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Characterization of claw lesions in gestating sows

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Introduction

Lameness is an economic and welfare concern in swine breeding herds. Lameness is known to be painful. Lame animals may not attain optimum breeding efficiency (Ritter et al., 1999) and may be culled before attaining peak production causing economic loss. Foot lesion is an important underlying cause for lameness (Dewey et al., 1993). Damage to claws is often the initiating factor for lameness in pigs. Claw lesions may cause lameness by acting as a source of pain. Infections may enter through the lesions and may spread upwards, leading to abscesses at the coronary band. Claw injuries during gestation may be associated with several factors including housing conditions and management on the farm. A combination of management, housing and behavioral factors are reported to be associated with development of foot lesions in pigs (Kroneman et al., 1993). The interaction between floor surface and the horn of the claw is responsible for the development of claw lesions (Simmins and Brooks 1988) and the severity of the lesion is influenced by the physical properties of floor (Jensen, 1979) and by the physical properties of the claw. Nutrition, especially related to biotin has been reported to have a major role in the development of claw lesions (Simmins and Brooks, 1988). The present study aims to characterize, and analyze the factors associated with claw lesions in a swine-breeding herd in the University of Minnesota.

Methodology

The study was conducted at the University of Minnesota Southern Research and Outreach Center, Waseca, MN. The research unit was an 800-sow (GAP, English Belle) breed-to-wean facility. This facility had 6 pens of 12.75 x 6.75 m size, with a central walk-through electronic sow feeders (ESF) (Osborne Industries TEAM electronic sow feeder, Kansas, USA, manufactured in 2001) in each pen and conventional gestation stalls (Crystal Spring Hog Equipment Ltd. St Agathe, MB, Canada). Pens and stalls had fully slatted flooring (12.7 cm solid portion, 2.54 cm slot, 12.7 cm deep). The study involved 184 sows (166 – 337 kg at 108 days of gestation) of parities 0-8. Of these sows, 102 were housed in group pens with ESF during gestation and others were in stalls during gestation. Sows were fed 2.2–3.0 kg of feed daily (CP content 15%), based on body weight and backfat at weaning. The sows were fed on an average 2.2 kg/ day and the quantity was increased by 0.1 kg/day every 30 days until day 109 of gestation. Sows were moved to farrowing crates (214 X 66 cm, excluding a creep area for the baby pigs, with cast iron flooring) on day 109 of gestation. The sows were then offered 3 kg feed per day until farrowing. Pigs were offered ad libitum feed during lactation. Data on daily lactation feed intake, body weight and backfat thickness were collected from sow cards. Daily lactation feed intake was recorded both in the morning and evening based on feed disappearance. Body weight and backfat thickness at day 108 and at weaning was recorded. All sows were weaned at 18.00 days of lactation.

The feet of the sows were individually examined for claw lesions on day 109 of gestation when they were in farrowing crates. Lesions comprised of erosions, cracks and overgrowths. The horny side wall and the volar / plantar surface of the hoof were examined for lesions. The medial (inner) and lateral (outer) claws of each foot were examined for lesions on a severity scale of zero (nil) to four (severe) (**Table 1**). For examination, areas on the claw were classified as side wall (composed of hard horn), heel (including overgrown heel), junction between heel and sole, sole, white line and toe. The final score on each area was obtained by multiplying number of lesions with corresponding severity of the lesions. Total score on each foot was obtained by adding scores for different areas of the claws in that foot. Scores on all areas on all feet were added together to obtain total claw lesion scores (TCLS). Sows were made to walk for short distance to detect lameness and degree of lameness was visually assessed if found lame. Degree of lameness was scored based on scale from zero to 6 on one or more legs (**Table 1**).

Statistical analysis

Median and range were used to describe the data collected on lesions and lameness. Logistic regression model (stepwise, Proc Logistic, SAS) was fitted to analyze (Wald statistic) the association of lameness (lame- L0 and non-lame L1), parity (P1- parity 0, P2 -parities 1 and 2, P3 - parities 3 to 5 and P4- parities 6), gestation housing systems (pens with ESF and stalls), body condition

(backfat and body weight) and average lactation feed intake with median TCLS (<median=0, > median=1) as the outcome variable. Lesions on different areas of claws, in different claws and in different limbs were compared among lame and non-lame sows by use of Kruskal-Wallis non-parametric one way ANOVA. Percentage distributions of sows with TCLS were calculated. Percentage distributions of sows with TCLS were also categorized among housing systems and among lame and non-lame sows. All the analyses were performed using computer software SAS version 9.1. A P value of < 0.05 was considered significant for all comparisons.

