EPIDEMIOLOGY OF LAMENESS IN BREEDING FEMALE PIGS

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To all the faculty and students of the swine group.

Dedication

This dissertation is dedicated to those who work hard to minimize welfare problems in intensive swine production systems...

Abstract

A low level of sow retention in the herd is a cause for both economic as well as welfare concerns. The results of the study confirmed that a low lactation feed intake, incidence of lameness or health problems, as well as sow-level characteristics such as higher parity and fewer piglets born alive per litter may adversely affect sow longevity. Sows retained with periparturient health problems had reduced longevity and fewer live-born piglets, and fewer such sows had another farrowing. A prospective data analysis indicated that the overall performance of lame sows in terms of the number of pigs born alive during the period of the study was less, compared with that for non-lame sows. Retaining sows with less severe lameness may enable the producer to meet immediate production targets. The findings suggest that sow removal decisions should be judiciously evaluated after farrowing considering the potential long-term losses. Lameness in swine herds should be minimized and if treatment is not an option lame sows should be culled as soon as possible to reduce long-term losses.

The results also confirmed the high prevalence of claw lesions in breeding female pigs and their association with lameness, specifically, white line and side wall lesions. The results indicate the possibility of nutritional intervention in minimizing claw lesions. However, there are other factors associated with claw lesion development in pigs. The quality of the floor as well as different bio-mechanical factors operating in lesion development are important here. The space between slats, roughness of the surface, and edge design are critical in claw lesion development. Those factors have not been addressed in this study. Further studies are required to understand the mechanism of lesion development in relation to the housing and management systems in place. This information is vital in formulating the appropriate intervention strategy to minimize the incidence of lameness and to improve sow longevity and performance. The studies in this thesis included data from single herds and therefore the generalization of the results may be restricted owing to the wide variations in management, housing and in genetic lines of sows.

Table of Contents

Acknowledge	ement	i
Dedication		ii
Abstract		iii
Table of contents		v
List of Tables		vii
List of Figure	S	X
Chapter I:	Literature review and justification of the problem	1
Chapter II:	Analysis of periparturient risk factors affecting sow	
	longevity in breeding herds	7
	2.1. Introduction	9
	2.2. Materials and methods	13
	2.3. Results	16
	2.4. Discussion	19
Chapter III:	Effects of lameness on sow longevity	32
	3.1. Introduction	34
	3.2. Materials and methods	36
	3.3. Results	39
	3.4. Discussion	40

Chapter IV: Factors associated with claw lesions in gestating

	Sows	50
	4.1. Introduction	52
	4.2. Materials and methods	53
	4.3. Results	55
	4.4. Discussion	57
Chapter V:	Nutritional intervention to minimize claw lesions and to	o improve
	performance and longevity in breeding female pigs	69
	5.1. Introduction	72
	5.1. Introduction5.2. Materials and methods	72 74
	5.2. Materials and methods	74
General discu	5.2. Materials and methods5.3. Results	74 77

List of Tables

Table 2.1.	Descriptive summary of data (number of sows/mean \pm SE) on	
	2066 commercial sows and a subset of data on 1357 sows with	
	lactation feed intake	24
Table 2.2 .	Performance variables (mean ± SE/number) of commercial sows	
	retained with (N=844) and without (N=936) health problems durin	g
	the periparturient period in the subsequent parity	25
Table 2.3 .	Odds ratios and confidence intervals of risk factors associated with	ı
	sow longevity within 35 d post-farrowing among commercial	
	sows (N=1357)	26
Table 2.4 .	Odds ratios and confidence intervals of risk factors associated	
	with sow removals before the next parity among commercial	
	sows (N=1357)	27
Table 3.1.	Results of survival analysis of the complete model for the	
	association between sow longevity and lameness, parity,	
	and farrowing performance	47
Table 4.1.	Scoring method for claw lesions in a study examining 184	
	sows at day 110 of gestation	62
Table 4.2.	Median and range of lesion scores on different areas of claws	
	across all limbs in 184 sows at day 110 of gestation	63
Table 4.3.	Proportions of sows with hoof lesions according to type of	
	gestation housing	64
Table 4.4.	Odds ratios (OR) and confidence intervals (CI) for association	
	of body condition (indicated by P2 back fat thickness and body	

	weight at gestation day 109), lameness, parity, and gestation housing systems with presence or absence of lesions on different	
Table 5.1.	claw areas in 184 sows Median and range of total lesion scores in control and treatment	65
	group sows housed in group pens with ESF for the entire study period	84
Table 5.2 .	Comparison of the number of group housed sows with claw lesion	04
	scores showing improvement or no change in different claw areas	
	with the number of sows showing worsening of lesion scores	85
Table 5.3.	Comparison of production performance in treatment (T) and	
	control (C) group sows housed in pens with ESF (2 sample	
	t test)- all farrowings	86
Table 5.4.	Spearman correlation between claw lesion scores and performance	
	variables among group housed sows	87
Table 5.5.	Median and range of total lesion scores in control and treatment	
	group sows housed in stalls during the study period	89
Table 5.6.	Comparison of the number of stall-housed sows with claw lesion	
	scores showing improvement or no change in different claw areas	
	with the number of sows showing worsening of lesion scores	
	(2 sample proportion test)	90
Table 5.7.	Comparison of the production performance of control and treatmen	nt group
	sows housed in stalls (2 sample t test)	91
Table 5.8.	Spearman correlation between claw lesion scores and performance	

Table 5.9.Odds ratios and 95% CI of the association of number of piglets
born alive per litter (≤ 10 vs. >10) with total scores for side wall and
white line lesions, housing system, parity, cycle, organic trace mineral
feeding, stillborn, mummies and preweaning mortality (outcome
evaluated is the likelihood for ≤ 10 piglets born alive per litter)94

Appendix 1

112

List of Figures

Figure 2.1.	Percentage of commercial sows culled and dead (including	
	euthanized) within 35 d post- farrowing (178 sows culled and	
	39 sows dead)	28
Figure 2.2.	Percentage of commercial sows culled and dead (including	
	euthanized) before the next parity (399 sows culled and	
	104 sows dead)	29
Figure 2.3.	Percentage of commercial sows removed (within 35 d	
	post-farrowing) with lameness (N=77) or health problems	
	(N=140) during the periparturient period	30
Figure 2.4.	Percentage of commercial sows removed (before the next parity)	
	with lameness (N=116) or health problems (N=387) during the	
	periparturient period	31
Figure 3.1.	Kaplan-Meier graph of the time to removal from the herd for lame	e (dotted
	line) and non-lame (solid line) sows after lameness	
	assessment	48
Figure 3.2.	Kaplan-Meier graph of the time to removal from the herd for sows	5
	of parity 1 and 2 (solid line), 3 through 5 (dotted line), and > 5	
	(dashed line)	49
Figure 4.1.	Proportions of 184 sows with and without lesions on different	
	claw areas	66

Figure 4.2.	Proportions of 184 sows with and without lesions on different area	.S
	of lateral and medial claws	67
Figure 4.3.	Proportions of 184 sows with and without lesions on lateral and	
	medial claws of front and hind limbs	68
Figure 5.1.	Proportion of group housed sows in the control and treatment	
	groups with different levels of total claw lesion scores	95
Figure 5.2.	Kaplan-Meier graph of the time to removal from the herd for	
	control (upper line) and treatment (lower line) sows housed	
	in groups	96
Figure 5.3.	Proportion of stall housed sows in the control and treatment groups	S
	with different levels of total claw lesion scores	97
Figure 5.4.	Kaplan-Meier graph of the time to removal from the herd for contr	ol
	(upper line) and treatment (lower line) sows housed in stalls	98

CHAPTER I

LITERATURE REVIEW AND JUSTIFICATION OF THE PROBLEM

CHAPTER I

LITERATURE REVIEW AND JUSTIFICATION OF THE PROBLEM

Reduced sow longevity is a cause for both economic and welfare concerns in breeding herds. The extent of sow retention is dependent on the level of culling and the mortality rates. Culling and mortality rates average near 50 and 9%, respectively in commercial swine herds in the US (Mote et al., 2009). Productivity is generally considered positively related to sow longevity (Dourmad et al., 1994; Xue et al., 1996; Xue et al., 1997). Production related problems, followed by locomotor problems are the major reported sow removal reasons. Studies focusing on lameness or locomotor problem as a sow removal reason are limited in the US. The classical French study by (Dagorn and Aumaitre, 1979) several decades ago indicated inefficient production as the major sow removal reason, with approximately 9% of sows removed for locomotor problems. A recent Swedish study (Engblom et al., 2008) also reported that 9% of all removals were due to lameness. Stalder et al., (2004), based on their review summarized that the percentage of sows removed for locomotor problems ranged from 6 to 15%. The reported rate of removal due to lameness in US herds is 15.2 % (USDA, 2006). A South American study (Saballo et al., 2007) indicated that 41.3% of removals were due to reproduction related reasons and locomotory problems accounted for 18.5% of the culling, especially in sows of parity 1 and 7-8. Engblom et al., (2008) also reported that the hazard for removal due to lameness was greater in first parity than in higher parities. However, these reports have not considered the indirect effect of lameness on sow longevity. Lameness is a putatively

painful condition. Pain can inhibit feed intake. The pro-inflammatory cytokines (interlukin 1, 6 and tumor necrosing factor alpha) can cause anorexia (Johnson, 1997). If a lactating sow becomes lame or is lame it may not consume enough feed. Inadequate lactation feed intake can affect the subsequent performance of the sow (Kirkwood et al., 1987). This effect may be more serious in younger parity sows as they need to consume for both lactation and growth. If we take this approach, lameness may not be the second most important removal reason. Lameness may be the most important risk factor for sow longevity.

It is also important to note that many sows removed for other reasons could also be lame. However, often studies on sow removal reasons are based only on recorded reasons. Even though lameness is a serious welfare concern, all lame sows may not be removed from the herd owing to market conditions or pressure to meet production targets. Other factors likely to influence this decision include parity number, production, reproductive status, herd structure and access to replacement gilts (Engblom et al., 2008). Lame sows may go unrecognized and are likely to remain in the herd longer because artificial insemination requires less structural soundness for breeding. This may result in retaining sows with mild but chronic lameness in the herd. However, **the long term effect on overall herd productivity of retaining sows with health problems such as lameness is not well understood**. A German study has reported fewer litters and higher piglet losses in lame sows than in healthy sows. Other reports have also suggested a possible negative relationship between lameness and production performance (Penny, 1980; English and Edwards, 1999). Despite the high prevalence of lameness in swine herds, potential measures to minimize lameness and its adverse effects are less thoroughly explored compared to other swine diseases. Sow removal decisions, especially culling decisions, are often not based on information about the sow/s at a single point of time. Given the limitation of offering therapeutic interventions and /or immediate removal of all lame sows upon diagnosis, it is important to understand the effects of lameness on sow performance and longevity prospectively.

Different factors (genetics, management, housing, nutrition) are reported to be associated with lameness in sows. However, assuming that suitable genetic lines have been selected and that housing system and management are less changeable, nutrition offers an opportunity for intervention to minimize incidence of lameness. Hoof (claw) lesions are reported to be an important underlying cause of lameness in pigs (Dewey et al., 1993). Housing conditions and management on the farm may be associated with the development of hoof lesions in pigs (Kroneman et al., 1993). Hoof lesions are very common in pigs (Gardner et al., 1990; Kirk et al., 2005). Although, pain in mild lesions is difficult to be confirmed through overt signs, severe hoof lesions may cause lameness by acting as a source of pain. Lesions in the hoof may permit entry of infections that may spread upwards, affecting joints and leading to lameness. **Despite a high prevalence of claw lesions in sows, studies focusing on claw lesions and their association with lameness and longevity in breeding sows are limited in the US. Considering the etiology of claw lesions, there is considerable limitation in extrapolating the results** of studies conducted elsewhere. It is important to characterize and understand the factors associated with claw lesions and their association with lameness to minimize incidence of these lesions and to reduce removal of sows due to lameness.

Decreasing the number of sows culled due to lameness has positive effects on overall production and welfare. Specific factors associated with hoof lesions in pigs are reported to include the interaction between floor surface and horn of the claw (Simmins and Brooks, 1988), physical properties of floor and nutrition, especially related to dietary biotin levels (Jensen, 1979). Though housing conditions and management on the farm are crucial as immediate causes for the development of hoof lesions in pigs (Kroneman et al., 1993), nutrition, especially of trace minerals may also act as a predisposing factor. Nutrition is vital in developing the hoof structure and integrity. Minerals such as Ca, Zn, Cu, Mn and vitamins A, D, and E, as well as biotin are reported to be important in the keratinization of hoof epidermis (Tomlinson et al., 2004). Improper nutrition can thus cause inferior quality horn tissue that is easily susceptible to damage from the environment. It is also important to maintain a balance of trace minerals in the diet given the interaction among the minerals (Nocek et al., 2000). The extent of bioavailability is important in trace mineral nutrition. Proteinating improves the bioavailability of minerals to target cells and organs. It has been shown that chelated mineral is more soluble and can cross the intestinal wall more easily (Rompala and Halley, 1995). Supplementation may have to be continued for at least 6 months (Potzsch et al., 2003). An advantage of proper mineral nutrition, in addition to the improvement in hoof health is the potential beneficial effect on reproduction. Although the role of minerals in the keratinization

process is well established, there has not been a detailed study in breeding female pigs on the effect of trace minerals like Zn, Mn and Cu in preventing or treating claw lesions.

Thesis objective

The general objective of this thesis was to increase the understanding about lameness as a risk factor of sow longevity and to evaluate the effect of nutritional intervention in minimizing the incidence of claw lesions, a major cause for lameness. This task was divided into the following specific aims:

- to identify the periparturient risk factors of sow longevity
- to analyze the effect of lameness on sow longevity and performance
- to characterize claw lesions in breeding female pigs housed in group pens and in gestation stalls and to analyze the association between claw lesions and lameness
- to analyze the effect of organic trace minerals in minimizing claw lesions and improving performance and longevity in sows housed in group pens and in gestation stalls

CHAPTER II

ANALYSIS OF PERIPARTURIENT RISK FACTORS AFFECTING SOW LONGEVITY IN BREEDING HERDS

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The association of periparturient risk factors with sow longevity and the validity of sow removal decisions made during the periparturient period were analyzed. Data pertaining to 2066 sows from a commercial breeding herd from the US Midwest were used in this study. The likelihood of removal from the herd within 35 d post-farrowing decreased with a younger parity, the absence of lameness or other health problems, a higher lactation feed intake (LFI) and a greater number of live-born piglets (P<0.05 for all). A greater number of piglets born alive, the absence of lameness and a younger parity lowered (P<0.05 for all) the likelihood of removal of sows from the herd before the next parity. The number of piglets born alive was higher (P<0.05) among sows without any health problems during the previous periparturient period. A greater (P<0.05) number of sows that were retained without any health problems during the periparturient period farrowed. More sows (P < 0.05) retained with health problems during the periparturient period were culled compared with sows retained without health problems during the periparturient period. In summary, periparturient factors such as LFI, the incidence of lameness or health problems, as well as sow-level characteristics such as higher parity and fewer piglets born alive predicted the removal of a sow from the breeding herd. Sows retained with periparturient health problems had reduced longevity and fewer live-born piglets, and fewer such sows had another farrowing.

2.1 Introduction

Sow longevity is an important issue in swine breeding herds, as there are economic and welfare impacts of a lower sow retention rate. Early removal of sows from the herd due to mortality, health problems and low productivity can involve animal welfare and economic concerns (Rodriguez-Zas et al., 2003) and can adversely affect employee morale (Deen, 2003a). The addition of new gilts into the system may increase health risks to the prevailing members in the system as well (Sanz et al., 2002). The benefits associated with improved sow longevity include larger litters with heavier pigs, fewer unproductive days, acquired immunity to herd diseases, higher sow salvage value, and lower replacements costs (D'Allaire et al., 1987; Lucia et al., 2000). The extent of sow retention in breeding herds is dependent on the level of culling and mortality rates. A mean death rate of 8.8% and a mean culling rate of 53.1% have been reported in the US herds during 2006 (PigCHAMP, 2006). Productivity is positively related to sow longevity (Dourmad et al., 1994; Xue et al., 1997), although, different opinions are also reported (Stein et al., 1990; Bilkei and Bolcskei, 1995; D'Allaire and Drolet, 1999). Factors influencing sow removals are not consistent and often the removal of a female pig is not dependent on its biological performance alone. The criterion used for deciding on sow removals may not be equally applied to all females present in the herd at a given point of time. For instance, although sow culling is a voluntary decision, producers may be reluctant to remove a periparturient/lactating sow considering the productivity losses associated with such removals. However, the effect of retaining sows with health problems in the subsequent overall performance of the herd is seldom evaluated. Therefore, it is important to evaluate sow removal decisions.

Reproductive performance is the major factor influencing voluntary culling of breeding females, since it is essential for the sow to remain productive to remain in the herd. A sow may be removed from the herd for production or health related reasons. Reproductive inefficiency directly results in poor sow longevity, whereas health problems can affect longevity both directly and indirectly. Severe health problems can cause immediate removal of a sow from the herd. Indirectly, health problems can affect feed intake or subsequent reproductive performance of the sow and cause sow removals. It is relatively easy to decide on the point at which a healthy animal is no longer economically valuable based on pre-fixed production criterion. However, there is great difficulty in assessing sick or injured animals as there is difficulty in objectively assessing the extent of welfare compromise due to health problems. While it is possible to validate the removal decisions based on production performance by evaluating the improvement in performance at the herd level, the same is not possible for removals associated with welfare reasons. The difficulty is not in identifying whether the welfare is poor or not, but in determining the level of compromise in welfare due to health problems so that a decision to retain, cull or euthanize an animal can be made. Further, the production related consequences of retaining a sow with compromised welfare are also less obvious. Sows are more likely to receive individual attention when they are in the farrowing crate as the common practice is to feed the lactating sows individually and to

perform the routine procedures for the piglets while they stay with the sow in the farrowing crates. The individual attention facilitates recording of different health problems such as lameness, downer and prolapses, in addition to disease symptoms such as off-feed, vulvar discharge, fever, diarrhea and respiratory problems while the sows are in the farrowing crates.

