Sow Claw Lesion Pathology

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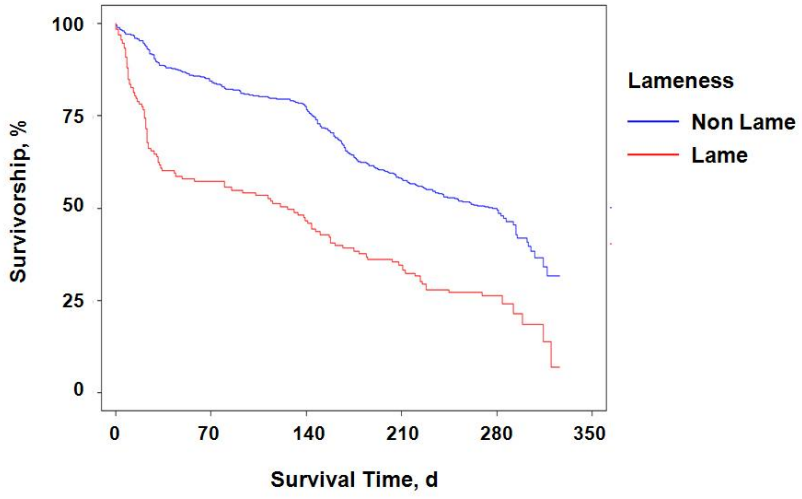
Pathology textbooks typically begin with the explanation that the direct translation of the term 'pathology' from the ancient Greek is the 'study of suffering.' We don't always think of our discipline in those terms, but in the case of sow lameness this concept seems particularly appropriate as there are several levels of suffering that can be considered. First and foremost is the suffering of the sow due to discomfort at the least and, more likely, some degree of pain and distress that manifests as lameness. Second, the farmer suffers from the reduced biological and financial performance in his or her operation as a result of lameness problems in breeding stock. Finally, one could argue, albeit feebly, that veterinarians can suffer from some measure of frustration as diagnostic investigations of lameness in sows can be quite challenging and laborious (Done, 1979). Our focus will be on the pathology experienced by the sow for purposes of this discussion.

Veterinary medicine has been embracing the concept of evidence based medicine in recent years to bolster the scientific basis for evaluating clinical diagnoses and response to treatment. The evidence used for such evaluations is the published scientific literature that relates to the specific disease or clinical presentation in question. An examination of scientific research on claw lesions as it relates to sow lameness provides a basis for understanding the clinical relevance of the lesions we observe. Working through a series of papers related to sow lameness and claw lesions is an opportunity to use some inductive reasoning to develop this understanding.

There should be no question about the significance of lameness as a key factor in sow herd performance since sow lameness has been shown repeatedly to be one of the lead factors associated with culling, euthanasia and even mortality for sows. In Figure 1 we see recent work illustrating the more rapid removal of sows that were lame at farrowing when compared to sows that were not lame (Anil, 2009). The dotted line in the graph represents sows that were lame in farrowing and the solid line represents sows that were not lame. Day 0 is the day of farrowing following the lameness assessment. As the lines indicate, the lame sows were removed from the herd more quickly than the non-lame sows, with a 50% survival rate two times longer for non-lame vs. lame sows. Many similar reports have been published that document the large contribution of lameness to sow culling and mortality.

Figure 1.



(Anil 2009)

So what about a link between feet and lameness? Taking it one step at a time, we can first examine the prevalence or incidence of hoof lesions in sows. Researchers have reported on claw lesions in sows as far back as 1950 when infected claw lesions observed in New Zealand pigs were described by Osborne as footrot (Osborne, 1950). The frequency of foot lesions has been reported several times over several decades. In Table 1, Penny (1963) reported on an extensive survey of foot lesions in a paper that first defined the classification of foot lesions along with reporting on the prevalence.

Table 1. Lesions of Pigs Feet: Lesions Seen as Percent of the Total Lesions

	Total	Distribution of lesions				
Survey	number of lesions	Heel erosion	Sole erosion	Toe erosion	White line lesion	False sand crack
H	2,349	31.1	10.4	20.2	34.6	3.8
C	6,799	30.6	21.5	24.2	21.2	2.7

(Penny, 1963)

A more recent report illustrates the extent to which the distribution of the lesions in sows has been examined in light of various risk factors that may affect the development of lesions. The results shown in Table 2 represent more than 2000 sows that were examined for foot lesions (Kilbride, 2008). The epidemiology of foot lesions will not be considered further here.

