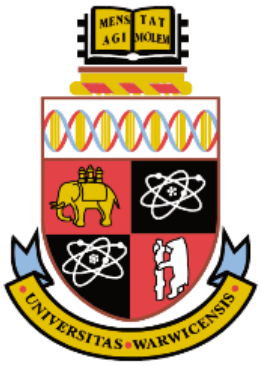
Limb health in pigs:

The prevalence and risk factors for lameness, limb lesions and claw lesions in pigs, and the influence of gilt nutrition on indicators of limb health

Amy Jean Quinn

MSc, BSc

Thesis submitted in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Veterinary Epidemiology



University of Warwick

School of Life Sciences

August 2014

Table of contents

List of tables

List of figures

Summary

This thesis examined the prevalence and risk factors for lameness, limb lesions and claw lesions in pigs, and the influence of gilt nutrition on indicators of limb health through a cross-sectional survey and two cohort studies. A cross-sectional survey of 68 integrated pig farms in Ireland on lameness, limb and claw lesions of 2948 piglets, 3368 weaners, 544 lactating sows, 1289 finishers, 525 replacement gilts, 518 pregnant gilts and 604 pregnant sows was conducted. The prevalence of foot lesions, limb lesions and lameness was determined for each appropriate group and data relating to environmental and management parameters were also collected to identify risk factors. There was a high prevalence of lameness in finishers, gilts and sows. Lameness prevalence is higher in group gestation housing systems than in gestation stalls. Slat void width and the frequency of pen washing increased the risk of lameness in finisher pigs. Floor type, particularly the floor material used influenced both limb and foot lesions. Two cohort studies were conducted to investigate the effect of three dietary regimes for replacement gilts on lameness, areal bone mineral density (aBMD), behaviour, limb, claw and joint lesions and carcass traits. In the first, a diet specifically formulated for developing gilts and fed restrictively from 70kg until 2 weeks before the gilts approximate weight at first service, reduced lameness, joint lesion prevalence and claw unevenness when compared to the two most commonly practiced feeding regimes for developing gilts. In the second, a diet specifically formulated for developing gilts fed *ad-libitum* from 65 kg reduced lameness and increased aBMD when compared to the two most commonly practiced feeding regimes for developing gilts. In conclusion, this study provides valuable information on lameness, foot and limb lesion prevalence and risk factors as well as providing information on nutritional strategies that could help to address the current high levels of lameness in replacement gilts.

Declaration

This thesis is submitted to the University of Warwick in support of my application for the degree of Doctor of Philosophy. It has been composed by myself and has not been submitted in any previous application for any degree. The work presented (including data generated and data analysis) was carried out by the author.

The following papers have been submitted for publication:

Quinn, A., Green, L., Lawlor, P., Boyle, L. (2014). The effect of feeding a diet formulated for developing gilts between 70kg and service at ~140 kg on lameness indicators and carcass traits. *Livest. Sci.*

Quinn, A., Boyle, L., KilBride, A., Green, L. (2014). A cross-sectional study on the prevalence and risk factors for limb and foot lesions in piglets on commercial farms in Ireland. *Prev. Vet Med.*

The following papers were presented at conferences:

Quinn, A., Green, L. and Boyle, L. (2011). A comparison of bone mineral density (BMD) measurements between thawed and frozen pig feet by dual-energy x-ray absorptiometry (DXA). In: *The University of Warwick Life Sciences Postgraduate Symposium,* Coventry, England, 26th – 27th May 2011, p 29.

Quinn, A., Boyle, L., Lawlor, P., Green, L. (2012). A study of the effect of a developer diet on behaviour, locomotory ability, claw lesions, limb lesions and bone mineral density in replacement gilts. In: *The University of Warwick Life Sciences Postgraduate Symposium*, Coventry, England, 26th – 28th March 2012, p 46.

Quinn, A., Green, L. and Boyle, L. (2012). The effect of feeding a developer diet to replacement gilts on lameness, claw lesions, bone mineral density and post-mortem joint lesions. In: *The Association for Veterinary Teaching and Research Work, Regional meeting,* Trim, Co. Meath, Ireland, 5th October 2012, p 20.

Boyle, L.A., Calderon-Diaz, J.A., and Quinn, A. (2012). Moving to group housing in 2013: Implications for lameness and sow welfare. In: *The 39th Pig Health Society Symposium,* Mullingar, Co. Meath, 22nd May 2012, pp 61-62.

Quinn, A. (2012). Feeding a gilt diet will improve sow longevity and productivity. In: *The Irish Pig Farmers' Conference,* Horse and Jockey, Co. Tipperary and Cavan, Ireland, 23rd and 24th of October 2012, pp 17-24.

Quinn, A., Green, L., Lawlor, P. and Boyle, L. (2013). The effect of three dietary regimes on lameness indicators in replacement gilts. In: *The Agricultural Research Forum*, Tullamore, Co. Offaly, Ireland, 12th March 2013, p 111.

Quinn, A., Green, L., Lawlor, P. and Boyle, L. (2013). Lameness in pigs: Prevalence, risk factors and possible prevention. In: *The University of Warwick Life Sciences Postgraduate Symposium*, Coventry, England, 18th – 20th March 2013, p 104.

Quinn, A., Green, L., Lawlor, P., Boyle and L. Boyle (2013). Lameness in pigs: Prevalence, risk factors and possible prevention. In: *Zinpro Feet First™ seminar 2013*, Horse and Jockey, Co. Tipperary, Ireland, 2nd May 2013, pp 4-11.

Quinn, A., Green, L., Lawlor, P., Boyle and L. Boyle (2013). The effect of feeding a developer, gestating sow and finisher diet to group housed gilts, in

terms of locomotory ability and lying behaviour. In: *The 47th Congress of the International Society for Applied Ethology*, Florianopolis, Brazil, 2nd – 6th June 2013, p 67.

Quinn, A., Green, L., Lawlor, P., Boyle and L. Boyle (2013). The effect of dietary regime on locomotory ability, claw lesions, limb lesions, body lesions and post-mortem joint lesions of developing gilts. In: *The 15th International Conference on Production Diseases in Farm Animals, Uppsala, Sweden*, 24th - 28th June 2013, p 90.

Boyle, L.A., Calderón Díaz, J.A., Quinn, A., Lawlor, P.G., KilBride, A., Green, L. (2013). Addressing lameness in group housed sows. In: *64th Annual Meeting of the European Federation of Animal Science*, Nantes, France, 26th-30th August 2013, p 475,

Boyle, L., Quinn, A., and Calderon Diaz, J (2013). Lameness in pigs. In: *The Irish Pig Farmers' Conference,* Horse and Jockey, Co. Tipperary and Cavan, Ireland, 22nd & 23rd October 2013, pp 47-50.

Quinn, A., Boyle, L., KilBride, A. and Green, L. (2014). A cross-sectional study on the prevalence and risk factors for limb and foot lesions in piglets on Irish farms. In: *The 65th Annual Meeting of the European Federation of Animal Science*, Copenhagen, Demark, 25th-29th August 2014.

Quinn, A., Boyle, L., KilBride, A. and Green, L. (2014). Prevalence and risk factors for limb and foot lesions in piglets. In: *The 6th international Conference on the assessment of Animal Welfare at Farm and Group Level*, Clermont-Ferrand, France, 3rd – 5th September 2014.

Contents of this thesis contributed to the following publications:

Boyle, L., Quinn, A., Calderon-Diaz, J., Kilbride, A., Green, L. and Fahey, A. (2012). Overcoming Lameness in Irish Sows. T Research, **7**; 36-37. (http://www.teagasc.ie/publications/2012/1527/TResearch-Autumn.pdf)

Quinn, A.J., Calderon Diaz, J.A., Boyle, L.A., (2013). Lameness in Pigs. Teagasc Research Dissemination Report, pp. 1-30. (http://www.teagasc.ie/publications/2013/2815/LamenessInPigs.pdf)

Boyle, L., Carroll, C., McCutchen, G., Clarke, S., McKeon, M., Lawlor, P., Ryan, T., Ryan, T., Fitzgerald, T, Quinn, A., Calderon-Diaz, J., Lemos Teixeira, D. (2012). Towards January 2013- Updates implications and options for group housing pregnant sows. Teagasc, IFA and IAPP publication, pp1-78. (http://www.teagasc.ie/publications/2012/1291/Towards_January2013_Updates_implicationsand_optionsfor_grouphousing_pregnantsows.pdf)

Acknowledgements

During this PhD I have been extremely lucky to have the guidance and wisdom of two great supervisors; Dr. Laura Boyle and Prof. Laura Green. Dr. Laura Boyle has always had an open door and nothing was ever a problem, I am extremely grateful for all the great opportunities she has given me. I have learnt so much from Prof. Laura Green throughout my PhD and despite the fact I was based in Ireland she never seemed that far away with an answer to every question and an encouraging word. I would like to acknowledge Dr. Amy KilBride for her invaluable statistical advice and Dr. Peadar Lawlor for his valued nutritional expertise. I would like to acknowledge the Teagasc Walsh fellowship scheme for my project funding.

I would like to acknowledge the staff of the Pig Development Department particularly Tomás, Henry and John and to all the placement students (Hazel, Aishling, Amelie, Caroline, Walter, Jenn, Padraig, Flora, Eric, Romain) for their care of experimental animals and assistance in data collection. I would also like to acknowledge the staff of Dawn Pork and Bacon for their assistance in data collection at the slaughtering facility. I would like to thank the pig specialists; Ciarán, Michael, Ger and Seamas, for their assistance throughout the farm survey, their help was invaluable. A massive thank you to all the farmers who participated in the farm survey, I met so many wonderful people and learned so much more than I could have anticipated. A special thank you to my head of department, Ciarán Carroll, for his support particularly over the past year, which has allowed me to undertake both my job and thesis. Throughout the course of this PhD I was given the opportunity to make some wonderful friends, and I would like to particularly thank Julia for her friendship and knowledge.

I would like to express my thanks to my brothers and sisters; Sarah, Tommy, Susie and Jack for all their support over the last few years, I really appreciate it and

consider myself extremely lucky to have you all. I also would like to thank Fergal for his support, endless encouragement and his wonderful ability to keep me calm just when I need it, I couldn't have asked for more. I am extremely lucky to have a wonderful mam and dad who I would like to thank a million times over for their love and support throughout this PhD and always, I really couldn't have done this without them. I finally wish to thank my very special granny May, for all of her encouragement and love that she given me, although she passed away before I completed this thesis I have comfort in the knowledge that she is with me always, sitting on my shoulder whenever I need her.

List of abbreviations

Abbreviation	Meaning
aBMD	Areal bone mineral density
AP	Anconeal process
ADG	Average daily gain
ADFI	Average daily feed intake
CI	Confidence interval
d	Day
DEV	Gilt developer dietary treatment
DXA	Dual X-ray absorptiometry
ESF	Electronic sow feeder
FIN	Finisher pig dietary treatment
GES	Gestating sow dietary treatment
FI	Feed intake
HC	Humeral condyle
IQR	Interquartile range
LS	Least square
OCD	Osteochondrosis
OR	Odds ratio
SE	Standard error

Chapter 1

General introduction

1.1 Background

The domesticated pigs in commercial pig farming are descendants of the Eurasian wild boar (*Sus scrofa*). Domestication of the wild boar occurred approximately 9000 years before present (Mills *et al.*, 2010). Substantial intensification of the pig industry occurred from the 1950's altering; farm productivity, pig physiology and animal welfare (Arey and Brooke, 2006; Mills *et al.*, 2010; Prunier *et al.*, 2010). Pigs are now the most abundant meat producing mammals worldwide with in excess of a billion pigs produced per year, with pork the highest per capita meat type consumed within the EU (Arey and Brooke, 2006; European Comission, 2013). While intensification had the positive effect of reducing production costs, labour requirement and improved farm biosecurity, it was accompanied by a heightened requirement for on-farm welfare monitoring due to the associated environmental and physiological challenges (Mills *et al.*, 2010; Prunier *et al.*, 2010).

Current commercial housing differs substantially from the extensive natural woodland habitat of the wild boar (Arey and Brooke, 2006; Mills *et al.*, 2010). The housing systems in operation vary between stage of production and governing legislation. The majority of intensive farms house pigs indoors, with slatted floors and no bedding (Prunier *et al.*, 2010). Weaners and finishers are entirely group housed, lactating sows are largely stalled within a farrowing crate, while gestating sows are either group or stalled housed. In relation to gestation housing in Europe, the EC Directive 2008/120/EC required that in all 25 member states, pregnant sows and gilts are group housed from 4 weeks after service to 1 week before farrowing from January 2013. Gestating sows are still housed in stalls in several major pig producing countries such as the USA, South America and Canada.

Genetic selection on growth traits has been a major factor contributing to the substantial increase in pig production levels in recent decades (Rauw *et al.*, 1998; Prunier *et al.*, 2010). Pigs were selectively bred for fast growth rates, high lean meat deposition and high feed conversion efficiency in slaughter pigs and for larger litters, reduced breeding intervals and increased weight gain in breeding pigs (Arey and Brooke, 2006; Prunier *et al.*, 2010). These genetic alterations increased the physiological demands on the body of the pig as well as altering its skeletal structure and the weight bearing capacity of the joints (Prunier *et al.*, 2010). A sow currently produces 25 pigs per year, in comparison to the 6 pigs produced per year by the ancestral wild boar, while the average daily gain in fattening pigs has increased in the past 20 years from 670g to 844g per day (Kirk *et al.*, 2005; Arey and Brooke, 2006; PigSys, 2013).

The high physiological demand in combination with close confinement in an unnatural and barren environment have resulted in health and welfare consequences such as an increases in leg weaknesses, injuries, development of stereotypies and increased disease susceptibility (Rushen, 1984; Arey and Brooke, 2006; Marchant Forde, 2008; Prunier *et al.*, 2010).

1.2 Limb health

Compromised limb health often results in an alteration to natural locomotion which may present as lameness which is a major cause of premature culling in all age categories of pigs, with both welfare and economic concern (Dewey *et al.*, 1993; Kirk *et al.*, 2005; Arnbjerg, 2007; Mustonen *et al.*, 2011). Disorders of the locomotory system were responsible for 16% of premature culling in weaner and finisher pigs and 11% in sows (Stein *et al.*, 1990; Baumann and Bilkei, 2002).

1.2.1 Lameness

Lameness in pigs is a major health problem on commercial pig farms (Deen *et al.*, 2007). It presents as an abnormal gait as a result of physical injury or infection in

the limbs or back (Velarde and Geers, 2007). Issues relating to limb pathology have previously been linked with lameness; these include infectious arthritis and osteochondrosis (Jensen *et al.*, 2007). Physical injury such as claw lesions, joint lesions, muscle damage, tendon damage and bone fractures have also been linked with lameness (Jensen and Toft, 2009).

The welfare of the pig is reduced because lameness is associated with pain and discomfort, it also affects a pigs ability to interact with its environment and ability to access resources (Dewey *et al.*, 1993; Anil *et al.*, 2002; Kirk *et al.*, 2005; Jensen *et al.*, 2007; Mustonen *et al.*, 2011). From an economic point of view, lameness results in a higher work load, increased veterinary expenses and an increase in the involuntary culling rate thus, a reduction in farm productivity (Dewey *et al.*, 1993; Jensen *et al.*, 2007; Anil *et al.*, 2009; Mustonen *et al.*, 2011; Pluym *et al.*, 2011).

1.2.1.1 Lameness in piglets

It is difficult to determine lameness levels in piglets due to their high activity levels resulting in a dearth of information on the prevalence of lameness in this age category (Gillman *et al.*, 2008). It has been reported however that 9.8% of piglets were treated for lameness with the main causes being arthritis, splayleg and injury (Straw *et al.*, 2006; Zoric, 2008). In the case of arthritis, bacteria often enter the blood stream at the site of skin and foot abrasions (Straw *et al.*, 2006; Zoric, 2008). Teeth clipping, castration and tail docking, all of which are performed on piglets at a very young age, are linked to arthritis and lameness in piglets (Nielsen *et al.*, 1975; Smith and Mitchell, 1976). Splayleg results in severe limb abduction and an inability to walk due to underdeveloped muscle fibres of the limb adductors (Straw *et al.*, 2006). It generally affects the hind limbs, affects 0.4% of new born piglets and results in 50% mortality in affected animals due to starvation and crushing (Ward, 1978; Straw *et al.*, 2006). Its presence is associated with gender, breed, induction and slippy floor surfaces (Sellier and Ollivier, 1982; Breuer *et al.*, 2005; Straw *et al.*, 2006; Papatsiros, 2012). The occurrence of painful injuries to the limbs also results

in lameness; they are more commonly caused by floor type, crushing and birth defects.

1.2.1.2 Lameness in weaner and finisher pigs

The prevalence of lameness in weaner and finisher pigs varies widely (2-20%) between studies (Petersen *et al.*, 2008; KilBride *et al.*, 2009a). Lameness is the third most common cause for treatment with antibiotics in weaner and finishing pigs and frequently results in involuntary culling (Christensen *et al.*, 1994). Euthanasia of a severely lame pig results in the loss of carcass value and imposes carcass disposal costs in addition to the actual euthanisation cost (Pluym *et al.*, 2011). In weaners and finishers lameness can also result in indirect effects such as a reduced feed intake (FI) which ultimately leads to a reduction in daily weight gain and so, a longer period to target slaughter weight (Anil *et al.*, 2008).

1.2.1.3 Lameness in gilts and sows

Lameness is a major cause of premature culling in the breeding herd, particularly in gilts and young sows (Dewey *et al.*, 1993; Boyle *et al.*, 1998; Anil *et al.*, 2009). Between 15 and 20% of gilts and first and second parity sows are culled due to lameness (D'Allaire *et al.*, 1987; Lucia *et al.*, 2000). As a sow does not become profitable until after she has had her third litter this represents a major economic concern for pig producers (Stalder *et al.*, 2000).

The prevalence of lameness in pregnant sows in Europe was reported as, 5% in England, 6 to 10% in Belgium and 9% in Finland in group housing systems (Heinonen *et al.*, 2006; Pluym *et al.*, 2011; Pluym *et al.*, 2013b; Willgert *et al.*, 2014). In England, KilBride *et al.* (2009a) observed a high prevalence of lameness in pregnant sows group housed on partially slatted flooring (45%). They also documented 12% abnormal gait in replacement gilts in a variety of housing systems, with a prevalence of 18.9% in gilts housed only on fully or partially slatted floors with no bedding. This figure included sows with lower (i.e. milder) scores indicative of abnormal gait. They argued that while it is impossible to determine whether

abnormal gait results in pain it does represent a biological cost to the animal because of the increased strain placed on the locomotive system. For this reason they took the view that abnormal gait is an indication of reduced quality of life and considered that lower scores should be included in the lameness prevalence.

Inflammation as a result of lameness increases metabolic activity and can alter nutrient utilisation in the body which can result in energy required for growth and litter development being diverted to the energy requirements of the immune response system (Ossent, 2010; Wilson *et al.*, 2010; Wilson and Ward, 2012). Cytokines released as a result of inflammation can result in reproductive hormone disruption and subsequently poor reproductive performance (Reichlin, 1999; Wilson *et al.*, 2009; Wilson *et al.*, 2010; Wilson and Ward, 2012). Cytokine release also results in hypersomnia and activates matrix metalloproteinase, resulting in connective tissue degradation, exacerbating lameness problems (Johnson, 1997; Reichlin, 1999; Mülling and Greenough, 2006). Reduced lactation feed intake as a result of inflammation stimulated anorexia is also associated with lameness and can have a negative impact on body condition and on further reproductive performance (Johnson, 1997; Lucia *et al.*, 2000; Anil *et al.*, 2009; Wilson *et al.*, 2010).

Lameness reduces the productivity of a farm by reducing sow longevity, increasing the involuntary culling rate of sows, increasing expenses as a result of the cost incurred in replacing sows and reducing the number of pigs produced per sow per year thus, reducing the numbers of finisher pigs reaching the factory (Dewey *et al.*, 1993; Grandjot, 2007; Anil *et al.*, 2008;2009; Wilson *et al.*, 2009; Pluym *et al.*, 2011). The latter occurs because an increase in the rate of sow culling results in a decrease in the average age of the herd and younger animals produce smaller litters (Dewey *et al.*, 1993; Wilson *et al.*, 2009).

1.2.2 Claw lesions

Claw lesions are a common injury in all categories of pigs and in some cases they may result in pain and lameness (Pluym *et al.*, 2011). Lesions commonly affect

various parts of the foot depending on the causal factor of the lesion (Figure 1.1). The outer heel is the portion of the foot that bears the most weight, the more it bears the thicker the horn of the sole becomes (Ossent, 2010). Increased pressure on the heel due to increased weight bearing results in haemorrhaging and inflammation followed by hyperkeratinisation and thus, heel overgrowth and increasing susceptibility to heel erosion (Ossent, 2010). Heel overgrowth or erosion are the most common claw lesions with the hind legs being most commonly and severely affected (Van Amstel, 2010; Pluym *et al.*, 2011). Heel overgrowth development is thought to be related to abnormal leg conformation or posture (Ossent, 2010). The junction of the heel and the sole of the foot (Figure 1.1) is vulnerable to separation particularly when heel overgrowth is present (Ossent, 2010; Van Amstel, 2010). The white line of the foot occurs at the joining between the claw wall and the heel of the foot (Ossent, 2010) (Figure 1.1). The horn of the white line is soft and therefore vulnerable to damage thus, injury may occur due to separation at the junction (Ossent, 2010; Van Amstel, 2010). Several causes of white line separation are known; overgrown heels, nutrition, laminitis and aggression (Ossent, 2010). Cracks may also occur in the claw wall as a result of trauma, with claws being particularly more susceptible when toes or dew claws are overgrown (Ossent, 2010; Van Amstel, 2010). Both the toes and the dew claws may become overgrown, it is thought this is an inflammatory response of increased metabolism and claw horn growth (Ossent, 2010). Its presence makes the claw more vulnerable to other claw injuries such as while line damage, wall cracks, claw breakages and amputations (Ossent, 2010; Van Amstel, 2010; Pluym *et al.*, 2011).

