day (weaning group, n = 21) or self-fed a commercial lamb ration on an ad libitum basis as a group. Remaining lambs were slaughtered in two groups (mid-finishing, 50 days post-weaning, n = 20, or finished, 71 days post-weaning, n = 21). Live weights were collected on lambs the day prior to slaughter and designated as slaughter weight. Following slaughter, lungs were visually examined and scored for presence of lung lesions. Lung samples were collected from all lambs and cultured for the presence of bacteria by the SDSU Veterinary Diagnostic Laboratory. Carcass traits and weights of the triceps brachii, biceps femoris, semitendinosus, infraspinatus, lumbar longissimus dorsi (the portion of the longissimus from the last thoracic to the last lumbar vertebrae) were recorded. A portion of the longissimus was then aged for 14 days and subsequently frozen until thawed and cooked for analysis of cooking shrink and shear force.

Experiment 2: Mannheimia (Pasteurella) haemolytica and Pasteurella multocida vaccination.

In spring of 2004, crossbred wethers born at SDSU Sheep Unit were assigned to either vaccinated or non-vaccinated groups. Vaccinated lambs were vaccinated with an avirulent *Pasteurella Haemolytica-Multocida* live culture (Once PMH serial no. 07973836 and 07973838, Intervet, Millsboro, Delaware) and a *Pasteurella Haemolytica-Multocida* bacterin (Lamb Pneumonia Vaccine serial no. 939B, Colorado Serum Company, Denver, Colorado) at 4 and 2 weeks prior to weaning, at weaning, and 2 and 6 weeks after weaning (a minimum of 21 days before slaughter). At weaning, lambs were assigned to one of six pens depending upon vaccination treatment. All pens were in the same barn, and lambs were prevented from having nose to nose contact with lambs in other pens. Lambs had feed available ad libitum. At slaughter, lamb lungs were examined for lung lesions.

Experiment 3: Impact of supplemental selenium on lung lesions.

One-hundred forty four wethers (74 ± 0.4 lbs initial BW) were blocked by weight and stratified by breed (western white-faced and western white-faced X Suffolk) and assigned randomly to one of 16 pens (9 lamb/pen) for an eighty-four day finishing phase. Pens were then assigned to one of 4 treatments (4 replications/treatment); supra-selenium supplementation for the final 56, 28, 14, or 0 days of feeding prior to harvest. Wethers were wormed on day 0 and managed for antibiotic-free status. Diets were 84% corn and 15% supplement provided daily at 8:00 am to ensure ad libitum intake (NRC, 1985). Diets were isonitrogenous and isocaloric. Feed offered was recorded daily and feed refusals collected and recorded when significant amounts of refused feed accumulated in the feeders. Selenium for supra-selenium supplementation treatments (14, 28, and 56 days) was provided as selenoyeast to provide 75 $\mu g \bullet kg^{-1}$ BW $\bullet$ d^{-1} Se (approximately 1.9 ppm ration concentration). At slaughter, lamb lungs were examined for lung lesions.

Lung lesion scoring: Lambs were determined to have severe lung lesions if over 50% of any lobe was consolidated. Lambs were considered to have moderate lung lesions if greater than 5% but less than or equal to 50% of a lobe was consolidated. Lambs were considered to have normal lungs if not more than 5% of any lobe was consolidated. Lungs were also examined for the presence of active abscesses or pleural adhesions.

Statistics: Effect of treatment on incidence and severity of lung lesions were tested by Chi-square comparison. Effect of lung lesions (presence and severity) on carcass traits in experiment 1 was determined using PROC GLM procedures of SAS for each slaughter group. Means separation were performed using the LSMEANS/PDIFF statement when the main effect was determined to be significant.

Results and Discussion:

Experiment 1: Timing of onset of lung lesions.

Lambs in weaning group had no severe lung lesions, and only 1 lamb had moderate lung lesions. All others had normal lungs. These data suggest lung lesions are a problem occurring in lambs post-weaning, although it is possible some pre-weaning event could predispose the lambs to develop lung lesions. The incidence of lung lesions had increased dramatically by 50 days post-weaning (Figure 1; $P<0.001$). At 71 days post-weaning, there was a shift to more lambs having severe lung lesions and fewer with moderate lung lesions. This is interpreted to indicate that as time passed, lung lesions become progressively more severe in those lambs affected.

Mycoplasma was not isolated from any lung samples. *Mannheimia (Pasteurella) haemolytica* and/or *Pasteurella multocida* were present in lambs with moderate or severe lung lesions, but not in lambs with normal lung tissue (Table 1). *Mannheimia haemolytica* and/or *Pasteurella multocida*, pathogens known to be capable of producing consolidative lesions and mild illness without clinical signs, were found commonly in lambs with lung lesions. (Sharp et al., 1978; Jones et al., 1982; Gilmour et al., 1988). Another common respiratory pathogen, *Mycoplasma spp*, was not recovered from any samples. Our findings suggest that these common respiratory pathogens are present in lung lesions, though they may not be the initiator of lesions.