The risk of removal for a breeding female is not the same throughout its life. Farrowing is generally considered as a high risk event for removal for both production and welfare reasons. Koketsu et al., (1996) has reported that the amount and pattern of feed intake during lactation can influence subsequent reproductive performance and can affect the amount of culling. A severe reduction in feed intake during lactation may be indicative of compromised welfare and may predispose a sow to removal following lactation. Stalder et al., (2004) noted that achieving maximum daily feed intake before the second week of lactation and having a maximum daily feed intake >8 kg will lower the risk of mortality. Anil et al., (2006) reported that sows consuming < 3.5 kg of feed per day during the first 2 wk of lactation were more likely to be removed from the herd before their next parity. Farrowing has been reported to be an important risk factor for sow mortality (Deen and Xue, 1999; Deen, 2003b). Karg and Bilkei (2002) reported that 40.2% of mortality of sows happened during lactation. The peripartum period is the risky period with 42% of all deaths occurring during this short period (Chagnon et al., 1991). Stalder et al., (2004) concluded that farrowing problems accounted for 1.6 to 7.2% of all culls made. Lameness is an important reason for health related removals. Among removed sows, 10 to 14 % of removals were due to locomotor problems (Stone, 1981; Friendship et al., 1986). In another study (Anil et al., 2005), it was observed that among sows that were removed, the proportion of sows that died (including euthanasia) was higher during lactation than during non-lactation. Uterine prolapse has also been reported as a major cause of sow removal, accounting for 6.6% of sow deaths (Chagnon et al., 1991). The chances for prolapses are higher around the time of farrowing. Although previous studies have evaluated the risk factors associated with sow longevity, most, if not all have focused on production or reproduction related problems. Similarly, none of them have evaluated the consequences of health related sow removal decisions made during the periparturient period on subsequent herd performance.

The objectives of the study described here were to identify the risk factors, including health related problems, operating at the periparturient period (i.e. while the sows were in the farrowing crate prior to farrowing and including lactation) and their association with sow longevity (within 35 days post-farrowing or before subsequent farrowing) and to validate the decisions to remove/retain a sow based on the performance of females retained with or without health problems during the periparturient period in the subsequent parity.

2.2 Materials and methods

Data pertaining to 2066 sows with data on LFI collected for a subset of 1357 sows from a commercial swine breeding herd in the US Midwest were used in this study. The sows belonged to commercial lines of Large White Yorkshire breed. Information on health problems (such as lameness, off-feed, vulvar discharge, prolapses, fever, diarrhea and respiratory problems), farrowing interventions (farrowing induction and farrowing assistance), feed intake and lactation length were collected from the sow cards while the sows were in the farrowing crate (prior to farrowing and during lactation). Lame animals were identified by the herd management based on sows' ability to bear weight on one or more limbs while moving them to the farrowing rooms and also while the sows were in the farrowing crates. Sows were hand-fed daily using a standardized scoop (1.36 kg capacity) while they were in the farrowing crate. Feed consumed was assumed to be equal to that fed if the feeder was empty. If any feed remained in the feeder from the previous delivery, the quantity of feed offered was reduced accordingly though the amount of feed remaining was not measured. If no feed remained from the previous delivery, the sow was offered one extra scoop of feed the following day. In some cases little feed wastage was noticed, however the quantity wasted was not measured. The average LFI for each sow was calculated by dividing the total quantity of feed consumed from the first day of lactation until weaning by the number of lactation days for that sow. Other information such as the parity of the sow, pre-weaning mortality, piglets born alive, mummies, stillborn, wean-to-service interval, status of the sow 35 days post-farrowing

(removed or retained), removal categories (cull, death or euthanasia) and removal reasons if removed and information on the farrowing performance of the retained sows in the subsequent parity were collected from the PigCHAMP database (PigCHAMP, Ames, Iowa) for the herd. The associations of the longevity of these sows 35 days postfarrowing or before the next parity with the data collected during the periparturient period were analyzed.

The production performances of the sows that were retained with and without health problems during the periparturient period in their subsequent parity were compared to validate removal decisions. However, not all sows from both groups could be included for the comparison owing to changes in the health status of these sows during the period subsequent to their first periparturient period. Development of any health problem other than the one originally reported in the periparturient period led to the exclusion of a sow from the group that was retained with health problems. When the new condition reported was a continuation or a complication of the condition reported during the periparturient period, such sows were not excluded from comparison. Similarly, those sows retained without any reported health problem were not included for comparison if they developed a condition before the subsequent parity. The Institutional Animal Care and Use Committee of the University of Minnesota approved this study.

Statistical analysis. All analyses were performed using SAS (SAS Institute Inc. 2003 v 9.1). A summary statistic (mean \pm SE or frequency) of the data collected was analyzed.

Univariate analyses were performed to identify the independent risk factors associated with sow longevity (Proc logistic) within 35 days post-farrowing or before next parity. For analysis, parity was categorized as parities 1 and 2, 3 to 5 and > 5. Health problems including lameness, off-feed, vulvar discharge, prolapses, fever, diarrhea and respiratory problems during lactation and immediately prior to farrowing were categorized as health problems reported or not. Lameness was categorized as lame or non-lame. Factors such as farrowing induction and farrowing assistance were categorized as induced or not, and as assisted or not, respectively. Mummies, stillborn and pre-weaning piglet mortality were categorized as present or absent. Lactation length, average LFI and piglets born alive were included in the model as continuous variables. The percentage distributions of sows removed within 35 days post-farrowing or before next parity were analyzed (Proc freq). Similarly, the percentage distributions of sows removed within 35 days postfarrowing or before next parity with reported incidences of lameness or health problems during the periparturient period were also analyzed (Proc freq). The frequency distributions of reported primary and secondary reasons for sow removals under different categories of removal (cull, death or euthanasia) within 35 days post-farrowing or before next parity were analyzed (Proc freq).

Multivariate logistic regression models (Proc logistic) were fitted to analyze the association of periparturient risk factors with sow longevity. Risk factors found significant ($P \le 0.05$) in the univariate analyses were only included in the multivariate models. The production performance of the sows that were retained with and without

health problems during the periparturient period in their subsequent parity was compared using t-test and 2-sample proportion test to validate the removal decisions. A P value of \leq 0.05 was considered significant in all analyses.

2.3 Results

A description of the data collected is presented in Table 2.1. Of the 217 sows (out of 2066) removed within 35 days post-farrowing, 178 sows were culled (voluntary removal), 17 sows were euthanized (involuntary removal for severe compromise in welfare) and 22 died. Among the sows removed within 35 d of farrowing, the majority were culled between 21-30 days post-farrowing, whereas most of the deaths and euthanasia were reported within the first week after farrowing (Figure 2.1). Among the 503 sows removed before attaining next parity (including the 217 removed within 35 days post-farrowing) 399 sows were culled, 70 died and 34 sows were euthanized. Figure 2.2 shows the percentage distribution of sow removals before next parity. In addition to an increase in removals during lactation there was an increase in culling during 70-80 d after farrowing. Similarly, there was an increase in sow deaths during 130-140 days post-farrowing.

Lameness was the primary reason for the removal of sows within 35 days post-farrowing. Among the sows culled within 35 days post-farrowing, 93 had lameness as the primary reason for removal and 28 had lameness as the secondary reason. Five hundred and three sows were removed before next parity, including those removed within 35 days of farrowing. Reproductive problems were the major reasons for sow removals before next parity.

Figure 2.3 and Figure 2.4 show the percentage distributions of removed sows (within 35 days post-farrowing or before next parity) with lameness or other health problems during the periparturient period. Among the sows removed within 35 days post-farrowing 35% had lameness during the periparturient period, whereas 23% sows removed before next parity had lameness during the periparturient period. The comparison of the subsequent performance of sows retained with and without health problems (lameness and other health problems) during the periparturient period is presented in Table 2.2. The results indicated that the number of piglets born alive was higher (P < 0.05) among the sows without any health problem during the previous periparturient period. The number of sows farrowed was also higher (P < 0.05) in the group without any health problems. A higher (P < 0.05) number of sows were culled from the group with health problems during the periparturient period than those without health problems. However, there was no difference between the groups in terms of wean-to-service interval, farrow-to-farrow interval, pre-weaning piglet mortality, lactation length, number of sows with mummies or stillborn and in the number of sow deaths.

A subset of the data collected with the information of LFI was used to analyze the association of LFI and sow level factors with sow longevity. Factors such as farrowing

interventions (farrowing induction and farrowing assistance) and presence of stillborn, mummies and pre-weaning piglet mortality and number of piglets weaned were not associated with sow longevity within 35 days post-farrowing based on the univariate analyses (P>0.05). However, in univariate analyses, both health problems during the periparturient period (which included lameness and other indications of compromised health) and lameness were found to be associated with sow longevity 35 days post-farrowing. Therefore, the associations between the identified factors and longevity of these sows within 35 days post-farrowing were analyzed using 2 separate logistic regression models, one with lameness and the other with health problems (including lameness) as one of the explanatory variables (Table 2.3). Unlike the models presented in Table 2.3, lactation length and the periparturient health problems were not associated with sow longevity before attaining subsequent parity in univariate analyses and therefore were not included in the multivariate model presented in Table 2.4.

The results (Table 2.3) indicated that the likelihood of removal from the herd within 35 days post-farrowing were associated with the number of piglets born alive, average LFI, health problems during the periparturient period and parity (P < 0.05 for all). The likelihood of removal from the herd decreased by approximately 18% with every additional piglet born alive. Similarly, the risk of removal from the herd decreased by 32 % with every additional kg increase in average LFI. Sows that did not have health problems during the periparturient period had a lower likelihood of removal from the herd to sows that had health problems. Sows of parity 1 and 2, and 3 to 5 were

approximately 82 % and 69% respectively, less likely to be removed from the herd compared to sows of parity >5. As in the model with health problems as an explanatory variable, the directions of associations and levels of significance were similar in the model with lameness during the periparturient period as one of the explanatory variable.

The risk of removal from the herd before next parity decreased by 8% with every additional piglet born alive (Table 2.4). Non-lame sows were 37% less (P < 0.05) likely to be removed from the herd before the next farrowing compared to lame sows. Sows of parity 1 and 2 had 45 % and sows of parity 3 to 5 had 44% lower (P < 0.05 for both) likelihood of removal from the herd before the next parity compared to sows of parity > 5. The likelihood of removal from the herd before the next parity tended to decrease with an increase in average LFI (P = 0.08).

2.4 Discussion

The main justification for retaining sows with considerable health problems during the periparturient period is to minimize productivity losses. Sows with poor performance and health problems are more likely to be culled after weaning as evident from a higher percentage of sow culling around 20 to 30 days post-farrowing in this study. Another peak in sow removals between 70 to 80 days post-farrowing may be associated with sow removals following pregnancy diagnosis after breeding or for other reproductive inefficiencies. Abortions, prolapses and problems such as 'not in pigs' (a female pig

expected to be pregnant and later identified as not pregnant after the expected farrow date) may have contributed to the increase in sow culling around 130-140 days. The higher percentage of sow deaths immediately after farrowing confirms that the periparturient period is a high risk period for sow mortality as reported in previous studies (Chagnon et al., 1991; Deen and Xue, 1999). It has been reported that 45-65% of mortality occurs before the due date for farrowing or within 21 d after farrowing (Deen, 2003b). The peripartum period and up to 3 weeks postpartum is the most sensitive time for sows with over 50% of sow deaths occurring in this period (Anil et al., 2005).

Locomotor problems have been reported to be a major reason for culling (Jorgensen, 2000). In the present study also lameness was the single most important reason for sow removals. A higher proportion of removed sows were reported to have lameness and other health problems during the periparturient period. A lame sow in the farrowing crate may be at a disadvantage. Lameness is a known painful condition and pain may reduce feed intake. Johnson (1997) has reported that cytokines (interleukin-1, interleukin-6 and tumor necrosis factor-alpha) released by the inflammatory process can induce anorexia and lethargy. Bach et al., (2007) have also suggested a negative relationship between dry matter intake and locomotion scores in dairy cattle. Inadequate LFI has been reported to undermine subsequent reproductive performance of the sow (Kirkwood et al., 1987; Baidoo et al., 1992) leading to a removal from the herd.

Retaining sows despite lameness and other health problems during the periparturient period may adversely affect the overall herd performance in the long term as evident in this study. The number of piglets born alive is an important performance variable in swine breeding herds as it influences the output from the unit. Adding to this is the effect of a lesser proportion of sows farrowed and a lower longevity among those sows retained with health problems during the periparturient period. This suggests that sow removal decisions should be reexamined after farrowing.

The results indicate the adverse effects of lameness and other health problems during the periparturient period on the longevity of sows within 35 days post-farrowing or before next parity. Lucia et al., (1996) reported that the lifetime number of piglets born alive was lower in females culled for reproductive problems, indicating the importance of number of live born piglets for higher sow longevity. This report is in agreement with the association between the number of piglets born alive and sow longevity observed in the study described here. Inadequate feed intake during lactation may adversely affect the subsequent reproductive performance of sows. Restricted LFI prolongs the wean-to-estrus interval (Baidoo et al., 1992) and is associated with a lower pregnancy rate and embryo survival (Kirkwood et al., 1987). As reproductive inefficiency is the most important reason for sow removals in breeding herds (Stalder et al., 2004), effects of low LFI may reduce longevity of sows as observed in this study. Hughes and Varley (2003) confirmed an adverse effect of inadequate nutrition on reproduction and longevity of females in breeding herds. Anil et al., (2006) also reported that the likelihood of removal

from the herd decreases by 30% with every additional kg increase in LFI. Lameness or health problems developed during the periparturient period can influence subsequent performance of the sows. Further, if severe, these conditions can lead to immediate removal from the herd. Lameness is the single most important reason for premature removal of sows from breeding herds (Anil et al., 2005). The finding of higher risk of removal of sows with diseases or lameness within 35 days post-farrowing is in agreement with this. Deen and Xue (1999) and Tiranti et al., (2003) reported increasing risk of mortality with increasing parity. Gilts had the lowest mortality risk and as parity increased, annual mortality risk also increased (Koketsu, 2000). It has been indicated that the risk of removal from the herd before another farrowing was 3 % and 24% lower (respectively), for sows of parity 1 and 2, and 3 to 5 compared to sows of parity >5 (Anil et al., 2006). It is likely that the risk of removal increases as the sow ages especially if the sow develops reproductive or health problems.

The present study indicated that periparturient factors such as LFI, incidences of lameness or health problems as well as sow level factors such as higher parity and lesser number of piglets born alive predict the removal of a sow from the breeding herd within 35 days post-farrowing or before another farrowing. Lameness appeared to be a major determinant in premature removal of sows. The results indicate the need for measures to ensure adequate feed intake during lactation and to minimize incidence of lameness to improve sow longevity. This study indicated that sows retained with periparturient health problems had lower longevity. In addition, fewer numbers of such sows had another

farrowing. Sows with the periparturient health problems also yielded fewer live born piglets if retained. The results suggest that retaining sows with health problems during the periparturient period may lower immediate production losses; however it may adversely affect herd performance in the long run. This observational study could identify some of the risk factors of sow longevity in a commercial farm setting. However, the results are limited to the extent that the recording of health related risk factors such as lameness and other disease symptoms were based exclusively on farm records that were not necessarily based on veterinary diagnosis or other standardized procedures. Further, the study included data from a single, large commercial herd and therefore the generalization of the results may be restricted owing to the wide variations in management, housing and in genetic lines of sows. **Table 2.1.** Descriptive summary of data (number of sows/mean \pm SE) on 2066 commercial sows and a subset of data on 1357

sows with lactation feed intake

Variables	Number of	Number of sows/ mean \pm SE
	Full data	Subset of the data
	(N=2066)	(N=1357) with the
		information of lactation
		feed intake
Number of sows with reported cases of lameness	298	176
Number of sows with reported cases of health problems other than lameness	649	465
Number of sows removed <35 days post-farrowing	217	77
Number of sows removed before next parity	503	269
Number of sows with assisted farrowing	1809	1195
Number of sows with induced farrowing	1468	991
Number of sows with stillborn piglets	908	580
Number of sows with mummies	698	477
Number of sows with reported pre-weaning piglet mortality	1043	711
Number of sows with wean-to-service interval < 5 days	1289	938
Number of sows not included in analysis ‡	156	56
Average parity (mean ± SE)	4.27 ± 0.05	4.12 ± 0.06
Average lactation length (mean \pm SE)	19.52 ± 0.09	20.67 ± 0.06
Average parity of sows removed within 35 days post-farrowing (mean \pm SE)	5.50 ± 0.17	4.12 ± 0.06
Average parity of sows removed before next parity (mean \pm SE)	4.82 ± 0.11	6.13 ± 0.30
Average number of piglets born alive (mean \pm SE)	10.48 ± 0.06	10.52 ± 0.07
Average number of piglets weaned (mean \pm SE)	8.54 ± 0.06	9.067 ± 0.041
Average daily lactation feed intake (kg) (mean \pm SE)		8.06 ± 0.01
‡ Information could not be obtained as these sows did not farrow until the completion of data collection period or were reported to be not in pig/or aborted.	pletion of data co	illection period or were

24

Table 2.2 . Performance variables (mean \pm SE/number) of commercial sows retained with
(N=844) and without (N=936) health problems during the periparturient period in the
subsequent parity

Variables	Sows without health problems (n=936)	Sows with health problems (n=844)	P value
	(mean ± SE/ Number)	(mean ± SE/ Number)	
Average number of piglets born alive	10.35 ± 0.13	9.79 ± 0.16	0.006 ^z
Average wean-to-service interval (days)	6.85 ± 0.40	7.66 ± 0.52	NS
Average farrow-to-farrow interval (days)	140.93 ± 0.38	140.72 ± 0.47	NS
Number of sows with pre-weaning piglet mortality (at least 1 piglet death)	417	343	NS
Average lactation length	19.10 ± 0.25	18.77 ± 0.29	NS
Number of sows with mummies	187	168	NS
Number of sows with stillborn	382	325	NS
Number of sows farrowed	663	555	0.021 ^y
Number of sows culled	163	200	0.001 ^y
Number of sows died (death and euthanasia)	44	42	NS

^z t-test; ^y 2-sample proportion test; NS- not significant.