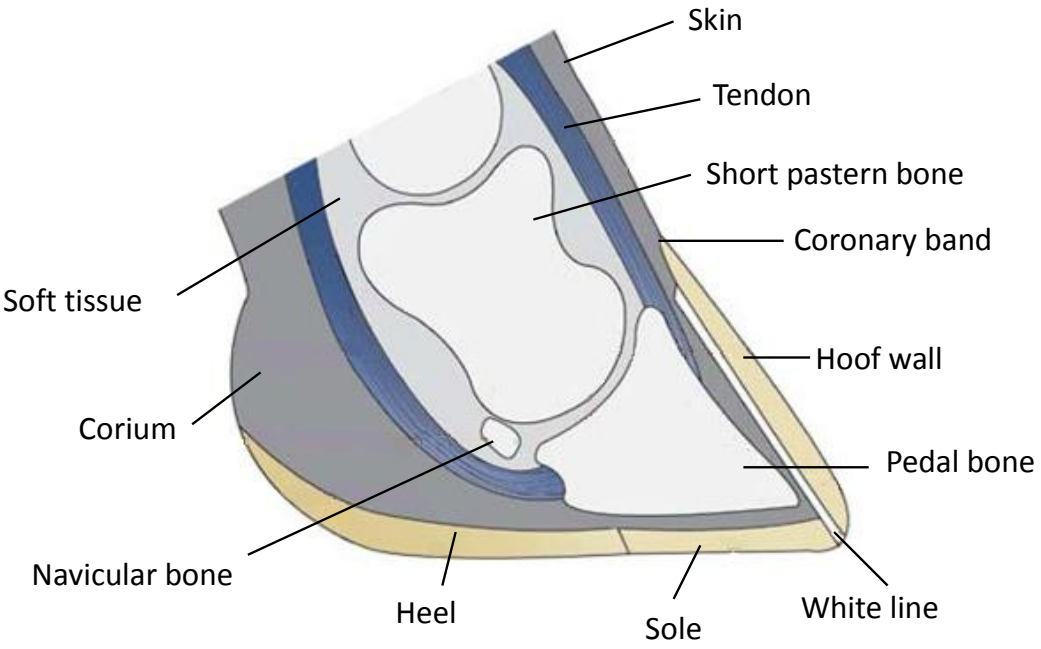


Figure 1.1. A cross-sectional view of pig claw (Figure adapted from http://www.zinpro.com/lameness/swine/)

The relationship between lameness and claw lesions may be dependent on the location and seriousness of the lesion, as some areas of the claw are more sensitive than others (Deen *et al.*, 2007). The corium is dense with nerves and blood vessels and is therefore a highly vulnerable and sensitive area of the foot (Brennan and Aherne, 1987; Stokka *et al.*, 1997; Van Amstel, 2010). Hence minor claw lesions which do not penetrate the corium may not result in pain and are therefore not associated with lameness (Anil *et al.*, 2007; Deen *et al.*, 2007). Claw lesions which penetrate the corium result in pain; they also allow for the entry of bacteria and may result in subsequent infection (Penny *et al.*, 1971; Gjein and Larssen, 1995; Mouttotou and Green, 1999b; Straw *et al.*, 2006). Van Amstel (2010) reported infections associated with the claw increase the risk of lameness. Infection may lead to heightened pain, inflammation, osteomyelitis with subsequent bone dissolution and tenosynovitis (Brennan and Aherne, 1987; Deen *et al.*, 2007; KilBride *et al.*, 2009b; Pluym *et al.*, 2011). Common injuries to the claw which penetrate the corium include damage to the coronary band region, white line disease and dew claw amputation (Deen *et al.*, 2007; KilBride *et al.*, 2009b; Pluym *et al.*, 2011). White

line separation has a strong association with lameness as it is prone to penetrating the corium (Anil *et al.*, 2007; Deen *et al.*, 2007). Dew claw amputations are extremely painful lesions as the corium becomes exposed and so may result in lameness (Pluym *et al.*, 2011). Additionally, the corium is responsible for oxygen and nutrient supply for hoof horn production and maintenance, disruption to the blood flow by inflammation can impair horn production and result in further lesions such as heel erosion, wall cracks and white line damage (Shearer *et al.*, 1996; Van Amstel, 2011).

1.2.2.1 Claw lesions in piglets

Piglets may develop several injuries to their feet during the suckling period. The susceptibility of the limbs and feet to injury is due to the vulnerability of the limb tissue at birth and this susceptibility to injury then diminishes over time (Smith and Mitchell, 1976; Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b). The sole of the foot of the piglet is affected by two main lesions, bruising and erosion, with bruising prevalence varying from 50 to 100% and an erosion incidence as high as 100% (Mouttotou and Green, 1999b; Mouttotou *et al.*, 1999c; Boyle *et al.*, 2000; KilBride *et al.*, 2009b). The coronary band (Figure 1.1) of the piglet is vulnerable to damage and infection (KilBride *et al.*, 2009b). Apparently superficial coronary band lesions can result in osteomyelitis and subsequent pedal bone dissolution, resulting in long term irreparable damage to the foot (KilBride *et al.*, 2009b).

1.2.2.2 Claw lesions in weaner and finisher pigs

About 50% of weaners and 94% of finishers have at least one claw lesion present (Mouttotou *et al.*, 1997; Mouttotou *et al.*, 1999a). Claw lesions are responsible for 14% of premature culling in weaner and finisher pigs (Baumann and Bilkei, 2002). Sole bruising, heel flaps and sole erosion are the most prevalent lesions in weaner pigs (Mouttotou *et al.*, 1999d). Sole erosion, toe erosion and white line damage are the most prevalent lesion in finisher pigs (Mouttotou *et al.*, 1999a). In a cross-sectional study of weaner and finisher pigs in 90 indoor and outdoor pig farms in England, Gillman *et al.* (2009) reported an overall lesion prevalence of 39.6% with

sole bruising (7.1%), sole erosion (10.8%), heel flaps (8.4%) and toe erosion (11.6%) the most prevalent lesions recorded.

1.2.2.3 Claw lesions in gilts and sows

Similar claw lesions have been recorded in gilts and sows where lesion prevalence can be as high as 99% (Pluym *et al.*, 2011). In a study of cull sows, Dewey *et al.* (1993)reported that claw lesions were responsible for between 5 and 20% of sows culled for lameness. The most commonly occurring claw lesions in sows are wall cracks, lesions to the heel and white line separation (Gjein and Larssen, 1994). Wall crack prevalence ranges from 5 to 80% (KilBride, 2008; Pluym *et al.*, 2011; Pluym *et al.*, 2013b), while heel overgrowth and white line lesions have a prevalence of 93% and 70% respectively (Pluym *et al.*, 2011; Pluym *et al.*, 2013b)

1.2.3 Limb lesions

Lesions to the limb are commonly occurring injuries in pigs at all stages in the production cycle. Their severity and location determine their influence on limb health. Lesion type varies considerably from calluses and alopecia to abscesses and swellings (Gillman *et al.*, 2008; KilBride *et al.*, 2008; KilBride *et al.*, 2009a). Lesions such as wounds, swellings, calluses, capped hock and bursitis are associated with lameness (Smith, 1988; Bonde *et al.*, 2004; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013). The association is not fully understood as painful limb injuries may result in lameness or lameness may cause animals to spend longer lying thereby, increasing contact time between the skin and the flooring and in turn increasing damage to the skin (Bonde *et al.*, 2004; KilBride et al 2009a; Calderon Diaz *et al.*, 2013). Calderón Díaz *et al.* (2013) reported an increased risk of lameness when wounds and severe lesions such as ulcers were present.

1.2.3.1 Limb lesions in piglets

The limbs of piglets become affected by abrasions and alopecia within the first few hours of life (Straw *et al.*, 2006). The prevalence of limb abrasions ranges between

36 and 70% with a prevalence of alopecia of 61% (Penny *et al.*, 1971; Svendsen *et al.*, 1979; Furniss *et al.*, 1986; Mouttotou *et al.*, 1999c; Boyle *et al.*, 2000; Straw *et al.*, 2006; KilBride *et al.*, 2009b; Zoric *et al.*, 2009).

1.2.3.2 Limb lesions in weaner and finisher pigs

Calluses, bursitis, capped hocks, abrasions and alopecia are the most commonly recorded limb lesions in weaner and finisher pigs (Mouttotou *et al.*, 1998; Mouttotou and Green, 1999a; Cagienard *et al.*, 2005; Gillman *et al.*, 2008; KilBride *et al.*, 2008). Adventitious bursitis is a fluid filled sac of the subcutaneous connective tissue of the limb and is referred to as capped hock when located on the tarsal joint, (Mouttotou *et al.*, 1998;1999b; Gillman *et al.*, 2008). KilBride (2008) reported a prevalence of calluses of 45.5% in weaner and finisher pigs in England. Gillman *et al.* (2008) and KilBride *et al.* (2008) reported a prevalence of bursitis and capped hock of 40.6% and 17.2% respectively in weaner and finisher pigs while Mouttotou *et al.* (1999b) reported a combined bursitis and capped hock prevalence of 63%. The prevalence of lesions such as calluses, alopecia, swellings and abscesses to the limbs has not been established.

1.2.3.3 Limb lesions in gilts and sows

Frequently documented limb lesions in sows include; calluses, bursitis, capped hock, swellings and wounds (KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013). KilBride *et al.* (2009a) reported a wide range of lesion prevalence of calluses (30-80%), bursitis (32-37%) and capped hock (32-57%) between replacement gilts, pregnant gilts, pregnant sows and lactating sows.

1.2.4 Osteochondrosis

Osteochondrosis has previously been identified as a cause of leg weakness in pigs and a contributor to premature culling (Dewey *et al.*, 1993; van Grevenhof *et al.*, 2011), other studies however have failed to find an association with lameness (Brennan and Aherne, 1986; Jørgensen, 1995; Jørgensen *et al.*, 1995; Stern *et al.*,

1995; Arnbjerg, 2007). It is defined as a non-infectious disease of the joint surface; it results in irreversible deterioration of articular cartilage quality and underlying bone (Kirk *et al.*, 2008; Busch and Wachmann, 2011; van Grevenhof *et al.*, 2011). This is due to disturbance to the endochondral ossification of the articular cartilage when the bones are still developing (Kirk *et al.*, 2008; Busch and Wachmann, 2011; van Grevenhof *et al.*, 2011). The articular cartilage has the essential role of reducing joint friction during movement and transferring the mechanical load to the underlying bone (van Riet *et al.*, 2013). Deterioration causes increased pressure on the surface of an affected joint in affected animals, resulting in inflammation and pain in some cases (Ytrehus *et al.*, 2007). The presence of osteochondrosis in the elbow joint of pigs results in postural changes, stiffness and lameness (Jørgensen and Sørensen, 1998; Kirk *et al.*, 2008; Jensen and Toft, 2009). Its presence has been associated with a reduction in sow longevity (Yazdi *et al.*, 2000).

It is most common and severe in the articular cartilage of the humeral condyle and anconeal process of elbow joint of pigs (Figure 1.2) and tends to be bilaterally symmetrical in the limbs (Jørgensen, 1995; Ytrehus *et al.*, 2007; Kirk *et al.*, 2008; Jensen and Toft, 2009). It affects more males than females; which may be as a result of differential growth rates between the sexes or hormonal differences (Grondalen, 1974; Nakano *et al.*, 1979). Osteochondrosis has a heritability of between 0.2-0.5 and purebred landrace pigs have a higher occurrence of osteochondrosis in comparison to other breeds (Grondalen and Vangen, 1974; Stern *et al.*, 1995; Jorgensen and Andersen, 2000; Jensen and Toft, 2009).

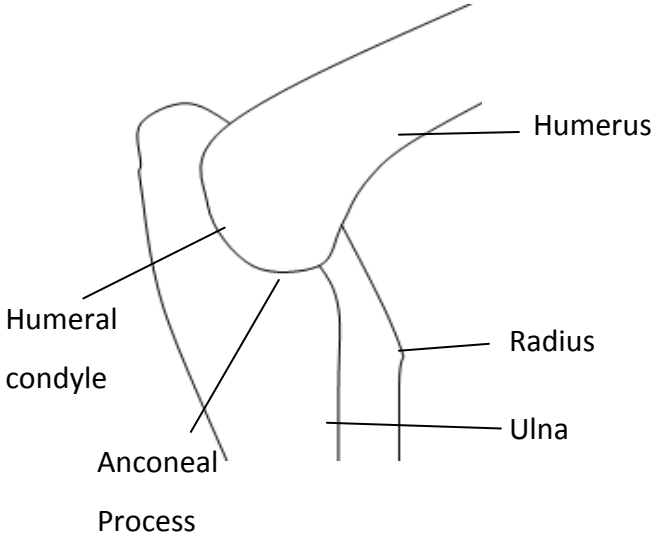


Figure 1.2. Diagram of the pig elbow joint

There is a dearth of information available on the prevalence of osteochondrosis in piglets and weaners. However lesions indicative of osteochondrosis were found in the joints of pigs as young as 15 days (Hill *et al.*, 1990). Nakano *et al.* (1979) reported a 100% prevalence of lesions associated with osteochondrosis in finisher pigs with Busch and Wachmann (2011) reporting 53% prevalence in the elbow joint of finisher pigs. Osteochondrosis is present in 84-95% of sows (Kirk *et al.*, 2008). Previous studies attributed osteochondrosis as the cause of 34% of culling's for lameness and 14% of mortality and cullings in sows (Dewey *et al.*, 1993; Engblom *et al.*, 2008). The majority of studies supporting the link between osteochondrosis and lameness are based on sows culled for lameness (D'Allaire *et al.*, 1987; Dewey *et al.*, 1993; Kirk *et al.*, 2005; Engblom *et al.*, 2007; Engblom *et al.*, 2008; Jensen *et al.*, 2010). Other studies have found no association between osteochondrosis and lameness/leg weakness (Brennan and Aherne, 1986; Jørgensen, 1995; Stern *et al.*, 1995; Arnbjerg, 2007). Hence the relationship is poorly understood (Dewey *et al.*, 1993; Heinonen *et al.*, 2006).

1.2.5 Bone mineral density

Severe trauma to the limb can result in bone fractures (Engblom *et al.*, 2008). Bone fractures are the most painful condition with the highest association with pain

(Jensen *et al.*, 2012). Fractures are associated with 16% of sow mortality (Kirk *et al.*, 2005). The strength of the bone determines the vulnerability to fractures and one of the major elements of this is bone mineral density (Ammann and Rizzoli, 2003; Mills *et al.*, 2010). Dual x-ray absorptiometry is the most common method of measuring bone mineral density and is widely used in pigs, which determines areal bone mineral density (aBMD), a two-dimensional measurement of bone minerals of a given area (Mitchell *et al.*, 1995; Mitchell *et al.*, 1998; Lorenz-Depiereux *et al.*, 2004; Nielsen *et al.*, 2004; Nielsen *et al.*, 2007; Ryan *et al.*, 2011). Calcium (Ca) and phosphorous (P) are essential minerals to bone strength, while age, production stage and mechanical stress are also influencing factors (Hall *et al.*, 1991; Mills *et al.*, 2010).

1.2.6 Active/inactive behaviours

Monitoring the frequency and duration of inactive behaviours may be used to indicate substandard health or environmental conditions (Velarde and Geers, 2007). Inactive behaviour may reflect limb health and pain levels as lame sows spend more time lying down than non-lame sows (Bonde *et al.*, 2004; Velarde and Geers, 2007; Valros *et al.*, 2009; Calderón Díaz and Boyle, 2014). Pigs with limb injuries such as sole erosion and limb abrasions spend less time in active behaviours (standing, walking, fighting, and playing) (Mouttotou and Green, 1999b; Valros *et al.*, 2009).

1.3 Factors influencing limb health and lameness

A number of parameters relating to the environment and management influence limb health, although the association is often multifactorial in nature. Such factors include; floor type, housing system, stocking density, group size, growth rate and nutrition.

1.3.1 Floor type

The floor conditions for pigs have a major impact on foot and limb health in relation to lameness, claw lesions, limb lesions and osteochondrosis. Features of the floor

that influence limb health include proportion of solid, fully slatted, partially slatted and fully solid flooring, flooring material (soil, concrete, metal, plastic, rubber), bedding provision/quantity and cleanliness.

Slatted flooring is the predominant floor type used in commercial pig farms as it allows excreta to pass through the voids, maintaining a cleaner environment and has a lower maintenance requirement (Marchant Forde, 2008). However slatted flooring is a major risk factor for lameness and claw lesions as the gap between the slats, the edge and the rough surface may provide an area of potential claw harm (Boon and Wray, 1989; Heinonen *et al.*, 2006; Velarde and Geers, 2007; Jensen and Toft, 2009). KilBride *et al.* (2009a) reported an increased risk of lameness in sows on slatted flooring when compared to outdoor soil systems and pens with fully solid concrete floors with bedding, no such effect was reported in replacement and pregnant gilts. The use of fully or partially slatted flooring is coupled with an increase in lameness, which might be due to an increase in claw lesions (Gjein and Larssen, 1994; KilBride *et al.*, 2009a). KilBride *et al.* (2009a) reported abnormal gait in 16.9%, 34.3% and 22.3% of finishers housed on partially slatted, fully slatted and fully solid flooring with sparse bedding respectively when lameness was defined as abnormal gait. The use of fully solid flooring has been associated with an increase in bursitis and capped hock prevalence weaners and finishers (Gillman *et al.*, 2008; KilBride *et al.*, 2008). A higher prevalence of sole erosion with partially slatted floors with little or no bedding than solid concrete pens with bedding (KilBride *et al.*, 2009b). In piglets, there is also an increased risk of sole bruising and joint swellings with slatted flooring in comparison to solid concrete bedded floors however, the opposite can be said for skin abrasions (KilBride *et al.*, 2009b).

The width of the slat and void and slat quality also impact limb health as slats that are too narrow do not provide sufficient support for the hoof structure (Fritschen, 1979; Baxter, 1984; Gjein and Larssen, 1994; Straw *et al.*, 2006; Marchant Forde, 2008). It is recommended that the slat void is no more than half the width of the sole of the foot and the solid area of the slat should be no narrower than the width of the foot (Baxter, 1984). The European Commission has outlined minimum slat

widths and maximum void widths for each category of pig to ensure suitability of floor type to each age class (Table 1.1).

Table 1.1 Slat width requirements as in EC Directive 2008/120/EC.

	Maximum slat void width (mm)	Minimum slat width (mm)
Piglets	11	50
Weaners	14	50
Finishers	18	80
Gilts (post service)	20	80
Sows (post service)	20	80

The effect of floor material on lameness, claw lesions and limb lesions has been widely investigated (Newton *et al.*, 1980; Gjein and Larssen, 1994; McKee and Dumelow, 1995; Andersen and Bøe, 1999; KilBride *et al.*, 2008; Tuyttens *et al.*, 2008; Gillman *et al.*, 2009; KilBride *et al.*, 2009a; KilBride *et al.*, 2009b; Elmore *et al.*, 2010; Calderón Díaz *et al.*, 2013; Calderón Díaz *et al.*, 2014; Westin *et al.*, 2014). Highly abrasive flooring can result in claw damage while floors with low abrasive properties can result in overgrown claws and increased slipping (Newton *et al.*, 1980; McKee and Dumelow, 1995).

A lower incidence of limb abrasions, sole bruising and sole erosion in piglets, and capped hock and bursitis in weaner and finisher pigs has been associated with outdoor production systems on soil compared with indoor housing systems (Gillman *et al.*, 2008; KilBride *et al.*, 2008; KilBride *et al.*, 2009b). A reduction in piglet limb abrasion is associated with plastic slatted floors compared with pens with rubber mats, solid concrete, metal slats and worn floors (Gravås, 1979; Furniss *et al.*, 1986; Mouttotou *et al.*, 1999c; Lewis *et al.*, 2005; KilBride *et al.*, 2009b; Zoric *et al.*, 2009). The prevalence of sole bruising is higher in piglets kept in pens with a part concrete and part round wire mesh floor, part concrete and part metal floors and exposed aggregate floors than in entirely concrete pens (Mouttotou *et al.*, 1999c). Concrete slats are associated with an increased risk of capped hock than metal or plastic slats,

an increased risk of bursitis than plastic slats and an increased risk of limb callus than metal stats in finisher pigs (KilBride *et al.*, 2008).

Several studies have investigated the use of rubber mats for sows as a means to improve pig health, through the assessment of its effect on lameness, claw and body lesions (Mouttotou *et al.*, 1999c; Tuyttens *et al.*, 2008; KilBride *et al.*, 2009b; Elmore *et al.*, 2010; Calderón Díaz *et al.*, 2013; Calderón Díaz *et al.*, 2014). Boyle *et al.* (2000) reported an increase in sow comfort and reduced slipping in farrowing crates when rubber mats were compared to metal slatted flooring. However they also reported an increase in piglet skin abrasions with the use of rubber mats. The use of rubber mats during gestation as opposed to concrete slats was found to reduce lameness levels but increased the risk of some claw lesions (overgrown claws, white line damage, heel/sole cracks and claw wall cracks) while reducing the risk of limb lesions (swellings and wounds) for pregnant sows (Calderón Díaz *et al.*, 2013).

The provision of bedding such as straw is known to reduce lameness, claw lesions and limb lesions in sows and growing pigs (Kroneman *et al.*, 1993; Andersen and Bøe, 1999; Tuyttens, 2005; KilBride, 2008). While the use of straw in farrowing crates reduces skin abrasion and sole erosion (Westin *et al.*, 2014). Contradictory studies have also found bedding is associated with reduced and increased prevalence of osteochondrosis in growing pigs (van Grevenhof *et al.*, 2011; de Koning *et al.*, 2014). The provision of bedding reduced body lesion prevalence in sows (Boyle *et al.*, 2002). The use of dirty straw is, however, associated with an increase in claw lesions due to weakening of the claw horn with increasing water content (Kroneman *et al.*, 1993; Tuyttens, 2005). However, despite some of its benefits, in intensive production systems, the use of straw is scarcely due to the liquid manure systems and predominant slatted floored pens as well as the increased cost and labour requirement (Tuyttens, 2005; Scott *et al.*, 2006).

Floor hygiene influences claw lesions because the presence of excess urine and manure on the floor surface can also contribute to claw lesions as they can weaken

claw hardness, increasing vulnerability to claw damage (Kroneman *et al.*, 1993; Carvalho *et al.*, 2009). Excess urine and manure may also increase slipping which can lead to injury such as limb and foot abrasions, bruising and bone fractures (McKee and Dumelow, 1995).

1.3.2 Group size and stocking density

There is high inter-farm variation in group size for both growing pigs and breeding sows. For breeding sows the group size is often determined by the housing type in operation, with large group sizes common in Electronic Sow Feeding (ESF) systems, but smaller group sizes are associated with floor feeding systems for example. The European Commission provides minimum space requirements (Table 1.2), depending on body weight for growing pigs from birth to slaughter and depending on group size in pregnant gilts and sows. Group size influences limb health as some studies found that pigs kept in larger groups tend to have higher limb lesion and lameness scores and fighting on slatted flooring results in more severe lesions to the claw and limbs (Gjein and Larssen, 1994;1995; Street and Gonyou, 2008). Jørgensen (2002) found higher percentages of pigs with abnormal gait and posture at higher stocking densities. In addition, the prevalence of osteochondrosis in finishers was reduced by providing a higher space allowance (van Grevenhof *et al.*, 2011). KilBride *et al.* (2009a) however reported that more pigs exhibited abnormal gait in loosely stocked pens in comparison to tightly stocked pens. The adverse effects in high stocking densities may increase particularly later in the finisher stage due to the increase in body size (Street and Gonyou, 2008).

Table 1.2 Minimum space requirements for weaners and finishers in EC Directive 2008/120/EC.

Live weight (kg)	m^2
≤10	0.15
11-20	0.20
21-30	0.30
31-50	0.40
51-85	0.55
86110	0.65
111+	1.00

Table 1.3 Minimum space requirements for gilts and sows in EC Directive 2008/120/EC.

Group size		m^2/gilt	m^2/sow
1-5	Minimum total floor area	1.81	2.5
	Minimum continuous solid area (i.e. lying area)	0.95	1.3
6-39	Minimum total floor area	1.64	2.25
	Minimum continuous solid area (i.e. lying area)	0.95	1.3
40+	Minimum total floor area	1.48	2.025
	Minimum continuous solid area (i.e. lying area)	0.95	1.3

1.3.3 Housing system

Pregnant sows can be either group housed in pens or individually housed in gestation stalls. In the European Union all member states were required to group house sows from 4 weeks after service until one week before farrowing from January 2013. This was expected to impact upon limb health both positively and negatively as a result of freedom of movement thus, the associated interaction with the pen environment and with social counterparts. Positively, the use of group housing allows for the expression of more natural behaviours as unrestricted movement facilitates social interactions and given the right environmental conditions allows the sow to make decisions regarding where to lie in order to

control her thermal and physical comfort (Gjein and Larssen, 1995; Pajor, 2002). Group housing also allows for a substantial increase in exercise with an associated increase in bone strength and muscle mass (Marchant Forde and Broom, 1996; Pajor, 2002). It also results in lower superficial joint damage and resting heart rate (Sather and Fredeen, 1978; Marchant Forde *et al.*, 1997). From a negative standpoint however individual stalls allow for individual feeding, ease of individual management and reduced physical aggression (Barnett *et al.*, 2000; Pajor, 2002).

One of the major disadvantages with group housing is the increased aggression between sows (Gjein and Larssen, 1995). The mixing of unfamiliar pigs results in an increase in aggressive behaviour as the animals attempt to establish a dominance hierarchy (Pajor, 2002). This aggression often results in injury, particularly an increase in claw lesions, body lesions and lameness (Gjein and Larssen, 1995). Normally, aggression decreases once the hierarchy has been established (Meese and Ewbank, 1973). However, in the case of dynamic groups and with certain competitive feeding systems high levels of aggression can persist throughout the production cycle (Andersen and Bøe, 1999; Jensen *et al.*, 2000).