Results

Median and range of lesion scores on different areas of claws are presented in **Table 2**.

Severe lesions were more on sidewalls and heel. The severity of lesions was more on lateral claws than on medial claws in both limbs. Similarly, severity of lesions was more on forelimbs than in hind limbs.

Figure 1 presents the distribution of different levels of lesion scores among the sows studied.

Of the total sows, only 3.8 % were without any claw lesion. Approximately 36% of sows had TCLS <10. More than half of the population had TCLS in the range of 10-20 and 8.7% had scores >20.

The proportion of lame and non-lame sows with different levels of TCLS is presented in **Figure 2**.

Among non-lame sows 4.2% had no claw lesions while 2.5% of lame sows had no claw lesions. Among lame sows, 20% had TCLS <10 whereas 40.3% of non-lame

Table 1: Description of scoring pattern for claw lesions and lameness.

Claw lesion and lameness scores	Description of the scoring
Claw lesion scores	
Score 0	No lesions
Score 1	Small, superficial cracks or lesions in the epidermis
Score 2	Serious lesions in the epidermis
Score 3	Deep lesions extended into the corium
Score 4	serious and deep cracks extended into corium or subcutis
Lameness scores	
Score 0	No visible limp
Score 1	Walks with a slight limp and fairly strong on legs
Score 2	Walks reasonably well but with a noticeable limp
Score 3	Walks with much difficulty and is often not weight-bearing or only slightly weight-bearing
Score 4	Can be made to stand but with great difficulty and in much pain
Score 5	Downer – cannot be made to stand and in much pain
Score 6	Splayed

Table 2: Median and range of lesion scores on different areas of claws in both limbs

Lesion scores	Median	Range
Side wall	3	0 - 12
Heel	3	0 - 12
Overgrown heel lesion	0	0 - 8
White line	1	0 - 10
Junction between heel and sole	1.5	0 - 10
Sole	0	0 - 6
Toe	0	0- 1
Front limb lateral claw	4	0 - 18
Front limb medial claw	1	0 - 11
Front limb total	6	0 - 23
Hind limb lateral claw	4	0 - 14
Hind limb medial claw	0	0 - 4
Hind limb total	5	0 - 14
Total claw lesion	11	0 - 31

Figure 1:Proportion of sows with different levels of total claw lesion scores.