Table 2.3. Odd	s ratios a	nd confidence	intervals	of risk	factors	associated	with	SOW
longevity within	35 d post-	farrowing amo	ong comme	ercial so	ws (N=1	357)		

Risk factors	Model with all health problems		Model with lameness	
	Odds ratio Confidence		Odds	Confidence
		interval	ratio	interval
Piglets born alive	0.817 ^z	0.750 - 0.890	0.813^{z}	0.745 - 0.887
Lactation length	0.935 ^x	0.846 - 1.034	0.957 ^x	0.866 - 1.058
Average lactation	0.679 ^y	0.470 - 0.981	0.656 ^y	0.454 - 0.947
feed intake				
No health problems	0.369 ^z	0.221 - 0.615	NI	NI
vs. health problems				
Non lame vs. lame	NI	NI	0.260^{z}	0.147 - 0.461
Parity 1 &2 vs. >5	0.175 ^z	0.079 - 0.384	0.181 ^z	0.082 - 0.397
Parity 3 to 5 vs. >5	0.305 ^z	0.176 - 0.529	0.285^{z}	0.163 - 0.498

z < 0.001; y < 0.05; x - not significant; NI= not included in the model

Table 2.4. Odds ratios and confidence intervals of risk factors associated with sow removals before the next parity among commercial sows (N=1357)

Risk factor	Odds ratio	Confidence interval
Piglets born alive	0.916 ^y	0.869 - 0.965
Average lactation feed intake	0.827 ^w	0.670 - 1.022
Non lame vs. lame	0.626 ^x	0.430 - 0.912
Parity 1 and 2 vs. >5	0.548 ^y	0.377 - 0.795
Parity 3 to 5 vs. >5	0.558 ^z	0.407 - 0.765

z < 0.001; y < 0.01; x < 0.05; mot significant (P=0.08).

Figure 2.1. Percentage of commercial sows culled and dead (including euthanized) within 35 d post- farrowing (178 sows culled and 39 sows dead).

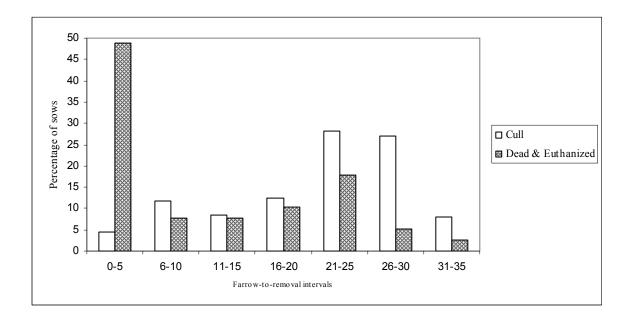


Figure 2.2. Percentage of commercial sows culled and dead (including euthanized) before the next parity (399 sows culled and 104 sows dead).

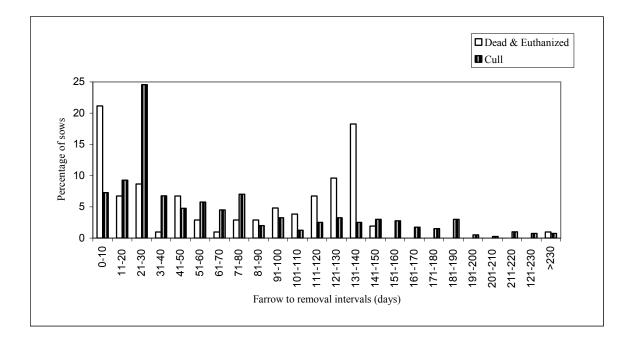


Figure 2.3. Percentage of commercial sows removed (within 35 d post-farrowing) with lameness (N=77) or health problems (N=140) during the periparturient period.

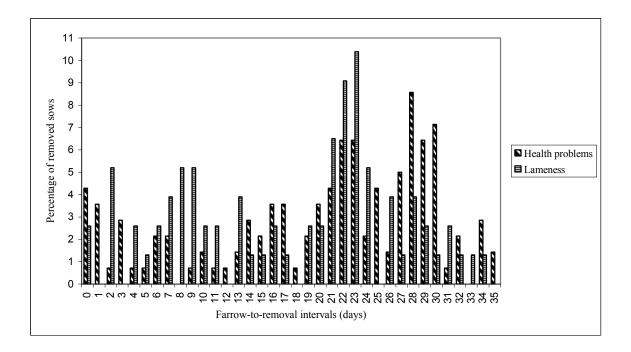
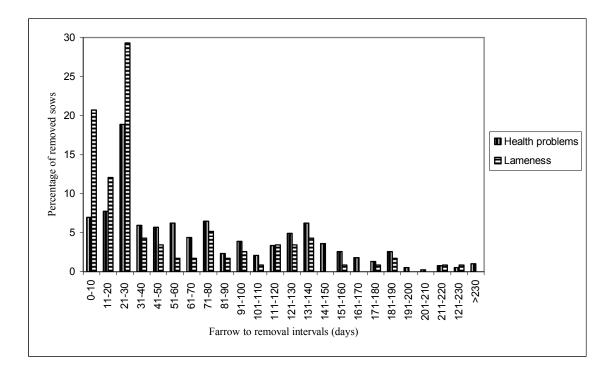


Figure 2.4. Percentage of commercial sows removed (before the next parity) with lameness (N=116) or health problems (N=387) during the periparturient period.



CHAPTER III

EFFECTS OF LAMENESS ON SOW LONGEVITY

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Association of risk factors (lameness, parity and farrowing performance) with sow longevity among 674 sows in a commercial herd was analyzed using survival analysis over three parities (Proc phreg, SAS v 9.1). The survival times were different among lame and non-lame sows and among parity categories (P <0.05 for both). Pre-weaning piglet mortality, stillborn piglets and mummies were negatively associated with sow longevity within 350 days of lameness assessment (P <0.05 for all). A higher number of piglets born alive appeared to be protective (P <0.05). Sows of parity 3-5 and >5 had approximately 1.58 and 1.35 times higher risk of removal from the herd respectively in comparison to sows of parity 1 and 2, keeping other variables constant (P<0.05 for both). Lame sows had 1.71 times higher risk of removal from the herd within 350 days of lameness assessment (P <0.05). The number of piglets born alive per day, survival at 350 days as well as the length of stay in the herd were lower in lame sows (P <0.05 for all).

3.1 Introduction

Lameness in sows is a common cause for compromised animal well-being and economic loss to producers. Lame animals are likely to be unable to attain optimal breeding efficiency (Ritter et al., 1999) and may be culled before they attain their peak production. Locomotor problems are a major reason for culling (Stalder et al., 2004) in swine herds, with a reported culling rate of 15.2% in US swine herds (USDA, 2006). Sows culled because of lameness are removed at a younger age than those removed for other reasons (Dagorn and Aumaitre, 1979; D'Allaire et al., 1997; Patterson et al., 1997). Early removal of sows from the herd results in lower mean litter size, number of litters per sow per year, and number of pigs weaned per sow per year, thus increasing the cost per weaned pig (D'Allaire et al., 1997). The addition of new gilts into the system may also increase health risks to the animals currently in the herd (Sanz et al., 2002). The author of a study (Grandjot, 2007) conducted in Germany reported fewer litters (< 3.0 litters for lame sows vs. 4.5 litters for non-lame sows) and higher baby pig losses (27.7% for lame sows vs 12.4% for non-lame sows) in lame sows than in healthy sows. In that study, the financial loss associated with a lame sow to account for fewer pigs born, baby pig mortality rate, and early slaughter of sows was estimated to be 37 euros/sow (\$52/ sow).

Furthermore, lame sows receive extra scrutiny when shipped to market, which decreases the salvage value. Lack of analgesics and the high labor requirement associated with providing medications for individual animals in large herds add to the fact that prevention of lameness is a better option than treatment. Despite the high prevalence of lameness in swine herds, potential measures to minimize lameness and its adverse effects have been less thoroughly explored, compared with evaluations conducted for other diseases of swine. Although lameness is extremely prevalent in breeding herds, diagnosis can be unreliable because of the lack of a criterion-referenced standard. A study (Anil et al., 2008) on the sensitivity and specificity of lameness identification in sows as determined on the basis of gait abnormalities indicated that it was relatively easy to distinguish a severely lame sow from a healthy sow, but it was difficult to correctly distinguish a mildly lame sow from a healthy sow. However, early detection is critical in preventing the condition from deteriorating. Additionally, lack of observation of individual pigs for gait abnormalities is another limitation in lameness assessment in large swine herds. However, the movement of sows to farrowing facilities offers an opportunity to observe the gait of sows on most farms. Given that a severely lame animal will be culled or euthanatized if necessary, sows with less severe, chronic lameness can continue to persist in herds. A better understanding of the effect of risk factors, including lameness, on sow productivity and longevity would augment efforts to minimize lameness in swine herds. The purpose of the study reported here was to analyze the association of lameness and performance variables with sow longevity by use of time-to-event analysis.

3.2 Materials and methods

Large White–Yorkshire crossbred sows (n = 674) that were part of a large (> 5,000 sows) commercial swine breeding herd in the Midwestern United States were used in the study. The 674 sows were selected on the basis of availability of records for longevity and farrowing performance. Each sow farrowed at least once during the study period. The Institutional Animal Care and Use Committee of the University of Minnesota approved this study. Sows were entered into the study at the time of farrowing (regardless of parity) during the study period. Information on lameness was recorded once for each sow at the time of the first farrowing during the study period.

Lameness assessment was performed prior to farrowing by caretakers, who evaluated the sows while moving them to the farrowing rooms and also while the sows were housed in farrowing crates. Lame sows were identified on the basis of the ability or willingness to bear weight equally on all limbs, and the information was manually recorded on a card located by the farrowing crate of each sow. Caretakers had not received training in lameness evaluation (other than their own experiences); the authors used farm records for data on lameness and did not control the procedures of lameness evaluation. Follow-up examinations were not performed by veterinarians to confirm the lameness evaluation conducted by the caretakers.

Each sow remained in the study for up to 3 parities after the lameness assessment. Data were collected on longevity and farrowing performance for up to 350 days after the first farrowing following lameness assessment. Other information, such as parity of the sow at the time of lameness assessment, farrowing performance (numbers of pigs born alive, mummified pigs, stillborn pigs, and baby pigs that died before weaning), and longevity (removal from the herd in the form of culling, death, or euthanasia and the date of removal from the herd), was collected from the PigCHAMP database for the herd (PigCHAMP Inc, Ames, Iowa).

Statistical analysis. All analyses were performed by use of SAS (SAS Institute Inc, Cary, NC, v 9.1). For analysis, sows were categorized as lame or non-lame sows, and parity of a sow at the time of lameness assessment was categorized as 1 and 2, 3 through 5, and > 5. Total numbers of pigs born alive, mummified pigs, stillborn pigs, and baby pigs that died before weaning (in 1 or more farrowings) during the period of \leq 350 days were calculated and included in analyses as continuous variables.

Univariate analyses were performed for categorical (Kaplan-Meier curves and a log-rank test of equality, Proc lifetest) and continuous (Cox proportional hazard regression, Proc phreg) variables to identify associations with sow longevity during the period \leq 350 days after lameness assessment. Kaplan-Meier curves were used (along with the log-rank test) to verify whether survival functions were approximately parallel among strata.

For the Cox regression model, it was assumed that the hazard ratio assessed remained constant during the study period. This assumption was assessed by use of the Wald test by adding interaction terms (i.e., total production days and covariates) to the model. This assumption was not violated because the hazard ratio of removal was constant for the duration of the monitoring period (P = 0.125; Wald χ 2 test). The likelihood ratio test was used to indicate whether a factor significantly improved the fit of the model, and significance of coefficients in the models was based on results of the Wald χ 2 test. Tests of all the time-dependent variables (individually or collectively) did not yield significant results; thus, there was no evidence to reject the proportionality assumption.

A Cox regression analysis was performed by use of the variables significantly ($P \le 0.1$) associated with sow longevity. Total production period (≤ 350 days after lameness assessment) was the time variable. Sow longevity was the censoring variable, and a sow was considered censored when it was not removed from the herd during or after the study period (longevity = 0 indicated that a sow was censored, and longevity = 1 indicated that a sow was removed within 350 days after the initial lameness assessment).

Also, the number of pigs per day (number of pigs born alive in the second and third farrowing after lameness assessment divided by total production days) and total production days were compared between lame and non-lame sows by use of a t-test. Number of lame and non-lame sows surviving at 350 days after lameness assessment was compared between lame and non-lame sows by use of a 2-sample proportion test.

The cumulative proportions of pigs born alive per day per sow farrowed among lame and non-lame sows during the production period were analyzed. For all analyses, a value of P ≤ 0.05 was considered significant.

3.3 Results

Univariate analyses indicated significant associations between risk factors (lameness, parity, and total numbers of pigs born alive, mummified pigs, stillborn pigs, and baby pigs that died before weaning) and sow longevity during the study. The reduction in survival function was proportional between lame and non-lame sows, and the Kaplan-Meier survivor curves were approximately parallel (Figure 3.1). Median survival time for lame sows was 140 days after first farrowing following the lameness assessment, whereas median survival time for non-lame sows was 302 days. Similarly, the reduction in survival functions was proportional among parity categories because the Kaplan-Meier survivor curves were approximately parallel (Figure 3.2). Median survival times for sows of parity 1 and 2, 3 through 5, and > 5 were 314, 302, and 167 days after first farrowing following lameness assessment, respectively. The test of equality indicated that the survival time differed significantly between lame and non-lame sows and among parity categories.

Results of survival analysis for the complete model were summarized (Table 3.1). There was a significant association between risk factors and sow longevity within 350 days

after lameness assessment. Lame sows had a higher risk (1.710 times as high) of removal from the herd within 350 days after lameness assessment, compared with the risk of removal for non-lame sows. As the total number of baby pigs that died before weaning increased by 1, the risk of removal from the herd decreased by approximately 11%. For each additional piglet born alive during the production period, risk of removal from the herd decreased by approximately 16%. The risk of removal from the herd decreased by approximately 12% for each additional stillborn pig. The risk of removal decreased by 13% for each additional mummified pig. Sows of parity 3 through 5 and > 5 had a higher risk (approximately 1.579 and 1.349 times as high, respectively) of removal from the herd, compared with the risk of removal for sows of parity 1 and 2. The cumulative sum of the number of pigs born alive per sow farrowed was less for lame sows than for nonlame sows; however, this comparison was not analyzed statistically. Mean \pm SE number of pigs born alive per day was significantly (P < 0.001) less for lame sows (0.028 \pm 0.003) than for non-lame sows (0.049 \pm 0.002). Survival at 350 days was significantly (P < 0.001) lower in lame sows (33/140 = 23.6%) than in non-lame sows (238/534=44.6%). Similarly, mean total number of days in the herd was significantly (P < 0.001) lower in lame sows (148.30 ± 10.67) than in non-lame sows (215.73 ± 4.45) .

3.4 Discussion

Sow removal from a herd may not depend on biological factors of the sow alone. Other factors, such as the number of gilts in the breeding herd and the market for culled sows,

may also influence culling of sows in breeding herds. Nonetheless, reproductive performance is a major determinant of sow longevity because it is essential for sows to remain productive to remain in the herd. A positive relationship between productivity and sow longevity has been suggested (Dourmad et al., 1994; Xue et al., 1997) because sow longevity is associated with the number of pigs produced per sow per year. The most commonly reported reason for unplanned sow removals is reproductive failure, which accounts for approximately one-third of all removals (Engblom et al., 2008). Reproductive performance is represented by farrowing performance (numbers of pigs born alive, stillborn pigs, mummified pigs, and baby pigs that died before weaning) and breeding performance (weaning-to-estrus interval and number of breedings per conception).

The number of pigs born alive is an important performance variable in swine breeding herds because it influences the output of the herd. A reduction in the risk of removal for sows with higher numbers of liveborn pigs in the study reported here was similar to a negative relationship reported between these 2 variables in other studies (Lucia et al., 1996; Anil et al., 2008). Anil et al., (2008) reported that the risk of removal from a herd before the next parity decreased by 8% with every additional pig born alive. The lifetime number of pigs born alive was reported (Lucia et al., 1996) to be lower in females culled for reproductive problems, which indicates the importance of number of liveborn pigs for increased sow longevity. The information in that report is also in agreement with the association between the number of pigs born alive of pigs born alive of pigs born alive in the information in that report is also in agreement with the

present study. Studies (Engblom et al., 2008; Yazdi et al., 2000; Serenius et al., 2004; Tarrés et al., 2006) conducted to evaluate the effect of litter size on longevity have also revealed a positive association between litter size and longevity in commercial herds. Obviously, the number of pigs born alive is the most influential component of litter size. The number of liveborn pigs reportedly has a genetic correlation with the need for assistance during parturition and duration of farrowing because the number of stillborn pigs was highly correlated with the need for assistance during parturition and the duration of farrowing (Holm et al., 2004). Litters of ≥ 12 pigs were 2 times as likely to contain a stillborn pig, compared with the likelihood of a stillborn pig in smaller litters (Lucia et al., 2002). It has been reported (Glastonbury, 1976) that litter size at birth has a significant effect on the frequency of stillbirths and number of baby pigs that die before weaning. In the study reported here, the finding of a higher survivability of sows with a higher number of baby pigs that died before weaning, higher number of stillborn pigs, and higher number of mummified pigs is linked to the association of these production variables with a larger litter size.

Lameness and associated pain will adversely influence performance of lame sows that are retained in herds. It has been suggested (Penny, 1980) that lameness can be associated with a reduction in fertility. A reduction in conception rate and an increase in the median number of days not pregnant have been reported in lame cattle (Lee et al., 1989). Lameness may also affect the ability of a sow to make postural changes within a farrowing crate and may cause death of baby pigs as a result of crushing. Another

analysis of a larger database (of which the data reported here are a subset) indicated that sows retained with periparturient health problems (including lameness) had reduced longevity and fewer liveborn pigs, and fewer numbers of such sows farrowed again (Anil et al., 2008).