Calderón Díaz *et al.* (2014) reported that group housed sows were more likely to be lame than individually stalled sows therefore lameness levels were expected to increase in the EU with the transition to group housing. Gjein and Larssen (1994) also reported that claw lesions are more prevalent in group housed herds (96% of sows affected) than in herds where sows are confined in gestation stalls (80%), on partially slatted flooring. Calderón Díaz *et al.* (2014) however reported a reduction in white line damage, horizontal wall cracks and dew claw injuries in group housed sows when compared to gestation stalls. Additionally they also reported a reduction in the limb lesions; calluses and bursitis.

1.3.4 Growth rates

Genetic selection in order to maximise growth rates results in negative physiological effects many of which have animal welfare and morbidity implications and consequential economic repercussions (Simonsen, 1993; Prunier *et al.*, 2010). Selection for fast growth is linked with several issues in relation to pig welfare including leg weaknesses, cardiovascular issues, increased muscle mass, skeletal injuries and modification of the release of various hormones as well as behavioural modifications (Grondalen and Vangen, 1974; Marchant Forde and Broom, 1996; EFSA, 2007; Prunier *et al.*, 2010).

Studies have found selection for fast growth rate is associated with postural defects and locomotory disorders in pigs and other species (e.g. broilers) (Jørgensen, 1995; Kestin *et al.*, 2001; Rauw, 2007). Arey and Brooke (2006) predicted that selection for fast growth rate would reduce the welfare of pigs as a result of leg and conformation abnormalities leading to lameness. In a study of Lacombe pigs, leg weakness was significantly more evident in pigs which were selected for higher lean tissue growth rates than in non-selected control pigs (Rauw *et al.*, 1998). A difference between sexes for leg weakness prevalence between the selected and control pigs was also noted (Rauw *et al.*, 1998). Many of these changes are mediated by osteochondritic changes in the cartilaginous structures of fast growing animals (Rauw *et al.*, 1998; Busch and Wachmann, 2011). A linkage between osteochondrosis and pigs selected for fast growth is widely reported. Busch and Wachmann (2011) found that during the finishing period, every 100g increase in daily gain results in an increase of 20% in the risk of exhibiting signs of osteochondrosis. Positive correlations between the presence of osteochondrosis and both growth rate and the percentage of lean tissue are documented in the literature (Lundeheim, 1987; Jorgensen and Andersen, 2000; Jørgensen and Nielsen, 2005; Busch and Wachmann, 2011). The European Food Safety Authority (EFSA, 2007) identified leg disorders, as a consequence of genetic selection, as a serious area of concern and recommended that the welfare implications, due to the prevalence of leg disorders as a result of breeding methods, should be determined.

1.3.5 Nutrition

Nutrition and limb health are inherently linked. Particularly in relation to diet composition, feed intake and nutritional management (Knauer *et al.*, 2007; Bradley, 2010; Crenshaw *et al.*, 2013; van Riet *et al.*, 2013). Nutrition is directly related to claw, bone and cartilage physiology and indirectly affects limb health through the effect of weight gain.

Supplying the correct vitamins and minerals in the correct quantities is essential to limb health in production animals (Mohammadina, 2008). Much work has been carried out on the importance of adequate supplies of various minerals and vitamins for claw health in commercially valuable animals, particularly in cattle with some work investigating this in pigs (Hedges *et al.*, 2001; Tomlinson *et al.*, 2004; Anil, 2011). Keratinisation of the hoof epidermal cells to make horn is reliant upon an adequate supply of minerals, as well as vitamins and trace elements (Tomlinson *et al.*, 2004). Hoof horn of reduced quality is produced if nutrient supply is inadequate, which results in the hoof being more vulnerable to physical, chemical and microbial damage from the surrounding environment (Tomlinson *et al.*, 2004).

Trace minerals such as zinc, manganese and copper are key minerals for hoof health, enzyme system and immune system functioning (Tomlinson *et al.*, 2004; Tomlinson *et al.*, 2008). Zinc is important in relation to horn production and the health of the skin due to its essential role in cellular repair and replacement and thus, the rate of wound healing (Mohammadina, 2008; Tomlinson *et al.*, 2008; van Riet *et al.*, 2013). In poultry, dietary zinc supplementation reduces scratches, cuts and other skin imperfections, while in cattle, zinc deficiency is associated with joint stiffness (Tomlinson *et al.*, 2008). Copper is crucial in the development of antibodies and the replication of lymphocytes (Tomlinson *et al.*, 2004). Copper deficient cows were found to have cracks in the heel and sole abscesses (Puls, 1994; Tomlinson *et al.*, 2008; NRC, 2012; van Riet *et al.*, 2013). Manganese is important for horn production and the formation and maintenance of cartilage and bone in cattle, deficiency is associated with joint stiffness and enlargement, and weak or short bones (Mohammadina, 2008; Petersen *et al.*, 2008; van Riet *et al.*, 2013). The

addition of an organic chelated trace mineral complex of zinc, copper and manganese to the diet of breeding sows has been investigated and heel erosion, heel overgrowth and white line lesions were reduced after supplementation (Anil, 2011). This suggests that such minerals may help to reduce the prevalence of lameness in sows. Ferket *et al.* (2009) also reported a reduction in leg abnormalities due to rapid growth rate after zinc, copper and manganese supplementation.

Vitamins also influence claw health. Biotin is one of the most widely investigated vitamins in relation to claw hardness, playing an important role in the formation of keratinised tissues which include the skin, claw horn and foot pads (Bryant *et al.*, 1980; Campbell *et al.*, 2000; Tomlinson *et al.*, 2004). Hedges *et al.* (2001) found a significant reduction in lameness in cattle after biotin supplementation. Biotin significantly reduced white line separation, vertical cracks and coronary band lesions in cattle (Campbell *et al.*, 2000; Hedges *et al.*, 2001). A reduction in toe lesions, heel cracks and side wall cracks in sows was also observed after biotin supplementation (Bryant *et al.*, 1980). Vitamins A, B and E also have an important role in the process of claw formation and health (Tomlinson *et al.*, 2004). A balance between these various minerals and vitamins are important as interactions can alter their bioavailability (Mohammadina, 2008; Anil, 2011). The addition of various minerals and vitamins improves claw health and might thereby improve sow longevity by meeting some of the nutrient demands of the sow during lactation (Goodband *et al.*, 1993).

Calcium is an essential determining factor of bone strength (Hall *et al.*, 1991). During gestation and lactation calcium requirements increase and calcium reserves in the bones may be used to meet this demand, if a deficiency results, long term bone strength is reduced making sows more vulnerable to bone fractures (Mahan, 1990; Mills *et al.*, 2010). The main nutritional causes of osteochondrosis are vitamin deficiency (Vitamin C, A, D and Biotin), copper deficiency and excess zinc in the diet (Nakano *et al.*, 1987). Research on the effect of dietary energy on osteochondrosis is contradictory (van Riet *et al.*, 2013). Several studies have found restrictive feeding positively affects joint lesions (Goedegebuure *et al.*, 1980; Savage *et al.*, 1993),

while others have reported no influence of dietary restriction (Jørgensen, 1995; Donabédian *et al.*, 2006; van Riet *et al.*, 2013). These studies however were carried out on multiple species (pigs and horses) at different stages of production.

1.3.5.1 Gilt and sow nutrition

Young replacement animals are particularly susceptible to lameness as a result of inadequate nutrition during the developmental phase. In a study by Calderón Díaz *et al.* (2013) it was reported that 39% of replacement gilts were lame on entry to the breeding herd. The majority of producers feed diets formulated for finisher pigs to replacement gilts until service (Boyd *et al.*, 2002). On some farms producers feed diets formulated for pregnant sows to growing gilts from selection at 100kg. Such regimes are nutritionally inadequate for the developing gilt; a gestating sow diet is formulated for a sow that has finished growing and a finisher diet is formulated for fast growth rates, high lean meat deposition and cost efficiency (Harper *et al.*, 2002). In contrast diets specifically formulated for the developing female animal include the nutrient requirements for bone development and fat deposition. High growth rates are linked with several pig welfare issues (Grondalen and Vangen, 1974; Marchant Forde and Broom, 1996; EFSA, 2007; Prunier *et al.*, 2010), and so are counter-productive for growing female breeding animals. Calcium and phosphorus are necessary for the development and maintenance of bone (van Riet *et al.*, 2013). Replacement gilts require higher levels of calcium and phosphorous or bone mineralisation to prevent bone weaknesses and as a backup source of Ca and P for the litter if needed during gestation and lactation (Mahan, 1990; Marchant Forde and Broom, 1996).

1.4 Pig production in Ireland

Ireland is one of the key pig producing countries within the EU with a total pig population of 1,403.600 on 290 pig production sites with a total herd size of 151,100 and an average herd size per farm of 520 sows (Teagasc, 2014, CSO, 2014 (CSO, 2014). Pig production is the third most important agricultural sector in Ireland, behind beef and milk production, accounting for 8% of the gross

agricultural output (GAO) (BordBia, 2014). Approximately 185,000 tonnes of pig meat were exported in 2013 with an estimated value of €525 million, 44% of which was exported to the UK market (Bordbia, 2013). The pig sector employs in excess of 7500 people, 1300 of which are employed directly on farms, with the remainder involved in areas such as transportation, meat processing and pig feed manufacturing (Teagasc 2014). Despite its importance to the national economy, on-farm profitability is volatile due to high production costs as it is estimated that pig feed alone currently makes up 70% of the production costs of pig meat (Teagasc, 2014). Tight margins coupled with the expense associated with compliance with the EU Directive 2001/88/EC in recent years makes it imperative to find ways to improve on farm productivity thus improving profitability. Lameness, limb and claw lesion prevalence has not previously been quantified in Ireland. Identification of the extent of on-farm welfare problems such as lameness and identifying causes and possible methods to improve limb health ultimately reducing lameness levels may improve sow longevity and the number and quality of finisher stock, improving both on-farm welfare and profitability.

1.5 Project sponsor

This project was carried out in conjunction with Teagasc, the Agriculture and Food Development Authority of Ireland. The Teagasc Pig Development Department Advisory Service facilitated the survey by providing farmer contact details from the advisory service database. The Pig Development Department research facilities were used to carry out the cohort study elements of the project. The funding for this project was provided by the Teagasc Walsh fellowship scheme and Teagasc core funding.

1.4 Conclusions

Compromised limb health is a serious concern in commercial pig farms. Determining the prevalence of lameness, limb and claw lesions and identifying the risk factors for

these with possible management methods to reduce such lesions would benefit productivity and pig welfare on commercial pig farms.

1.5 Research objectives

- To determine the prevalence and identify risk factors for limb and foot lesions in piglets and weaners in a cross-sectional study of commercial farms in Ireland.
- To determine the prevalence and identify risk factors for lameness, limb lesions, claw lesions and body lesions in finishers, gilts and sows in a cross-sectional study of commercial farms in Ireland.
- To determine the effect of limit feeding a diet formulated for developing gilts on lameness, limb and claw lesions, bone mineral density, growth performance and carcass traits.
- To determine the effect of *ad-libitum* feeding a diet formulated for developing gilts on lameness, limb and claw lesions, bone mineral density, carcass traits and behaviour.

Chapter 2

A cross-sectional study on the prevalence and risk factors for limb and foot lesions in piglets on commercial farms in Ireland

2.1 Abstract

A cross-sectional survey of 68 integrated Irish pig farms was conducted to determine the prevalence and risk factors for foot and limb lesions in 2948 piglets from 272 litters. One litter was selected per age category 3–7 days (d), 8–14d, 15–21d and 22–28d per farm. All piglets were examined for limb abrasions, limb swellings, sole bruising, sole erosion, foot swellings and coronary band injuries and scored from 0–3 based on relative size. Environmental parameters were recorded for each litter examined. A questionnaire was completed on management, health and performance factors for each farm. The overall prevalence of each lesion was calculated and multilevel mixed effect logistic regression models were used to elucidate risk factors. The prevalence (farm range) of lesions were: sole bruising 61.5% (7-100%), sole erosion 34.1% (0-100%), coronary band injuries 11.3% (0-46%), limb abrasions 55.7% (11-98%), swollen limbs 2.4% (0-11%) and swollen feet 4.4% (0-14%). Age was negatively associated with sole bruising (OR 0.42; CI 0.37, 0.50) and coronary band injury (OR 0.69; CI 0.60, 0.81) and positively associated with limb abrasions (OR 1.54; CI 1.12, 2.14). There was a reduced risk of sole bruising in piglets in pens with plastic slats with oval voids in the piglet area of the pen with a plastic solid area for piglets and metal slats under the crate (OR 0.32; CI 0.15, 0.70) compared with plastic stats throughout and a plastic solid area for piglets. There was an increased risk of sole erosion (OR 1.81; CI 1.07, 3.09) and foot and limb swellings in pigs in pens with metal slats only and a solid plastic area for piglets (OR 1.90; CI 1.01, 3.57) compared with plastic stats throughout and a solid plastic area for piglets. There was an increased risk of coronary band injury in pens with metal slats throughout and a metal solid area for piglets (OR 4.25; CI 1.96,

3.57) compared with plastic stats throughout and a plastic solid area for piglets. We conclude no single floor type was ideal for piglet foot and limb health with all floors influencing different lesions in different ways, however, the positive association between sole erosions, coronary band injury and foot and limb swellings and metal slats suggest that this floor type was most detrimental and most likely to be associated with joint infections that lead to severe health and welfare concerns.

2.2 Introduction

Nursing piglets can develop various injuries to their limbs and feet (KilBride *et al.*, 2009b). Prevalence and severity vary by lesion type and environment (Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b). However foot and limb injuries impair welfare, reduce performance and may cause death, with joint ill or lameness reported as the cause of 2.8% of preweaning mortality (Mouttotou and Green, 1999a; Johansen *et al.*, 2004; Kilbride *et al.*, 2012; Westin *et al.*, 2014).

Limbs are affected by skin abrasions, alopecia and swellings. Abrasions are removal of the epidermis with either an open wound or healing scab present (Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b). They predominantly affect the carpal joint bilaterally and are caused by friction between limb and floor during feeding (Lewis et al., 2005). Abrasions develop within hours of birth and their incidence typically reduces with age (Straw *et al.*, 2006; KilBride *et al.*, 2009b; Zoric *et al.*, 2009). Alopecia can arise from mild abrasions to the skin or from scar tissue which is non-hairy and typically reflects healed abrasions (Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b).

Lesions to the foot include sole bruising and sole erosion, coronary band injuries and swellings. Sole bruising is haemorrhaging into the solar corium (Mouttotou and Green, 1999a; KilBride *et al.*, 2009b). It is most prevalent in the first week of life when the epidermis is very thin (Zoric, 2008; KilBride *et al.*, 2009b). Erosion arises when the sole epidermis is removed (Mouttotou and Green, 1999a; KilBride *et al.*, 2009b). Both lesions are associated with a reduction in suckling and active

behaviour and a slower growth rate; probably because of the pain associated with such injuries (Mouttotou and Green, 1999a). Little is known about coronary band injuries but it is thought to arise when the toe or, in very young piglets, the entire claw, wedges in the slat void leading to pressure and necrosis (KilBride *et al.*, 2009b).

As abrasions, sole erosion and injury to the coronary band penetrate the epidermis they provide an entry site for pathogens that can lead to secondary infection in the tarsal, carpal, carpophalangeal, or digital joints, which results in swelling (inflammation) in the joints of the limbs and feet (Penny *et al.*, 1971; Zoric *et al.*, 2004; KilBride *et al.*, 2009b). Such bacterial infections can cause osteomyelitis, arthritis, endocarditis, or meningitis (Penny *et al.*, 1971; Mouttotou and Green, 1999a; Zoric *et al.*, 2004; Straw *et al.*, 2006; KilBride *et al.*, 2009b). KilBride *et al.* (2009b) found a relatively low prevalence (5.5%) of swellings but their effect on piglet welfare and performance is severe and so even low prevalence is of concern (Kilbride *et al.*, 2012).

In general a lower incidence of foot and limb injuries is associated with outdoor systems and indoor systems with straw bedding (Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b; Zoric *et al.*, 2009). Indoors, in the absence of bedding, plastic floors (Furniss *et al.*, 1986; Lewis *et al.*, 2005) and rubber covered floors are also associated with a lower incidence of lesions (Gravås, 1979). KilBride *et al.* (2009b) investigated the prevalence and risk factors for foot and limb lesions in piglets in England in a number of types of production system including outdoor and indoor systems with straw and indoor systems without bedding. However, in most other countries outside of the UK production systems are more homogenous and intensive; in 95% of EU farms sows farrow in crates whilst the equivalent figure in the UK is 70% (BPEX, 2004; Johnson and Marchant-Forde, 2009). To date, there has been no large scale study of piglet foot and limb injuries in indoor, predominately slatted systems without the use of straw, as used across the EU. Hence, the aim of the current study was to investigate the prevalence and risk factors for foot and

limb lesions in piglets reared in commercial intensive systems in Ireland which may additionally provide valuable information for the main EU pig producing countries.

2.3 Methods

2.3.1 Farm selection and sample size

Data were collected as part of a cross-sectional survey examining the prevalence and risk factors for lameness, foot and limb lesions in pigs at all stages of the production cycle on Irish farms (Chapter 2, 3 and 4). Sample size was estimated at 59 farms based on an expected overall prevalence of foot and limb lesions of 95%, a population size of 297 integrated (produced pigs from 'farrow to finish', >100 sows on a single production site) pig farms in Ireland, 95% confidence interval and precision of 5% using Win Episcope 2.0. A total of 68 integrated pig farms were sampled between March 2011 and December 2012. No farms in Northern Ireland were sampled. Farms were selected from a database containing information on c. 98 farms which were availing of the Teagasc advisory service with a mean herd size of 654 sows. The database consisted of farmers who sought advice from trained agricultural advisors in 2009 and 2010. Farms within the database were divided into geographical regions. The numbers of farms to be sampled per region was determined based on farm density within a region. Farms were then randomly selected by assigning a number to each farm and using a random number generator to identify a farm for selection. Farms were contacted by phone and were invited to participate in the survey.

2.3.2 Training and biosecurity

All farms were visited by one trained researcher (Amy Quinn [AQ]) and 1 to 2 research assistants. All animal based measures were scored by the same person (AQ). The main observer (AQ) was trained by an experienced researcher (Laura Boyle [LB]) over 28d. Training involved repeated scoring until over 90% repeatability was reached. A total of 7 research assistants also participated in data recording.

Training was conducted with each assistant by AQ, which included a detailed review of the protocols, demonstrations and practice data collection sessions.

A maximum of 2-3 farms were visited each week depending on the biosecurity practices of the farms. The majority of farmers required the research team to have been away from pigs for 24 - 72 hours prior to visiting their farm. Disposable equipment (e.g. overalls, masks earplugs, gloves) was used where possible and all other equipment (clip boards, torch, measuring tape, electronic distance measure) was thoroughly cleaned and disinfected between each farm visit.

2.3.3 Measurements

A tour of the farm was provided by a member of the farm staff to familiarise the research team with the farm layout. On each farm, four litters were selected for examination, one from each of the following age groups; 3–7 days (d), 8–14d, 14d, 15–21d and 22–28d and every piglet in each selected litter was examined. All pens of each age category on the farm were counted and assigned a number; a random number generator was then used to determine the pens to be examined. Piglets were lifted for examination of their limbs and feet (Appendix 1). Lesion definitions can be found in Appendix 2.

2.3.3.1 Limb lesions

All four limbs were examined for abrasions, alopecia and swellings and scored as per KilBride *et al.* (2009b) (Appendix 3). Abrasions and alopecia were scored from 0–3 as follows: 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the size of the nearest joint on the affected limb. Limb and foot swellings were scored from 0–3 as follows: 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the size of the opposing unaffected joint or foot, if joint or foot were bilaterally affected a pig of the same size was used for comparison.

2.3.3.2 Foot lesions

All four feet were examined for swellings, sole bruising and sole erosion and scored as per KilBride *et al.* (2009b) (Appendix 3). Sole bruising and erosion were scored from 0–3 as follows: 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the heel affected. Coronary band damage was defined as disruption to the epidermis at the coronary band presenting as an open or healing wound and it was scored as follows: 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the total coronary band area affected by a lesion. These scoring systems took variation in the piglets size into account as the score of the lesion was relative to the size of the pig.

2.3.3.3 Environmental parameters

Environmental measurements were recorded in the pens in which selected litters were housed (Table 2.1). A detailed diagram of each pen was also drawn indicating the location of resources in each pen (Appendix 1).

Table 2.1 Environmental measurements for piglets.

Pen measurements		Option
Area	Pen	m^2
	Fully slatted	m^2
	Fully solid	m^2
	Crate	m^2
	Heat pad	m^2
	Feeder	m^2
Wall composition	Material	Concrete, plastic, metal, wood, other
	Structure	Fully solid, part solid +50%, part solid <50%
Flooring	Structure	Solid, fully slatted, partially slatted
	Material	Concrete, plastic, metal, other
	Flooring dimensions	Slat void width, length, inter-void area, shape, profile, edge profile, surface texture)
Slopes and steps		Present, absent
Feeders	Sow feeder	Present, absent
	Piglet feeder	Present, absent
Drinker	Piglet drinker	Present, absent
Supplementary heat	Heat pads	Present, absent
	Heat lamps	Present, absent
Bedding		Present, absent

2.3.3.4 Management parameters

A questionnaire was completed on each farm with the farm manager (Appendix 1). It comprised of 147 questions on the following topics; animal management routines, breeding policy and genetics, farm performance records, hygiene practices, nutrition and pig health.

2.3.4 Data analysis

Databases were created in Microsoft Access 2003 to store data, to maintain data accuracy all data was inputted by AQ. Each farm was assigned a random number for

database identification and no identifiable information was stored in the database in order to ensure data security. Data were checked for outliers prior to analysis. Outliers were checked against the raw data and impossible values were removed.

2.3.4.1 Calculation of prevalence of lesions

The prevalence of foot and limb lesions was calculated by maximum severity score and by presence / absence of each lesion per pig. The following formula was used to calculate prevalence of the lesions:

$$\frac{\text{No. piglets with lesion score} \geq 1}{\text{No. piglets examined on all farms}} \times 100$$

The herd prevalence was also calculated to determine the range of lesions between farms. The following formula was used to calculate herd lesion prevalence to determine the range between herds:

$$\frac{\text{No. piglets on farm with lesion score} \geq 1}{\text{No. piglets examined on the farm}} \times 100$$

Chi-squared statistics were used to test differences in prevalence and Pearson's correlation coefficient was used to examine correlations between lesions.

2.3.4.2 Descriptive analysis of flooring environments

Slat shape and flooring material used for the slatted and solid areas were correlated. As a consequence floor characteristics were grouped into 6 categories. Descriptions and the distribution of the floor characteristics are shown in Tables 2.2 and 2.3. Foot and limb swellings were combined for regression analysis because of the low number of animals affected (70 and 129 respectively). The solid area of a pen was determined by adding the area of the heat pad, if present, and any other solid area (e.g. piglet solid area) within the pen. The farrowing pen was coded into three areas: sow lying area, piglet slatted area, piglet solid area (Figure 2.1).

Table 2.2 The six categories of pen floors for piglets.

Floor type category	Material used		
	Piglet area slat	Sow area slat	Solid piglet area
Plastic slat, solid plastic	Plastic	Plastic	Plastic
Plastic (oval) and metal slats, solid plastic	Plastic	Metal	Plastic
Plastic (rectangular) and metal slats, solid plastic	Plastic	Metal	Plastic
Metal slat, solid plastic	Metal	Metal	Plastic
Metal slat, metal solid	Metal	Metal	Metal
Other slats, solid concrete	Metal, concrete, plastic	Metal, concrete, plastic	Concrete

Table 2.3 Number (n) and percentage (%) of piglets by type of floor.