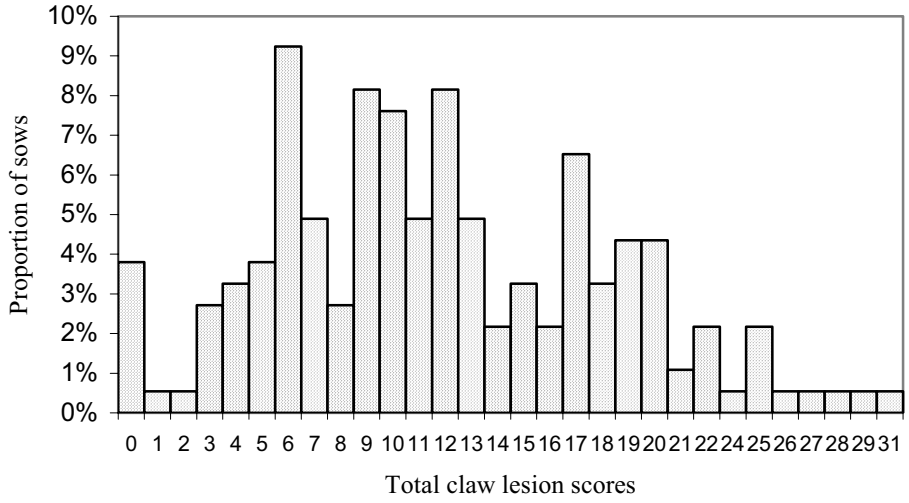
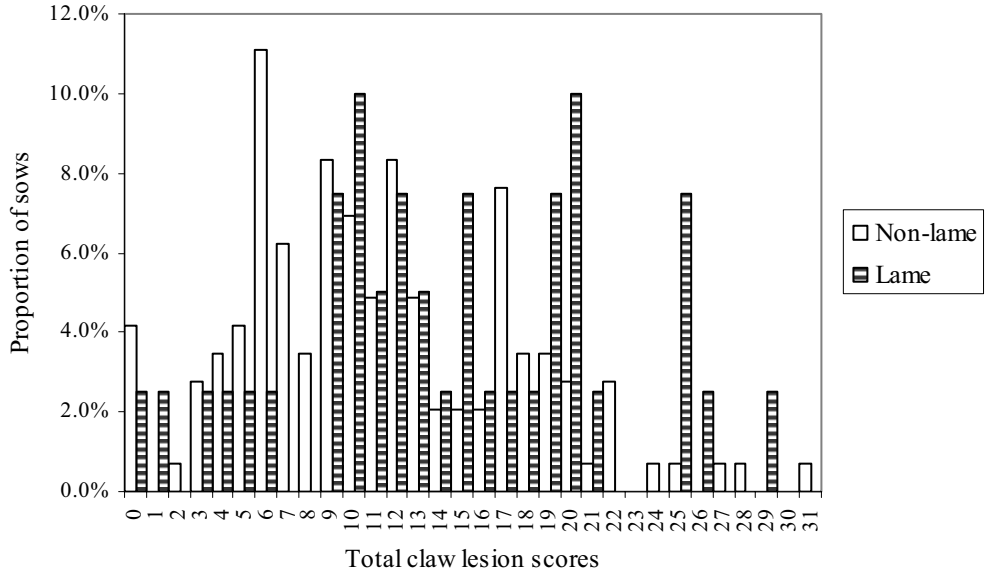


Figure 2: Proportion of lame and non-lame sows with different levels of total claw lesion scores.



sows had TCLS <10. While 62.5 % of lame sows had TCLS of 10-20, only 48.6% of non-lame sows had similar TCLS. Approximately 7 % of non-lame sows and 15% of lame sows had TCLS >20.

Figure 3 presents the distribution of sows in pens with ESF and stalls with different levels of TCLS.

Approximately 9% of sows in stalls had no claw lesions whereas there was no sow without claw lesion in pens with ESF. The proportions of sows with a TCLS of <10 were 14.7 and 62.2 in pens with ESF and stalls respectively while 14.7 % of sows in pens with ESF and 1.2% sows in stalls had TCLS >20.

An area-wise distribution of claw lesions among the sows is presented in **Figure 4**.

Figure 4 shows that most of the sows had lesions on sidewall and heel. Another area where lesions were common was at the junction between heel and sole. Toe lesions were relatively uncommon. The distribution of sows with and without lesions on lateral and medial claws in fore and hind limbs is presented in **Figure 5**.

Figure 5 shows that lesions were more common in lateral claws than in medial claws in both forelimb and hindlimb. More than 90% of sows had lesions on lateral claws. The association of different factors with >median TCLS is presented in **Table 3**.

Sows with higher backfat thickness at day 108 of gestation had a lower likelihood of >median TCLS. The likelihood of >median TCLS was lower among non-lame sows compared to lame sows. Sows in pens with ESF had 22 times higher likelihood of having >median TCLS com-

Figure 3: Proportion of sows with different levels of total lesion scores among pens with ESF and stall gestation housing.