Locomotor problems are a major reason for culling (Stone, 1981; Friendship et al., 1986; Jorgensen, 2000). Lameness appears to influence sow longevity directly and indirectly. Acute, severe lameness can result in immediate removal of sows from herds. However, a chronic, less severe form of lameness can affect the performance of sows and indirectly lead to sow removals. Lameness is a painful condition, and pain can influence feed intake (Oldham, 1985). Cytokines released during the inflammatory process can induce anorexia and lethargy (Johnson, 1997). A negative relationship between dry-matter intake and locomotion scores in dairy cattle has been reported (Bach et al., 2007). If a lame sow is lactating, inadequate feed intake during lactation can adversely affect the subsequent reproductive performance of that sow and eventually cause her removal from the herd. Inadequate feed intake during lactation can undermine subsequent reproductive performance of sows (Baidoo et al., 1992; Kirkwood et al., 1987) such as an increase in the weaning-to-estrus interval, which can lead to removal from the herd. Sows consuming \leq 3.5 kg (7.7 lb) of feed/d during the first 2 weeks of lactation are more likely to be removed from the herd before their next parturition (Anil et al., 2006).

The adverse effect of lameness on the lifetime output of pigs was also evident in the study reported here, although causality could not be established. Survival curves illustrated that the largest differences were at approximately 20 to 25 days after the farrowing following the lameness assessment, which suggested that lame sows were removed after lactation. Therefore, the reduction in the number of pigs born alive per day among lame sows could partially be attributable to fewer sows remaining in that group for subsequent farrowings. However, the median survival time in the study for lame and non-lame sows was 140 and 302 days after first farrowing, respectively. Therefore, it is clear that many lame sows had a chance to farrow a second time or, at least, were still in the herd for up to 140 days.

The higher risk of removal for lame sows detected in this study agreed with the aforementioned reports on the effects of lameness on sow longevity. The time at which lameness was diagnosed could have influenced removal decisions in this study. Even if identified as lame, a sow in advanced gestation is less likely to be removed prior to farrowing. Also, manifestations of lameness are likely to be less pronounced at the time of weaning because of the reduction in body weight during lactation, compared with effects evident during the pre-farrowing period. Thus, a sow may be bred again. However, repeated incidences of lameness may result in removal from the herd as a direct effect of the severity of lameness or because of the indirect effect of lameness on reproductive performance. Although a causal link cannot be suggested, analysis of the results reported here indicated a negative effect of lameness on the number of pigs born

alive over the long term. This finding contradicted the observation in another study (Kroneman, 1993b) that lameness in sows during the last month of gestation was not associated with the numbers of pigs born alive, number of stillborn pigs, or birth weights of the pigs, although the sows were only monitored until the next mating in that study.

Each farrowing is a high-risk event with regard to sow removal. In addition, farrowing can be an important risk factor for death of sows (Deen and Xue, 1999; Deen, 2003b). In another study Karg and Bilkei, (2002), 40.2% of the sow deaths were during lactation. The peripartum period is a risky period, with 42% of all deaths occurring during this short time frame (Chagnon et al., 1991). Other studies (Deen and Xue, 1999; Koketsu, 2000; Tiranti et al., 2003; Engblom et al., 2008) have also revealed an increasing risk of death of sows with increasing parity. The risk of culling increases for older parities (Tarrés et al., 2006) and with decreasing litter size at weaning. The higher risk of removal for older-parity sows in the study reported here agreed with results in the aforementioned reports with regard to effects of parity on sow longevity.

Several causal factors have been reported for lameness, with a common one being the flooring of the housing system (Jensen, 1979; Svendsen et al., 1992; Heinonen et al., 2006). Injuries to the forelimbs and hind limbs, including lesions to the claws, are a major cause of lameness (Anil et al., 2005), and the risks of these injuries are higher in group-housed sows (especially if there is frequent regrouping of sows) because of aggressive interactions and increased mobility (Anil et al., 2005). The importance of this risk factor

is likely to increase with the industry trend toward use of group pens for housing of gestating sows.

In the study reported here, time-to-event data were analyzed by use of survival analysis and Cox regression methods. Results indicated significant differences between survivability of lame and non-lame sows in a commercial herd. Other factors influencing survivability of sows in this commercial herd were parity and farrowing performance (i.e., numbers of pigs born alive, mummified pigs, stillborn pigs, and baby pigs that died before weaning). The overall performance of lame sows in terms of the number of pigs born alive during the period of the study was also less, compared with that for non-lame sows. Therefore, this study indicated the need to minimize the incidence of lameness and to remove lame sows from a herd as early as possible when treatment and recovery have a low likelihood. However, this study was based on farm records, and the diagnosis of lameness was not confirmed by a veterinarian. Similarly, there was no training program for lameness diagnosis, and the validity and reliability of the lameness assessment were not verified. However, the differences were large and suggested considerable validity for the scoring methods. **Table 3.1**. Results of survival analysis of the complete model for the association between

 sow longevity and lameness, parity, and farrowing performance.

Risk factor	Hazard ratio and CI	P value
Total pre-weaning mortality	0.893 (0.832-0.958)	0.0017
Lameness (lame vs. non-lame)	1.710 (1.346-2.174)	< 0.0001
Total piglets born alive	0.839 (0.825-0.853)	< 0.0001
Total stillborn	0.879 (0.829-0.932)	< 0.0001
Total mummies	0.866 (0.813-0.922)	< 0.0001
Parity 3-5 vs. Parity 1 and 2	1.579 (1.186-2.102)	< 0.0018
Parity >5 vs. Parity 1 and 2	1.349 (1.011-1.801)	< 0.0420

Figure 3.1. Kaplan-Meier graph of the time to removal from the herd for lame (dotted line) and non-lame (solid line) sows after lameness assessment. Day 0 is the day of farrowing following lameness assessment.

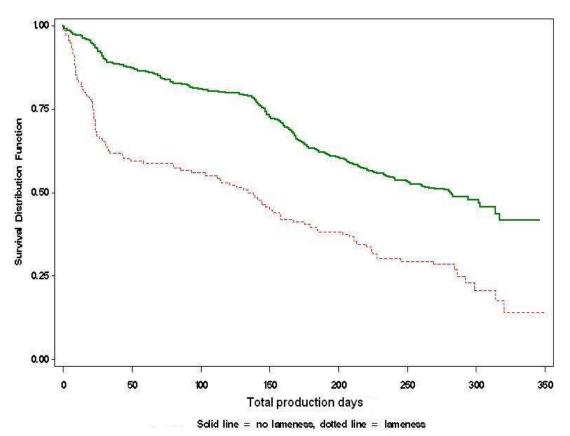
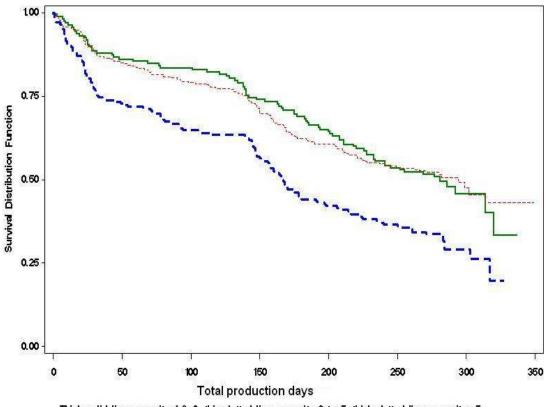


Figure 3.2. Kaplan-Meier graph of the time to removal from the herd for sows of parity 1 and 2 (solid line), 3 through 5 (dotted line), and > 5 (dashed line).



Thick solid line = parity 1 & 2, thin dotted line = parity 3 to 5, thick dotted line = parity>5

CHAPTER IV

FACTORS ASSOCIATED WITH CLAW LESIONS IN GESTATING SOWS

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A study was conducted to characterize claw lesions and to analyze the factors associated with types of claw lesions in sows. Claw lesions were scored for 184 sows in farrowing stalls on day 110 of gestation, and associations with body condition (body weight and back fat thickness on day 109 of gestation), parity, gestation housing system and lameness were analyzed using logistic regression models. At least one lesion was present on at least one claw on the wall and the heel areas of 88.6 and 86.4% of sows, respectively, with more severe lesions on forelimbs and on lateral claws. The likelihood of wall lesions in any claw increased with body weight on day 109 of gestation. For any claw, the likelihood of heel lesions increased and the likelihood of overgrown heels decreased with higher back fat. Non-lame sows were less likely than lame sows to have white-line lesions on any claw. Sows of parity ≤ 5 were more likely to have white-line lesions in any claw than sows of parity >5. Sows housed in pens with electronic sow feeders (ESF) were more likely than stall-housed sows to have all types of lesions in any claw. Under the conditions of this study, lameness and younger parity were associated with white-line lesions. Use of ESFs was associated with more severe lesions. Measures to minimize claw lesions in sows are needed to reduce the incidence of lameness and probably removal rates, especially for younger parities.

4.1 Introduction

Hoof lesions are very common in pigs and are an important underlying cause of lameness (Dewey et al., 1993). Although sows with mild lesions may show no overt signs of pain, severe hoof lesions may cause lameness. In addition, lameness may result when hoof lesions permit entry of infections that spread upwards, affecting joints. Lameness is a welfare concern and a major reason for early removal of sows from breeding herds (Anil et al., 2005), causing producers considerable economic loss. Housing conditions and management practices may be associated with development of hoof lesions in pigs (Kroneman et al., 1993a).

Specific factors associated with hoof lesions in pigs include the interaction between the floor surface and the horn of the claw (Simmins and Brooks, 1988), physical properties of the floor (Jensen, 1979), and nutrition, especially related to dietary biotin levels (Simmins and Brooks, 1988). Studies on hoof lesions in pigs are relatively rare. It is important to characterize and understand the factors associated with claw lesions to minimize the incidence of such lesions and to reduce removal of sows for lameness. The objectives of this study were to characterize claw lesions and to identify factors associated with different types of claw lesions in sows in a breeding herd in Minnesota.

4.2 Materials and methods

The study was conducted at the University of Minnesota, Southern Research and Outreach Center, Waseca, Minnesota. All protocols were approved by the Institutional Animal Care and Use Committee of the University of Minnesota. One hundred and eighty-four sows (Genetically Advanced Pigs, GAP Genetics, Winnipeg, Manitoba, Canada) of parities 0 to 8 and weighing 166 to 337 kg at 109 days of gestation were included in the study. Sows were housed either in pens (12.75 m \times 6.75 m) with one electronic sow feeder (ESF) per pen (TEAM electronic sow feeder; Osborne Industries, Osborne, Kansas) or in stalls (Crystal Spring Hog Equipment Ltd, St Agathe, Manitoba, Canada; length 200 cm, width 60 cm, height 97 cm). Pens and stalls had fully slatted flooring (solid portion 12.7 cm wide and 12.7 cm deep; slots 2.54 cm wide). Among the study sows, 102 had been housed in group pens during gestation throughout their life in the herd, and 82 had been housed in stalls during gestation. Twenty one parity-one sows, 32 parity-two sows, 26 sows of parities 3 to 5, and 23 sows of parities > 5 were housed in pens, and 16 parity-one sows, 27 parity-two sows, 18 parity of parities 3 to 5, and 21 sows of parities > 5 were housed in stalls. Distributions of parities in group-housed and stall-housed sows did not differ (chisquare test; P > 0.05). Until day 109 of gestation, sows were fed 2.2 to 3.0 kg of feed daily (crude protein content 15%) on the basis of body weight and back fat at weaning. On gestation day 109, sows were weighed on a digital Ag Alliance scale (Altoona, Iowa) and back fat was measured at the last rib (5.08 cm from the midline of the back on both left and right sides) with a Lean-Meater ultrasound unit (Renco, Minneapolis, Minnesota). Mean back fat thickness for group-housed sows (19.76 mm; SE, 0.38 mm) and stall-housed sows (18.55 mm; SE, 0.60 mm) did not differ (two-sample t-test; P > 0.05). Sows were then washed and moved into farrowing stalls with cast-iron flooring (214 cm × 66 cm, excluding a creep area for the piglets). Sows in the farrowing stalls were offered 3 kg of feed per day until farrowing, and ad libitum feed during lactation. Sows were weaned at an average of 18.8 days of lactation (SE, 0.14 day).

Claws were individually examined for lesions on day 110 of gestation when sows were in the farrowing stalls. Lesions included erosions, cracks, and overgrowths. The horny wall and the volar (plantar) surface of the hoof were examined. The medial and lateral claws of each foot were examined for lesions on a severity scale (Gjein and Larssen, 1995) of 0 (no lesions noted) to 4 (severe) (Table 4.1). Areas on the claw were classified as wall (composed of hard keratinized epidermis), heel (soft keratinized epidermis on the ventral aspect of the claw towards the posterior end, including overgrown heel), sole (hard keratinized epidermis anterior to the heel on the ventral aspect of the claw), junction between heel and sole, white line (junction between sole and wall), and toe (anterior part of the sole). The final score on each area was obtained by multiplying the number of lesions by the severity of these lesions. The total score for each claw was obtained by summing the scores for different areas of the claw. Total score on each foot was obtained by summing scores for different areas of the two claws. Scores on all areas of all feet were summed to obtain total claw-lesion scores. Sows were made to walk a short distance within the farrowing room and were observed for difficulty in bearing weight on one or more limbs. Sows were categorized as either lame or non-lame. The same individual scored lesions and evaluated lameness in all sows, and was unaware of the gestation housing system at the time of scoring.

Statistical analysis. Median and range were used to describe the data collected on lesions. All analyses were performed using SAS software (SAS, v 9.1; SAS Institute Inc, Cary, NC). The proportions of sows with and without lesions on different areas in the lateral and medial claws of front and hind limbs were compared using one-sample and two-sample proportion tests. Separate logistic regression models (Proc logistic) were fitted (Wald statistic) to analyze the association of lameness, parity, gestation housing system, and body condition (back fat and body weight) at 109 days of gestation with the presence or absence of lesions on different areas of the claw. For analysis, parity of the sows was categorized as P1 (parity 1), P2 (parity 2), P3-5 (parities 3 to 5), and P > 5 (parities > 5). Gestation housing system was categorized as pens with ESF or stalls, and lameness was categorized as lame or non-lame. A P value of ≤ 0.05 was considered significant in all analyses.

4.3 Results

Median and range of lesion scores on different areas of claws are presented in Table 4.2. Only seven of the 184 sows had no lesions on any claw area, and these sows were

all housed in stalls during gestation. More severe lesions were seen on walls and heels. Among the 184 sows examined and considering all claws, 88.6% had at least one wall lesion and 86.4% had at least one heel lesion (Figure 4.1). Other areas where lesions were common were the junction between heel and sole (66.3% of sows affected) and the white line (60.9% of sows affected). Toe lesions were relatively uncommon (0.5%). The proportions of sows with lesions were greater than the proportions without lesions (P < 0.05) in all claw areas except the sole. Lesions were more severe on forelimbs than on hind limbs (Table 4.2), and the proportions of sows with lesions on different areas of the lateral claws were greater (P < 0.05 for all except toe lesions) than the proportions of sows with lesions on different areas of the medial claws (Figure 4.2). The proportions of sows with lesions on the lateral claws were greater (P < 0.05) for both front and hind limbs (Figure 4.3). The proportions of sows with claw lesions were greater (P < 0.05) among sows housed during gestation in group pens with ESF than in those housed in stalls (Table 4.3), with the exception of toe lesions. Among the sows with claw lesions, 60.7% with wall lesions, 60.0% with heel lesions, 63.4% with white line lesions, 67.0% with lesions at heel-sole junction, 57.8% with sole lesions, and 75.4% with overgrown heels were housed in group pens during gestation. The proportion of lesions with severity > 2 was numerically larger in the wall and heel areas than in other areas of the foot, but this proportion was not statistically analyzed.

The odds ratios and confidence intervals indicating the association of lesions on different areas of the claw with body condition, lameness, parity, and gestation housing

system are presented in Table 4.4. Since the overall prevalence of toe lesions was low, they were not included in the regression analysis. The likelihood of wall lesions increased 3% with an increase of 1 kg of body weight on day 109 of gestation. Sows with higher back fat on day 109 of gestation had a 15% greater likelihood of having heel lesions (P < 0.05) and a 12% lesser likelihood of having overgrown heels (P < 0.05) 0.05). Non-lame sows had a 62% lesser likelihood of having white-line lesions (P <0.05) than lame sows. The likelihood of having white-line lesions was 5.5, 4.5, and 2.9 times greater in sows of parities P1, P2, and P3 to P5, respectively, than in sows of parity > 5 (P < 0.05). The likelihood of having all types of claw lesions (overgrown heel and lesions on wall, heel, white line, junction between sole and heel, and sole) was greater in sows housed in pens with ESF (P < 0.05). The likelihood of having wall lesions, heel lesions, overgrown heel, white-line lesions, and lesions at the junction between sole and heel were approximately 10.0, 3.5, 5.0, 2.0, and 5.0 times greater, respectively, in sows housed in pens with ESF during gestation than in sows housed in stalls.

4.4 Discussion

The smaller number of sows without any claw lesions may be linked to the intensive selection and management in today's swine industry that has made pigs grow rapidly to a large body weight. It has been suggested that this rapid growth affects the soundness of legs and feet (Kroneman et al., 1993). The findings of the present study

are thus in agreement with a previous report (Gjein and Larssen, 1995) that more than 96% of loose-housed sows and 80% of confined sows had at least one lesion on the lateral hind claws. The most prevalent lesions reported in that study (Gjein and Larssen, 1995) were hoof-wall cracks, heel lesions, cracks in the white line, and overgrown heels. In the present study, the majority of sows had lesions on the wall, heel, and white line. In another study (Kirk et al., 2005), it was reported that hyperkeratinization of soft heel tissue was observed in 74% to 84% of sows autopsied. Other common claw lesions observed (Kirk et al., 2005) were cracks in the hoof wall (44% to 49%), cracks in the sole (75% to 77%), and cracks in the white line (63% to 65%).