Floor type	n	%
Plastic slat, solid plastic	675	22.9
Plastic (oval) and metal slats, solid plastic	283	9.6
Plastic (rectangular) and metal slats, solid plastic	545	18.5
Metal slat, solid plastic	548	18.6
Metal slat, metal solid	320	10.9
Other slats, solid concrete	577	19.6

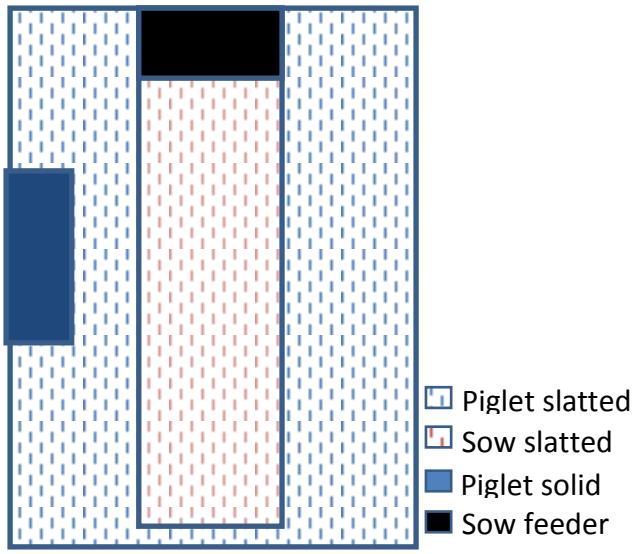


Figure 2.1 Farrowing crate "Typical design" based on mean dimensions

2.3.4.3 Risk factors

Data were analysed using MlwiN 2.27 (Rasbash *et al.*, 2012). Multilevel mixed effect logistic regression was used to allow for pigs clustered in litters within farms and therefore a two level random effect model was used. All continuous variables were transformed to categorical variables and checked for linearity, if a linear association was found the continuous variable was used, otherwise the categorical version was used. Predictor variables were screened in the univariable analysis and those with a significance of $P<0.2$ were used to develop the multivariable model. They remained in the final model if they were significant at $P\leq0.05$. If variables were highly correlated the variable that made the most biological sense was left in the model. Then to check for residual confounding all variables not in the model, including those with $p>0.2$ in the univariable analysis were retested in the model. The following model was used:

$$\text{Logit}(p_{ijk}) = \beta_0 + \Sigma\beta x_{ijk} + \Sigma\beta x_{jk} + \Sigma\beta x_k + v_k + u_{jk}$$

p_{ij} = the proportion of the litter that were affected (score ≥1) with the lesion being investigated, Logit= logit link function, β0= constant, βx = vector of fixed effects varying at level 1 (ijk), level 2 (jk), or level 3 (k), i =Piglet, j=pens (i.e. litter), K= farms, v_k= level 3 residual variance, u_{jk}= the level 2 residual variance. When the prevalence

of lesions was low or high (<5% or >95%) logistic regression was not carried out. For each model the predicted and observed data were combined and sorted in ascending sequence according to the predicted data set. The predicted and observed data were then divided into 6 categories and summed and these values were visually assessed with model fit acceptable for all models.

2.4 Results

2.4.1 Farm features

A total of 2948 piglets were examined from 272 litters (3-7d = 757; 8–14d = 753; 15–21d = 727 and 22–28d = 711 piglets). The overall mean litter size was 10.9 (SD ±1.5), with a mean litter size per age category of 3-7d = 11.1 (SD ±1.4); 8–14d = 11.1 (SD ±1.4); 15–21d = 11.0 (SD ±1.4) and 22–28d=10.5 (SD ±1.7) piglets. All piglets were housed indoors in farrowing pens consisting of a farrowing crate and a piglet area on a variety of floor types, bedding was not provided in any form on any of the farms. The mean pen area was 4.0 m^2 (±0.3) with an mean slat width of 11 mm (±3) and void width of 10 mm (±1.5) in the piglet area and a slat width of 18 mm (±10) and void width of 12 mm (±5) in the sow area.

Slat shape and flooring material used for the slatted and solid areas were correlated. As a consequence floor characteristics were collapsed into 6 categories (Table 2.2 and Figure 2.1). Descriptions and the distribution of the floor characteristics are shown in Table 2.2 and 2.3, and examples of the floor types can be seen in Figure 2.2 and 2.3. Limb and foot swellings were combined for regression analysis because of the low number of animals affected (70 and 129 respectively).

Figure 2.2. Image of farrowing crate showing the combination "plastic slat, solid plastic" i.e. plastic slats in the sow and piglet areas and a solid plastic piglet area.

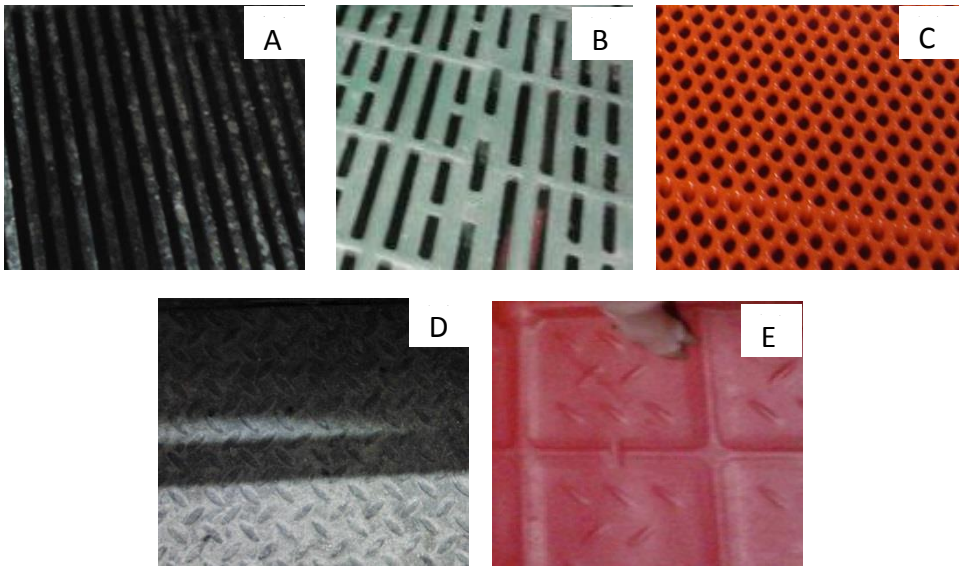


Figure 2.3 Image of types of piglet flooring: (A) Metal slat, (B) Plastic (rectangular) slat, (C) Plastic (oval) slat, (D) Solid metal, (E) Solid plastic.

2.4.2 Prevalence of limb and foot lesions

The prevalence of sole bruising, sole erosion, coronary band injury, limb abrasions, alopecia, swollen limbs and swollen feet in 2948 piglets was 61.5%, 34.1% , 11.3%,

55.7%, 24.8%, 2.4% and 4.4% respectively (Table 2.4). There was a wide range in the prevalence of foot and limb lesions between farms: sole bruising (7-100%), sole erosion (0-100%), coronary band damage (0-46%), foot swelling (0-14%), limb abrasions (11-98%), alopecia (0-83%) and limb swelling (0-11%).

The prevalence of foot and limb lesions varied between the front and hind limbs and feet (Table 2.4). Limb abrasions were more prevalent on front (54.7%) than on hind (6.7%) limbs. Injury to the coronary band was slightly more prevalent on front than on hind feet (8.4% vs. 4.9%) as was sole bruising (55.1% vs. 48.4%). There was no significant difference in the prevalence of lesions between the left and right limbs and feet. The distribution of lesions by severity score varied by lesion type. When present, sole bruising, sole erosion and coronary band injuries were mainly score 1 while, the modal score for limb abrasions and alopecia was 2. The prevalence of foot and limb lesions varied by age and pen floor type (Table 2.5).

Table 2.4 Number (n) and prevalence (%) of lesions in front and hind limbs in piglets with lesion present (score ≥ 1), and each of scores 1, 2 and 3.

	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Sole bruising	1863	61.5	1277	41.6	487	16.5	99	3.4
Front limbs	1624	55.1	1167	39.6	392	13.3	65	2.2
Hind limbs	1426	48.4	1044	35.4	329	11.2	53	1.8
Sole erosion	1005	34.1	576	19.5	321	10.9	108	3.7
Front limbs	689	23.4	437	14.8	195	6.6	57	1.9
Hind limbs	793	26.9	475	16.1	241	8.2	77	2.6
Coronary band injury	333	11.3	185	6.3	91	3.1	57	1.9
Front limbs	248	8.4	135	4.6	71	2.4	42	1.4
Hind limbs	143	4.9	94	3.2	32	1.1	17	0.6
Foot swelling	129	4.4	44	1.5	47	1.6	38	1.3
Front limbs	84	2.9	28	1.0	33	1.1	23	0.8
Hind limbs	49	1.7	17	0.6	15	0.5	17	0.6
Limb abrasion	1641	55.7	424	14.4	757	25.7	460	15.6
Front limbs	1612	54.7	416	14.1	752	25.5	444	15.1
Hind limbs	198	6.7	117	4.0	53	1.8	28	1.0
Alopecia	731	24.8	176	6.0	352	11.9	203	6.9
Front limbs	704	23.9	168	5.7	338	11.5	198	6.7
Hind limbs	70	2.4	23	0.8	23	0.8	24	0.8
Swollen limb	70	2.4	27	0.9	30	1.0	10	0.3
Front limbs	44	1.5	13	0.4	16	0.5	15	0.5
Hind limbs	29	1.0	11	0.4	7	0.2	11	0.4

Table 2.5 Number (n) and prevalence (%) of piglets affected (score ≥1) by sole bruising, sole erosion, coronary band damage, foot swellings, limb abrasions, alopecia and limb swellings for the different categories of age and floor characteristics.

	Sole bruising		Sole erosion		Coronary band damage		Foot swelling		Limb abrasion		Alopecia		Limb swelling	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Age														
3-7d	667	87.9	264	34.8	146	19.2	32	4.2	321	42.3	57	7.5	8	1.1
8–14d	521	69.3	265	35.2	82	10.9	30	4.0	370	49.2	129	17.2	9	1.2
15–21d	348	47.9	234	32.2	47	6.5	24	3.3	421	58.0	260	35.8	24	3.3
22–28d	277	39.0	242	34.0	58	8.2	43	6.1	529	74.4	285	40.1	29	4.1
Floor types														
Plastic slat, solid plastic	427	63.3	209	31.0	58	8.6	23	3.4	351	52.0	130	19.3	11	1.6
Plastic (oval)and metal slats, solid plastic,	103	36.4	51	18.0	16	5.7	11	3.9	109	38.5	38	13.4	6	2.1
Plastic (rectangular) and metal slats, solid plastic	377	69.2	131	24.0	51	9.4	26	4.8	308	56.5	103	18.9	16	2.9
Metal slat, solid plastic	372	67.9	252	46.0	78	14.2	32	5.8	336	61.3	135	24.6	19	3.5
Metal slat, metal solid	183	57.2	139	43.4	75	23.4	15	4.7	174	54.4	126	39.4	6	1.9
Other slats, solid concrete	351	60.8	223	38.7	55	9.5	22	3.8	363	62.9	199	34.5	12	2.1

2.4.3 Risk factors for limb and foot lesions

2.4.3.1 Sole bruising

The risk of sole bruising decreased with age. There was a lower risk of sole bruising in piglets in pens with plastic oval slats in the piglet area and metal slats under the sow with a solid plastic area when compared with piglets in pens with plastic stats throughout and a plastic solid area for piglets (Table 2.6).

2.4.3.2 Sole erosion

There was no association between sole erosion and age. There was an increased risk of sole erosion in piglets in pens with metal slats throughout with a piglet plastic solid area and for piglets in pens which had an other slatted area and a concrete solid area when compared to pigs in pens with plastic stats throughout and a piglet plastic solid area. The other category comprised of a variety of concrete, metal and plastic combinations. The risk of sole bruising decreased when a heat pad was present (Table 2.6).

2.4.3.3 Coronary band damage

The risk of coronary band injury decreased with age. There was an increased risk of coronary band injury in piglets in pens with metal slats throughout with a piglet metal solid area when compared to pigs in pens with plastic stats throughout and a piglet plastic solid area (Table 2.6).

2.4.3.4 Limb abrasion

The risk of limb abrasions increased with age. There was no significant association between limb abrasions and floor type (Table 2.6).

2.4.3.5 Correlations between lesions

Several foot and limb lesions were correlated to one another (Table 2.7). Key associations were a positive association between limb abrasions and limb joint swelling and a positive association between sole erosion and coronary band injury and foot swelling.

Table 2.6 Multilevel mixed effect binomial regression models of the risks associated with presence of sole bruising, sole erosion, coronary band injury, limb abrasions and foot and limb swellings in piglets.

	Sole bruising			Sole erosion			Coronary band damage			Limb abrasion			Alopecia			Limb & foot swellings		
Intercept coefficient	2.67			0.3			-1.56			-0.29			-3.3			-0.82		
	OR	CI		OR	CI		OR	CI		OR	CI		OR	CI		OR	CI	
Age	0.42	0.37	0.50	0.97	0.88	1.06	0.69	0.60	0.81	1.54	1.12	2.14	1.19	1.04	1.36	1.25	1.06	1.47
Floor type																		
Plastic slat, plastic solid																		
Plastic (oval) & metal slats, solid plastic	0.32	0.15	0.70	1.19	0.69	2.07	0.59	0.22	1.59				0.68	0.30	1.53	1.32	0.58	3.00
Plastic (rectangular) & metal slats, solid plastic	1.70	0.88	3.28	1.93	1.13	3.30	1.02	0.46	2.24				0.17	0.09	0.34	1.59	0.82	3.08
Metal slat, plastic solid	1.32	0.72	2.41	1.92	0.62	5.93	1.74	0.87	3.51				1.16	0.63	2.12	1.90	1.01	3.58
Metal slat, metal solid	0.95	0.47	1.91	1.54	0.82	2.90	4.25	1.96	9.22				2.59	1.30	5.15	1.33	0.62	2.86
Other slats, solid concrete	1.24	0.69	2.23	1.79	1.04	3.08	1.23	0.61	2.50				1.62	0.91	2.90	1.28	0.67	2.46
Heat pad present																		
Yes				0.3	0.2	0.6												
No																		
Random effects	Var	SE		Var	SE		Var	SE		Var	SE		Var	SE		Var	SE	
Farm	0.6	0.17		1.2	0.24		0.8	0.23		0.5	0.12		0.7	0.17		0.3	0.15	
Pen	0.9	0.14		0.3	0.07		0.8	0.18		0.5	0.09		0.7	0.13		0.6	0.21	

Table 2.7 Significant correlations between piglet foot and limb lesions.

	Limb swelling	Foot swelling	Sole bruising	Sole erosion	Coronary band injury	Limb abrasion	Alopecia
Limb swelling	1						
Foot swelling	0.09***	1					
Sole bruising	-0.06**	0.02	1				
Sole erosion	0.02	0.11***	0.19***	1			
Coronary band injury	0.01	0.21***	0.01	0.12***	1		
Limb abrasion	0.08***	0.04*	-0.01	0.07**	0.05*	1	
Alopecia	0.8***	0.11***	-0.06**	0.09***	-0.03	0.05*	1

* = <0.05, ** = <0.005, *** = -<0.0001

2.5 Discussion

This study is the first to examine the prevalence and risk factors for foot and limb lesions in piglets on commercial farms in Ireland and it is the largest cross-sectional study to date of indoor housing systems. The information provided in this study is valuable to all pig producing countries as the majority use indoor accommodation without bedding for piglets.

This is the first cross-sectional study to determine the prevalence and risk factors of coronary band injuries. However a previous study has indicated that straw provision reduces the occurrence of this lesion (Westin *et al.*, 2014). In the current study, the negative association with increasing age is likely due to a combination of healing lesions and increasing resilience over time and reduced incidence of occurrence with increasing size and resilience of the foot as the piglet gets older which makes it less likely to become trapped in the voids between the slats (KilBride *et al.*, 2009b). There was an increased risk of coronary band injury in pens with metal slats throughout with a metal solid area for piglets when compared to the other floor types. Metal is a more unyielding material than plastic and therefore applies more pressure to the coronary band if the foot becomes caught in the void between the slats and this might explain the increased risk of injury (Gregory and Grandin, 2007; KilBride, 2008).

As reported in other studies, sole bruising was the most prevalent foot lesion, although there was an even higher prevalence of sole bruising in the current study (62%) than in Mouttotou et al. (1999c) (50%) and KilBride et al. (2009b) (49%). This might be explained by the absence of outdoor farms and farms with solid floors with bedding in the current study, which was associated with a reduced risk of sole bruising in the previous studies and that all the floors in the current study were slatted, at least in part, which was associated with a higher prevalence of sole bruising (Mouttotou et al., 1999c; KilBride et al., 2009b; Westin et al., 2014). The negative association between sole bruising and age is probably because the epithelium of the sole at birth is only 1 – 2 mm deep and thickens as the piglets age

thereby making the sole less susceptible to bruising (KilBride *et al.*, 2009b). In the current study, the risk of sole bruising decreased when the floors had plastic oval slats in the piglet area, metal slats in the sow area and a piglet solid plastic area when compared to pens with fully plastic rectangular slats with a piglet plastic solid area. This is possibly due to a more even distribution of weight to the foot pad provided by an oval compared to a rectangular shaped void, reducing pressure on specific points of the foot. The risk of sole bruising also decreased when a heat pad was present. Mouttotou and Green (1999a) reported that the protective benefit of a heat pad is related to the fact that piglets use these areas of the pen more because of increased comfort and warmth and therefore spend less time in the potentially more injurious parts of the pen i.e. the slatted sow area.

KilBride et al. (2009b) also suggested an alternative hypothesis to reduction in sole bruising, which is that sole bruising is replaced by sole erosion (when the epidermal layers of skin are removed) in harsh environments and so bruising will no longer be present. There was a high prevalence of sole erosion in the current study (34%) in comparison to KilBride et al. (2009b) (17%) and it was most strongly associated with sole bruising. This is a serious welfare concern as this injury, along with injury to the coronary band, is associated with pain and both can act as an entry site for bacteria and result in infection and joint swelling (Penny *et al.*, 1971; Mouttotou and Green, 1999a; Straw *et al.*, 2006). Metal slats are considerably more abrasive than plastic slats (Gregory and Grandin, 2007), so it is not surprising that the risk for sole erosion and swellings increased in pigs in pens with metal slats throughout with a small plastic solid floor area for piglets.

Given the high susceptibility of piglets to coronary band injuries and sole bruising at young ages it is possible that such lesions might be prevented by housing piglets on softer flooring for the first week of life. On the other hand this could merely delay the onset of bruising and coronary band injury to a later age (KilBride *et al.*, 2009b).

Skin abrasions were the most prevalent limb lesion and they occurred most commonly bilaterally on the carpus of the front limbs, as previously reported

(Gravås, 1979; Svendsen *et al.*, 1979; Mouttotou and Green, 1999a; Mouttotou *et al.*, 1999c; Zoric *et al.*, 2004; Zoric *et al.*, 2008; KilBride *et al.*, 2009b). In the current study limb abrasions increased with age, this is in contrast to previous studies (Svendsen *et al.*, 1979; Phillips *et al.*, 1996; Mouttotou and Green, 1999b; Mouttotou *et al.*, 1999c; Zoric *et al.*, 2004; KilBride *et al.*, 2009b). However, the lack of bedding provision and floor types is different from these studies. The unusual trend in the current study might also be because any epidermal disruption was considered a skin abrasion and the stage of the lesion (i.e. fresh or healing) was not recorded. In the current study, there was no association between the prevalence of limb abrasions and pen floor type. This agrees with KilBride et al. (2009b) where a lower risk of occurrence was only associated with outdoor systems and Mouttotou et al. (1999c) and Zoric et al. (2009) where a lower risk of skin abrasions on solid concrete floors and with bedding as opposed to pens with slatted floors, which included all the farms in the current study. That is, there was insufficient variation in floor type to observe a difference in prevalence of limb abrasions.

In the current study alopecia was more common on front than hind limbs, similar to Mouttotou and Green (1999a) and KilBride et al. (2009b), and increased with age. One explanation for alopecia is that it is formed from hairless scar tissue after a limb abrasion has healed (Wechsler *et al.*, 2000; KilBride, 2008; Zaffino, 2012). Hairless patches may also present on piglets as a mild lesion when pen conditions lead to less damage to the skin than an abrasion. The former explanation is more likely in piglets in the current study.

Overall, in the current study, there was a relatively low prevalence of foot and limb swellings (6.8%) when compared with the other lesions observed. The prevalence is similar to KilBride et al. (2009b). KilBride et al. (2009b) reported that internal pathological damage was often more severe than external injuries suggested and internal infection often did not result in noticeable swelling and so swellings may underestimate the prevalence of internal infections in the foot and limb. The severity of swellings therefore makes them a production and welfare concern despite their low prevalence (Penny et al., 1971; Zoric et al., 2004; KilBride et al.,

2009b). There was an increased risk of foot and limb swellings when sole erosion, coronary band injuries or limb abrasions were present. These three lesions penetrate the epidermis and so provide an entry site for pathogens which can result in infection and associated swellings (Penny et al., 1971; Mouttotou and Green, 1999a; Straw et al., 2006).

Whilst no one floor type was ideal in the current study, avoiding the use of metal slats in both the piglet and sow areas of the farrowing pen could reduce the occurrence of coronary band lesions, sole bruising, sole erosion and foot and limb swellings. These lesions are associated with the most potential for negative welfare impact. Sampling of a range of ages of piglets and observing trends with age indicate that for some environments there was a dose effect (time exposed), which increases the concern about the damage caused by some environments. There were no management variables significantly associated with foot and limb lesions in the current study. This is probably due to the lack of variation in management and general housing of piglets on commercial pig units in Ireland.

2.6 Conclusions

The high prevalence of foot and limb lesions in commercial farms in Ireland is a substantial welfare concern. Whilst no single floor type in this sample of Irish pig farms was consistently associated with low levels of piglet foot and limb lesions, metal slats were associated with an increased risk of coronary band injury, sole bruising and erosion and these in turn were associated with swellings of foot and limb joints. We conclude that avoiding the use of metal slats in both the piglet and sow areas of the farrowing pen could reduce the occurrence of these lesions which can contribute to infection in foot and limb joints.

Chapter 3

A cross-sectional study on the prevalence and risk factors for limb and claw lesions in weaner and finisher pigs, and lameness in finisher pigs on 68 commercial farms in Ireland

3.1 Abstract

A cross-sectional survey of 68 integrated Irish pig farms was conducted to determine the prevalence and risk factors for limb lesions in 3368 weaner and 1289 finisher pigs and lameness in finisher pigs in Ireland. On each farm 5 pens of weaners, one each aged 6, 8, 10, 12, 14 weeks (w) and 2 pens of finishers, one each aged 18w and 22w were examined for limb lesions and foot lesions and additionally finishers were examined for locomotory ability. Limbs were examined for scratches, wounds, swellings, abscesses, calluses, alopecia, bursitis and capped hock, which were scored from 0–3 based on severity. Overgrown, broken and fully amputated dew claws and toes were also scored as present or absent. Locomotory ability was scored from 0 to 5 based on severity. Environmental parameters were recorded for each pen examined. A questionnaire was completed on management, health and performance factors for each farm. The overall prevalence of each lesion was calculated and multilevel mixed effect logistic regression was used to elucidate risk factors. The risk of scratches, swellings, bursitis wounds and calluses in weaners increased with age when compared with pigs aged 6w. There was a higher risk of scratches, wounds and alopecia in pigs in pens with concrete slats and alopecia in pigs in pens with metal slats when compared with pigs in pens with plastic slats. The risk of scratches in finisher pigs increased with age when pigs aged 18w was compared with pigs of 22w. A reduced risk of scratches, wounds, alopecia and bursitis was associated with pigs in pens which were partially slatted when

compared with fully slatted pens. Pigs that were stocked at 0.84 - 3.04 m^2 per pig when compared to pigs stocked at 0.35 - 0.7 m^2 per pig had a reduced risk of limb scratches. The prevalence of lameness in finisher pigs was 32% and the risk of lameness increased with age from 18 to 22 weeks. An increased risk was also associated with a slat void of greater than 20 mm when compared to less than 20 mm. These findings have implications for weaner and finisher management because weaners and finishers had a high prevalence of a variety of lesions associated with age and environment. There was a very high prevalence of lameness in finisher pigs which was associated with the width of the slat void and the frequency of pen cleaning.