Table 2. Number and percent of multiparous sows with foot lesions score 1-3 by parity, reproductive stage and breed line

	Any lesion		Over-grown claws		Wall damage		White line lesions		Toe erosion		Heel/ sole erosion		Heel flap		Heel corru-gation		Total
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n
Parity																	
1st	276	65.1	36	8.5	40	9.4	18	4.2	110	25.9	126	29.7	58	13.7	28	6.6	424
2nd	309	69.1	49	11	36	8.1	28	6.3	124	27.7	124	27.7	77	17.2	26	5.8	447
3rd	275	68.1	54	13.4	32	7.9	16	4	99	24.5	151	37.4	64	15.8	21	5.2	404
4th	295	75.3	66	16.8	40	10.2	20	5.1	122	31.1	135	34.4	81	20.7	16	4.1	392
5th	199	70.1	42	14.8	21	7.4	15	5.3	62	21.8	99	34.9	51	18	21	7.4	284
6th	51	71.8	12	16.9	6	8.5	4	5.6	16	22.5	22	31	12	16.9	2	2.8	71
7th plus	56	75.7	4	5.4	7	9.5	3	4.1	17	23	25	33.8	18	24.3	7	9.5	74
Reproductive stage																	
Lactating	716	109	140	21.2	104	15.8	44	6.7	221	33.5	303	45.9	214	32.4	112	17	660
Pregnant	865	54.7	152	9.6	96	6.1	65	4.1	357	22.6	448	28.3	177	11.2	29	1.8	1581
Breed line																	
Non-pigmented	1061	74.1	219	15.3	115	8	78	5.5	415	29	532	37.2	233	16.3	76	5.3	1431
Pigmented	424	63.5	63	9.4	64	9.6	26	3.9	133	19.9	185	27.7	123	18.4	49	7.3	668
Indoor vs. outdoor																	
Indoor	1303	73.4	160	9	143	8.1	145	8.2	472	26.6	562	31.6	256	14.4	125	7	1776
Outdoor	124	62	11	5.5	15	7.5	0	0	29	14.5	44	22	42	21	11	5.5	200

For our purposes, the relative contribution of foot lesions to lameness and reduced performance in sows is important to understand, and this has been examined repeatedly. Dewey (1993) reported on the prevalence of foot lesions as the primary cause of lameness among sows that were culled for lameness in a Canadian herd (Table 3). In Table 4, similar work in 15 Norwegian herds provided more information on the contribution of claw lesions to lameness.

Table 3. The causes of lameness on 50 sows culled for lameness as determined by clinical and gross postmortem examination

	Number of sows	
Diagnosis	Primary cause	Additional lesions
Osteochondrosis	17	4
Arthrosis	6	4
Infectious arthritis	11	2
Foot lesions	10	11
Other	6	

(Dewey, 1993)

Table 4. Lameness in relation to major claw lesions (score >= 3) and claw infections in 15 loose herds

		Lame – Left hind leg			
		Yes	No	%	RR (95%CI)
Major claw lesions	Yes	62	487	11.3	1.3 (0.8-1.9)
	No	27	272	9.0	1.0
Claw infection	Yes	12	13	48.0*	5.2 (3.3-8.2)
	No	75	734	9.3	1.0

RR = Relative risk. *Significantly more lame sows ($p < 0.05$). (Gjein, 1995)

More recent work in Denmark (Table 5) ,Sweden (Table 6) and Finland (Table 7) further characterize the relative contribution of lameness generally and foot lesions specifically to the reduced performance of sows.

Table 5. Primary causes of killing (n=172) and spontaneous death (n=93) of sows in Denmark

	Killed sows		Spontaneously dead sows	
Primary causes	**No.**	**%**	**No.**	**%**
Locomotive system				
Bone fractures [13 cases (48%) of fractures in the physis of proximale humerus or femur (epiphysiolysis)]	27	16	0	0
Arthroses	15	8	0	0
Arthritis	41	24	0	0
Osteomyelitis, other locations	12	7	0	0
Vertebral osteomyelitis	19	11	0	0
Other lesions (claw lesions, rupture of ligament etc.)	9	5	0	0
Total	123	72	0	0
Reproductive system				
Endometritis, retained fetuses, rupture of uterus	16	9	22	24
Gastrointestinal system and spleen				
Torsion of liver lobuli	0	0	11	12
Torsion of spleen	0	0	8	9
Haemorrhagic gastritis	0	0	6	6
Proliferative haemorrhagic enteropathy	0	0	7	8
Rupture of liver, perforation of oesophagus,intestinal volvulus	7	4	10	11
Total	7	4	42	45
Urinary system				
Pyelonephritis, cystitis	3	1	5	5
Miscellaneous				
Septicaemia, endocarditis, trauma because of fighting, pneumonia, pleuritis, tumour	12	7	12	13
Not stated	11	6	12	13

(Kirk, 2005)

Table 6. Descriptive statistics on pathological-anatomical findings, including most incidental findings, in 96 post mortem examined sows/gilts from a large Swedish herd.