The weight distribution of sows may be an important factor determining development of lesions on different claws and different limbs (Kroneman et al., 1993). Lesions may not develop equally on all claws, as observed in the present study. A previous study (Simmins and Brooks, 1988) also indicated more lesions on fore and hind lateral claws than fore and hind medial claws, as in the present study. Hoof lesions may be more common in lateral claws because they have a greater weight-bearing surface than medial claws (Tubbs, 1988). More than 75% of the weight of the pig is born by the lateral digits, and 80% of injuries affect these digits (Webb, 1984). The strength of different parts of the foot may also vary, and junctions between hard and soft areas 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 the heel bulb and the abaxial hoof wall, whereas the tip of the toe is the greatest weight-bearing region in the inner digit (Webb, 1984). In agreement with these reports, greater proportions of sows in this study had lesions at the heel-sole junction and on lateral rather than medial claws.

The floors in the stalls and pens in this study were fully slatted. The greater freedom of movement among sows housed in pens with ESF, and especially the greater activity associated with aggression at mixing and on entry into the feeder, may have been associated with the greater proportion of claw lesions in these sows. Severe lesions (lesion score > 2) on the wall and heel might also be associated with the slatted floor, i.e., a foot catching between slats is a source of claw lesions. A higher incidence of claw problems in sows housed on partially slatted concrete floors than in sows on straw-bedding or solid concrete floors has already been reported (Holmgren et al., 2000). The space between slats, roughness of the surface, and edge design are crucial factors in deciding the extent of injury (Boon and Wray, 1989). When body weight increases, the pressure exerted per unit area of the claw is greater, increasing the chance of injury. This might explain the greater likelihood of wall lesions with an increase in body weight on gestation day 109 and greater likelihood of heel lesions with an increase in back fat thickness on gestation day 109. However, the negative association between back fat thickness and overgrown heel could not be explained.

Foot lesions have long been recognized as a cause of lameness (Penny et al., 1963). In this study, analysis suggested a link between lameness and white-line lesions. Other claw lesions were not significantly related to lameness. While the wall and sole consist of reinforced tubular horn, the white line is the cemented junction of wall and sole and is an inherent point of weakness (Budras et al., 1996). In cattle, it has been reported that injuries to the weak white line may easily penetrate the corium and facilitate spread of infection, causing lameness (Kempson and Logue, 1993). Therefore, whiteline lesions are more likely to cause lameness than other types of claw lesions. Although the relationship between claw lesions and lameness is obvious, occurrence of claw lesions is only one factor causing lameness in pigs. It has been reported (Logue et al., 1989) that in cows, lameness may occur in the absence of lesions and that observed lesions may not necessarily cause lameness. The greater likelihood of white-line lesions in lower parities in this study may have occurred because older sows with severe white-line lesions might have already been removed from the herd for lameness or old age, excluding them from analysis.

The amount and type of activity of sows may determine the type and severity of claw lesions, and these two factors varied greatly in gestation stalls and group pens with ESF. The ESF allows only one sow at a time to eat, resulting in a highly competitive situation, especially since the sows were on a restricted diet during gestation. Fighting and aggressive activity at mixing and subsequently at feeding time may increase the likelihood of claw lesions in sows housed in group pens with ESF systems (Anil et al., 2005). The greater proportion of sows with claw lesions in pens with ESF compared to that in sows housed in stalls may have been due to the greater mobility of sows in the ESF system. Although a greater proportion of sows with claw lesions in pens is partly attributable to the ESF and associated aggression at feeding time, the extent of this effect has not been differentiated in this study.

Measures to minimize the incidence of claw lesions may have to focus on nutrition and management. Biotin supplementation of barley-based diets may reduce claw lesions (Bryant et al., 1985; Simmins and Brooks, 1988). Claw problems have been reported as more common in sows housed on slatted floors than in sows on solid floors with straw bedding, and also when stocking density is higher (Jorgensen, 2003). Rough concrete, worn or uneven slats, and sharp protruding objects increase the risk of lesions. In slatted floors, the void ratio also influences development of claw lesions (Webb, 1984). Stable group structure in group pens may reduce opportunities for aggression and trauma (Anil et al., 2007). Similarly, minimizing chances of upward infection through claw lesions helps to reduce incidence of lameness.

Table 4.1. Scoring method for claw lesions in a study examining 184 sows at day 110 of gestation

Score	Description of lesions
0	No lesions
1	Small, superficial cracks or lesions in the epidermis
2	Serious lesions in the epidermis
3	Deep lesions extended into the corium
4	Serious and deep cracks extended into corium or subcutis

 Table 4.2. Median and range of lesion scores on different areas of claws across all limbs

 in 184 sows at day 110 of gestation

Area affected	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
Тое	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

The final score for each area = number of lesions \times lesion score. Total score for each claw = sum of scores for different areas of the claw. Total score per foot = sum of scores for both claws. Total claw lesions = sum of scores for all four feet.

	Sows in pens	s (n=102)		n stalls =82)
			No	
Claw area	No Lesions	Lesions	lesions	Lesions
Wall (%)	2.94	97.06	21.95	78.05
Heel (%)	6.86	93.14	21.95	78.05
Overgrown heel (%)	49.02	50.98	79.27	20.73
White line (%)	30.39	69.61	50.00	50.00
Heel-sole junction (%)	19.61	80.39	51.22	48.78
Sole (%)	52.94	47.06	57.32	42.68
Toe (%)	99.02	0.98	100	0.00

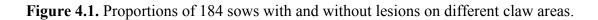
Table 4.3. Proportions of sows with hoof lesions according to type of gestation housing

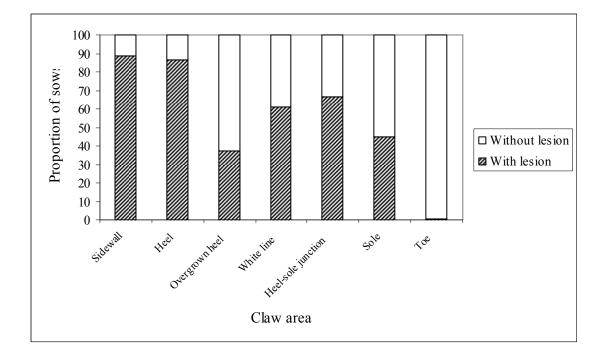
For all claw areas except the toe, proportions of sows with and without lesions differed for pen housing and stall housing (P < 0.05; two-sample proportion test).

Table 4.4. Odds ratios (OR) and confidence intervals (CI) for association of body condition (indicated by P2 back fat thickness and body weight at gestation day 109), lameness, parity, and gestation housing systems with presence or absence of lesions on different claw areas in 184 sows

Explanatory		Wall		Heel	Ονε	Overgrown heel	٨	White line	Sole-]	Sole-heel junction
variables	OR	CI	OR	CI	OR	CI	OR	CI	OR	CI
Weight (kg)	1.03 ^a	1.000-1.054	°99°	0.969-1.014	1.01 [°]	0.994-1.026	1.01 ^c	0.997-1.029	1.00°	0.984-1.017
Backfat (mm)	1.03°	0.911-1.162	1.15 ^a	1.021-1.295	0.88 ^b	0.804-0.968	0.95°	0.869-1.031	0.99°	0.911-1.082
Lameness	0.55°	0.140-2.180	1.31 [°]	0.447-3.816	0.99°	0.444-2.212	0.38 ^a	0.163-0.880	1.15°	0.514-2.573
Pen or stall	9.71 ^b	2.609-36.112	3.49 ^b	1.330-9.161	5.40 ^b	2.590-11.275	2.43 ^b	1.265-4.658	4.53 ^b	2.301-8.928
Parity										
1 vs. > 5	0.53°	0.082-3.403	0.38 ^c	0.055-2.568	0.67°	0.185-2.417	5.54 ^b	1.602-19.123	0.70°	0.202-2.458
2 vs. > 5	0.91°	0.201-4.131	0.32°	0.083-1.234	1.00°	0.393-2.551	4.52 ^b	1.749-11.667	1.02°	0.391-2.640
3-5 vs. > 5	0.79°	0.170-3.647	0.33°	0.081-1.296	0.71 ^c	0.271-1.850	2.92 ^a	1.134-7.514	0.44°	0.165-1.173
^{abc} Level of significance (Wald	enificar		stic): a	$P < 0.05 \cdot h P$	0 < 0.01	statistic): a. $P < 0.05$: b. $P < 0.01$ and c. $P > 0.05$ (variables did not differ). Sole lesions did)5 (varia	bles did not di	ffer) So	le lesions did

U.U. (Valiaules ulu IIUI ulliel). DUIE lesiulis ulu \sim 0.01, and c, Γ v.v.yv. F</u> Level of significance (wald statistic): a, P not differ among variables (P > 0.05).





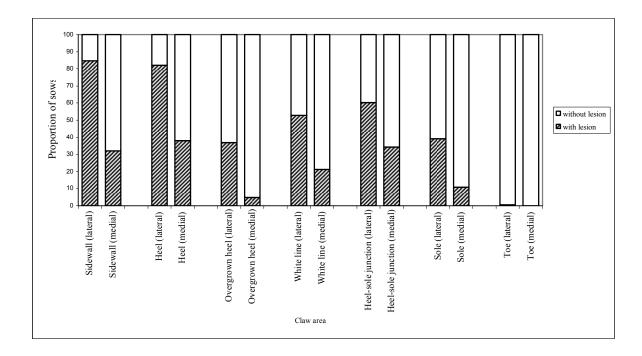
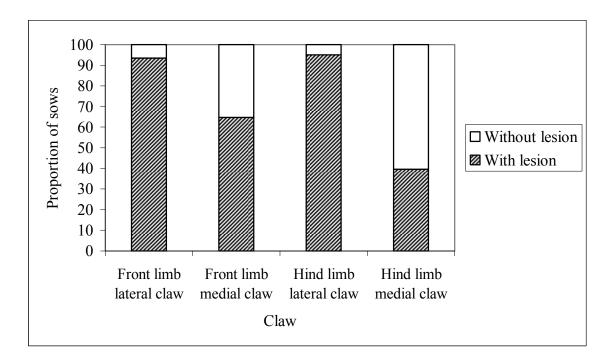


Figure 4.2. Proportions of 184 sows with and without lesions on different areas of lateral and medial claws.

Figure 4.3. Proportions of 184 sows with and without lesions on lateral and medial claws of front and hind limbs.



CHAPTER V

NUTRITIONAL INTERVENTION TO MINIMIZE CLAW LESIONS AND TO IMPROVE PERFORMANCE AND LONGEVITY IN BREEDING FEMALE PIGS

A study involving 615 sows (229 in stalls and 386 in group pens with electronic sow feeders (ESF)) was conducted to study the effect of supplementing complexed trace minerals (Cu, Zn and Mn) on claw lesions, reproductive performance and longevity of breeding female pigs. Of these sows, 197 group-housed sows and 116 stall-housed sows received treatment diet containing complexed trace minerals. Lesions on different claw areas among control and treatment group sows in both housing systems were recorded in subsequent parities (up to 4 parities). The proportions of sows showing a reduction (or no change) in lesion scores were compared to the proportions showing worsening of lesions using 2-sample proportion tests in both housing systems. The longevity (2-sample proportion test) and reproductive performance (2-sample t-test) of treatment and control group sows in the two housing systems were also compared. Logistic regression was used to analyze the association of the number of live born piglets per litter (\leq median vs. > median) with sow-level factors, claw lesions on side wall and white line and dietary supplementation of complexed trace minerals. All analyses were performed using SAS (v 9.1). A higher proportion (P < 0.05) of group housed sows fed treatment diet (91%) had either a reduction or similar lesion score compared to those fed with control diet. A higher (P<0.05) proportion of stall housed sows fed treatment diet (95 vs. 82%) had a decrease in the severity or no change in severity of heel-sole junction lesion score compared to the control group sows. The treatment group sows had lower (P < 0.05) stillborn piglets/ litter, sow weight at weaning and farrow to removal interval and higher (P < 0.05) wean litter weight and average wean weight per pig. The logistic regression analysis indicated that sows in the control group were 40% more likely to have ≤ 10

piglets born alive per litter compared to the sows in the treatment group controlling for various sow-level factors and claw lesions.

5.1 Introduction

Claw lesions are very common in pigs. Gjein and Larssen (1995) reported that more than 96% of loose-housed sows and 80% of confined sows had at least one lesion on the lateral hind claws. A study in a Minnesota herd also showed that 96% of breeding females have claw lesions (Anil et al., 2007). Claw lesions are an important underlying cause for lameness in pigs (Dewey et al., 1993). Claw lesions may cause lameness by acting as a source of pain, an obvious welfare concern. Besides this, claw lesions may permit entry of infections that may spread upwards, affecting joints and cause lameness. Locomotor problems have been reported as a major reason for culling of sows. It has been reported that approximately 6 to 35 % of sows are culled because of lameness (Rowles, 2001). Lame breeding stock may not attain optimum breeding efficiency (Ritter et al., 1999) and may be culled before attaining peak production causing economic loss. In addition, lame animals, while in the herd may require extra labor for management. Decreasing the number of sows culled due to lameness therefore has positive effects on overall production and welfare.

Though housing conditions and management on the farm are crucial as immediate causes for the development of claw lesions in pigs (Kroneman et al., 1993), nutrition, especially of trace minerals may also act as a predisposing factor. Nutrition is vital in developing the hoof structure and integrity. Minerals such as Ca, Zn, Cu, Mn, and vitamins A, D, and E, as well as biotin are reported to be important in the keratinization of hoof epidermis (Tomlinson et al., 2004). Improper nutrition can thus cause inferior quality horn tissue that is easily susceptible to chemical, physical, or microbial damage from the environment. Zinc is an important intracellular trace element and is vital to keratinization process (Smart and Cymbaluk, 1997; Mulling et al., 1999). It is also important to maintain a balance of trace minerals in the diet given the interaction among the minerals (Nocek et al., 2000).

The extent of bioavailability is crucial in trace mineral nutrition. Proteinating improves the bioavailability of the mineral to target cells and organs. During digestion, the weak bonds of conventional inorganic oxides and sulfates are broken down by the low pH of the stomach. The released ions become susceptible to the formation of insoluble and unavailable complexes that are excreted in feces. Though the mechanism is not well understood, it has been shown that chelated minerals are more soluble and can cross the intestinal wall more easily (Rompala and Halley, 1995). The epidermal cells in the hoof receive nutrition through the dermis by diffusion since epidermis is not vascular. However, the control mechanism of this nutrient flow is not well-understood and is likely to be influenced by nutritional, management and genetic factors. It is probable that supplementation may have to be of sufficient duration to have a visible effect on the integrity of the horn tissue.

An advantage of proper mineral nutrition, in addition to the improvement in hoof health is the potential beneficial effect on reproduction. The effects of added minerals in the diet on the reproductive performance of pigs have also been reported extensively. The beneficial role of trace minerals in improving the performance of breeding females (Mahan, 2005; Peters and Mahan, 2008), in minimizing stress deaths (Hagen et al., 2000) and in improving immunity (Tengerdy, 1986) has already been described. Although the role of minerals in the keratinization process is well established, there has not been a detailed study in breeding female pigs on the effect of trace minerals like Zn, Mn and Cu in preventing or treating claw lesions. Therefore, a study was formulated to evaluate the effect of complexed trace minerals on the prevalence and/or healing of claw lesions in breeding female pigs. It was also proposed to assess the effect of feeding complexed trace minerals on the performance and longevity of female pigs. The compounds used were trace mineral (Cu, Zn and Mn) amino acid complexes. The specific objectives were to determine the effect of supplementing complexed trace minerals in diets on the prevalence and severity of claw lesions in breeding female pigs and to determine the effect of supplementing complexed trace minerals in the diets on subsequent reproductive performance and longevity of breeding female pigs.

5.2 Materials and methods

The study was conducted at the Southern Research and Outreach Center (SROC), University of Minnesota, Waseca (800 sows, breed to wean facility) with IACUC approval. The study involved gilts and sows of multiple parities, randomly allocated to two treatments and two housing systems (gestation stalls and group pens with electronic sow feeders- ESF). This study involved 615 sows (229 in stalls and 386 in group pens with ESF). The sows from group housing system were followed till removal or the end of the trial at 24 months. Stall housed sows were followed till removal or the end of the trial at 18 months. The sows were fed either a diet containing complexed trace minerals (Zn, Mn and Cu) or a control diet without supplementation (regular diet fed in the research unit with inorganic trace minerals). One hundred and ninety seven group-housed sows and 116 stall-housed sows received treatment diet. Both the control and treatment groups received a gestation diet from the date of weaning till approximately day 110 of gestation. Animals were then moved to the farrowing room and fed a lactation diet till weaning. The lactation diet was given ad libitum post-farrowing. The sows received the same diet throughout the study period.

Number and severity of lesions on the claws were recorded on approximately day 60 of gestation using the format as laid out in Appendix 1. Claw examination at mid-gestation was performed with the help of a mechanical restraint developed for the purpose. The total score for each area was obtained by adding the scores for that area in the lateral and medial claws of front and hind limbs. The same individual conducted the evaluation of claw lesions at all occasions. All animals were assessed for lameness by the same individual on the days of claw lesion scoring.

Body weight and P2 back fat of all animals in the study groups were recorded on day 110 of gestation and at weaning. The level of lameness (lame or not-lame) in all animals was

assessed on the day of claw lesion scoring by making them walk for a short distance in an alleyway. Average daily feed consumption by individual animals in the study groups during lactation was collected. Representative feed samples were routinely collected and assayed for mineral composition.

Information on farrowing performance (born alive, mummies and stillborn, litter birth weight, litter weaning weight, preweaning mortality, number of piglets weaned per litter), lactation length and wean to first service interval in all parities during the study period were also collected for the sows in the study groups. Fostering was done within the treatments and within the housing system only. Removal (culling, death and euthanasia) reasons of all animals removed from the study groups and the parity at removal were recorded.

Statistical analysis. The data were analyzed using descriptive statistics. The proportions of sows showing either no change in lesion scores or a reduction in lesion scores in different claw areas in the control and treatment groups were compared using 2-sample proportion test. The production performances and longevity of sows in the treatment and control groups in both housing systems were compared using 2 sample t-test and 2 sample proportion tests. Spearman rank correlation was used to analyze the relationship between claw lesions and performance variables in group housed and stall-housed sows. Kaplan-Meier graphs with log-rank test / Wilcoxon test were used to compare the survivability of sows in the control and treatment groups in both housing systems.