3.2 Introduction

Lameness, limb and claw lesions are commonly observed in weaner and finisher pigs (Mouttotou *et al.*, 1997; Mouttotou *et al.*, 1998;1999b;a;d; Cagienard *et al.*, 2005; Gillman *et al.*, 2008; KilBride, 2008; KilBride *et al.*, 2008). In severe cases they are a health concern and cause of impaired productivity and negatively impact pig welfare due to the association with pain and discomfort (Dewey *et al.*, 1993; Kirk *et al.*, 2005; Deen *et al.*, 2007; Jensen *et al.*, 2007; Mustonen *et al.*, 2011).

Calluses, bursitis, capped hocks, abrasions and alopecia are the most commonly recorded limb lesions in weaner and finisher pigs (Mouttotou *et al.*, 1998;1999b;d; Cagienard *et al.*, 2005; Gillman *et al.*, 2008; KilBride *et al.*, 2008; KilBride *et al.*, 2009a). A callus is hyperkeratinosis of the epidermis in response to frequent contact with an abrasive surface, resulting in a thickened protective area (Cagienard *et al.*, 2005; KilBride, 2008). It commonly occurs at the carpus and tarsus in weaner and finisher pigs (Cagienard *et al.*, 2005; KilBride, 2008). KilBride (2008) reported a prevalence of calluses of 45.5% in weaner and finisher pigs in England. Cagienard *et al.* (2005) compared pigs housed in high welfare pens and traditional pens and reported a prevalence range of 57.4% to 89.2% at the carpus and 42% to 99.3% at the tarsus respectively in high welfare and traditional pens respectively. KilBride (2008) noted an increased risk of calluses as the depth of bedding reduced.

Adventitious bursitis is a fluid filled sac of the subcutaneous connective tissue of the limb. Adventitious bursitis, when located on the tarsal joint, is referred to as capped hock and bursitis when present elsewhere on the limb (Mouttotou *et al.*, 1999b; Gillman *et al.*, 2008). In England, Gillman *et al.* (2008) and KilBride *et al.* (2008) reported a prevalence of bursitis and capped hock of 40.6% and 17.2% respectively in weaner and finisher pigs while Mouttotou *et al.* (1999b) reported a combined bursitis and capped hock prevalence of 63%. Bursitis was more prevalent in the hind limbs in both studies. A lower risk of bursitis is associated with the presence of bedding and outdoor systems, while wet pen floors are associated with an increased risk (Mouttotou *et al.*, 1999b). Concrete pens with sparse bedding are associated with an increased risk of capped hock compared with outdoor soil systems (KilBride *et al.*, 2008). Other lesions such as abrasions, alopecia, swellings and abscesses to the limbs have been investigated to a lesser extent in weaner and finisher pigs and risk factors have not yet been elucidated.

Lameness exhibits as an abnormal gait as a result of physical injury or infection in the limbs, pelvis or back (Velarde and Geers, 2007). KilBride *et al.* (2009a) reported abnormal gait in 16.9%, 34.3% and 22.3% of finishers housed on partially slatted, fully slatted and fully solid flooring with sparse bedding respectively when lameness was defined as abnormal gait. Limb pathologies such as osteochondrosis and infectious arthritis have previously been linked with lameness (Jensen *et al.*, 2007; KilBride *et al.*, 2009a). Physical injury such as claw lesions, joint lesions, muscle or tendon damage and bone fractures have also been previously associated lameness (Jensen and Toft, 2009). KilBride *et al.* (2009a) reported a reduced risk of lameness in finisher pigs on solid concrete floors with deep bedding in all areas when compared with other indoor floor types. Slatted flooring is a risk factor for lameness due to the slat void, the slat edge and the rough surface produced by slat aging as these provide areas for potential claw harm (Boon and Wray, 1989; Velarde and Geers, 2007; Jensen and Toft, 2009). Furthermore, narrow slates provide a poor weight bearing surface for the claw (Mouttotou *et al.*, 1999a).

Gillman *et al.* (2008), KilBride (2008), KilBride *et al.* (2008) and KilBride *et al.* (2009a) examined the prevalence and risk factors for bursitis, capped hock and calluses only in weaner and finishers and lameness in finishers in both indoor and outdoor English pig farms. The aims of the current study were to determine the prevalence and risk factors for limb lesions in weaner and finisher pigs and lameness in finisher pigs on commercial farms in Ireland. This is the first such study in Ireland and the largest study to assess weaner and finisher pigs housed entirely indoors in intensive production systems. In addition, the current study is the first study of a wider range of limb lesions in weaner and finisher pigs.

3.3 Methods

3.3.1 Farm selection and sample size

Data were collected as part of a cross-sectional survey as described in Chapter 2.

3.3.2 Measurements

On each farm 5 pens of weaners, one pen of each aged: 6 weeks (w), 8w, 10w, 12w and 14w and 2 pens of finishers, one of 18w and one of 22w were examined in total. Pens were randomly selected by assigning a number to each pen of each age group then using a random number generator to identify pens for selection. Every pig in each selected pen was examined if less than 10 pigs per pen and if greater than 10 pigs per pen only 10 were randomly selected and examined (Appendix 1). Pigs were randomly selected by firstly walking through the pen to ensure all pigs moved from their initial location, then starting at the entrance of the pen going in a clockwise direction every 5^{th} pig was marked with a number (from 1-10) using a spray colour marker. Lesion definitions are supplied in Appendix 2.

3.3.2.1 Limb lesions

All four limbs were examined for the lesions; scratches, wounds, swellings, abscesses, calluses, alopecia and bursitis, and capped hock in the hind limbs only and defined and scored as per KilBride *et al.* (2009a) (Appendix 3), that is, lesions

were scored as area affected from 0–3 where 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the size of the nearest joint on the affected limb.

3.3.2.2 Claw lesions

Claw lesions were scored as present or absent, only gross claw lesions (overgrown, broken and fully amputated dew claws and toes) which were easily observable were recorded as the pigs were not restrained (Appendix 3).

3.3.2.3 Locomotory ability

Locomotory ability was scored in finishers only using the posture and gait components of the protocol proposed by Main *et al.* (2000) (Appendix 4). The locomotory ability of the pigs was scored according to severity from 0 (not lame) to 5 (severely lame) after observing the pigs for 6 consecutive steps (Calderón Díaz *et al.*, 2013). A pig was deemed as lame if it received a score of ≥2 (i.e. uneven posture, abnormal gait and caudal swagger). Locomotory ability was not assessed in weaners because their agility and rapid movements make it too difficult to establish accurate results.

3.3.2.4 Manure on the body

Manure on the body was recorded for all pigs as per the Welfare Quality Consortium (2009) Assessment Protocol for Pigs. It was scored from 0 to 2 as follows; 0 = up to 20% of body surface soiled, 1= 20-50% of body surface soiled and 2 = >50% of body surface soiled.

3.3.2.5 Floor cleanliness score

The cleanliness of each pen was scored from 0 (clean, dry and no excreta) to 4 (excreta very wet over 50% coverage) adapted from Hacker *et al.* (1994) (Appendix 4).

3.3.2.6 Environmental parameters

Environmental measurements were recorded from the pens in which selected pigs were housed similarly to Chapter 2 (Table 3.1). A detailed diagram of each pen was also drawn indicating the location of resources (drinker, feeder).

Table 3.1 Environmental measurements for weaner and finisher pigs.

Pen measurements	Unit/categories of measurement
Area	
Pen	m^2
Fully slatted	m^2
Fully solid	m^2
Heat pad	m^2
Feeder	m^2
Wall composition	
Material	Concrete, plastic, metal, wood, other
Structure	Fully solid, part solid +50%, part solid <50%
Flooring	
Type	Solid, fully slatted, partially slatted
Material	Concrete, plastic, metal, other
Flooring dimensions	Slat width, slat void width, slat void length, shape, profile, edge profile, surface texture
Slopes and steps	Present, absent
Feeders	Present, absent
	Type
Drinker	Present, absent
	Type
Other	
Heat pads	Present, absent
Environmental enrichment	Present, absent
Bedding	Present, absent

3.3.2.7 Management parameters

A questionnaire was completed as per Chapter 2. It included questions on weaner and finisher husbandry and feeding routines, hygiene practices and genetics (Appendix 1).

3.3.5 Data analysis

Data were stored in Microsoft Access 2003. The prevalence of each limb and claw lesion for both weaners and finishers and for lameness in finishers only was calculated by maximum severity score and presence/absence of each lesion per pig using the same method as Chapter 2. The farm prevalence of each lesion and lameness was calculated as described in Chapter 2. Data were analysed using MlwiN 2.27 (Rasbash *et al.*, 2012). Multilevel mixed effect logistic regression using a two level random effect model was used to determine risk factors as per Chapter 2. Due to the low prevalence of abscesses in weaners (<1%) and to the high prevalence of calluses in finishers (>99%) logistic regression was not carried out for these lesions. Model fit was assessed as per Chapter 2, model fit was acceptable for all models.

3.4 Results

3.4.1 Farm features

A total of 3368 weaners and 1289 finishers were examined from 335 weaner pens and 132 finisher pens (6w = 67; 8w = 67; 10w = 67; 12w =67; 14w = 67; 18w = 65 and 22w = 67 pigs) on 68 farms. The overall mean group size was 35.9 (SD ±24.5) for weaners and 20.3 (SD ±8.9) for finishers. All weaners were housed indoors in pens consisting largely of fully slatted or partially slatted flooring made from a variety of materials. Bedding was not provided in any form on any of the farms visited (Table 3.2). The mean pen area was 13.7 m^2 (±10.3) for weaners and 17 m^2 (±8.5) for finishers. Descriptions and the distribution of the floor characteristics are shown in Table 3.2. The mean slat width was 44 mm (±3.7) and the slat void width was 14 mm (±5) for weaners and the mean slat width was 92 mm (±2.2) and slat void width was 20 mm (±4) for finishers.

Table 3.2 Distribution of weaner and finisher pigs across the 6 categories of flooring type.

Floor type	Weaner		Finisher	
	n	%	n	%
Fully slatted	2409	71.5	864	67
Fully solid	60	1.8	0	0.0
Partially slatted	899	26.7	425	33

3.4.2 Limb lesion prevalence

The prevalence of scratches, wounds, alopecia, abscesses, swellings, calluses, bursitis and capped hock in 3368 weaners was 83%, 20.3%, 31%, 0.5%, 14.7%, 94.2%, 21.2%, and 3.6% respectively and for 1289 finishers was 80.8%, 25.4%, 54.6%, 0.8%, 28.9%, 99.5%, 29.6%, and 7.1% respectively (Tables 3.3 and 3.4). The prevalence of limb lesions varied between the front and hind limbs and feet (Table 3.3). The prevalence of limb and foot lesions also varied with age (Table 3.5).

3.4.3 Claw lesion prevalence

Very low levels of claw lesions were observed in both weaner and finisher pigs. No cases of overgrown dew claws, amputated toes, amputated dew claws or overgrown dew claws were recorded in weaner pigs. A low prevalence of overgrown toes and broken toes were found, 0.2% (n = 8) and 0.1% (n = 4) respectively in weaner pigs. No cases of amputated toes were recorded in finisher pigs. Amputated dew claws and broken toes and broken dew claws were all 0.08% prevalent (n = 1) and overgrown toe was detected in 0.8% (n = 10) pigs.

3.4.4 Lameness prevalence

The prevalence of lameness in finisher pigs was 32% (417 scored ≥2 for locomotory ability). This increased with age with: 27.8% (n = 179) of finishers lame at 18w and

36.8% (n = 238) at 22w (Table 3.6). Scores 4 and 5 had a prevalence of 0.3% (n =4) and 0.06% (n = 1) respectively.

There was a wide range in the prevalence of lesions in weaners between farms: scratches (35-100%), wounds (0-80%), alopecia (0-80%), abscesses (0-8%), swellings (0-39.6%), calluses (76-100%), bursitis (0-60%) and capped hock (0-24%). This was also the case for the prevalence of lesions on finisher pigs: scratches (10-100%), wounds (10-85%), alopecia (0-100%), abscesses (0-10%), swellings (0-75%), calluses (90-100%), bursitis (0-100%) and capped hock (0-45%). The prevalence range in lameness in finisher pigs between farms was 5 to 75%.

Table 3.3 Number (n) and prevalence (%) of weaners with lesions absent (score 0), present (score ≥ 1) and scores 1, 2 and 3 for scratches, wounds, swellings, abscesses, calluses, alopecia and bursitis and capped hock by front and hind limb.

	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	2796	83.0	2623	77.9	163	4.8	10	0.3
Front limbs	1558	46.3	1509	44.8	44	1.3	5	0.2
Hind limbs	2595	77.1	2462	73.1	128	3.8	5	0.2
Wound	685	20.3	608	18.1	68	2.0	9	0.3
Front limbs	277	8.2	239	7.1	32	1.0	6	0.2
Hind limbs	496	14.7	456	13.5	37	1.1	3	0.1
Alopecia	1044	31.0	237	7.0	654	19.4	153	4.5
Front limbs	529	15.7	137	4.1	326	9.7	66	2.0
Hind limbs	603	17.9	129	3.8	390	11.6	84	2.5
Abscess	15	0.5	1	0.0	6	0.2	8	0.2
Front limbs	8	0.2	0	0.0	1	0.0	7	0.2
Hind limbs	7	0.2	1	0.0	5	0.2	1	0.0
Swelling	495	14.7	189	5.6	221	6.6	85	2.5
Front limbs	263	7.8	103	3.1	117	3.5	43	1.3
Hind limbs	276	8.2	105	3.1	124	3.7	47	1.4
Callus	3173	94.2	428	12.7	1739	51.6	1006	29.9
Front limbs	3134	93.1	504	15.0	1792	53.2	838	24.9
Hind limbs	2341	69.5	681	20.2	1268	37.7	392	11.6
Bursitis	715	21.2	379	11.3	284	8.4	52	1.5
Front limbs	128	3.8	76	2.3	42	1.3	10	0.3
Hind limbs	632	18.8	339	10.1	250	7.4	43	1.3
Capped hock	120	3.6	98	2.9	20	0.6	2	0.1

Table 3.4 Number (n) and prevalence (%) of finishers of absent (score 0), present (score ≥ 1), Score 1, 2 and 3 for scratches, wounds, swellings, abscesses, calluses, alopecia and bursitis and capped hock by front and hind limb.

	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	1041	80.8	976	75.7	63	4.9	2	0.2
Front limbs	614	47.6	580	45.0	33	2.6	1	0.1
Hind limbs	932	72.3	896	69.5	34	2.6	2	0.2
Wound	327	25.4	294	22.8	28	2.2	5	0.4
Front limbs	144	11.2	130	10.1	11	0.9	3	0.2
Hind limbs	238	18.5	218	16.9	17	1.3	3	0.2
Alopecia	704	54.6	107	8.3	402	31.2	195	15.1
Front limbs	553	42.9	81	6.3	323	25.1	149	11.6
Hind limbs	354	27.5	75	5.8	216	16.8	63	4.9
Abscess	10	0.8	2	0.2	2	0.2	6	0.5
Front limbs	6	0.5	2	0.2	0	0.0	4	0.3
Hind limbs	4	0.3	0	0.0	2	0.2	2	0.2
Swelling	373	28.9	119	9.2	175	13.6	79	6.1
Front limbs	268	20.8	90	7.0	129	10.0	49	3.8
Hind limbs	182	14.1	54	4.2	87	6.8	41	3.2
Callus	1282	99.5	12	0.9	436	33.8	834	64.7
Front limbs	1262	97.9	33	2.6	540	41.9	689	53.5
Hind limbs	1251	97.1	78	6.1	672	52.1	501	38.9
Bursitis	381	29.6	172	13.3	169	13.1	40	3.1
Front limbs	95	7.4	48	3.7	37	2.9	10	0.8
Hind limbs	322	25.0	150	11.6	141	10.9	31	2.4
Capped hock	92	7.1	82	6.4	7	0.5	3	0.2

Table 3.5 Number (n) and prevalence (%) of weaners and finishers by age with wounds, swellings, abscesses, calluses, alopecia and bursitis and capped hock.

Age	Scratch		Wound		Alopecia		Abscess		Swelling		Callus		Bursitis		Capped hock	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Weaner																
6 w (n = 678)	454	67.0	71	10.5	143	21.1	2	0.3	28	4.1	574	84.7	54	8.0	5	0.7
8 w (n = 680)	549	80.7	108	15.9	154	22.7	7	0.7	51	7.5	609	89.6	86	12.7	26	3.8
10 w (n = 670)	598	89.3	152	22.7	200	29.9	4	0.6	103	15.4	665	99.3	163	24.3	19	2.8
12 w (n = 670)	596	89.0	186	27.8	284	42.4	2	0.3	145	21.6	664	99.1	202	30.2	35	5.2
14 w (n = 670)	599	89.4	168	25.1	263	39.3	0	0.0	168	25.1	662	98.8	210	31.3	35	5.2
Finisher																
18 w (n = 643)	536	83.4	154	24.0	330	51.3	4	0.5	167	26.0	642	99.8	197	30.6	40	6.2
22 w(n =646)	505	78.2	173	26.8	374	57.9	6	0.8	206	31.9	640	99.1	184	28.5	52	8.1

Table 3.6 Number (n) and prevalence (%) of lame finishers recorded as lame (score ≥ 2) and with scores 0, 1, 2, 3, 4 and 5 for locomotory ability.

Age	Lame		Score 0		Score 1		Score 2		Score 3		Score 4		Score 5	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
18w	179	28	102	16	362	56	162	25	16	2.5	1	0.2	0	0
22w	238	37	67	10	341	53	206	32	28	4.3	3	0.5	1	0.2

3.4.5 Risk factors for limb lesions in weaner pigs

The risk of scratches, swellings and bursitis in weaner pigs increased linearly with increasing age when compared with pigs aged 6 weeks (Table 3.7). The risk of wounds and calluses also increased with age when compared to pigs aged 6 weeks. There was a higher risk of scratches, wounds and alopecia in pigs in pens with concrete slats, and alopecia in pigs in pens with metal slats when compared with pigs in pens with plastic slats. There were insufficient numbers of pigs affected to determine risk factors for abscesses and capped hocks (Table 3.7). From the questionnaire, sorting pigs by sex at weaning reduced the risk of scratches (OR 0.36, CI 0.18-0.74) and wounds (OR 0.36, CI 0.16-0.85) when compared with pigs that were not sorted by sex.

3.4.6 Risk factors for limb lesions in finisher pigs

The risk of scratches in finisher pigs increased with age when pigs aged 18 weeks were compared with pigs aged 22 weeks. A reduced risk of scratches, wounds, alopecia and bursitis was associated with pigs in pens which were partially slatted when compared to fully slatted pens. There was a reduced risk of scratches to the limbs when pigs were stocked at 0.84 to 3.04 m^2 per pig compared with pigs stocked at between 0.35 to 0.7 m^2 per pig (Table 3.8).

3.4.7 Risk factors for lameness in finisher pigs

The risk of lameness in finisher pigs increased with age from 18 weeks to 22 weeks. An increased risk was also associated with a slat void of > 20mm when compared to

< 20mm (Table 3.9). From the questionnaire, pigs in pens washed more than 4 times a year had a reduced risk of being lame (OR 0.62, CI 0.40-0.98).

3.4.8 Correlations between lesions

Several weaner and finisher limb lesions were correlated to one another and lameness was correlated to several finisher limb lesions (Table 3.10 and 3.11).

Table 3.7 Multilevel binomial models of the risks associated with 3368 weaner pigs from 68 farms with scratches, wounds, alopecia, swelling and bursitis.

	Scratch			Wound			Alopecia			Swelling			Bursitis		
Intercept coefficient	0.7			-2.2			-1.4			-3.1			-2.4		
	OR	CI		OR	CI		OR	CI		OR	CI		OR	CI	
Age															
6w															
8w	2.15	1.57	2.94	1.55	1.09	2.22	1.42	1.03	1.95	1.88	1.13	3.11	1.67	1.16	2.42
10w	5.34	3.47	8.22	1.97	1.32	2.93	1.09	0.77	1.56	4.20	2.64	6.69	3.69	2.62	5.19
12w	5.12	3.30	7.97	2.47	1.65	3.69	1.11	0.77	1.60	6.39	3.54	11.55	4.95	3.54	6.94
14w	5.76	3.57	9.28	2.02	1.31	3.11	1.43	0.97	2.12	7.74	4.94	12.14	5.24	3.75	7.32
Slat type															
Plastic															
Concrete	1.54	1.04	2.28	1.49	1.07	2.08	1.72	1.25	2.36						
Metal	1.04	0.48	2.24	1.05	0.50	2.23	2.44	1.29	4.62						
Plastic & metal	1.01	0.31	3.33	2.49	0.88	7.04	1.10	0.35	3.45						
Random effects		Var	SE		Var	SE		Var	SE		Var	SE		Var	SE
Farm		0.94	0.20		0.79	0.17		1.22	0.24		0.33	0.10		0.46	0.10
Pen		0.28	0.90		0.17	0.07		0.18	0.06		0.22	0.09		0.05	0.06

Table 3.8 Multilevel binomial models of the risks associated with finisher pigs with scratches, wounds, alopecia, abscesses, swellings and bursitis and capped hock.

	Scratch			Wound			Bursitis		
Intercept coefficient	1.20			-1.10			-0.50		
	OR	CI		OR	CI		OR	CI	
Age	0.69	0.51	0.95	1.12	0.87	1.50	0.86	0.68	1.10
Floor type									
Fully slatted									
Partially slatted	0.36	0.20	0.65	0.59	0.36	0.95	0.60	0.39	0.93
Number of pigs/pen									
7-16									
17-20	3.11	1.62	5.95						
21-54	3.16	1.60	6.25						
Stocking density(m^2/pig)									
0.35-0.7									
0.71-0.83	1.67	0.93	2.98						
0.84-3.04	2.38	1.25	4.54						
Random effects	Var	SE		Var	SE		Var	SE	
Farm	1.33	0.31		0.93	0.21		0.71	0.17	
Pen	0.07	0.13		0.00	0.00		0.00	0.00	

Table 3.9 Multilevel binomial models of the risks associated with finisher pigs with lameness.

		Lameness		
Intercept coefficient		-1.5		
		OR	CI	
Age		1.48	1.16	1.87
Slat width				
	<20 mm			
	20 mm	1.3	0.89	1.92
	>20 mm	1.7	1.06	2.73
Random effects			Var	SE
Farm			0.25	0.09
Pen			0.00	0.00

Table 3.10 Significant correlations between limb lesions for weaner pigs.

	Scratch	Wound	Alopecia	Abscess	Swelling	Callus	Bursitis	Capped hock
Scratch	1							
Wound	0.13***	1						
Alopecia	0.15***	0.15***	1					
Abscess	0	0.17***	0.03	1				
Swelling	0.08***	0.16***	0.17***	0.17***	1			
Callus	0.15***	0.13***	0.14***	0.011	0.12***	1		
Bursitis	0.06**	0.06**	-0.01	0.04*	0.12***	0.069***	1	
Capped hock	-0.02	0.03*	-0.03	-0.01	0.03*	0.01	0.03	1

* = <0.05, ** = <0.001, *** = <0.0001

Table 3.11 Significant correlations between limb lesions for finisher pigs.