	Found dead (n = 17) No.	Euthanized (n = 79) No.	Total (n = 96) No.	Total %
Arthritis	2	41	43	44.8
Abscess, at least one	3	34	37	38.5
Teeth injuries	6/15	21/72	27/85	31.0
Osteochondrosis/ epiphysiolysis	0	21	21	21.9
Kidney/urinary bladder failure	4	12	16	16.7
Pneumonia (App, SEP)	1	11	12	12.5
Mastitis	4	7	11	11.5
Fracture	0	10	10	10.4
Gastritis and/or ulceration	1	9	10	10.4
Heart disorders	5	5	10	10.4
Claw disorders	2	6	8	8.3
Abscess in spinal cord	0	7	7	7.3
Liver disorders	2	2	4	4.2
Reproductive organs	0	3	3	3.1
Spleen disorders	1	1	2	2.1

(Engblom, 2008)

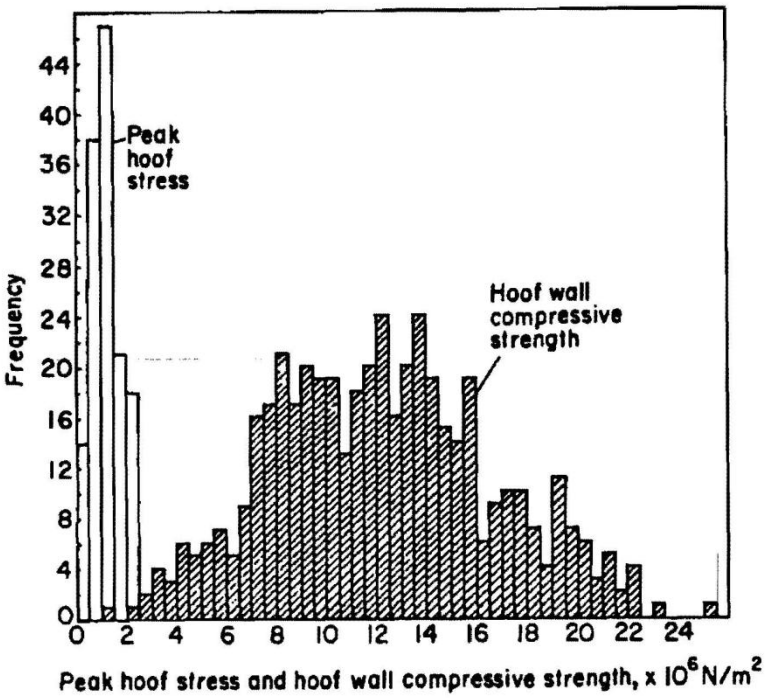
Table 7. Distribution of clinical lameness among 646 sows and gilts in 21 loose-housed herds.	
Lameness diagnosis	Percentage of sows and gilts affected
OC/OA	4.3%
Infected skin wound	1.2%
Arthritis	0.8%
Claw lesions	0.9%
Infected claw lesions	0.6%
Overgrown claws	0.6%
Nervous signs	0.3%
Total	8.8%

(Heinonen, 2006)

The data demonstrates associations of foot lesions with sow lameness, performance and mortality. The prevalence of foot lesions and the extent to which the lesions are associated with herd problems obviously has a fairly broad range in the published literature. Multiple factors affect this variability and several of these factors were explored by the different researchers, but will not be considered further here.

However one factor that is specific to the foot is the variability in hoof wall strength. This variability is illustrated in Figure 2, which show a tremendous range of hoof wall compression strength test results for growing pigs.

Figure 2. Histogram showing the frequency of occurrence of measurements of peak hoof stress and of the compression strength of the hoof wall (Webb, 1984)