Multivariate logistic regression analysis was used to describe the association of number of piglets born alive per litter ($\leq 10 \text{ vs.} > 10$) with total scores for side wall and white line lesions (considering the association of lameness with side wall and white line lesions reported), housing system, parity, complexed trace mineral supplementation, length of supplementation (cycle) stillborn, mummies and preweaning mortality. All analyses were performed using SAS (V 9.1). In all analyses a P value of ≤ 0.05 was considered significant.

5.3. Results

5.3.1 Group housing system (pens with ESF)

The median and range of total claw lesion scores (all observations) in control and treatment groups among sows housed in group pens with ESF is presented in Table 5.1. The median and range of total lesion scores and the lesion scores in different claw areas appeared to be similar in control and treatment groups among group housed sows. Figure 5.1 presents the proportion of group housed sows in the control and treatment groups with different levels of total claw lesion scores. The proportion of sows with claw lesion score > 10 appeared to be similar in both treatment and control groups (80.27 and 78.45% respectively). A comparison of the proportion of group-housed sows receiving control and treatment diets with claw lesion scores showing improvement or no change in different claw areas with the number of sows showing worsening of lesion scores in the first and last lesion evaluations is presented in Table 5.2. A higher proportion (P<0.05) of

sows in the treatment group (91 vs. 73%) had a reduction or similar lesion score for vertical side wall lesion in comparison to the control group.

Table 5.3 presents the comparison of production performance, lactation feed intake, body condition and longevity between treatment and control groups among sows housed in pens with ESF. The results indicated lower (P<0.05) stillborn piglets/ litter and sow weight at weaning for sows in the treatment group. Farrow to removal interval tended to be lower among sows in the treatment group (P=0.08). The treatment group sows also had higher (P<0.05) wean litter weight and average wean weight per pig. The Kaplan-Meier graph (Figure 5.2) showing the longevity of group-housed sows 200 days post-farrowing (first farrowing during the study period) indicated no significant difference in the survivability of sows between treatment and control groups (log-rank test chi square = 0.0006, P = 0.9802).

The correlations between claw lesion scores and performance variables among group housed sows are presented in Table 5.4. The number of piglets born alive was negatively correlated with the score for long dew claw and positively correlated with vertical side wall scores and total side wall scores. Stillborn was positively correlated with scores for long dew claws and elongated toes and negatively correlated with total white line lesion scores. Mummies were positively correlated with the score for elongated claws. Litter weight was negatively correlated with the score for long dew claws and positively correlated with vertical side wall scores and total side wall scores. Number of piglets weaned had negative correlation with total medial claw score, score for long dew claw and total overgrown heel score and positively correlated with total white line lesion score. Wean litter weight was negatively correlated with the score for long dew claws and total overgrown heel score. Piglet death was positively correlated with total lesion score, total lateral claw lesion score and total scores for long dew claw, heel and overgrown heel. Wean to service interval was negatively correlated with the score for long dew claws and positively correlated with the score for total side wall lesions. Average piglet birth weight was negatively correlated with total lateral claw lesion score. Average piglet wean weight was negatively correlated with the score for long dew claws and over grown heel. Although these correlations were significant (P<0.05), none of these correlation coefficients was of a large magnitude.

5.3.2 Stall housing system

The median and range of lesion scores appeared to be similar among the control and treatment group sows housed in stalls (Table 5.5). A comparison of the proportion of stall housed sows receiving control and treatment diets with claw lesion scores showing improvement or no change in different claw areas with the number of sows showing worsening of lesion scores in the first and last lesion evaluations is presented in Table 5.6. A higher (P<0.05) proportion of treatment group sows housed in stalls (95 vs. 82%) showed reduction in the severity (or no change in severity) of heel-sole junction lesion score. Figure 5.3 presents the proportion of stall housed sows in the control and treatment

groups with different levels of total claw lesion scores. The proportion of sows with claw lesion score > 10 were 38.5 and 28.7% in treatment and control groups respectively.

Comparison of the production performances of treatment and control group sows housed in stalls (Table 5.7) indicated differences (P<0.05) in terms of the number of piglets born alive (10.4 and 11.1 in control and treatment respectively) and stillborn (0.7 and 1.0 in control and treatment respectively) per litter. Litter birth weight also tended to be higher (P = 0.07) in the treatment group (35.6 in control vs. 37.5 lbs in treatment group). The Kaplan-Meier graph (Figure 5.4) showing the longevity of sows 150 days post-farrowing (first farrowing during the study period) indicated no difference in the survivability of sows between treatment and control groups (Wilcoxon test chi square =0.1415, P = 0.7068) among stall-housed sows.

Table 5.8 shows the correlations between claw lesions and performance variables. The number of piglets born alive was negatively associated with the total scores for long toes and dew claws. Litter weight was negatively associated with the total scores for lateral claw, medial claw, total lesions, dew claw and overgrown heels. Number of piglets weaned showed a negative association with scores for medial claw, dew claw, and heel-sole lesions whereas it was positively associated with the score for long toes. Wean litter weight was negatively associated with the total score for medial claw lesions and dew claw. Preweaning piglet death was positively associated with total medial claw scores and negatively associated with the score for long toes. Wean-service interval was

positively associated with total heel lesions, vertical side wall lesions and total side wall lesions. It had a negative association with white line lesion scores. Average piglet birth weight was negatively associated with total medial claw lesion scores. Though significant (P<0.05), none of these correlation coefficients was of a large magnitude.

The associations of number of piglets born alive per litter ($\leq 10 \text{ vs.} > 10$) with total scores for side wall and white line lesions, housing system, parity, cycle, organic trace mineral feeding, stillborn, mummies and preweaning mortality are presented in Table 5.9. The likelihood of having <10 piglets born alive per litter decreased (P<0.05) with an increase in scores for total side wall lesions (Table 5.9). Sows in the control group were 40% more likely (P<0.05) to have ≤ 10 piglets born alive per litter compared to the sows in the treatment group. Sows with no mummies were 57% more likely to have ≤ 10 piglets born alive per litter (P<0.05). Similarly, sows with no preweaning piglet mortality were 3 times more likely to have ≤ 10 piglets born alive per litter (P<0.05). Sows of parity ≤ 5 were less likely (P<0.05) to have ≤ 10 piglets born alive per litter than sows of parity > 5.

5.4 Discussion

The results of the study indicate beneficial effects of the supplementing complexed trace minerals in heeling claw lesions among sows housed in both group pens and in stalls. The proportions of group-housed sows with either similar lesion scores or lower lesion scores for heel lesions and vertical side wall lesions were higher among sows in the treatment group compared to the sows in the control group. The present results are similar to a previous trial which indicated a negative relationship between complexed trace mineral supplementation and number of lesions in different claw areas (Anil et al., 2009). However, that study had not considered the severity of lesions. They also reported that sows receiving inorganic trace minerals had more lesions on the hind limbs than the sows receiving complexed trace minerals. However, the present results were not consistent with lesion scores on different claw areas. The exact mechanism behind the development of claw lesions, especially the effect of different bio-mechanical factors has not yet been fully understood. These factors were not the same in the two housing systems studied. This being an observational study, other potential causal factors for claw lesion development were not controlled for. This may partially explain the inconsistency in the beneficial effect of trace mineral supplementation on claw lesions. The survivability of treatment and control group sows was the same in both housing system, suggesting the influence of other causes as well as the influence of factors (e.g. availability of gilt pool, market factors) not related to the sow on sow removal decisions. The present result suggesting a higher number of live born piglets in the treatment group is in agreement with the previous studies indicating a beneficial effect of complexed trace mineral supplementation on the reproductive performance of sows. A previous study involving supplementation with complexed trace minerals (Bradley et al., 2009) indicated higher litter birth weights in supplemented sows though the number of live born piglets was not different (P=0.2) in control (12.89) and treatment (13.37) groups. A higher number of stillborn piglets per litter in the treatment group sows observed in the present study could be related to the higher number live born piglets per litter. Similarly, the higher litter birth weight in the treatment group sows could also be related to higher number of live born piglets. The results also indicated that with an increase in side wall lesion score the likelihood of having ≤ 10 live born piglets per litter decreased. This is suggestive that high producing sows are more susceptible to lesions. Similar relationship between milk production and hoof lesions in dairy cattle have been reported (Manske, 2002; Hultgren et al., 2004). Though it is hard to extrapolate that relationship in this case, it clearly points to the need to have further studies in this area.

Table 5.1. Median and range of total lesion scores in control and treatment group sows housed in group pens with ESF for the entire study period

Total lesion score	Control (n=746)	Treatment (n=721)
Lateral claw	12(1-33)	12(0-28)
Medial claw	2(0-15)	2(0-14)
Total lesions	15(3-42)	14(4-37)
Long toe	0(0-13)	0(0-15)
Dew claw	5(0-16)	5(0-18)
Heel	5(0-14)	5(0-13)
Overgrown heel	3(0-9)	4(0-9)
Heel-sole junction	0(0-5)	0(0-9)
White line	0(0-8)	1(0-9)
Side wall-horizontal	2(0-10)	2(0-10)
Side wall- vertical	2(0-12)	2(0-12)
Total side wall	4(0-16)	4(0-15)
Front limb	7(0-29)	7(0-25)
Hind limb	7(1-16)	7(0-17)

Table 5.2. Comparison of the number of group housed sows with claw lesion scores showing improvement or no change in different claw areas with the number of sows showing worsening of lesion scores

Total lesion score		ervation vs. servation	
	Control	Treatment	Р
	n=59	n = 45	value
Lateral claw	41	27	NS
Medial claw	39	34	NS
Total lesions	45	28	NS
Long toe	25	14	NS
Dew claw	27	18	NS
Heel	53	42	NS
Overgrown heel	19	13	NS
Heel-sole junction	56	37	NS
White line	47	33	NS
Side wall-			
Horizontal	41	30	NS
Side wall- vertical	43	41	0.037
Total side wall	46	37	NS
Front limb	36	24	NS
Hind limb	47	34	NS

NS= non-significant

Table 5.3. Comparison of production performance in treatment (T) and control (C) group sows housed in pens with ESF (2 sample t test)- all farrowings

Variables	C (parity records	T (parity records =529)	Р
	=527) mean \pm SE	Mean \pm SE	value
Average parity	5.3251±0.1274	5.2155 ± 0.1293	0.5460
Born alive	10.0473±0.1396	10.2197 ± 0.1416	0.3862
Still born	1.2903±0.0755	1.0568 ± 0.0661	0.0202
Mummies	0.2311±0.0241	0.2557 ± 0.0262	0.4891
Average birth wt/pig (lbs)	3.6980 ±0.0268	3.6953 ± 0.0260	0.9416
Litter weight (lbs)	36.1973±0.4572	37.1190 ± 0.4838	0.1665
Wean litter weight (lbs)	129.7520±1.3310	133.5520 ± 1.3122	0.0423
Average wean wt/pig (lbs)	14.0336 ± 0.0870	14.2832 ± 0.0806	0.0357
Pre weaning mortality	1.0753 ± 0.0601	0.9439 ± 0.0563	0.1110
Wean to service interval (days)	6.3803 ± 0.2844	6.7761 ± 0.3475	0.3775
Number weaned	9.2136 ± 0.0927	9.2581 ± 0.0841	0.7228
Weight at day 109 of gestation, (lbs)	585.9440 ± 2.7635	578.3670± 2.9144	0.0595
(n=521)			
Back fat – L at farrowing (mm)	18.9310±0.2843	20.743±2.30083	0.4347
Back fat - R at farrowing (mm)	18.7310±0.1847	19.647±1.22743	0.4609
Weight at weaning (lbs)	543.4040 ± 3.1922	531.5900 ± 3.5147	0.0130
Back fat – L at weaning (mm)	16.1846 ± 0.1534	15.8012 ± 0.1657	0.0896
Back fat - R at weaning (mm)	16.0096 ± 0.1537	15.7290 ± 0.1684	0.2184
Average lactation feed intake, (lbs)	12.8231±0.15535	12.5409 ± 0.15333	0.1963
(n=520)			
Average LFI – 14days (lbs) (n=520)	12.4008 ± 0.16030	12.0466±0.15598	0.1136
Remove parity (C:n= 93, T:n= 94)	7.9032 ± 0.3795	7.5851 ± 0.3764	0.5525
Farrow to removal interval	114.1400 ± 10.2697	90.2690 ± 9.0730	0.0832
Proportion of sows removed ^o	93/189 (49.20%)	95/197 (48.22%)	0.9273

[°] 2 sample proportion test

et	ght	-0.06	0.138	0.05	185	-0.01	0.754	-0.06	233	-0.09	283	-0.02	104	-0.13	014	0.02	783	0.00	995	0.07	
Ave piglet wean	weight		0.1		0.2185		0.7	0-	0.1233		0.0283		0.6104		0.0014	0	0.6783	0	0.9995	0	
Ave piglet hirth	weight	-0.08	0.0532	0.04	0.3066	-0.04	0.3418	-0.01	0.721	-0.03	0.3944	-0.04	0.3157	-0.02	0.6429	-0.01	0.7154	-0.01	0.8057	0.01	
Wean- service	interval	0.02	0.6878	0.03	0.4582	0.03	0.5347	-0.05	0.2602	-0.10	0.0193	-0.06	0.1425	-0.01	0.8488	-0.03	0 4746	0.03	0.5285	0.07	
Pre-weaning	mortality	0.10	0.0192	0.02	0.6455	0.09	0.0316	-0.01	0.8929	0.13	0.0019	0.12	0.0045	60:0	0.0213	0.02	0 6868	-0.04	0.3459	-0.04	
Wean	WT	-0.05	0.2061	0.00	0.9799	-0.03	0.4491	-0.06	0.1334	-0.14	0.0004	-0.03	0.4	-0.15	0.0002	0.02	0.7047	0.07	0.0881	0.05	
Number	weaned	-0.02	0.568	-0.09	0.0266	-0.06	0.1735	-0.02	0.6808	-0.13	0.0018	-0.06	0.1652	-0.09	0.0304	-0.03	0 455	0.10	0.0135	0.00	
Litter	weight	0.03	0.5079	0.06	0.1571	0.06	0.1406	-0.05	0.1847	-0.17	<0.0001	-0.01	0.7744	-0.05	0.2496	-0.01	0 7943	0.07	0.0751	0.03	
	Mummies	-0.01	0.8906	0.02	0.5425	0.00	0.9409	0.08	0.0501	0.06	0.1315	0.01	0.7146	0.04	0.2993	-0.04	0.3769	-0.01	0.828	0.05	
	Stillborn	0.05	0.2324	0.02	0.5473	0.04	0.2678	0.11	0.0052	0.08	0.0458	0.07	0.1039	0.05	0.1827	0.01	0.7752	-0.09	0.0198	0.07	
Born	alive	0.06	0.1302	0.03	0.5047	0.07	0.0875	-0.02	0.6052	-0.12	0.0022	0.01	0.8883	-0.04	0.3571	-0.01	0.8504	0.06	0.1443	0.03	
		Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Corr. Coef	P value	Сопт. Coef	
Total score/	variable	Lateral claw	Lateral claw	Medial claw	Medial claw	Total score	Total score	Long toe	Long toe	Long dew claw	Long dew claw	Heel	Heel	Overgrown heel	Overgrown heel	Heel-sole lesion	Heel-sole lesion	White line	White line	Side wall- horizontal	

Table 5.4. Spearman correlation between claw lesion scores and performance variables among group housed sows

87

Side wall-											
vertical	Corr. Coef	0.09	-0.03	-0.07	0.09	0.03	0.03	-0.03	0.03	-0.03	0.01
Side wall-											
vertical	P value	0.0317	0.3918	0.0686	0.0267	0.4876 0.4147	0.4147	0.4939	0.4559	0.5202	0.7275
Side wall- total Corr. Coef 0.08	Corr. Coef	0.08	0.02	-0.02	0.08	0.01	0.06	-0.04	0.10	-0.01	0.07
Side wall- total P value	P value	0.044	0.5632	0.5577	0.0414	0.7981 0.1728	0.1728	0.325	0.0264	0.8062 0.1134	0.1134

Table 5.5. Median and range of total lesion scores in control and treatment group sows housed in stalls during the study period

Total lesion score	C (n	T (n =208)
	=206)	
Lateral claw	7 (0-17)	6(0-17)
Medial claw	2(0-7)	1(0-7)
Total lesions	8(0-18)	7(0-21)
Long toe	0 (0-4)	0(0-4)
Long toe	2 (0-8)	1(0-8)
Heel	1 (0-6)	1(0-6)
Overgrown heel	2(0-4)	2(0-5)
Heel-sole junction	0(0-2)	0(0-2)
White line	0(0-5)	0(0-3)
Side wall-horizontal	0(0-4)	1(0-5)
Side wall- vertical	1(0-6)	1(0-4)
Total side wall	1 (0-6)	1(0-6)
Total sole	0 (0-5)	0 (0-4)
Front limb	1(0-7)	1(0-5)
Hind limb	4(0-10)	4(0-8)

Table 5.6 Comparison of the number of stall-housed sows with claw lesion scores

 showing improvement or no change in different claw areas with the number of sows

 showing worsening of lesion scores (2 sample proportion test)

		pservation vs. 1 st bservation	
	Control	Treatment	P value
Total lesion score	n =66	n = 63	
Lateral claw	22	29	NS
Medial claw	37	30	NS
Total lesions	26	21	NS
Long toe	47	46	NS
Dew Claw	37	30	NS
Heel	34	37	NS
Overgrown heel	37	30	NS
Heel-sole junction	54	60	0.0355
White line	53	56	NS
Side wall-Horizontal	48	41	NS
Side wall- vertical	40	41	NS
Total side wall	39	33	NS
Front limb	32	28	NS
Hind limb	31	29	NS

NS= non-significant

Table 5.7. Comparison of the production performance of control and treatment group

sows housed in stalls (2 sample t test)