	Locomotory ability	Scratch	Wound	Alopecia	Abscess	Swelling	Callus	Bursitis	Capped hock
Locomotory ability	1.00								
Scratch	-0.09**	1.00							
Wound	0.02	0.19***	1.00						
Alopecia	0.04	0.22***	0.14***	1.00					
Abscess	0.12***	-0.04	0.20***	-0.01	1.00				
Swelling	0.16***	0.00	0.14***	0.17***	0.13***	1.00			
Callus	0.05	0.01	0.02	0.19***	0.01	0.1**	1.00		
Bursitis	0.01	-0.01	0.04	-0.07**	0.02	0.06*	-0.09**	1.00	
Capped hock	0.07*	-0.02	0.01	-0.09**	0.04	0.00	0.03	0.02	1.00

*= <0.05, **=<0.001, ***-<0.0001

3.5 Discussion

This is the first study to examine the prevalence and risk factors for limb lesions in weaner and finisher pigs on commercial farms in Ireland and the largest cross-sectional study to date of indoor housing systems. The information produced by this study is valuable to all pig producing countries throughout the world, the majority of which use indoor systems without bedding for both weaner and finisher pigs.

In previous literature a broad spectrum of scoring systems have been used and the definition of lameness varied greatly therefore variation in lameness levels is to be expected (Petersen *et al.*, 2008; KilBride *et al.*, 2009a). In this study the prevalence of lameness in finishers was greater than previously recorded and increased with age which highlights a severe welfare concern (Petersen *et al.*, 2008; KilBride *et al.*, 2009a). In the current study however the cut off level for lameness was lower than in other studies as in accordance with KilBride (2008) and Calderón Díaz *et al.* (2013). At this point abnormal gait and posture negatively affects the locomotory ability of the animal thus, impairing their ability to undertake normal behaviours, for domesticated pigs, such as competing for resources and other social interactions (KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013).

An important factor to consider also is that it is possible that within this and other studies lameness may still be underestimated because severely lame pigs (i.e. score 4 and 5) are often removed from the pen for treatment and isolation and so may not be recorded. As there were no soil or bedded underfoot conditions in the pens examined, it was expected that higher levels of lameness would be reported when compared to alternative housing systems (KilBride *et al.*, 2009a). KilBride *et al.* (2009a) identified slatted unbedded floors as a risk factor for lameness as opposed to soil or bedded pens. The width of the slat void was identified as a risk factor for lameness in the current study as slat voids that are too wide do not provide sufficient support for the claw making it more susceptible to injury (Fritschen, 1979; Straw *et al.*, 2006). On farms where pens were washed more frequently there were lower levels of lameness. This may be due to a reduction in environmental

pathogen levels and therefore a reduction in infection of lesions causes of lameness (Heinonen *et al.*, 2006; Cook and Nordlund, 2009).

Similar to KilBride (2008) and Cagienard *et al.* (2005), calluses were the most prevalent limb lesions, for both weaners and finishers in the current study (94.2%, 99.5%). KilBride (2008) however reported half the level reported by this study and Cagienard *et al.* (2005). This is most likely explained by the different housing systems in operation, as there were no outdoor farms or indoor pens with bedding sampled in the current study or in the study by Cagienard *et al.* (2005) dissimilar to KilBride (2008).

There is substantial variability in bursitis prevalence in both the literature and this study for both weaner and finisher pigs. A study by Savary *et al.* (2009) reported similar levels to the current study (15-30%), while Mouttotou *et al.* (1999d) reported substantially lower levels. Gillman *et al.* (2008) (40.6%) and Temple *et al.* (2011) (50%) reported higher levels of bursitis in both weaner and finisher age groups respectively. Furthermore Mouttotou *et al.* (1999b) (63%) and Mouttotou *et al.* (1998) (51%) reported higher levels in finisher pigs only, however capped hock was included in these values which would be expected to contribute to the higher prevalence. Prevalence differences may be a result of differences in the scoring systems used; in this study, bursitis was only recorded when an abnormal area was manipulated and a fluid filled sac was palpable otherwise this was recorded as a swelling, also several studies do not differentiate between bursitis and swellings. In agreement with other studies, bursitis was most prevalent in the hind limbs (Gillman *et al.*, 2008; Savary *et al.*, 2009). In the current study bursitis increased with age in weaner pigs, similarly to findings of Gillman *et al.* (2008). However dissimilarly, in Gillman *et al.* (2008) the increase in prevalence of bursitis persisted in the finisher pigs. Partially slatted flooring as opposed to fully slatted floors was associated with a reduced risk of bursitis in finisher pigs similar to Gillman *et al.* (2008); however the reason for this is unknown. Gillman *et al.* (2008) also reported a similar finding for weaner pigs which was not found in the current study.

Low levels of capped hock were recorded in weaner pigs in the current study in agreement with Mouttotou *et al.* (1999d). Mouttotou *et al.* (1998) reported a slightly lower prevalence in finisher pigs than in the current study (3.7%), while KilBride *et al.* (2008) reported a much higher prevalence in a cross-sectional study of English farms in both weaner and finisher pigs (17%). Data collection methods for this lesion differed between the studies. In this study, in a similar method to bursitis, capped hock was recorded if a fluid filled sac could be felt on the hock while KilBride *et al.* (2008) included any swelling to the hock which in some instances for the lower level scores, pathology revealed as being collagenous connective tissue and not capped hock. This could partially explain the prevalence variability between the current study and that of KilBride *et al.* (2008). Due to the low prevalence of these lesions in this study no risk factors were identified. However as Irish production systems operate indoors, without bedding largely on fully slatted floors prevalence would have been expected to be higher. A higher risk of capped hock in finisher pigs is associated with floor material (soil/concrete), floor type (fully solid/fully slatted) and bedding depth (KilBride *et al.*, 2008).

In the current study scratches were the second most prevalent limb lesion recorded in both weaner and finisher pigs with over 80% of pigs affected by this lesion. The prevalence of this common lesion has not previously been quantified for these groups. Scratches can be an indication of either aggression between pigs or of a poor physical environment (Velarde and Geers, 2007). Wounds were less prevalent but they are a more severe injury as the epidermis is broken and are often associated with more severe pain (Calderón Díaz *et al.*, 2013). Its presence is also associated with aggression and the pen environment (Velarde and Geers, 2007). Scratches and wounds to the fore quarter of the body are widely associated with aggression however limb scratches and wounds have not previously been documented (Turner *et al.*, 2006). It is thought that they arise largely due to the environment or scrambling behaviour of young pigs. Interestingly, in finishers in the current study; as the number of pig per pen increased the risk of scratches increased, however, finisher pigs at a lower stocking density had a higher risk of scratches. KilBride *et al.* (2009a) reported similar findings relating to lameness in

high stocking densities and they hypothesised that the increased space allowance led to increased activity making them more likely to receive injuries. The relationship to the number of pigs per pen may be associated with aggression levels as previous studies have found aggression is proportionate to group size at mixing (Mujuni *et al.*, 1985; Arey and Edwards, 1998; Mills *et al.*, 2010). Both scratches and wounds were most prevalent in the hind limbs and their risk increased with age in weaners. In weaners, there was a higher risk of wounds in pigs in pens with concrete slats compared to plastic slats, indicating that these are linked to the more abrasive floor type. Sorting pigs by sex at weaning reduced the risk of both scratches and wounds in weaners when compared with pigs in mixed-sex pens. Previous studies found that all female pens have less antagonistic behaviours such as mounting and nudging behaviours than mixed or entire male pens (Björklund and Boyle, 2006; Boyle and Björklund, 2007). Entire male pens however have higher levels of some of the antagonistic behaviours such as mounting which could result in an increase in injuries (Björklund and Boyle, 2006; Boyle and Björklund, 2007). Unfortunately as gender was not recorded in this study, it is possible that on farms where pigs were kept in single sex pens, more pens of females than of males were recorded which would reflect the finding that sorting pigs by sex at weaning is associated with fewer scratches and wounds. In finishers, there was a higher risk of both scratches and wounds in pens with fully slatted floors when compared to partially slatted floors. This may be related to the establishment of functionally distinct zones in partially slatted pens as solid areas encourages the establishment of designated lying areas which results in more uninterrupted lying and inactive behaviour in a distinct areas away from areas of competition for desired resources (feed, water, environmental enrichment) thus, making them less likely to be interrupted (stood upon or encourage aggression) (Boyle *et al.*, 2012; Levis *et al.*, 2013).

The prevalence of alopecia in weaner and finisher pigs was previously not documented. While this lesion was highly prevalent in both weaner (31%) and finisher (55%) pigs, it is regarded as superficial. Nevertheless it may be used as an indicator of historical injuries or identify areas of the limb which are frequently

exposed to abrasion such as the carpus and the flank in older pigs (Wechsler *et al.*, 2000; KilBride, 2008; Zaffino, 2012). Therefore alopecia may be used as a long term indicator of welfare. A higher risk was associated with concrete flooring in weaner pens as opposed to plastic which may indicate its association with healed lesions (scratches and wounds) or its association with more highly abrasive concrete floors. Indeed Savary *et al.* (2009) found a reduced likelihood of alopecia when pigs were housed on straw as opposed to concrete. The increase with age in weaners may be related to longer lying times as weight increased and ultimately an increase in pressure on contact points of the limb with increasing body weight (Ekkel *et al.*, 2003; KilBride, 2008).

This is the first cross-sectional study to date to determine the prevalence of limb swellings in weaner and finisher pigs. The few studies which have record swellings, recorded hock swellings solely, one of which also included bursitis within the category of swellings (Mouttotou *et al.*, 1999b; Cagienard *et al.*, 2005). Limb swellings can reduce pig welfare due to triggering an inflammatory response which is associated with pain. Inflammation can also alter nutrient utilisation in the body which can result in the energy required for growth being diverted to the energy requirements of the immune response system (Wilson *et al.*, 2010; Wilson and Ward, 2012). Importantly, swellings increased with age in weaner pigs in the current study. Similarly to the association of alopecia and age, this may too be explained by the associated increase in time spent lying with age and additional increase in pressure on contact points of the limb with increasing body weight (Ekkel *et al.*, 2003; KilBride, 2008).

In the current study correlations between locomotory ability and limb and claw lesions were found. In finishers there was a negative association between scratches and locomotory ability. This may be a result of reduced activity as a result of impaired locomotive ability (Kestin *et al.*, 1992; Weeks *et al.*, 2000; Anil *et al.*, 2009) which may subsequently reduce interaction with the environment and other pigs such that the risk of receiving scratches is reduced. A positive correlation with increasing locomotory ability score and abscess and swellings was found. Both

abscesses and swellings to the limbs are painful injuries which can impair locomotion (Jensen *et al.*, 2007). A study by Nielsen *et al.* (2001) supports this link as swellings were observed in 62% of lame pigs and in only 18% of non-lame pigs. Wounds were also positively correlated with abscesses and swellings in weaners and finishers. As wounds penetrate the epidermis they provide an entry site for pathogens which can lead to infection, which may result in swelling and abscessation (Penny *et al.*, 1971). Alopecia was positively correlated to scratches and wounds for both weaners and finishers, Alopecia commonly occurs in areas which were previously occupied by lesions or an area of frequent abrasion (Velarde and Geers, 2007). The prevalence of scratches and wounds was high throughout all age ranges. It is likely that these lesions heal over time and new ones appear and alopecia may remain in areas where there was a scratch or wound. Calluses and bursitis were also positively associated with swellings. As swellings tend to be painful they may increase the pigs time spent lying down increasing the risk of these injuries, as calluses and bursitis are linked to contact with hard floors (Mills *et al.*, 2010).

3.6 Conclusions

There was a high prevalence of a variety of limb lesions in weaners and finishers. The most prevalent lesions were mild. Lesions were associated with age and the environment. Age of the pig influenced the prevalence of lesions in weaners (scratches, wounds, alopecia, swellings, calluses and bursitis) and finishers (scratches) as well as floor material (scratch wound and alopecia) in weaners and proportion of the floor slatted in finishers (bursitis scratches and wounds). Very high levels of lameness in finisher pigs were found which is affected by the width of the slat void and the frequency of pen cleaning. This high level of lameness is a welfare concern as lameness is associated with pain.

Chapter 4

A cross-sectional study of the prevalence and risk factors for limb, claw and body lesions and lameness in gilts and breeding sows on 68 commercial farms in Ireland

4.1 Abstract

A cross-sectional survey of 68 integrated pig farms was conducted to determine the prevalence and risk factors for limb lesions in 525 replacement gilts, 518 pregnant gilts, 604 pregnant sows and 544 lactating sows and lameness in replacement gilts, gestating gilts and pregnant sows in Ireland. On each farm 1 pen of replacement gilts, 1 pen or 10 individual stalls of pregnant gilts, 1 pen or 10 individual stalls of pregnant sows and 8 lactating sows in farrowing crates, were examined for body lesions, limb lesions, claw lesions and all with the exception of lactating sows were examined for locomotory ability. Limbs were examined for scratches, wounds, swellings, abscesses, calluses, alopecia, bursitis and capped hock, which were scored from 0–3 based on severity. Overgrown, broken and fully amputated dew claws and toes were also scored as present or absent. Body lesions were examined at the ear, shoulder, flank, hind-quarter, anogenital, tail and vulva and scored from 0-6 based on severity. Locomotory ability was scored from 0 to 5 based on severity. Environmental parameters were recorded for each pen examined. An interview questionnaire was completed by the farmer on management, health and performance factors for each farm. The overall prevalence of each lesion was calculated and multilevel mixed effect logistic regression model was used to elucidate risk factors. Lameness prevalence was 38.9% (scored ≥2 for locomotory ability) in replacement gilts, 41.1% in pregnant gilts and 41.7% in pregnant sows. There was an increased risk of lameness in pregnant gilts and sows housed in groups compared with in individual stalls (OR 3.66, CI 1.23-3.66), pregnant gilts and

sows had a lameness prevalence of 48.1% in groups and 30.4% in individual stalls. There was an increased risk of swellings in replacement gilts separated from terminal stock at >90kg compared with those separated at <50 kg (OR 3.1; CI 1.6, 7.7). There was a reduced risk of lesions on the shoulder, flank and hindquarter in replacement gilts at low stocking densities (2.1-5.2 m^2/pig) and an increased risk of anogenital lesions in replacement gilts in pens with partially slatted floors when compared to fully slatted floors (OR 2.02; CI 1.08, 3.78). There was an increased risk of lesions at the ear (OR 4.83; CI 1.53, 15.23), shoulder (OR 10.29; CI 4.15, 25.53), middle (OR 5.50, CI 2.92-10.36), hindquarter (OR 6.30; CI 3.34, 11.87) and anogenital (OR 4.94; CI 1.22, 20.00) regions in group housed pregnant gilts when compared with individually housed pregnant gilts (OR 3.66, CI 1.23, 2.12). There was an increased risk of scratches to the limbs (OR 2.15; CI 1.25, 3.69) and a reduced risk of overgrown toes (OR 0.29; CI 0.14, 0.61) in pregnant gilts and sows in groups when compared with animals in individual stalls. The percentage of lactating sows unable to stand was 1.2% (n = 8); a further 3.5% (n = 19) had difficulty rising. There was an reduced risk of lesions on the hindquarters and the time since the sow farrowed and a reduced risk of alopecia on the flank and capped hock with farrowing crate area. These findings have implications for replacement gilt, pregnant gilt, pregnant sow and lactating sow management and welfare.

4.2 Introduction

Sow longevity is a key component of an efficient and profitable pig farming enterprise. However disorders of the locomotory system are a key contributor to premature culling, with 11% of sows culled for issues relating to limb health (Dewey *et al.*, 1993). Lameness, claw and body lesions are commonly occurring abnormalities relating to limb health in both gilts and sows (D'Allaire *et al.*, 1987; Lucia *et al.*, 2000; Leeb *et al.*, 2001; Deen *et al.*, 2007; KilBride *et al.*, 2009a; Pluym *et al.*, 2011; Calderón Díaz *et al.*, 2013). These injuries are associated with a direct reduction in sow performance and welfare and also act as an indicator for reduced production and welfare (Deen *et al.*, 2007; Velarde and Geers, 2007).

Lameness in gilts and sows is a major health problem on commercial pig farms (Deen *et al.*, 2007). The prevalence of lameness in pregnant sows was reported as 5 to 17% in England, 6 to 10% in Belgium and 8.8% in Finland in group housing systems (Heinonen *et al.*, 2006; KilBride *et al.*, 2009a; Pluym *et al.*, 2011; Pluym *et al.*, 2013a; Willgert *et al.*, 2014). Housing system is one of the major factors influencing lameness in sows. It was expected that the EC Directive 2008/120/EC would result in increased lameness levels as Calderón Díaz *et al.* (2013) reported that group housed sow were more lame than individually stalled sows. Other risk factors identified for lameness include: slatted flooring, group size, stocking density, genetics and parity (Boon and Wray, 1989; Straw *et al.*, 2006; Velarde and Geers, 2007; Jensen and Toft, 2009).

A high proportion of sows have at least one claw lesion (99%) although only 9.7% of affected animals are lame (Pluym *et al.*, 2011). Risk factors for claw lesions include pen design, flooring type, gestation housing type and management (Kroneman *et al.*, 1993; Gjein and Larssen, 1994; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013). The use of fully or partially slatted flooring is coupled with an increase in claw lesions as slats that are too narrow do not provide sufficient support for the hoof structure (Fritschen, 1979; Gjein and Larssen, 1994). Gestation housing type influences claw lesion prevalence, with claw lesions of various types being more prevalent in group housed herds (96% of sows affected) than in herds where sows are confined (80% of sows affected), on partially slatted flooring (Gjein and Larssen, 1994).

Limb lesions in gilts and sows have been investigated to a lesser extent than in piglets, weaners and finishers (Mouttotou *et al.*, 1997; Mouttotou *et al.*, 1998;1999a;d;b; Cagienard *et al.*, 2005; Gillman *et al.*, 2008; KilBride *et al.*, 2008). The lesions previously examined include calluses, bursitis, capped hock, swellings and wounds (Boyle *et al.*, 1999; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013; Calderón Díaz *et al.*, 2014). KilBride *et al.* (2009a) reported a wide range of lesion prevalence of calluses (30-80%), bursitis (32-37%) and capped hock (32-57%) between replacement gilts, pregnant gilts, pregnant sows and lactating sows. Risk

factors for limb lesions include flooring, pen design and housing system (grouped or stalled) (Boyd *et al.*, 2002; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013). Several studies linked lesions such as wounds, swellings, calluses, capped hock and bursitis to lameness (Smith, 1988; Bonde *et al.*, 2004; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013). This link is multidirectional as painful injuries may result in an alteration to locomotion or lameness may result in increased lying behaviour thus, increased contact time with flooring and a rise in associated limb lesions (Bonde *et al.*, 2004; KilBride *et al.*, 2009a; Calderón Díaz *et al.*, 2013).

Lesions to the body arise due to social interactions or environmental interactions and can be used as indicators of aggression and harsh pen environments (Gjein and Larssen, 1995; Velarde and Geers, 2007). Socially they can arise as a result of aggressive behaviour due to establishing social ranking such as the mixing of unfamiliar pigs or aggressive interactions at feeding events (Gjein and Larssen, 1995; Pajor, 2002). Lesions to the front (head, ears and shoulder) of the body arise due to social ranking aggression and lesions to the hind of the body are associated with feeding aggression (Luescher *et al.*, 1990; Geverink *et al.*, 1996; Leeb *et al.*, 2001; Velarde and Geers, 2007). Body lesion prevalence varies with location and housing system. These lesions, depending on severity, may result in pain to the animal and also act as indicator of stress (Velarde and Geers, 2007). The prevalence of body lesions varies greatly between farms and groups (pregnant or lactating sows) (Boyle *et al.*, 1999). Pen design, pen areas and group size are known to influence the prevalence and severity of body lesions (Leeb *et al.*, 2001).

While lameness is the second most important contributor to involuntary culling on Irish pig farms (Boyle *et al.*, 1998), the prevalence and risk factors of lameness in replacement gilts and pregnant gilts and sows has not yet been determined. Lameness and leg problems are expected to escalate as a result of the change from individual stalls to group housing in the EU, particularly in countries where the use of fully slatted flooring predominates, such as in Ireland. The aims of the current study were two fold, to determine the prevalence and risk factors for limb lesions, claw lesions, and body lesions in replacement gilts, pregnant gilts, pregnant sows

and lactating sows and secondly lameness in replacement gilts, pregnant gilts, pregnant sows, on commercial farms in Ireland. This study will provide the first information on the possible effects of the transition to group housing on pregnant gilt and sow limb health and the first to provide information on a wider catalogue of limb lesions affecting breeding female pigs.

4.3 Methods

4.3.1 Farm selection and sample size

Data were collected as part of a cross-sectional survey as described in Chapter 2.

4.3.2 Measurements

On each farm 1 pen of replacement gilts, 1 pen or 10 individual stalls of pregnant gilts, 1 pen or 10 individual stalls of pregnant sows and 8 lactating sows (2 each farrowed; 3–7 days (d), 8–14d, 15–21d and 22–28d) in farrowing crates were examined in total. If in gestation stalls or farrowing crates 10 gilts or sows in stalls/crates were randomly selected and examined. If in group pens of ≤ 10 pigs per pen all pigs were examined and if > 10 pigs per pen 10 were randomly selected and examined (Appendix 1).

4.3.2.1 Limb lesions

All four limbs were examined for the following lesions: scratches, wounds, swellings, abscesses, calluses, alopecia, alopecia flank and bursitis. Capped hock was scored in the hind limbs only. All limb lesions were defined and scored as per KilBride *et al.* (2009a) as described in Chapter 3, that is, lesions were scored as area affected from 0–3 where 0 = no lesion, 1 = < 25%, 2 = 25–50%, 3 = > 50% of the size of the nearest joint on the affected limb.

4.3.2.2 Claw lesions

Claw lesions (overgrown, broken and amputated toes and dew claws) were scored as present or absent as per Chapter 3, only gross claw lesions which were easily observable were recorded as the pigs were not restrained.

4.3.2.3 Body lesions

Body lesions were examined at the ear, shoulder, flank, hindquarter, anogenital, tail and vulva region and scored from 0 (no lesion) to 6 (>1 extensive lesion) based on severity as per O'Driscoll *et al.* (2013) (Appendix 4).

4.3.2.4 Body condition score

The body condition of gilts and sows was scored from 1 (visually thin) to 5 (excessively fat) according to the DEFRA guidelines for the condition scoring of pigs (Appendix 3).

4.3.2.5 Locomotory ability

Locomotory ability was scored from 0 to 5 in replacement gilts, pregnant gilts and pregnant sows using the posture and gait components of the protocol proposed by Main *et al.* (2000) as per Chapter 3. A pig was deemed as lame if it received a score of ≥2 (i.e. uneven posture, abnormal gait and caudal swagger).

4.3.2.6 Ability to stand

The ability of lactating sows to stand up was assessed in the farrowing crate and scored from 0 to 3: 0 = Already standing; 1 = stands within 60 seconds of encouragement (applied back pressure) to stand, 2 = stands after over 60 seconds of encouragement and 3 = will not stand.

4.3.2.7 Manure on the body

Manure on the body was scored from 0 to 2 for all pigs as per the Welfare Quality consortium assessment protocol for pigs (Welfare Quality Consortium, 2009) and as described in Chapter 3.

4.3.2.8 Floor cleanliness score

The cleanliness of each pen was scored from 0 to 4, adapted from Hacker *et al.* (1994) and as described in Chapter 3.

4.3.2.9 Environmental parameters

Environmental measurements of the pens/stalls/crates in which selected pigs were housed were recorded as described in Chapter 3 for replacement gilts and pregnant gilts and sows as described in Chapter 2 for lactating sows. A detailed diagram of each pen was also drawn indicating the location of resources (drinker, feeder).

4.3.2.10 Management parameters

A survey questionnaire was delivered as per Chapter 2. It included questions on gilt and sow management practices and feeding routines, vaccinations, hygiene practices and genetics.

4.3.5 Data analysis

Data were stored in Microsoft Access 2003. The prevalence of each limb and claw lesion for each group was calculated using maximum severity score and presence/absence of each lesion per pig as in Chapter 2. The farm prevalence of each lesion and lameness was calculated as described in Chapter 2. Data were analysed using MlwiN 2.27 (Rasbash *et al.*, 2012). A two level or three level mixed effect logistic regression model was used to determine risk factors as per Chapter 2. Model fit was assessed as per Chapter 2, with model fit acceptable for all models.