The presence or severity of lung lesions did not significantly affect any of the carcass traits examined in lambs slaughtered mid-finishing. Although there was a tendency for lambs with severe lung lesions to have a higher marbling score than lambs with normal lungs or moderate lung lesions (388 ± 7 vs 325 ± 29 and 334 ± 13 respectively; $P=0.0902$). In lambs slaughtered at finishing, the presence of lung lesions resulted in lambs having smaller ribeye areas and lighter semitendinosus muscles ($P<0.05$; Table 2). Furthermore, lambs with lung lesions tended to have lighter biceps femoris and infraspinatus muscles when slaughtered at finishing ($P<0.10$).

Experiment 2: Mannheimia (Pasteurella) haemolytica and Pasteurella multocida vaccination.

Vaccination did not alter the incidence of lung lesions (82% vs 84% incidence of lung lesions in vaccinated vs not vaccinated lambs; $P=0.76$). Nor was the incidence of severe lung lesions altered (63% incidence of severe lung lesions in vaccinated and not vaccinated lambs; $P=0.80$). Based on these results, it does not appear vaccination against *Mannheimia (Pasteurella) haemolytica* and *Pasteurella multocida* will prevent lung lesions. These results support the possibility these common pathogens are not the initiators of lung lesions.

Experiment 3: Impact of supplemental selenium on lung lesions.

Feeding supplemental selenium did not alter the incidence of lung lesions ($P>0.23$) or severity of lung lesions ($P=0.25$). However, these lambs were from two different sources which utilized different breeding strategies. Each source raised lambs using farm flock type management in western North Dakota on different sites. The breed of lamb also differed. Lambs from the source using Rambouillet rams on western whitefaced ewes had a lower percentage of severe lung lesions than lambs from the source using Suffolk rams on western whitefaced ewes (Figure 2; $P=0.0195$). In previous studies, we have also observed breed to alter the incidence of lung lesions. Dorper-sired lambs tended to have a greater incidence of lung lesion than Hampshire-sired (Daniel et al., 2003). Together these data suggest susceptibility to lung lesions may have a genetic component.

Implications

Lungs lesions not only reduce gain as has been demonstrated previously but also decrease lean tissue growth as indicated by smaller ribeyes and lighter muscle weights. Lung lesions are a problem which develops post-weaning, indicating we should target intervention strategies to this time period in the sheep's life. Although *Mannheimia (Pasteurella) haemolytica* and *Pasteurella multocida* are associated with lung lesions, vaccination against these organisms does not reduce the incidence of lung lesions. Furthermore, supplementation with selenium also does not reduce the incidence of lung lesions. However, susceptibility to lung lesions may have a site or genetic component.

Literature Cited

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Table 1: Percentage of lambs with normal lungs, moderate (>5 and ≤50% of any one lobe consolidated) or severe (>50% of any one lobe consolidated) lung lesions from experiment 3 which culture positive for *Mannheimia haemolytica, Pasteurella multocida,* or both.

	Normal	Moderate	Severe
Mannheimia haemolytica positive	0	13% (2/15)	67% (8/12)
Pasteurella multocida positive	0	20% (3/15)	50% (6/12)
M. haemolytica and *P. multocida* positive	0	7% (1/15)	33% (4/12)
M. haemolytica or *P. multocida* positve	0	27% (4/15)	83% (10/12)

Table 2. Effect of Lung Lesions on Carcass Traits in Experiment 3 (mean ± std err).

	Normal	Consolidated Lung Lesions
Number of lambs	9	12
Slaughter weight (lb)	118.1 ± 3.3	110.5 ± 4.6
Hot carcass weight (lb)	66.4 ± 2.1	61.0 ± 2.8
Backfat thickness (in)	0.19 ± 0.02	0.20 ± 0.03
Adjusted backfat thickness (in)	0.25 ± 0.02	0.23 ± 0.02
Ribeye area (sq in)	2.67 ± 0.06[a]	2.43 ± 0.08[a]
Bodywall thickness (in)	1.12 ± 0.05	1.00 ± 0.06