Variables	C (n=199) mean \pm	T (n=200) Mean \pm SE	P value
	SE		
Average parity	4.9146 <u>+</u> 0.2126	4.8150 <u>+</u> 0.2086	0.7383
Born alive	10.4372 <u>+</u> 0.2170	11.0700 <u>+</u> 0.2052	0.0347
Still born	0.6985 <u>+</u> 0.0852	0.9950 <u>+</u> 0.1135	0.0375
Mummies	0.1859 <u>+</u> 0.0371	0.1850 <u>+</u> 0.0333	0.9851
Average birth wt/pig (lbs)	3.4701 <u>+</u> 0.0391	3.5022 <u>+</u> 0.1068	0.7787
Litter weight (lbs)	35.6382 <u>+</u> 0.7164	37.4523 <u>+</u> 0.6761	0.0663
Wean litter wt (lbs)	40.5160 <u>+</u> 1.7403	138.7450 <u>+</u> 1.6783	0.4642
Average wean wt/pig (lbs)	14.1458 <u>+</u> 0.1276	14.0110 <u>+</u> 0.1283	0.4566
Pre weaning mortality	0.9045 ± 0.0877	1.1106 <u>+</u> 0.0999	0.1221
Wean to service interval (days)	6.2071 <u>+</u> 0.4092	7.5617 <u>+</u> 0.8900	0.1619
Number weaned	9.8694 <u>+</u> 0.0919	9.8350 <u>+</u> 0.0868	0.7859
Weight at day 109 of gestation	534.3858 ± 3.4843	527.5365±4.0748	0.2014
(lbs)			
Back fat – L at farrowing (mm)	15.2843±0.3263	14.6458±0.3713	0.1966
Back fat – R at farrowing (mm)	15.3300±0.3327	14.7292±0.3750	0.2309
Weight at weaning (lbs)	511.9082±4.7705	504.2552±3.7656	0.2099
Back fat – L at weaning (mm)	13.7398±0.2571	13.2813±0.2833	0.2310
Back fat – R at weaning (mm)	13.6225±0.2576	13.2031 ± 0.2825	0.2730
Average lactation feed intake (lbs)	13.8547±0.2177	14.2810±0.2465	0.1950
AVE LFI – 14days (lbs)	13.5627±0.2181	13.8441±0.2693	0.4161
Remove parity (C: n=64, T: n=76)	7.9688 <u>+</u> 0.3965	7.7500 <u>+</u> 0.3393	0.6739
Farrow to removal interval (days)	150.6940 <u>+</u>	139.3430 <u>+</u> 10.9792	0.4982
	12.7169		
Proportion of sows removed ⁶	64/113 = 56.64%	76/116 = 65.52%	0.2140

 $^{\circ}2$ sample proportion test

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orrelation between claw lesion scores and performance variables among stall housed sows	
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							Wean		Wean-	Ave piglet	Ave piglet
Total score /		Born			Litter	Number	litter	Pre-weaning	service	birth	wean
variable		alive	Stillborn	Mummies	weight	weaned	weight	mortality	interval	weight	weight
Lateral claw	Corre. Coeff	-0.08	-0.01	-0.08	-0.12	-0.02	-0.04	0.00	0.03	-0.03	-0.04
Lateral claw	P value	0.1172	0.8458	0.1035	0.0149	0.6733	0.4805	0.955	0.5725	0.4924	0.4328
Medial claw	Corre. Coeff	-0.06	0.02	-0.06	-0.14	-0.13	-0.13	0.10	0.04	-0.10	-0.06
Medial claw	P value	0.2356	0.6737	0.2667	0.0055	0.0074	0.0116	0.0443	0.4345	0.0501	0.2677
Total score	Corre. Coeff	-0.08	0.00	-0.09	-0.15	-0.07	-0.08	0.03	0.03	-0.07	-0.05
Total score	P value	0.1012	0.9635	0.0688	0.0034	0.1619	0.1339	0.5351	0.5562	0.1898	0.3059
Long toe	Corre. Coeff	-0.10	0.02	0.02	-0.01	0.10	0.06	-0.14	-0.06	0.07	0.03
Long toe	P value	0.0394	0.7036	0.6734	0.8342	0.0491	0.2321	0.0068	0.2782	0.1482	0.5801
Long dew claw	Corre. Coeff	-0.14	0.01	-0.04	-0.22	-0.12	-0.14	0.05	-0.01	-0.09	60.0-
Long dew claw	P value	0 0055	0 7901	0 473	< 0001	0.0184	0 0047	0 3437	0 8938	0 0774	0 077
Heel	Corre. Coeff	-0.06	-0.04	-0.08	-0.06	-0.07	-0.03	0.02	0.11	0.05	-0.01
Heel	P value	0.24	0.4398	0.1285	0.2338	0.1393	0.6069	0.6834	0.0483	0.3311	0.836
Overgrown heel	Corre. Coeff	-0.05	-0.01	-0.08	-0.10	-0.02	-0.03	-0.01	0.03	-0.03	-0.02
Overgrown heel	P value	0.2789	0.7652	0.1272	0.0525	0.7525	0.5729	0.9091	0.5392	0.5043	0.6212
Heel-sole lesion	Corre. Coeff	0.03	0.01	-0.02	0.03	-0.10	-0.08	-0.02	0.03	-0.05	-0.04
Heel-sole lesion	P value	0.5373	0.7877	0.7279	0.6025	0.0532	0.1066	0.7219	0.5272	0.3479	0.4144
White line	Corre. Coeff	0.09	0.03	0.00	0.05	0.04	0.03	0.02	-0.16	-0.02	-0.01
White line	P value	0.0759	0.5455	0.9895	0.2733	0.3927	0.562	0.6192	0.0029	0.7111	0.8735
Side wall- horizontal	Corre. Coeff	0.00	0.05	-0.08	-0.03	-0.02	-0.08	0.00	-0.01	-0.01	-0.08

Side wall-											
horizontal	P value	0.9677	0.3187	0.0952	0.531	0.632	0.104	0.9373	0.8419	0.8455	0.1318
Side wall-											
vertical	Corre. Coeff	0.09	-0.07	0.00	0.06	0.08	0.08	0.02	0.14	-0.01	0.04
Side wall-											
vertical	P value	0.0871	0.1673	0.9806	0.9806 0.2029	0.1308	0.1031	0.7158	0.009	0.8027	0.4798
Side wall-											
total	Corre. Coeff	0.06	-0.04	-0.06	0.02	0.04	0.02	0.03	0.11	-0.02	-0.01
Side wall-											
total	P value	0.1963	0.4485	0.2471	0.2471 0.6383	0.4763	0.7359	0.5382	0.0479	0.7155	0.8586

Table 5.9. Odds ratios and 95% CI of the association of number of piglets born alive per litter ($\leq 10 \text{ vs.} > 10$) with total scores for side wall and white line lesions, housing system, parity, cycle, organic trace mineral feeding, stillborn, mummies and preweaning mortality (outcome evaluated is the likelihood for ≤ 10 piglets born alive per litter)^a

Effect	OR	95 %Confidence Limits	P value
Total white line			
lesion score	0.95	0.811 - 1.113	0.5269
Total side wall lesion			
score	0.939	0.882 - 1.000	0.0498
Control vs. Treatment	1.402	1.070 - 1.837	0.0142
Cycle 1.5 vs. 3.5	0.863	0.534 - 1.396	0.5481
Cycle 2.5 vs. 3.5	0.791	0.483 - 1.295	0.3509
Parity 1&2 vs. >5	0.386	0.258 - 0.578	< 0.0001
Parity 3-5 vs. >5	0.29	0.213 - 0.396	< 0.0001
Group vs. stall	1.18	0.832 - 1.673	0.3528
Stillborn: no vs. yes	0.865	0.654 - 1.143	0.3079
Mummies: no vs. yes	1.566	1.087 - 2.256	0.016
Preweaning			
mortality: no vs. yes	3.065	2.311 - 4.064	< 0.0001

^a only sows with mid gestation claw lesion scores and subsequent production performance details

Figure 5.1: Proportion of group housed sows in the control and treatment groups with different levels of total claw lesion scores

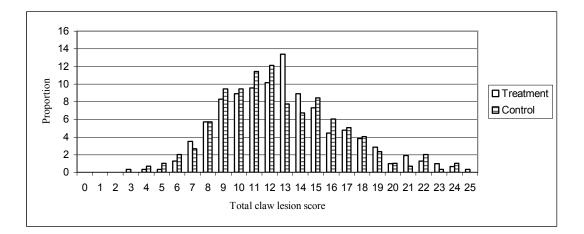


Figure 5.2: Kaplan-Meier graph of the time to removal from the herd for control (upper line) and treatment (lower line) sows housed in groups

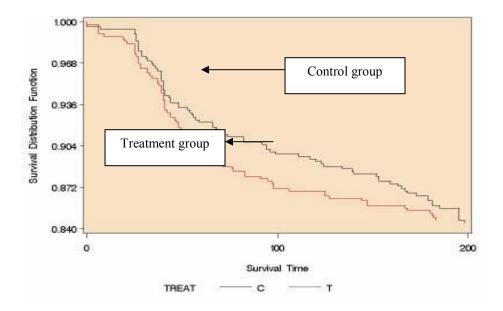


Figure 5.3: Proportion of stall housed sows in the control and treatment groups with different levels of total claw lesion scores

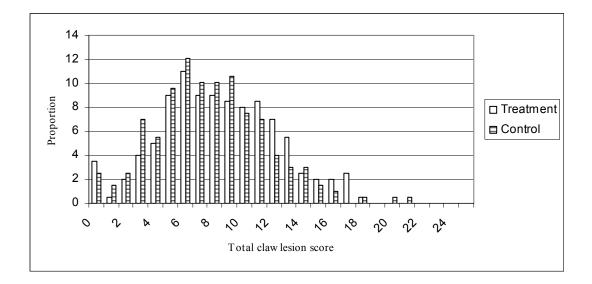
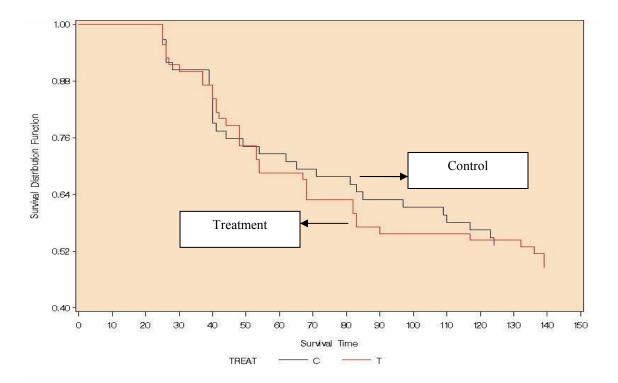


Figure 5.4: Kaplan-Meier graph of the time to removal from the herd for control (upper line) and treatment (lower line) sows housed in stalls



GENERAL DISCUSSION AND CONCLUSION

Culling and replacement rate continue to be the major vardsticks for sow longevity assessment in US swine breeding herds. Reproductive inefficiency is the single most important reason leading to sow removals in most herds. Another most commonly reported cause for premature sow removals is locomotor problem. Regardless of the removal reason, a low level of sow retention in the herd is a cause for both economic as well as welfare concerns. Breeding females are removed from the herd for production or health reasons. The importance of the problem of poor longevity increases when the cause is associated with compromised welfare as it attracts wide criticisms from the public. Although a sow may be removed from the herd at any point in time during its reproductive cycle, the risk of removal is not the same throughout its life. Farrowing is known to be a high risk event for removal for both production and welfare reasons. The results of the study described in the second chapter confirmed that LFI, incidence of lameness or health problems, as well as sow-level characteristics such as higher parity and fewer piglets born alive were important risk factors. This study also showed that sows retained with periparturient health problems had reduced longevity and fewer liveborn piglets, and fewer such sows had another farrowing. Often the retention of sows with considerable health problems during the periparturient period is justified to minimize productivity losses. However, the findings suggest that sow removal decisions should be judiciously evaluated after farrowing considering the potential long-term losses.

Most of the studies and thus the research conclusions on sow longevity are based on producer-recorded data. It is an undeniable fact that in many instances not all reasons or even the most significant reason may not be the one that gets recorded. As a result, the underlying causes leading to the removal of sows are likely to be underestimated. Lameness/ locomotor problem appears to be one such cause given that it is a painful condition and that pain can initiate a whole set of undesirable physiologic responses capable of affecting the reproductive performance of sows. Nevertheless, lameness is likely to be a reported removal reason only when it acute and severe in nature. Identification of less severe, chronic cases of lameness is a practically challenge in commercial operations. Painful conditions like lameness occurring around farrowing certainly affects lactation feed intake with subsequent adverse effect on reproduction. This thesis comprises of a series of studies to identify and address the risk factors affecting sow longevity with special emphasis on lameness. The second chapter was an attempt to delineate the periparturient risk factors of sow longevity. The most important finding of that study was the effect of retaining sows with painful conditions like lameness on subsequent herd performance. Retaining sows with less severe lameness may enable the producer to meet immediate production targets. However, the results clearly indicate the long-term cost associated with such retention. The results of the prospective analysis explained in chapter three also indicated that the overall performance of lame sows in terms of the number of pigs born alive during the period of the study was less, compared with that for non-lame sows. Therefore, lameness in swine

herds should be minimized and if treatment is not an option lame sows should be culled as soon as possible to reduce long-term losses. The third chapter was a continuation of the study described in the second chapter to prospectively analyze the effect of lameness on longevity and performance, using time to event analysis methodology, as it happens in commercial swine breeding herds. The results of this study confirmed the adverse effects of lameness on sow longevity, controlling for the effect of other sow level factors.

The fourth chapter described the distribution of claw lesions in a breeding herd and the association of claw lesions with lameness. The results confirmed the high prevalence of claw lesions and their association with lameness. Specifically, white line lesions were associated with lameness. The finding with a long-term implication in this study was the high prevalence of claw lesions of all types among group-housed sows. This result points to the need to formulate strategies to minimize claw lesions and lameness when the industry moves to group housing system for breeding female pigs. Obviously, the amount and type of activity of sows determine the type and severity of claw lesions, and these two factors vary between group and confined systems.

The results described in the fifth chapter indicate the possibility of nutritional intervention in minimizing claw lesions. However, there are other factors associated with claw lesion development in pigs. The quality of the floor as well as different bio-mechanical factors operating in lesion development are important here. The space between slats, roughness of the surface, and edge design are critical in claw lesion

development. Those factors have not been addressed in this study. Further studies are required to understand the mechanism of lesion development in relation to the housing and management systems in place. The inconsistent pattern of results suggests that this information is vital in formulating the appropriate intervention strategy. Similarly, though the relationship between claw lesions and lameness is well-supported, occurrence of claw lesions is only one factor causing lameness in pigs.

This thesis was based on observational studies. The results are limited to the extent that the recording of health related risk factors such as lameness and other disease symptoms were based exclusively on farm records that were not necessarily based on veterinary diagnosis or other standardized procedures. Similarly, there was no training program for lameness diagnosis, and the validity and reliability of the lameness assessment were not verified. However, the differences were large and suggested considerable validity for the scoring methods. Further, the studies included data from single herds and therefore the generalization of the results may be restricted owing to the wide variations in management, housing and in genetic lines of sows.

References

Anil L., Anil, S.S. and Deen, J. (2008). Sensitivity and specificity of lameness assessment in sows. Proceedings of the 20th International Pig Veterinary Society Congress, Durban, South Africa, pp.615.

Anil, L., Anil, S.S., Deen, J., Baidoo, S.K. and Walker, R.D. (2006). Effect of group size and structure on the welfare and performance of pregnant sows in pens with electronic sow feeders. Can. J. Vet. Res. **70**:128–136.

Anil, L., Anil, S.S., Deen, J., Baidoo, S.K. and Wheaton, J.E. (2005). Evaluation of wellbeing, productivity, and longevity of pregnant sows housed in groups in pens with an electronic sow feeder or separately in gestation stalls. Am. J. Vet. Res. **66**:1630–1638.

Anil, S.S., Anil, L. and Deen, J. (2005). Evaluation of patterns of removal and associations among culling because of lameness and sow productivity traits in swine breeding herds. J. Am. Vet. Med. Assoc. **226**:956–961.

Anil, S.S., Anil, L. and Deen, J. (2005). Evaluation of patterns of removal and associations among culling because of lameness and sow productivity traits in swine breeding herds. J. Am. Vet. Med. Assoc. **226**: 956-961.

Anil, S.S., Anil, L. and Deen, J. (2007). Factors associated with claw lesions in gestating sows. J. Swine. Health. Prod.15: 78-83.

Anil, S.S., Anil, L. and Deen, J. (2008). Analysis of periparturient risk factors affecting sow longevity in breeding herds. Can. J. Anim. Sci. **88**: 381-389.

Anil, S.S., Anil, L., Deen, J., Baidoo, S.K. and Walker, R.D. (2006). Association of inadequate feed intake during lactation with removal of sows from the breeding herd. J. Swine. Health. Prod. **14**: 296-301.

Anil, S.S., Deen, J., Anil, L., Baidoo, S.K., Wilson, M.E., and Ward, T.L. (2009). Evaluation of the supplementation of complexed trace minerals on the number of claw lesions in breeding sows. Manipulating Pig Production XII, Twelfth Biennial Conference of the Australasian Pig Science Association.

Bach, A., Dinares, M., Devant, M. and Carre, X. (2007). Associations between lameness and production, feeding and milking attendance of Holstein cows milked with an automatic milking system. J. Dairy. Res. **74**: 40-46.

Baidoo, S. K., Aherne, F. X., Kirkwood, R.N. and Foxcroft, G. R. (1992). Effect of feed intake during lactation and after weaning on sow reproductive performance. Can. J. Anim. Sci. **72**: 911-917.

Bilkei, G. and Bolcskei, A. (1995). Production related culling strategy in a large pig production unit. The Pig Journal. **35**: 140-149.

Boon, C.R. and Wray, C. (1989). Building design in relation to the control of diseases of intensively housed livestock. J. Agric. Eng. Res. **43**:149–161.

Boyd, R.D., Castro, G. and Aherne, F.X. (1998). Nutrition and metabolism – how they influence sow viability. Proceedings of the 16th International Pig Veterinary Society Congress, Melbourne, Australia. pp. 292.

Boyle, L., Leonard, F.C., Lynch, B. and Brophy, P. (1998). Sow Culling Patterns and Sow Welfare. Irish Vet. J. **51**: 354 – 357.