4.4 Results

In total 525 replacement gilts, 518 pregnant gilts, 604 pregnant sows and 544 lactating sows were inspected on 68 farms. Of the 68 farms visited, 4 did not produce their own replacement gilts and so replacement gilts were not inspected on these farms. Pen floor characteristics for replacement gilts, pregnant gilt, pregnant sows and lactating sows varied between farms and between groups within farms. The distribution of the floor characteristics are shown in Table 4.1.

Table 4.1. Distribution of floor type (fully slatted, partially slatted or solid floors) for replacement gilts, pregnant gilts, pregnant sows and lactating sows.

Floor type	Replacement gilt		Pregnant gilt		Pregnant sow		Lactating sow	
	n	%	n	%	n	%	n	%
Fully slatted	251	47.8	284	54.8	245	40.6	190	34.9
Partially slatted	238	45.3	230	44.4	359	59.4	338	62.1
Fully solid	36	6.9	4	0.8	0	0	16	3.0

4.4.1 Replacement gilts

4.4.1.1 Farm features

A total of 525 replacement gilts were examined from 64 pens on 64 farms (with a mean body condition score of 2.29 (SD ±0.5). The median group size was 10 (IQR 8-13). All replacement gilts were housed indoors in pens largely consisting of fully slatted or partially slatted floors (Table 4.1), all slats were made of concrete and any solid areas were made from concrete, plastic or metal. The distribution of the floor materials are shown in Table 4.2. The mean pen area was 17.11 m^2 (±13.0). The mean slat width was 95 mm (±26) and mean slat void was 20 mm (±3.1). Bedding was not provided on any of the farms visited.

Table 4.2. Distribution of replacement gilts by floor characteristics.

Floor type	n	%
Slatted concrete only	263	50.1
Solid concrete only	15	2.9
Slatted concrete, solid concrete	192	36.5
Slatted concrete, solid other (plastic, metal)	55	10.5

4.4.1.2 Limb lesion prevalence

The prevalence of scratches, wounds, alopecia, alopecia on the flank, abscesses, swellings, calluses, bursitis and capped hock in 525 replacement gilts was 78.1%, 34.7%, 47.8%, 16.8%, 0.19%, 30.3%, 100%, 25.0%, and 11.6% respectively (Table 4.3). The prevalence of limb lesions varied between the front and hind limbs (Table 4.3). There was a wide range in the prevalence of lesions between farms: scratches (0-100%), wounds (0-80%), alopecia (0-100%), alopecia flank (0-80%), swellings (0-60%), calluses (80-100%), bursitis (0-90%) and capped hock (0-25%).

Table 4.3. Number (n) and prevalence (%) of replacement gilts with lesions present (score ≥ 1) and scores 1, 2 and 3 for scratches, wounds, alopecia, alopecia flank, swellings, abscesses, calluses and bursitis and capped hock overall and by front and hind limb.

Lesion Type	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	410	78.1	369	70.3	39	7.4	2	0.4
Front	248	47.2	221	42.1	25	4.8	2	0.4
Hind	359	68.4	338	64.4	21	4.0	0	0.0
Wound	182	34.7	146	27.8	35	6.7	1	0.2
Front	89	17.0	72	13.7	16	3.1	1	0.2
Hind	133	25.3	111	21.1	22	4.2	0	0.0
Alopecia	251	47.8	39	7.4	137	26.1	75	14.3
Front	204	38.9	34	6.5	119	22.7	51	9.7
Hind	150	28.6	26	5.0	91	17.3	33	6.3
Alopecia flank	88	16.8	20	3.8	54	10.3	14	2.7
Front	88	16.8	20	3.8	54	10.3	14	2.7
Abscess	2	0.4	1	0.2	1	0.2	0	0.0
Front	1	0.2	1	0.2	0	0.0	0	0.0
Hind	1	0.2	0	0.0	1	0.2	0	0.0
Swelling	159	30.3	72	13.7	75	14.3	12	2.3
Front	99	18.9	47	9.0	48	9.1	4	0.8
Hind	79	15.1	35	6.7	35	6.7	9	1.7
Callus	525	100.0	4	0.8	169	32.2	352	67.1
Front	519	98.9	17	3.2	210	40.0	292	55.6
Hind	514	97.9	22	4.2	265	50.5	227	43.2
Bursitis	131	25.0	64	12.2	57	10.9	10	1.9
Front	38	7.2	27	5.1	10	1.9	1	0.2
Hind	105	20.0	49	9.3	47	9.0	9	1.7
Capped hock	61	11.6	53	10.1	8	1.5	0	0.0
Hind	58	11.1	50	9.5	8	1.5	0	0.0

4.4.1.3 Claw lesion prevalence

Very low levels of claw lesions were observed in replacement gilts (Table 4.4). There were 3.1% of replacement gilts with at least one overgrown dew claw and 1.7% of replacement gilts with at least one overgrown toe. Low percentages of broken dew claws and toes were observed: 0.6% and 0.2%. No cases of amputated dew claws or toes were recorded.

Table 4.4 Number (n) and prevalence (%) of replacement gilts, pregnant gilts, pregnant sows and lactating sows with lesions present for broken dew claw, broken toe, amputated dew claw, amputated toe, overgrown dew claw and overgrown toe.

Lesion Type	Replacement gilts		Pregnant gilts		Pregnant sows		Lactating sows	
	n	%	n	%	n	%	n	%
Broken dew claw	3	0.6	12	2.3	74	12.3	130	23.9
Front	3	0.6	5	1.0	13	2.2	28	5.2
Hind	1	0.2	8	1.5	68	11.3	112	20.6
Broken toe	1	0.2	7	1.4	9	1.5	19	3.5
Front	1	0.2	1	0.2	2	0.3	2	0.4
Hind	1	0.2	6	1.2	7	1.2	17	3.1
Amputated dew claw	0	0.0	6	1.2	8	1.3	3	0.6
Front	0	0.0	0	0.0	0	0.0	0	0.0
Hind	0	0.0	6	1.2	8	1.3	3	0.6
Amputated toe	0	0.0	0	0.0	3	0.5	3	0.6
Front	0	0.0	0	0.0	1	0.2	1	0.2
Hind	0	0.0	0	0.0	2	0.3	2	0.4
Overgrown dew claw	9	1.7	29	5.6	146	24.2	135	24.8
Front	6	1.1	12	2.3	57	9.4	58	10.7
Hind	9	1.7	23	4.4	128	21.2	108	19.9
Overgrown toe	16	3.1	80	15.4	187	31.0	183	33.6
Front	8	1.5	29	5.6	37	6.1	66	12.1
Hind	16	3.1	66	12.7	173	28.6	164	30.2

4.4.1.4 Body lesion prevalence

The prevalence of body lesions at the ear, shoulder, flank, hindquarter, anogenital, tail and vulva in 525 replacement gilts was 94.3%, 83.8%, 78.5%, 76.8%, 44.0%, 34.3% and 11.6%, respectively (Table 4.5).

Table 4.5 Number (n) and prevalence (%) of replacement gilts with body lesions present (score ≥ 1) and scores 1, 2, 3 and ≥4 to the ear, shoulder, flank, hindquarter, anogenital, tail and vulva region.

	Present		Score 1		Score 2		Score 3		Score ≥4	
Body location	n	%	n	%	n	%	n	%	n	%
Ear	495	94.3	128	24.4	307	58.5	57	10.9	3	0.6
Shoulder	440	83.8	105	20.0	256	48.8	74	14.1	5	1.0
Flank	412	78.5	124	23.6	243	46.3	44	8.4	1	0.2
Hind quarter	403	76.8	142	27.1	230	43.8	31	5.9	0	0.0
Anogenital	231	44.0	132	25.1	95	18.1	4	0.8	0	0.0
Tail	180	34.3	128	24.4	42	8.0	7	1.3	3	0.6
Vulva	61	11.6	46	8.8	12	2.3	0	0.0	3	0.6

4.4.1.5 Lameness prevalence

The prevalence of lameness (score ≥2) in replacement gilts was 38.9% (204 scored ≥2 for locomotory ability). Scores ≥3 had a prevalence of 4.95% (n = 26). The prevalence range in lameness between farms was 0 to 100% of replacement gilts (Table 4.6).

Table 4.6. Number (n) and prevalence (%) of locomotory ability scores (0-5) and lameness (Score ≥2) in replacement gilts, pregnan gilts and pregnant sows.

Group	Score 0		Score 1		Lame(≥2)		Score 2		Score 3		Score 4+	
	n	%	n	%	n	%	n	%	n	%	n	%
Replacement gilts	42	8.0	279	53.1	204	38.9	178	33.9	25	4.8	1	0.2
Pregnant Gilts	37	7.1	268	51.7	213	41.1	189	36.5	23	4.4	1	0.2
Pregnant sows	47	7.8	305	50.5	252	41.7	224	37.1	25	4.1	3	0.5

4.4.1.6 Risk factors for lameness, limb, claw and body lesions

No environmental risk factors were identified for lameness in replacement gilts. There was an increased risk of limb swellings in replacement gilts that were separated from terminal stock when they reached weights of >90kg when compared with replacement gilts separated from terminal stock when they weighed less than 50 kg (OR 3.1; CI 1.6, 7.7). Due to the low prevalence of claw lesions it was not possible to assess risk factors in replacement gilts. There was a lower risk of body lesions on the shoulder, flank and tail in replacement gilts kept at low stocking densities (2.1-5.2 m^2/pig). There was also a higher risk of anogenital lesions in replacement gilts in pens with partially slatted floors when compared to fully slatted floors (Table 4.7).

4.4.1.7 Correlations between lesions

Several limb lesions were correlated to one another other and with lameness in replacement gilts (Table 4.8).

Table 4.7. Multilevel binomial mixed effect models of the risks associated with replacement gilts for body lesions of the shoulder, flank, tail and anogenital.

	Shoulder			Flank			Tail			Anogenital		
Intercept coefficient	3.01			2.50			1.60			-0.63		
	OR	CI		OR	CI		OR	CI		OR	CI	
Stocking density area per pig												
<1m^2												
1-1.4 m^2	0.27	0.05	1.43	1.05	0.06	17.77	0.36	0.15	0.90			
1.41-1.7 m^2	0.25	0.04	1.75	1.04	0.03	33.03	0.55	0.18	1.73			
1.71-2 m^2	0.20	0.03	1.18	1.03	0.10	10.72	0.32	0.11	0.91			
2.1-5.2 m^2	0.13	0.02	0.68	1.02	0.26	3.97	0.30	0.13	0.69			
Floor type												
partially slatted, yes versus no										2.02	1.08	3.78
Random effects		Var	SE		Var	SE		Var	SE		Var	SE
Farm		1.6	0.45		0.8	0.28		0.85	0.27		0.96	0.26

Table 4.8. Significant correlations between lameness and limb lesions for replacement gilts.

	Lameness	Scratch	Wound	Alopecia	Alopecia flank	Swelling	Callus	Bursitis	Capped hock
Lameness	1								
Scratch	0.02	1							
Wound	-0.04	0.15**	1						
Alopecia	0.04	0.23***	0.31***	1					
Alopecia flank	0.09*	0.05	0.05	0.18***	1				
Swelling	0.06	0.02	0.04	0.01	-0.09*	1			
Callus	0.05	0.01	0.06	0.23***	0.16**	-0.04	1		
Bursitis	-0.03	-0.01	0.09	-0.05	-0.01	0.01	-0.11*	1	
Capped hock	-0.04	-0.06	0.00	0.06	0.02	-0.04	-0.08	0.01	1

* = <0.05, ** = <0.001, *** = <0.0001

4.4.2 Pregnant gilts and sows

4.4.2.1 Farm features

A total of 518 pregnant gilts were examined; 337 group housed gilts from 48 pens and 181 gilts kept in individual stalls on 68 farms with a mean body condition score of 2.7 (SD ±0.8). A total of 604 pregnant sows were examined; 364 from 44 pens and 240 from individual stalls on 68 farms, with a mean body condition score of 2.7 (SD ±0.7) respectively. Group housed pregnant gilts and sows were housed in four housing system types as seen in Figure 4.1 and as described in Tables 4.9. The distribution of pregnant gilts and sows amongst housing system are described in Table 4.10. There was a median group size of 7 (IQR 6-11) for pregnant gilts group housed in long trough pens, short feeder pens and free access stall pens and 80 (IQR 25-100) in electronic sow feeder (ESF) pens (Table 4.11). The mean group size for pregnant sows was 10.5 (IQR 6-12) in long trough pens, short feeder pens and free access stall pens and 170 (IQR 100-180) in ESF pens.

All group housed pregnant gilts and sows were housed indoors in pens consisting of fully slatted or partially slatted floors, all slats were concrete and solid areas were largely concrete or plastic, bedding was not provided in any form (Table 4.9). The mean pen area was 17.8 m^2 (±8.54) for pregnant gilts group housed in long trough pens, small feeder pens and free access stall pens. The mean pen area was 164.4 m^2 (SD ±45.65) for pregnant gilts in ESF pens. For pregnant sows group housed in long trough pens, small feeder pens and free access stall pens the mean pen area was 22.3 m^2 (SD ±10.48). The mean pen area was 352 m^2 (SD ±106.75) for pregnant sows in ESF pens. The mean slat width was 101.9 mm (±25) and mean slat void was 20 mm (±2.3) in group housed pregnant gilt pens and mean slat width was 103.9 mm (±27) and mean slat void was 20.9 mm (±3.4) in pregnant sow pens.

All pregnant gilts and sows housed in gestation stalls were housed on fully slatted or partially slatted floors, all slats and solid areas were concrete, bedding was not provided in any form (Table 4.9). The mean individual stall area was 1.28 m^2 (±0.21) for pregnant gilts and 1.30 m^2 (SD ±0.26) for pregnant sows. In pregnant gilt pens,

the mean slat width was 90.3 mm (±31.6) and the mean slat void was 20.8 mm (±3.7). In pregnant sow pens the mean slat width was 93.4 mm (±28.3) and the mean slat void was 21.3 mm (±3.7) in pregnant sows in gestation stalls.

Table 4.9. Group housing systems used for pregnant gilts and sows.

Type	Description (Group size range in this study)
Long trough	Unobstructed pen area with a long trough feeder (2-19)
Short feeder	Unobstructed pen area with a single or a double pig space feeder (2-21)
Free access stall	A pen containing partial or full length stalls for each pig at the feeder on one or both sides of the pen with a common open space (4-20)
Electronic sow feeder	Large groups, sows fed individually by an electronic sow feeder using electronic tags (17-206)

Table 4.10. Distribution of group housed pregnant gilts and sows by floor charateristics.

Floor type	Pregnant gilts		Pregnant sows	
	n	%	n	%
Slatted concrete only	204	60.5	141	38.7
Slatted concrete, solid concrete	126	37.4	215	59.1
Slatted concrete, solid other (plastic, metal)	7	2.1	8	2.2

Figure 4.1 Group housing systems used for pregnant gilts and sows; a) long trough system; b) short feeder system, c) free access stall system and d) electronic sow feeding system.

Table 4.11. Distribution of pregnant gilts and sows in each of the group housing.

	Pregnant gilt		Pregnant sow	
	n	%	n	%
Group housed	337	65.1	364	60.3
Long trough	156	46.3	145	39.8
Short feeder	52	15.4	38	10.4
Free access stalls	69	20.5	111	30.5
Electronic sow feeder	60	17.8	70	19.2
Fully slatted	200	59.4	141	38.7
Partial slatted	133	39.5	223	61.3
Fully solid	4	1.2	0	0.0
Individual stalls	181	34.9	240	39.7
Fully slatted	84	46.4	104	43.3
Partial slatted	97	53.6	136	56.7

4.4.2.2 Limb lesion prevalence

The prevalence of scratches, wounds, alopecia, alopecia flank, abscesses, swellings, calluses, bursitis and capped hock in 518 pregnant gilts was 62.7%, 28.8%, 48.7%, 27.6%, 0.2%, 20.2%, 99.8%, 19.3%, and 12.7% respectively (Table 4.12). There was a wide range in the prevalence of pregnant gilt limb lesions between farms: scratches (0-100%), wounds (0-100%), alopecia (0-100%), abscesses (0-90%), swellings (0-75%), calluses (60-100%), bursitis (0-100%) and capped hock (0-100%). The prevalence of limb lesions varied between group and gestation stall housing systems (Table 4.12).

The prevalence of scratches, wounds, alopecia, abscesses, swellings, calluses, bursitis and capped hock in 604 pregnant sows was 63.4%, 37.3%, 51.7%, 45.7%,

3.3%, 30.1%, 99.2%, 21.9%, and 15.4% respectively (Table 4.13). There was a wide range in the prevalence of pregnant sow limb lesions between farms: scratches (10-100%), wounds (0-100%), alopecia (0-100%), abscesses (20-0%), swellings (0-100%), calluses (80-100%), bursitis (0-100%) and capped hock (0-87.5%). The prevalence of limb lesions also varied between group and gestation stall housing systems (Table 4.13).

Table 4.12. Number (n) and prevalence (%) of pregnant gilts with lesions present (score ≥ 1) and scores 1, 2 and 3 for scratches, wounds, alopecia, alopecia flank, swellings, abscesses, calluses and bursitis and capped hock overall and by front and hind limb.

Lesion type	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	325	62.7	277	53.5	43	8.3	5	1.0
Individual stall	87	48.1	75	41.4	8	4.4	4	2.2
Group housed	238	70.6	202	59.9	35	10.4	1	0.3
Wound	149	28.8	122	23.6	26	5.0	1	0.2
Individual stall	42	23.2	37	20.4	5	2.8	0	0.0
Group housed	107	31.8	85	25.2	21	6.2	1	0.3
Alopecia	252	48.7	27	5.2	135	26.1	90	17.4
Individual stall	99	54.7	9	5.0	63	34.8	27	14.9
Group housed	153	45.4	18	5.3	72	21.4	63	18.7
Alopecia flank	143	27.6	26	5.0	78	15.1	39	7.5
Individual stall	52	28.7	14	7.7	24	13.3	14	7.7
Group housed	91	27.0	12	3.6	54	16.0	25	7.4
Abscess	1	0.2	0	0.0	1	0.2	0	0.0
Individual stall	0	0.0	0	0.0	0	0.0	0	0.0
Group housed	1	0.3	0	0.0	1	0.3	0	0.0
Swelling	115	22.2	45	8.7	55	10.6	15	2.9
Individual stall	30	16.6	11	6.1	16	8.8	3	1.7
Group housed	85	25.2	34	10.1	39	11.6	12	3.6
Callus	517	99.8	1	0.2	137	26.5	379	73.2
Individual stall	181	100	1	0.6	69	38.1	111	61.3
Group housed	336	99.7	0	0.0	68	20.2	268	79.5
Bursitis	100	19.3	58	11.2	37	7.1	5	1.0
Individual stall	41	22.7	24	13.3	13	7.2	4	2.2
Group housed	59	17.5	34	10.1	24	7.1	1	0.3
Capped hock	66	12.7	60	11.6	6	1.2	0	0.0
Individual stall	22	12.2	20	11.1	2	1.1	0	0.0
Group housed	44	13.1	40	11.9	4	1.2	0	0.0

Table 4.13. Number (n) and prevalence (%) of pregnant sows with lesions present (score ≥ 1) and scores 1, 2 and 3 for scratches, wounds, alopecia, alopecia flank, swellings, abscesses, calluses and bursitis and capped hock overall and by front and hind limb.

Lesion Type	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	419	69.4	365	60.4	48	8.0	6	1.0
Individual stall	150	62.5	127	52.9	18	7.5	5	2.1
Group housed	269	73.9	238	65.4	30	8.2	1	0.3
Wound	225	37.3	168	27.8	47	7.8	10	1.7
Individual stall	75	31.3	48	20.0	22	9.2	5	2.1
Group housed	150	41.2	120	33.0	25	6.9	5	1.4
Alopecia	312	51.7	25	4.1	146	24.2	141	23.3
Individual stall	143	59.6	9	3.8	62	25.8	72	30.0
Group housed	169	46.4	16	4.4	84	23.1	69	19.0
Alopecia flank	276	45.7	19	3.2	134	22.2	123	20.4
Individual stall	127	52.9	9	3.8	50	20.8	68	28.3
Group housed	149	40.9	10	2.8	84	23.1	55	15.1
Abscess	20	3.3	0	0.0	3	0.5	17	2.8
Individual stall	7	2.9	0	0.0	1	0.4	6	2.5
Group housed	13	3.6	0	0.0	2	0.6	11	3.0
Swelling	182	30.1	51	8.4	83	13.7	48	8.0
Individual stall	62	25.8	14	5.8	27	11.3	21	8.8
Group housed	120	33.0	37	10.2	56	15.4	27	7.4
Callus	599	99.2	0	0.0	83	13.7	516	85.4
Individual stall	240	100.0	0	0.0	27	11.3	213	88.8
Group housed	359	98.6	0	0.0	56	15.4	303	83.2
Bursitis	132	21.9	66	10.9	50	8.3	16	2.7
Individual stall	48	20.0	26	10.8	18	7.5	4	1.7
Group housed	84	23.1	40	11.0	32	8.8	12	3.3
Capped hock	93	15.4	77	12.8	16	2.7	0	0.0
Individual stall	39	16.3	34	14.2	5	2.1	0	0.0
Group housed	54	14.8	43	11.8	11	3.0	0	0.0

4.4.2.3 Claw lesion prevalence

There were 5.6% pregnant gilts with at least one overgrown dew claw and 15.5% of pregnant gilts with at least one overgrown toe. There were 1.2% pregnant gilts with at least one amputated dew claw, 1.4% of pregnant gilts with at least one broken toe and 2.3% of pregnant gilts with at least one broken dew claw (Table 4.4). No cases of amputated toes were recorded. In pregnant sows, overgrown dew claws and toes were prevalent at 24.2% and 31.0% respectively. Amputated dew claws, amputated toes, broken toes and broken dew claws were 1.3%, 0.5%, 1.5% and 12.25% prevalent (Table 4.4). The range of claw lesion prevalence across farms in pregnant gilts varied for broken dew claws (0-33.3%), broken toes (0-20%), amputated dew claws (0-20%), overgrown dew claws (0-100%) and overgrown main claws (0-90%). The range of claw lesion prevalence across farms in pregnant sows varied for broken dew claws (0-60%), broken toes (0-20%), amputated dew claws (0-20%), amputated toes (0-14.3%), overgrown dew claws (0-100%) and overgrown toes (0-100%). The prevalence of claw lesions varied between housing systems for both pregnant gilts and sows (Table 4.14).

4.4.2.4 Body lesion prevalence

The prevalence of body lesions at the ear, shoulder, flank, hindquarter, anogenital, tail and vulva in 518 pregnant gilts was 82.8%, 71.8%, 65.1%, 65.7%, 31.7%, 26.1% and 7.5% respectively (Table 4.15). The prevalence of body lesions at the ear, shoulder, flank, hindquarter, anogenital, tail and vulva in 604 pregnant sows was 85.6%, 75.0%, 70.4%, 66.6%, 34.3%, 28.3% and 12.6% respectively (Table 4.16). The prevalence of body lesions was higher in group housed gilts and sows for all areas of the body when compared to gestation stalls (Table 4.15 and 4.16).

Table 4.14. Number (n) and prevalence (%) of pregnant gilts and pregnant sows in group housing or individual stalls with lesions present for broken dew claw, broken toe, amputated dew.