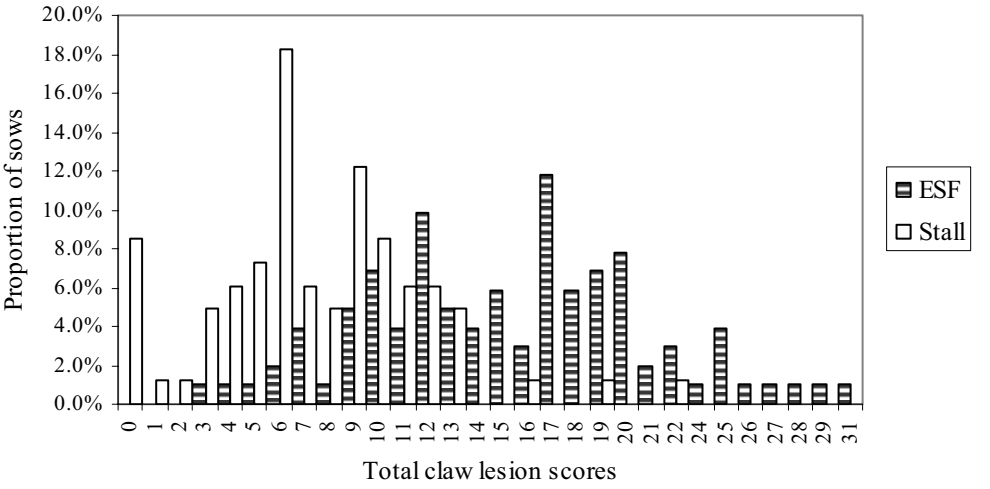
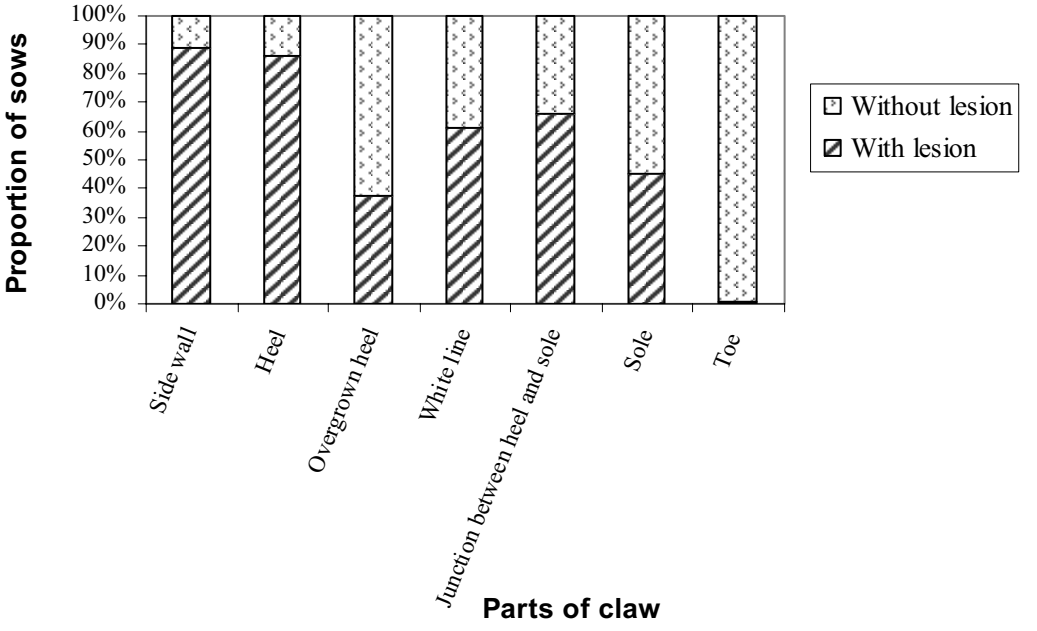


Figure 4: Proportion of sows with and without lesions on different areas of claw.



pared to sows housed in stalls. Parity, body weight and average lactation feed intake did not have significant association with claw lesion scores.

Analysis (Kruskal-Wallis ANOVA) indicated that claw lesions on the sidewall and white line were higher ($P<0.05$) among lame sows than in non-lame sows, whereas there was no difference on lesions on other areas of claw among lame and non-lame sows. Similarly, claw lesions in front and hind limbs and overall lateral claw lesions were higher ($P<0.05$) among lame sows than in non-lame sows. However, medial claw lesions were similar among lame and non-lame sows.