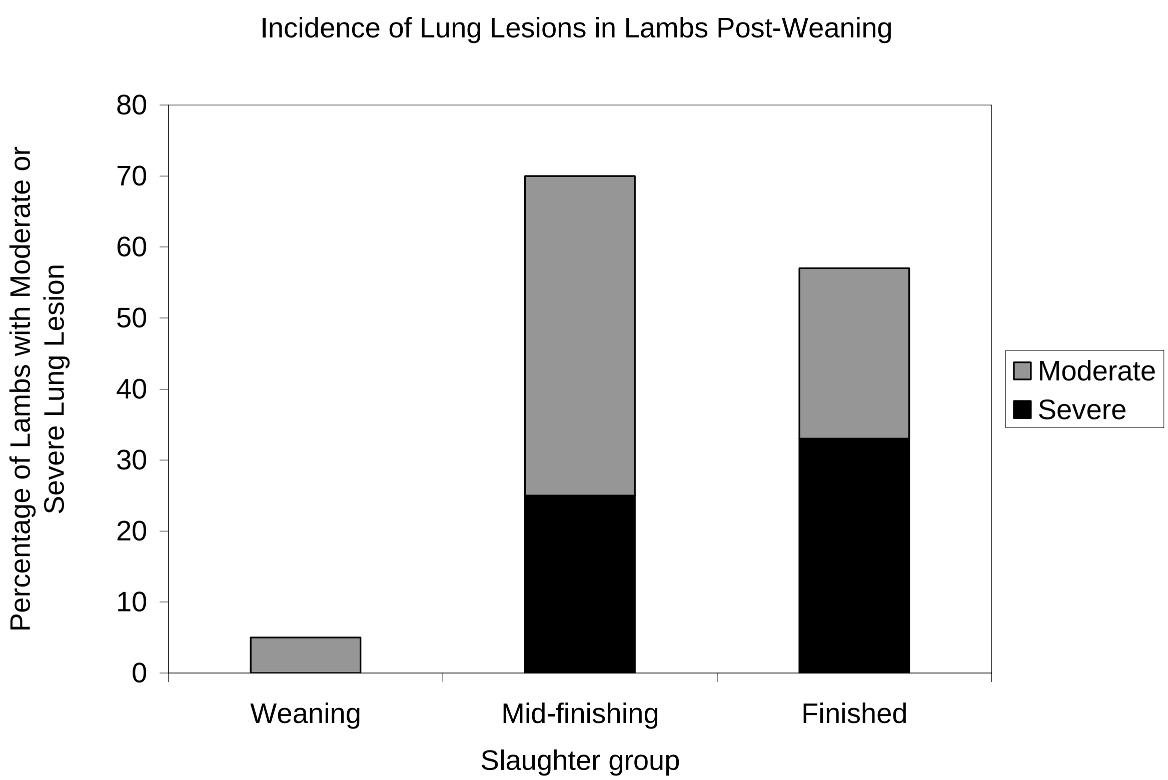

Figure 1: Percentage of lambs with moderate (>5 and ≤50% of any one lobe consolidated) or severe (>50% of any one lobe consolidated) lung lesions slaughtered at 1 (Weaning), 50 (Mid-finishing) or 71 (Finished) days after weaning in Experiment 1. Percentages indicate percentage of all lambs slaughter at each time point with either moderate or severe lung lesions.

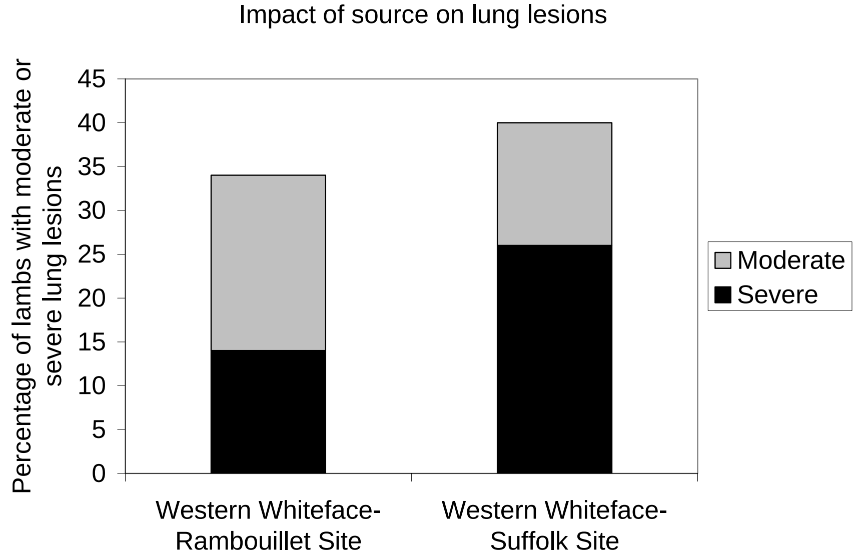

Figure 2: Percentage of lambs with moderate (>5 and ≤50% of any one lobe consolidated) or severe (>50% of any one lobe consolidated) lung lesions from two different sources in Experiment 3. Lambs from different sources were of different breeds and raise on different sites in western North Dakota.