Bradley, C.L., Frank, J.W., Maxwell, C.V., Johnson, Z.B., Ward, T.L. and Wilson, M.E. (2009). The influence of complexed minerals fed to developing maternal gilts through three parities on reproductive and lactation performance. Midwest ASAS meeting, March 16-18. Abstract 219.

Bryant, K.L., Kornegay, E.T., Knight, J.W., Veit, H.P. and Notter, D.R. (1985). Supplemental biotin for swine. 3. Influence of supplementation to corn- and wheat-based diets on the incidence and severity of toe lesions, hair and skin characteristics and structural soundness of sows housed in confinement during four parities. J. Anim. Sci. **60**:154–162.

Budras, K.D., Mülling, C. and Horowitz, H. (1996). Rate of keratinization of the wall segment of the hoof and its relation to width and structure of the zona alba (white line) with respect to claw disease in cattle. Am. J. Vet. Res. **57**:444–455.

Chagnon, M., D'Allaire, S. and Drolet, R. (1991). A prospective study of sow mortality in breeding herds. Can. Vet. J. **55**: 180–184.

D'Allaire, S. and Drolet, R. (1999). Culling and mortality in breeding animals. In A. D. Leman., B. E. Straw., W. L. Mengeling., S. D'Allaire and D. J.Taylor, eds. Diseases of swine, 7th edition. Iowa State University Press, Ames. pp. 1003-1016.

D'Allaire, S., Stein, T. E. and Leman, A. D. (1987). Culling patterns in selected Minnesota swine breeding herds. Can. J. Vet. Res. **51**: 506-512.

Dagorn, J. and Aumaitre, A. (1979). Sow culling: reasons for and effect on productivity. Livest. Prod. Sci. 6:167–177.

Deen, J. (2003a). Sow longevity measurement. Proc. Allen D. Leman Swine Conference., St Paul, Minnesota **30**:192–193.

Deen, J. (2003b). Periparturient mortality. Proc. Allen D. Leman Swine Conference., St Paul, Minnesota **30**: 203–204.

Deen, J. and Xue, J. (1999). Sow mortality in the U.S.: An industry-wide perspective. Proc. Allen D. Leman Swine Conference., St Paul, Minnesota **26**: 91–94.

Dewey, C.E., Friendship, R.M. and Wilson, M.R. (1993). Clinical and postmortem examination of sows culled for lameness. Can. Vet. J. **34**:555-556.

Dourmad, J. Y., Etienne, M., Prunier, A. and Noblet, J. (1994). The effect of energy and protein intake of sows on their longevity: a review. Livest. Prod. Sci. **40**: 87-97.

Engblom, L., Lundeheim, N., Strandberg, E., del P. Schneider, M., Dalin, A.M and Andersson, K. (2008). Factors affecting length of productive life in Swedish commercial sows. J. Anim. Sci. **86**:432–441.

English, P.R., Edwards, S.A. (1999). Animal welfare. In: Straw, B.E., D'Allaire, S., Mengeling,
W.L. and Taylor, D.J. (eds). *Diseases of Swine*. Iowa State University Press, Ames, Iowa, pp. 1067-1076.

Friendship, R. M., Wilson, M. R., Almond, G. W., McMillan, I., Hacker, R. R., Pieper, R. and Swaminathan, S. S. (1986). Sow wastage: Reasons for and effect on productivity. Can. J. Vet. Res. **50**: 205–208.

Gardner, I. A. Hird, D.W. Sullivan, N. M. and Pierce, R. J. (1990). Clinical, pathologic, and microbiologic findings of foot abscess in neonatal pigs. J. Am. Vet. Med. Assoc. **196**: 1791-1794.

Gjein, H. and Larssen, R.B. (1995). Housing of pregnant sows in loose and confined systems-a field study. 2. Claw lesions: morphology, prevalence, location and relation to age. Acta. Vet. Scand. **36**:433-442.

Glastonbury, J.R.W. (1976). A survey of preweaning mortality in the pig. Aust. Vet. J. **52**:272–276.

Grandjot, G. (2007). Claw problems cost money. SUS - Schweinezucht und Schweinemast. Landwirtschaftsverlag GmbH, Munster-Hiltrup, Germany **5:** 28-31.

Hagen, C.D., Lindemann, M.D. and Purser, K.W. (2000). Effect of dietary chromium tripicolinate on productivity of sows under commercial conditions. J. Swine. Health. Prod. **8**: 59-63.

Heinonen, M., Oravainen, J., Orro, T., Seppä-Lassila, L., Ala-Kurikka, E., Virolainen, J., Tast, A. and Peltoniemi, O.A.T. (2006). Lameness and fertility of sows and gilts in randomly selected loose-housed herds in Finland. Vet. Rec. **159**:383–387.

Hill, M.A. (1992). Skeletal system and feet. In: Leman, A.D., Straw, B.E., Mengeling, W.L., D'Allaire, S. and Taylor, D.J. (eds). *Diseases of Swine*. 7th ed. Iowa State University Press, Ames, Iowa, pp.163–195.

Holm, B., Bakken, M., Vangen, O. and Rekaya, R. (2004). Genetic analysis of litter size, parturition length, and birth assistance requirements in primiparous sows using a joint linear-threshold animal model. J. Anim. Sci. **82**:2528–2533.

Holmgren, N., Eliasson-Selling, L. and Lundeheim, N. (1998). Claw and leg injuries in group housed dry sows. Proceedings of the 16th International Pig Veterinary Society Congress, Melbourne, Australia. pp. 352.

Hughes, P. E. and Varley, M. A. (2003). Lifetime performance of the sow. In J. Wiseman., M. A. Varley and B. Kemp, eds. Perspectives in Pig Science. Nottingham University Press, Nottingham. pp. 333-355.

Hultgren, J., Manske, T. And Bergsten, C. (2004). Associations of sole ulcer at claw trimming with reproductive performance, udder health, milk yield, and culling in Swedish dairy cattle. Prev. Vet. Med. 62: 233–251.

Jensen, A.H. (1979). The effects of environmental factors, floor design and materials on performance and on foot and limb disorders in growing and adult pigs. Proc. Pig. Vet. Soc. **5**:85-94.

Johnson, R.W. (1997). Inhibition of growth by pro-inflammatory cytokines: an integrated view. J. Anim. Sci. **75**: 1244-1255.

Jorgensen, B. (2000). Longevity of breeding sows in relation to leg weakness symptoms at six months of age. Acta. Vet. Scand. **41**: 105-121.

Jorgensen, B. (2003). Influence of floor type and stocking density on leg weakness, osteochondrosis and claw disorders in slaughter pigs. Anim. Sci. 77:439–449.

Karg, H. and Bilkei, G. (2002). Causes of sow mortality in Hungarian indoor and outdoor pig production units. Berliner-und-Munchener-Tierarztliche-Wochenschrif **115**: 366-368.

Kempson, S.A. and Logue, D.N. (1993). Ultrastructural observations of hoof horn from dairy cows: changes in the white line during the first lactation. Vet. Rec. **132**:524–527.

Kirk, R.K., Svensmark, B., Ellegaard, L.P. and Jensen, H.E. (2005). Locomotive disorders associated with sow mortality in Danish pig herds. J. Vet. Med. A. **52**:423–428.

Kirkwood, R. N., Baidoo, S. K., Aherne, F. X. and Sather, A. P. (1987). The influence of feeding level during lactation on the occurrence and endocrinology of the post-weaning estrus in sows. Can. J. Anim. Sci. **67**: 405-415.

Koketsu, Y. (2000). Retrospective analysis of trends and production factors associated with sow mortality on swine breeding farms in USA. Prev. Vet. Med. **46**: 249–256.

Koketsu, Y., Dial, G. D., Pettigrew, J. E. and King, V. L. (1996). Feed intake pattern during lactation and subsequent reproductive performance of sows. J. Anim. Sci. **74**:2875-2884.

Kroneman, A., Vellenga, L., Van der Wilt, F.J. and Vermeer, H.M. (1993a). Review of health problems in group-housed sows, with special emphasis on lameness. Vet. Quart. **15**: 26-29.

Kroneman, A., Vellenga, L., van der Wilt, F.J. and Vermeer, H.M. (1993b). Field research on veterinary problems in group-housed sows—a survey of lameness. Zentralbl Veterinarmed. A. **40**:704–712.

Lee, L.A., Ferguson, J.D. and Galligan, D.T. (1989). Effect of disease on days open assessed by survival analysis. J. Dairy. Sci. **72**:1020–1026.

Logue, D.N., Lawson, A., Roberts, D. and Hunter, E.A. (1989). The effect of two different protein sources in the diet upon the incidence and prevalence of lameness in dairy cattle. Anim. Prod. **48**:636.

Lucia, T., Corrêa, M.N., Deschamps, J.C., Bianchi, I., Donin, M.A., Machado, A.C., Meincke, W. and Matheus, J.E.M. (2002). Risk factors for stillbirths in two swine farms in the south of Brazil. Prev. Vet. Med. **53**:285–292.

Lucia, T., Dial, G. D. and Marsh, W. E. (2000). Lifetime reproductive performance in female pigs having distinct reasons for removal. Livest. Prod. Sci. **63**: 213-222.

Lucia, T., Dial, G. D. Marsh, W. E. (1996). Patterns of female removal. II. Longevity and lifetime performance for females with different reasons for removal. Proceedings of the 14th International Pig Veterinary Society Congress, Bologna, Italy, pp.541.

Mahan, D. (2005). Feeding the sow and piglet to achieve maximum antioxidant and immunity protection. [Book chapter] *Re-defining mineral nutrition. Nottingham University Press, Nottingham, UK:* 63-73.

Manske, T. (2002). Hoof Lesions and Lameness in Swedish Dairy Cattle. Prevalence, risk factors, effects of claw trimming, and consequences for productivity. Doctoral thesis Swedish University of Agricultural Sciences, Skara.

Morris, J.R., Hurnik, J.F., Friendship, R.M. and Evans, N.M. (1998). The effect of the Hurnik-Morris (HM) system on sow reproduction, attrition, and longevity. J Anim Sci. **76**: 2759-2762.

Morrow, M. and Meyer, M. (2002). Which pigs should you euthanize and when? <u>http://www.thepigsite.com/articles/6/production-and-mgmt/738/which-pigs-should-you-euthanize-and-when accessed on 2/1/2007</u>.

Mote, B.E., Mabry, J.W., Stalder, K.J. and Rothschild, M.F. (2009). Evaluation of current reasons for removal of sows from commercial farms. Professional Animal Scientist **25**: 1-7.

Mulling, C., Bragulla, H., Reese, S., Budras, K.D. and Steinberg, W. (1999). How structures in bovine hoof epidermis are influenced by nutritional factors. Anat. Hist. Embryol. **28**:103–108.

Nocek, J.E., Johnson, A.B. and Socha, M.T. (2000). Digital characteristics in commercial dairy herds fed metal-specific amino acid complexes. J. Dairy. Sci. **83**:1553-1572.

Oldham, J.G. (1985). Clinical measurement of pain, distress and discomfort in pigs Proc. Br Vet Assoc Anim Welfare Found 2nd Symp. pp. 89–91.

Paterson, R., Cargill, C. and Pointon, A. (1997). Lameness in breeding stock. Proceedings. Pig Prod A. T. Reid Course Vet, C247–C300.

Penny, R.H.C. (1980). Locomotor dysfunction causing reproductive failure. In: Morrow, D.A. (ed). *Current Therapy in Theriogenology*. W. B. Saunders Company Philadelphia, pp. 1042-1045.

Penny, R.H.C., Osborne, A.D. and Wright, A.I. (1963). The causes and incidence of lameness in store and adult pigs. Vet. Rec. **75**:1225–1235.

Peters, J.C. and Mahan, D.C. (2008). Effects of dietary organic and inorganic trace mineral levels on sow reproductive performances and daily mineral intakes over six parities. J. Anim. Sci. **86**:2247-2260.

PigCHAMP. (2006). http://www.pigchampinc.com

Potzsch, C.J., Collis, V.J., Blowey, R.W., Packington, A.J. and Green, L.E. (2003). The impact of parity and duration of biotin supplementation on white line disease lameness in dairy cattle. J. Dairy. Sci. **86**: 2577-2582.

Ritter, L.A., Xue, J., Dial, G.D., Morrison, R. B. and Maesh, W.E. (1999). Prevalence of lesions and body condition scores among female swine at slaughter. J. Am. Vet. Med. Assoc. **214**: 525–528.

Rodriguez-Zas, S. L., Southey, B. R., Knox, R. V., Connor, J. F., Lowe, J. F. and Roskamp, B. J. (2003). Bioeconomic evaluation of sow longevity and profitability. J. Anim. Sci. **81**: 2915–2922.

Rompala, R. E. and Halley, J.T. (1995). Explaining the absorption of chelated trace minerals: the Trojan horse of nutrition. Feed Management. **46**:52.

Rowles, C. (2001). Sow Lameness. J. Swine. Health. Prod. 9: 130-131.

Saballo, A. J., Lopez-Ortega, A. and Marquez, A. A. (2007). Causes of discarding pigs in farms of central western region of Venezuela during the 1996-2002 period. Zootecnia Tropical. **25**: 179-187.

Sanz, M., Roberts, J, Almond, G., Alvarez, R., Donovan, T. and Perfumo, C. (2002). What we see with sow mortality. Proc. Allen D. Leman Swine Conference., St Paul, Minnesota **29**: 181-184.

SAS Inst, Inc. (2003). SAS version 9.1, 2003. SAS Inst, Inc.Cary, NC.

Serenius, T. and Stalder, K.J. (2004). Genetics of length of productive life and lifetime prolificacy in the Finnish Landrace and Large White populations. J. Anim. Sci. **82**:3111–3117.

Shuman, R.D. and Ross, R.F. (1975). Streptoccosis. In: Dunn, H. and Leman, A. (eds). *Diseases of Swine*. 4th ed. Iowa State University Press, Ames, Iowa, pp. 630–631.

Simmins, P.H. and Brooks, P.H. (1988). Supplementary biotin for sows: Effect on claw integrity. Vet. Rec. **122**:431-435.

Smart, M. and Cymbaluk, N. F. (1997). Role of nutritional supplements in bovine lameness in *Lameness in Cattle*. 3rd ed. In: Greenough, P. R. and Weaver, A. D. (eds). W. B. Sanders Co., Philadelphia, PA.

Stalder, K. J., Knauer, M., Baas, T. J., Rothschild, M. F. and Mabry, J. W. (2004). Sow longevity. Pig News Inf. **25**: 53N-74N.

Stein, T. E., Dijkhuizen, A., D'Allaire, S. and Morris, R. S. (1990). Sow culling and mortality in commercial swine breeding herds. Prev. Vet. Med. **9**: 85-94.

Stone, M. W. (1981). Sow culling survey in Alberta. Can. Vet. J. 22: 363.

Svendsen, J., Olsson, A.C. and Svendsen, L. (1992). Group housing system for sows. 3. The effect on health and reproduction. A literature review. Swed. J. Agric. Res. **22**:171–180.

Tarrés, J., Bidanel, J.P., Hofer, A. and Ducrocq, V. (2006). Analysis of longevity and exterior traits on Large White sows in Switzerland. J. Anim. Sci. **84**:2914–2924.

Tengerdy, R. P. (1986). Nutrition, immunity and disease resistance. In Proc 6th Int Conf Prod Dis in Farm Anim, Sept. 1986, Belfast, Northern Ireland, 175-182.

Tiranti, K., Hanson, J., Deen, J. and Morrison, B. (2003). Description of removal patterns in a selected sample of sow herds. Proc. Allen D. Leman Swine Conference, St Paul, Minnesota **30**: 194–198.

Tomlinson, D. J. Mulling, C. H. and Fakler, T. M. (2004). Formation of keratins in the bovine claw: roles of hormones, minerals, and vitamins in functional claw integrity. J. Dairy. Sci. **87**: 797-809.

Tubbs, R.C. (1988). Lameness in sows: Solving a preventable problem. Vet. Med. **83**:610–616.

USDA. Swine 2006, part I: reference of swine health and management practices in the United States, 2006. Fort Collins, Colo: USDA, APHIS, Veterinary Services, Centers for Epidemiology and Animal Health, 2007.

Vitfoss (2009). Lameness costs up to 100 Euro per sow. http://www.vitfoss.com/NR/rdonlyres/044102D0-33A0-448C-AB44-1EB11C518CD3/0/FebruaryVitOmicweb.pdf accessed on August 21, 2009.

Webb, N.G. (1984). Compressive stresses on, and the strength of, the inner and outer digits of pig's feet and the implications for injury and floor design. J. Agric. Eng. Res. **30**:71–80.

Xue, J. L., Dial, G. D. and Bahnson, P. (1996). An evaluation of productivity of commercial swine: lifetime production efficiency. In: Proceedings of the 14th International Pig Veterinary Society Congress, Bologna, Italy, pp.546.

Xue, J. L., Dial, G. D., Marsh, W. E. and Lucia, T. (1997). Association between lactation length and sow reproductive performance and longevity. J. Am. Vet. Med. Assoc. **210**: 935-938.

Yazdi, M.H., Rydhmer, L., Ringmar-Cederberg, E., Lundeheim, N. and Johansson, K. (2000).Genetic study of longevity in Swedish Landrace sows. Livest. Prod. Sci. **63**:255–264.

Appendix 1

Lesion scoring form

	SOW ID	DATE			
		Lateral	Medial		
LEGS:	Location	Score	Score		
Right-					
Hind	HE				
	OH				
	HSJ				
	WL				
	CWH				
	CWV				
	ET				
	DC				
	Sole				
Left-Hind	HE			HE	Heel Erosion
	OH			OH	Overgrown Heel
	HSJ			HSJ	Heel/Sole Junction
	WL			WL	White Line
	CWH			CWH	Claw Wall Horizontal
	CWV			CWV	Claw Wall Vertical
	ET			ET	Elongated Toes
	DC			DC	Dew Claw
	Sole			Sole	
Right-					
Front	HE				
	OH				
	HSJ				
	WL				
	CWH				
	CWV				
	ET				
	DC				
	Sole				
Left-Front	HE				
	OH				
	HSJ				
	WL				
	CWH				
	CWV				

112

ET	
DC	
Sole	