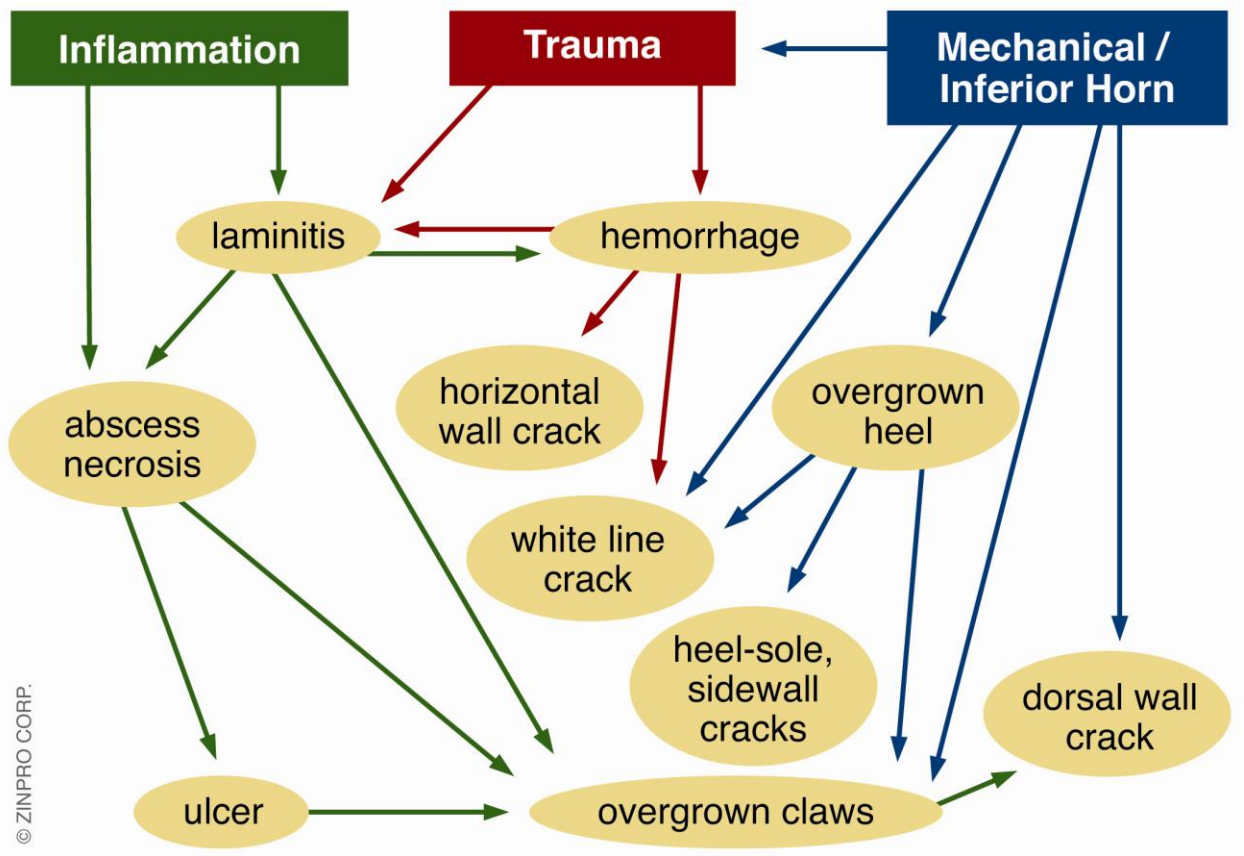


Multiple factors are also associated with hoof wall strength, including genetics, nutrition and environmental conditions. In a paper by Webb and others published in the same year (Webb *et al* 1984), feeding supplemental biotin was shown to affect the compressive strength and hardness of the hoof wall of pigs. Previously, Brooks (1977) had demonstrated a reduction in foot lesions in sows by feeding supplemental biotin. Taken together, we see evidence that hoof wall strength and hardness can affect the development of lameness in pigs and are influenced by nutrition.

The evidence from the published literature, finally, can be fairly summed as follows regarding the role of foot lesions in lameness: 1) Lameness affects sow performance; 2) foot lesions are associated with lameness in sows; 3) hoof wall strength is variable in sows, and; 4) hoof wall strength affects foot lesion development.

This information is helpful as we seek to develop an understanding of pathogenesis of foot lesions in pigs. Ossent (2010) spent considerable time exploring the lesions observed in sow feet, and considerable thought as to the pathogenesis of these lesions. Figure 3 illustrates the three main causative factors that result in foot lesions, and provides a flow diagram for how the ten primary lesion forms can develop.

Figure 3.



Ossent, 2010

Brooks (1977) published a schematic for lesion categorization scheme that has formed the basis for much of the subsequent work on foot lesion pathology. The following pictures taken by Pete Ossent summarize the primary foot lesions pathology according to the scheme he modified from Brooks.

A. Trauma

Haemorrhage in the claw capsule

Horizontal crack

Double sole/ heel/ wall

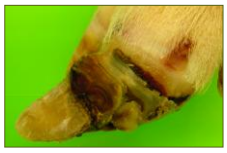
B. Inflammation

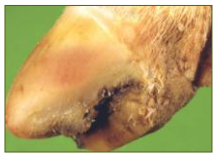
Overgrown toe

Laminitis

Abscess in corium

Overgrown heel





C. Mechanical / Inferior Horn

White line crack

Side wall crack

Heel-sole crack

Dorsal wall crack

D. Excessive / Inadequate wear

Excessive wear

Inadequate wear

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