Discussion

The intensive selection and management in the present swine industry has made pigs grow rapidly to a large body weight and this may affect the soundness of legs and feet (Kroneman et al., 1993). The fewer number of sows without any claw lesions observed in the present study (**Figure 1**) may be indicative of the adverse effect of this rapid growth on feet health. Gjein and Larssen (1995) based on a study in slaughtered sows have reported that more than 96% of loose-housed sows and 80% of confined sows had at least 1 lesion on the lateral hind claws. The most prevalent lesions reported in that study were sidewall

Figure 5: Proportion of sows with and without lesions on different claws of front and hind limb.

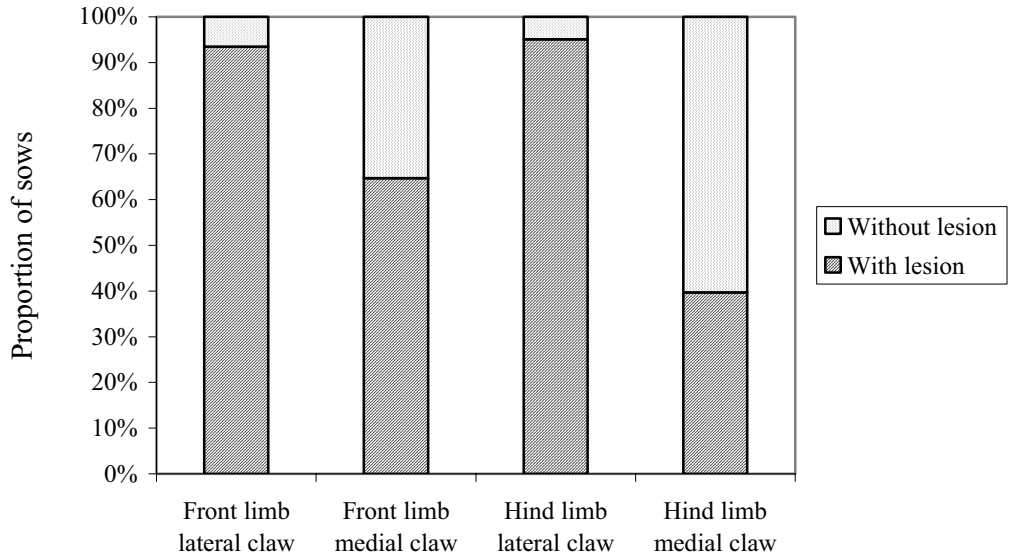


Table 3: Odds ratios (OR) and confidence interval (CI) for the association of backfat thickness (mm), lameness and gestation housing system with > median total claw lesion scores.

Factors	Odds ratio	95 %CI
Backfat at 108 day of gestation	0.876	0.802 - 0.957
Non lame vs. Lame	0.310	0.115 - 0.834
Pens with ESF vs. Stall	22.426	9.471 - 53.101

cracks, heel lesions, cracks in the white line and overgrown heels. The flooring has a contributing role in the development of claw lesions. Most of the claw lesions observed in this study was developed while the sows were in their gestation housing systems. Flooring has been viewed as an important factor causing claw lesions in pigs (Brennan and Aherne, 1987; Applegate et al., 1988). Claw lesions develop as a result of complex interactions between the floor surface and the claw horn of the foot. The extent of abrasion and trauma are influenced by the physical properties of floor (Jensen, 1979) and by the physical properties of the claw. A slippery floor may cause cracks in the sidewall. Similarly, excessive roughness may lead to lesions on heel or at the junction between heel and toe. In slatted floors, foot catching in between slats is a source of claw lesions. Holmgren et al., (1998) have reported a higher incidence of claw problems when sows were housed in partially slatted concrete floor than on strawbedding or on solid concrete floor. The space between slats, roughness of the surface and the edge design are all crucial factors in deciding the extent of injury (Boon and Wray, 1989).

Foot lesion has long been known as a cause for lameness (Penny et al., 1963); it may act as a source of pain and as an entry point for infection, which spreads upwards. Dewey et al., (1993) have also reported that foot lesions were major cause for lameness in sows. The finding in the present study of higher proportion of sows with severe claw lesions among lame sows than in non-lame sows (**Figure 2**) and the higher likelihood of >median TCLS in lame sows (**Table 3**) are in agreement with previous reports on foot lesions as a cause for lameness. Sidewall lesions and lesions on the white line are reported to be among the most prevalent claw lesions (Gjein and Larssen, 1995). The score for these lesions and overall lesion scores were higher among the lame sows than in non-lame sows in the present study indicating that claw lesions are one of the major causes for lameness as reported previously. Although the relationship between the presence of claw lesions and exhibition of lameness is obvious, claw lesion is only one of the factors leading to lameness in pigs. It has been reported (Logue et al., 1989) in cows that lameness may occur in the absence of lesions, or that the presence of lesions may not necessarily cause lameness in a cow.

The amount and type of movements of a sow may determine the type and severity of claw lesions and these 2 factors vary greatly in gestation stalls and group pens with ESF. Fighting and chasing following mixing can increase the chance of claw lesions in group pens. The lower pro-

portion of sows without claw lesions and with less severe claw lesions in stalls compared to sows in pens with ESF may be due to the increased mobility of sows in the ESF system. According to Kroneman et al., (1993) most of the cases of lameness in group-housed pigs are caused by foot problems. Anil et al., (2005) have also reported higher injuries on leg among sows in pens with ESF than stalls because of competition to enter the feeder, queuing and aggression. The higher incidence of claw lesions among sows housed in pens with ESF (**Figure 3**) and the higher likelihood of >median TCLS (**Table 3**) observed in the present study are in agreement with the above reports.

The development of lesions on different claws and different limbs is determined mostly by the weight distribution of the sows (Kroneman et al., 1993). Lesions may not develop equally on all claws. Simmins and Brooks (1988) observed more lesions on fore and hind outer claws (lateral) than fore and hind inner claws (medial). Hoof lesions are more common in lateral claws because of a greater weight-bearing surface than the medial claws (Tubbs, 1988). More than 3/4th of the weight of the pig is born by the lateral digits and 80 % of injuries are also on these digits (Webb, 1984). The strength of different parts of the foot may also vary and a region between hard and soft parts may be more susceptible to injuries (Kroneman et al., 1993). In the lateral digit, maximum weight is born by the heel bulb followed by the junction between heel bulb and the abaxial hoof wall whereas the tip of the toe is the most weight-bearing region (Webb, 1984). The findings in the present study of a higher proportion of sows with lesions in the heel, and at the junction between heel and sole (**Figure 4**) and a higher proportion of sows with lesions on the lateral claws compared to medial claw (**Figure 5**) are in agreement with the above reports.

Nutrition plays an important role in the health of the claw. Biotin intake has reported to increase the compressive strength of mid-abaxial sidewall of the claw (Webb et al., 1984). De Jong et al., (1983) had reported that after 2.5 months of biotin supplementation the claw lesion score of the gilts had decreased by 28% towards the end of gestation. It has been reported that thin sows are more likely to become lame than fat sows and that lameness was associated with skin lesions on the hock and hind feet (Bonde et al., 2004). However, the reported study had not specifically considered claw lesions. Another report on cows has indicated a higher risk of sole lesions in those with low body condition scores (Bell et al., 2005). Tubbs (1988) concluded that adequate but not excessive feed intake during gestation and high feed intake during lactation help to avoid downers and trauma related lameness problems. Though the nutrients in the feed were not analyzed in the present study, the above reports may explain the lower likelihood of >median claw lesions associated with higher backfat thickness at the end of gestation observed in this study (**Table 3**). Backfat thickness at the end of gestation may be considered as an indication of gestation feed intake though feed intake during gestation was not assessed in this study. Although feed intake may be affected when severity pain associated with claw lesions is high, there was only one sow with very severe claw lesion and a lameness score of 4 in this study. This explains the lack of association between claw lesions and lactation feed intake. The horn tissue in pigs grows faster than that of other animals. It has been reported that claw lesions can appear and disappear quickly (Kroneman et al., 1993). Mild lesions thus may not be carried over to subsequent parities and this may explain the lack of association between parity and claw lesions.

Conclusion

The results of this study indicated that claw lesions were common among sows and were associated with lameness. Lame sows had higher sidewall and white line lesion scores. Sows in pens were likely to have higher claw lesion scores than stall-housed sows. Sows with better body condition in terms of backfat had lower claw lesion scores. Claw lesion scores were not associated with parity, body weight at the end of gestation or average lactation feed intake.

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