	Pregnant gilt				Pregnant sow			
	Individual stall		Group housing		Individual stall		Group housing	
	n	%	n	%	n	%	n	%
Broken dew claw	2	1.1	10	3.0	42	17.5	32	8.8
Front	0	0.0	5	1.5	4	1.7	9	2.5
Hind	2	1.1	6	1.8	40	16.7	28	7.7
Broken toe	2	1.1	5	1.5	6	2.5	3	0.8
Front	1	0.6	0	0.0	1	0.4	1	0.3
Hind	1	0.6	5	1.5	5	2.1	2	0.6
Amputated dew claw	0	0.0	6	1.8	2	0.8	6	1.7
Front	0	0.0	0	0.0	0	0.0	0	0.0
Hind	0	0.0	6	1.8	2	0.8	6	1.7
Amputated toe	0	0.0	0	0.0	1	0.4	2	0.6
Front	0	0.0	0	0.0	0	0.0	1	0.3
Hind	0	0.0	0	0.0	1	0.4	1	0.3
Overgrown dew claw	7	3.9	22	6.5	83	34.6	63	17.3
Front	4	2.2	8	2.4	24	10.0	33	9.1
Hind	3	1.7	20	5.9	83	34.6	45	12.4
Overgrown toe	40	22.1	40	11.9	121	50.4	66	18.1
Front	6	3.3	23	6.8	14	5.8	23	6.3
Hind	36	19.9	30	8.9	119	49.6	54	14.8

Table 4.15. Number (n) and prevalence (%) of pregnant gilts with body lesions present (score ≥ 1) and scores 1, 2, 3 and ≥4 to the ear, shoulder, flank, hindquarter, anogenital, tail and vulva region overall and in group housing or individual stalls.

Lesion type	Present		Score 1		Score 2		Score ≥3	
	n	%	n	%	n	%	n	%
Ear	429	82.8	140	27.0	256	49.4	33	6.4
Individual stall	117	64.6	55	30.4	55	30.4	7	3.9
Group housed	312	92.6	85	25.2	201	59.6	26	7.7
Shoulder	372	71.8	107	20.7	220	42.5	45	8.7
Individual stall	80	44.2	40	22.1	34	18.8	6	3.3
Group housed	292	86.7	67	19.9	186	55.2	39	11.6
Flank	337	65.1	120	23.2	182	35.1	35	6.8
Individual stall	72	39.8	44	24.3	24	13.3	4	2.2
Group housed	265	78.6	76	22.6	158	46.9	31	9.2
Hindquarter	340	65.6	125	24.1	175	33.8	40	7.7
Individual stall	71	39.2	37	20.4	25	13.8	9	5.0
Group housed	269	79.8	88	26.1	150	44.5	31	9.2
Anogenital	164	31.7	93	18.0	67	12.9	4	0.8
Individual stall	23	12.7	17	9.4	6	3.3	0	0.0
Group housed	141	41.8	76	22.6	61	18.1	4	1.2
Tail	135	26.1	95	18.3	36	7.0	4	0.8
Individual stall	35	19.3	26	14.4	9	5.0	0	0.0
Group housed	100	29.7	69	20.5	27	8.0	4	1.2
Vulva	39	7.5	33	6.4	5	1.0	1	0.2
Individual stall	10	5.5	9	5.0	0	0.0	1	0.6
Group housed	29	8.6	24	7.1	5	1.5	0	0.0

Table 4.16. Number (n) and prevalence (%) of pregnant sows with body lesions present (score ≥ 1) and scores 1, 2, 3 and ≥4 to the ear, shoulder, flank, hindquarter, anogenital, tail and vulva region overall and in group housing or individual stalls.

	Present		Score 1		Score 2		Score ≥3	
	n	%	n	%	n	%	n	%
Ear	518	85.8	201	33.3	289	47.9	28	4.7
Individual stall	183	76.3	88	36.7	90	37.5	5	2.1
Group housed	335	92.0	113	31.0	199	54.7	23	6.3
Shoulder	453	75.0	105	17.4	253	41.9	95	15.7
Individual stall	115	47.9	41	17.1	48	20.0	26	10.9
Group housed	338	92.9	64	17.6	205	56.3	69	19.0
Flank	425	70.4	115	19.0	250	41.4	60	9.9
Individual stall	117	48.8	49	20.4	60	25.0	8	3.3
Group housed	308	84.6	66	18.1	190	52.2	52	14.3
Hindquarter	402	66.6	132	21.9	211	34.9	59	9.8
Individual stall	101	42.1	48	20.0	47	19.6	6	2.5
Group housed	301	82.7	84	23.1	164	45.1	53	14.6
Anogenital	207	34.3	124	20.5	75	12.4	8	1.3
Individual stall	45	18.8	33	13.8	11	4.6	1	0.4
Group housed	162	44.5	91	25.0	64	17.6	7	1.9
Tail	171	28.3	124	20.5	43	7.1	4	0.7
Individual stall	58	24.2	43	17.9	14	5.8	1	0.4
Group housed	113	31.0	81	22.3	29	8.0	3	0.8
Vulva	76	12.6	45	7.5	16	2.7	15	2.5
Individual stall	17	7.1	15	6.3	2	0.8	0	0.0
Group housed	59	16.2	30	8.2	14	3.9	15	4.1

4.4.2.5 Lameness prevalence

The prevalence of lameness in pregnant gilts was 41.1% with 213 animals scored ≥2 for locomotory ability (Table 4.6). Scores 4 and 5 both had a prevalence of 0.2%. (n = 1). The prevalence of lameness in pregnant sows was 41.7% with 252 sows scored ≥2 for locomotory ability (Table 4.6). Scores 4 and 5 had a combined prevalence of 0.5%. (n = 3). Prevalence of lameness varied between housing system, pregnant gilts and sows in group housed systems had a lameness prevalence of 48.1% (n = 337/701) while in individual stalls lameness prevalence was 30.4% (128/421). The prevalence range in lameness between farms was 0 to 100% for both pregnant gilts and sows.

4.4.2.6 Risk factors for lameness, limb, claw and body lesions

There was an increased risk of lameness in group housing systems when compared with individual stalls for pregnant gilts and sows (OR 3.66; CI 1.23, 3.66). No risk factors for lameness in pregnant gilts and sows in individual stalls were found. The use of one genetic companies maternal line reduced the risk of lameness in gilts and sows in group housed systems compared with the only other genetic company observed in this study (OR 0.38; CI 0.19, 0.74). No other risk factors were identified.

There was a higher risk of lesions to the body at the ear (OR 4.83; CI 1.53, 15.23), shoulder (OR 10.29; CI 4.15,25.53), flank (OR 5.50; CI 2.92, 10.36), hindquarter (OR 6.30; CI 3.34, 11.87) and anogenital (OR 4.94; CI 1.22, 20.00) regions in sows and gilts in group housing systems when compared with individual stalls (OR 3.66; CI 1.23, 2.12). In individual stalls only, pregnant sows had a lower risk of tail lesions (OR 0.22; 0.11, 0.44) and a higher risk of vulvar lesions (OR 5.98; 2.87, 12.46) than pregnant gilts. In group housed system pregnant sows had a higher risk of lesions to the ear, shoulder flank and hindquarter than pregnant gilts (Table 4.17). In group housing systems there was a reduced risk of having lesions to the flank and hindquarter when pigs were in housed in group sizes 60-206 when compared with pigs housed in groups < 8 (Table 4.17). There was also a reduced risk of having lesions to the ear when pigs were housed in pens >25m^2 when compared to pigs

housed with smaller pen areas (<15m^2) (Table 4.17). There was an increased risk of shoulder lesions when concrete walls were used in the pen compared with other wall materials (Table 17).

There was an increased risk of scratches to the limbs (OR 2.15; CI 1.25, 3.69) in pregnant gilts and sows in group housing systems compared with individual stalls. In both individual stalls (OR 3.53; CI 2.21, 5.65) and group housing (OR 2.03; CI 1.45, 2.85) pregnant sows had a higher risk of having alopecia of the flank than pregnant gilts. In sows kept in individual stalls there was a higher risk of bursitis (OR 2.29; CI 1.06, 4.96) when there was partially slatted flooring beneath the crate compared with fully slatted flooring only. In group housed pregnant sows, there was a higher risk of bursitis in sows than gilts (Table 4.18). There was also a higher risk of having scratches and wounds on the limbs in ESF group systems compared with long trough systems and a lower risk of alopecia when plastic walls were used in the pen (Table 4.18). There was a lower risk of capped hock in group housed systems when the pen floor was partially slatted when compared to pens that were entirely slatted (Table 4.18).

There was a reduced risk of overgrown toes (OR 0.29; CI 0.14, 0.61) in pregnant gilts and sows housed in groups compared with individual stalls. In individual stalls, pregnant sows had a higher risk of having overgrown toes (OR 4.45; CI 2.65, 7.46), overgrown dew claws (OR 18.49, CI 7.54, 45.33) and broken dew claws (OR 17.55; CI 4.23, 72.87) than pregnant gilts. In group housing, pregnant sows also had a higher risk of having overgrown toes (OR 1.84; CI 1.02, 3.31), overgrown dew claws (OR3.53; CI 1.02, 3.31) and broken dew claws (OR 2.75; CI 1.32, 5.75) than pregnant gilts.

4.4.2.7 Correlations between lesions

Several limb and claw lesions were correlated to one another other and lameness in pregnant gilts and sows (Table 4.19).

Table 4.17. Multilevel binomial models of the risks associated with group housed pregnant gilts and sows from for body lesions of the shoulder, flank, tail and anogenital.

	Ear			Shoulder			Flank			Hindquarter		
Intercept coefficient	-1.38			-1.10			1.02			1.11		
	OR	CI		OR	CI		OR	CI		OR	CI	
Pregnant gilt												
Pregnant sow	1.02	0.45	2.33	2.22	1.16	4.24	1.54	0.85	2.79	1.11	0.64	1.94
Pen area												
<15m^2												
15-25m^2	0.53	0.17	1.60									
>25m^2	0.29	0.09	0.97									
Number/group												
<8												
8-25							1.41	0.76	2.62	1.41	0.76	2.63
26-206							0.34	0.12	0.92	0.28	0.10	0.80
Concrete wall				2.69	1.28	5.66						
Random effects		Var	SE		Var	SE		Var	SE		Var	SE
Farm		0.00	0.00		0.32	0.40		0.00	0.00		0.16	0.29
Pen		1.58	0.53		0.71	0.47		0.96	0.28		0.76	0.37

Table 4.18. Multilevel binomial models of the risks associated with group housed pregnant gilts and sows for the limb lesions; scratches, wounds, alopecia, alopecia flank, bursitis and capped hock.

	Scratch			Wound			Alopecia			Alopecia flank			Bursitis			Capped hock		
Intercept coefficient	0.45			-1.33			0.59			-1.00			-1.60			-1.50		
	OR	CI		OR	CI		OR	CI		OR	CI		OR	CI		OR	CI	
Pregnant gilt																		
Pregnant sow	1.10	0.76	1.60	1.34	0.95	1.90	1.07	0.76	1.50	2.03	1.45	2.85	1.55	1.04	2.31	1.43	0.72	2.82
Feeder																		
Long trough																		
Short feeder	1.51	0.72	3.16	1.65	0.79	3.44												
Free access stalls	2.73	1.48	5.03	2.52	1.43	4.44												
ESF	3.71	1.62	8.50	3.46	1.64	7.28												
Flooring																		
Fully slatted																		
Partially slatted																0.28	0.14	0.57
Plastic wall							2.03	1.45	2.85									
Random effects		Var	SE		Var	SE		Var	SE		Var	SE		Var	SE		Var	SE
Farm		0.49	0.21		0.47	0.19		1.53	0.38		1.00	0.27		1.34	0.34		0.81	0.33
Pen		0.04	0.16		0.04	0.14		0.00	0.00		0.00	0.00		0.00	0.00		0.02	0.24

Table 4.19. Significant correlations between lameness and limb lesions for pregnant gilts and sows.

	Lameness	Scratch	Wound	Alopecia	Alopecia flank	Abscess	swelling	Callus	Bursitis	Capped hock	Broken Dew claw	Broken toe	Amputated dew claw	Amputated toe	Overgrown dew claw	Overgrown toe
Lameness	1															
Scratch	0.02	1														
Wound	0.02	0.27***	1													
Alopecia	0.01	0.23***	0.14***	1												
Alopecia flank	0.03	0.04	0.05	0.18**	1											
Abscess	0.10**	0.01	0.11**	0.02	0.01	1										
Swelling	0.05	0.10**	0.15***	0.22**	0.00	0.24***	1									
Callus	0.06	0.05	0.06	0.08*	0.20***	0.01	0.03	1								
Bursitis	0.00	0.04	0.09*	-0.01	0.00	0.01	0.10**	-0.11**	1							
Capped hock	0.00	-0.03	-0.02	-0.1**	-0.01	-0.01	-0.05	-0.02	0.07*	1						
Broken dew claw	0.02	-0.03	0.05	0.04	0.11**	0.05	0.08*	0.03	0.00	0.01	1					
Broken toe	0.00	-0.04	0.05	0.04	0.12	-0.02	0.00	0.04	-0.01	-0.01	0.19***	1				
Amputated dew claw	0.09	-0.01	0.05	0.04	-0.06*	-0.02	0.05*	0.04	0.05*	-0.02	0.09*	0.12***	1			
Amputated toe	0.07*	0.02	0.03	0.02	-0.01	-0.01	0.01	0.00	-0.02	-0.02	0.05	-0.01	-0.01	1		
Overgrown dew claw	-0.04	0.06	0.03	0.08*	0.15***	0.05	0.11**	0.11**	-0.01	0.03	0.22**	0.13***	0.04	0.02	1	
Overgrown toe	-0.05	-0.10**	0.07*	0.00	0.20***	0.00	0.00	0.07*	-0.04	-0.04	0.19***	0.13***	0.07*	0.01	0.40***	1

* = <0.05, ** = <0.001, *** = <0.0001

4.4.3 Lactating sows

4.4.3.1 Farm features

A total of 544 lactating sows were examined on 68 farms (farrowed 3-7 d [n = 136], 8-14d [n = 136], 15-21d [n = 136] and 22-28d [n = 136]), with a mean body condition score of 2.7 (SD ±0.7). All lactating sows were housed indoors in steel farrowing crates with mainly fully slatted or partially slatted floors under the crate made from concrete or metal. Bedding was not provided. The mean crate area was 1.24 m^2 (±0.20). The mean slat dimensions for the slat positioned below the sow were; slat width 13.7 mm (±1.03) and slat void 10.8 mm (±2.3).

4.4.3.2 Limb lesion prevalence

The prevalence of scratches, wounds, alopecia, alopecia flank, abscesses, swellings, calluses, bursitis and capped hock in 544 lactating sows was 27.0%, 42.1%, 57.0%, 38.6%, 2.9%, 29.4%, 99.8%, 25.7% and 22.4% respectively (Table 4.20). There was a wide range in the prevalence of lesions between farms: scratches (0-87.5%), wounds (0-87.5%), alopecia (0-100%), alopecia flank (0-87.5%), abscesses (0-25%), swellings (0-87.5%), calluses (87.5-100%), bursitis (0-75%) and capped hock (0-100%). The prevalence of limb lesions varied between the front and hind limbs (Table 4.20).

Table 4.20. Number (n) and prevalence (%) of lactating sows with lesions present (score ≥ 1) and scores 1, 2 and 3 for scratches, wounds, alopecia, alopecia flank, swellings, abscesses, calluses, bursitis and capped hock overall and by front and hind limb.

Lesion type	Present		Score 1		Score 2		Score 3	
	n	%	n	%	n	%	n	%
Scratch	147	27.0	138	25.4	8	1.5	1	0.2
Front	94	17.3	89	16.4	4	0.7	1	0.2
Hind	78	14.3	74	13.6	4	0.7	0	0.0
Wound	229	42.1	149	27.4	69	12.7	11	2.0
Front	149	27.4	109	20.0	32	5.9	8	1.5
Hind	134	24.6	85	15.6	45	8.3	4	0.7
Alopecia	310	57.0	34	6.3	141	25.9	135	24.8
Front	293	53.9	28	5.2	139	25.6	126	23.2
Hind	77	14.2	19	3.5	37	6.8	21	3.9
Alopecia flank	210	38.6	20	3.7	103	18.9	87	16.0
Front	210	38.6	20	3.7	103	18.9	87	16.0
Abscess	16	2.9	2	0.4	3	0.6	11	2.0
Front	14	2.6	1	0.2	3	0.6	10	1.8
Hind	3	0.6	1	0.2	1	0.2	1	0.2
Swelling	160	29.4	35	6.4	75	13.8	50	9.2
Front	122	22.4	30	5.5	52	9.6	40	7.4
Hind	62	11.4	14	2.6	33	6.1	15	2.8
Callus	543	99.8	0	0.0	69	12.7	474	87.1
Front	542	99.6	3	0.6	122	22.4	417	76.7
Hind	536	98.5	7	1.3	156	28.7	373	68.6
Bursitis	140	25.7	70	12.9	59	10.9	11	2.0
Front	71	13.1	39	7.2	24	4.4	8	1.5
Hind	88	16.2	45	8.3	39	7.2	4	0.7
Capped hock	122	22.4	91	16.7	27	5.0	4	0.7
Hind	122	22.4	91	16.7	27	5.0	4	0.7

4.4.3.3 Claw lesion prevalence

There were 24.8% pregnant sows with at least one overgrown dew claw and 33.7% of pregnant sows with at least one overgrown toe. There were 0.6% pregnant sows with at least one amputated dew claw, 0.6% pregnant sows with at least one amputated toe, 3.5% of pregnant sows with at least one broken toe and 23.9% of pregnant sows with at least one broken dew claw (Table 4.4).

4.4.3.4 Body lesion prevalence

The prevalence of body lesions at the ear, shoulder, flank, hindquarter, anogenital, tail and vulva in 544 lactating sows was 46.7%, 37.3%, 18.4%, 14.2%, 5.3%, 5.7% and 3.1% respectively (Table 4.21).

Table 4.21. Number (n) and prevalence (%) of lactating sows with body lesions present (score ≥ 1) and scores 1, 2, 3 and ≥4 to the ear, shoulder, flank, hindquarter, anogenital, tail and vulva region.

Lesion location	Present		Score 1		Score 2		Score 3		Score 4+	
	n	%	n	%	n	%	n	%	n	%
Ear	254	46.7	165	30.3	86	15.8	3	0.6	0	0.0
Shoulder	203	37.3	73	13.4	62	11.4	33	6.1	35	6.4
Flank	100	18.4	63	11.6	34	6.3	2	0.4	1	0.2
Hindquarter	77	14.2	47	8.6	25	4.6	3	0.6	2	0.4
Anogenital	29	5.3	18	3.3	9	1.7	2	0.4	0	0.0
Tail	31	5.7	23	4.2	6	1.1	2	0.4	0	0.0
Vulva	17	3.1	14	2.6	3	0.6	0	0.0	0	0.0

4.4.3.5 Ability to stand

The prevalence of lactating sows which would not stand up, were slow to stand, were fast to stand and were already standing were 1.2% (n = 8), 3.5% (n = 19), 65.6% (n = 357) and 29.4% (n = 160) respectively. If categories 1 and 2 are combined the range in farm prevalence was 0-25%.

4.4.3.6 Risk factors for lameness, limb, claw and body lesions

No environmental or management risk factors were identified for ability to stand and claw lesions in lactating sows. There was a reduced risk of lesions to the hindquarter of the body and the time since the sow farrowed (Table 4.22). There was a reduced risk of alopecia on the flank with higher farrowing crate area (>1.2 m^2) and a reduced risk of capped hock (>1.34 m^2) when compared to lower farrowing crate areas (<1.19 m^2) (Table 2.22).

4.4.3.7 Correlations between lesions

Several limb and claw lesions were correlated to one another other and lameness in lactating sows (Table 4.23).

Table 4.22. Multilevel binomial models of the risks associated with lactating sows sows for body lesions of the hindquarter and the limb lesions scratch, alopecia flank and capped hock.

	Hindquarter			Limb scratch			Alopecia flank			Capped hock		
Intercept coefficient	-1.18			0.70			0.14			-0.93		
	OR	CI		OR	CI		OR	CI		OR	CI	
Days post farrowing												
d 3-7												
d 8–14	0.50	0.26	0.94	0.73	0.43	1.23	0.98	0.59	1.62	1.23	0.69	2.21
d 15–21	0.38	0.19	0.76	0.70	0.41	1.19	0.87	0.53	1.45	1.00	0.55	1.84
d 22–28	0.34	0.17	0.69	0.57	0.33	0.99	0.79	0.47	1.30	0.70	0.37	1.33
Crate area												
<1.19 m^2												
1.2-1.34 m^2							0.51	0.29	0.91	0.59	0.34	1.04
1.34-1.94 m^2							0.39	0.21	0.71	0.39	0.21	0.73
Random effects		Var	SE		Var	SE		Var	SE		Var	SE
Farm		0.06	0.19		0.95	0.27		0.77	0.23		0.25	0.18

Table 4.23. Significant correlations between lameness limb and claw lesions for lactating sows sows.

	Standing ability	Scratch	Wound	Alopecia	Alopecia flank	Abscess	swelling	Callus	Bursitis	Capped hock	Broken Dew claw	Broken toe	Amputated dew claw	Amputated toe	Overgrown dew claw	Overgrown toe
Standing ability	1															
Scratch	0.03	1														
Wound	0.02	0.10*	1													
Alopecia	-0.02	0.13**	0.31***	1												
Alopecia flank	-0.04	-0.02	0.08	0.24***	1											
Abscess	0.06	-0.06	1.6	0.04	0.04	1										
Swelling	0.00	0.09*	0.36***	0.27***	0.05	0.25***	1									
Callus	-0.04	0.03	0.095*	0.20***	0.22**	0.06	0.08	1								
Bursitis	0.01	0.01	0.06	-0.05	0.04	-0.02	0.07	0.06	1							
Capped hock	0.03	0.06	0.07	0.03	0.04	-0.02	0.06	0.03	0.07	1						
Broken dew claw	-0.05	0.04	0.15**	0.17***	0.13**	-0.04	0.02	0.11	0.09	0.08	1					
Broken toe	0.08	-0.05	0.01	0.07	0.03	0.04	0.00	0.04	-0.06	-0.01	0.10**	1				
Amputated dew claw	0.09	-0.04	0.04	0.00	0.03	-0.01	0.08	-0.04	0.06	-0.04	-0.04	-0.01	1			
Amputated toe	0.02	0.06	0.01	-0.08	-0.06	-0.01	0.00	0.03	0.03	0.05	0.07	-0.01	-0.01	1		
Overgrown dew claw	0.04	0.02	0.04	-0.05	0.14	0.06	0.00	-0.01	-0.01	0.06	0.23***	0.19***	0.01	-0.04	1	
Overgrown toe	0.04	-0.02	0.06	0.04	0.09*	0.04	0.09*	0.10*	0.03	0.09*	0.17***	0.20***	-0.05	0.00	0.32***	1

* = <0.05, ** = <0.001, *** = <0.0001

4.5 Discussion

This is the first study to examine the prevalence and risk factors for limb lesions, claw lesions, body lesions and lameness in gilts and sows on commercial farms in Ireland and the largest cross-sectional study to date of indoor intensive housing systems. The information produced by this study is valuable to all pig producing countries throughout the world, the majority of which use indoors systems for gilts and sows. The examination of group and stalled systems for pregnant animals provides valuable information for all pig producing countries both within the EU and internationally. Within EU pig producing countries pregnant sows may still be stalled up until 28 days post service after which they must be group housed until 1 week before farrowing. Several pig producing countries such as the United States, Canada and Australia predominantly house their pregnant sows in stalls for their entire pregnancy however there is a shift towards transitioning to group housing due to government and stakeholder pressure. The information provided by this study provides information on the effect of each system on limb health and may highlight issues to be considered when converting system or undergoing development.

The prevalence of lameness in this study is much higher than previously reported for replacement gilts, pregnant gilts and sows (Heinonen *et al.*, 2006; KilBride *et al.*, 2009a; Pluym *et al.*, 2011; Pluym *et al.*, 2013a; Willgert *et al.*, 2014). This may indicate that lameness levels on Irish farms are higher than other European countries, perhaps as a result of an over reliance of fully slatted concrete floors and absence of bedding in Irish systems. Some of the difference may also be a result of the underestimation of lameness due to the different lameness thresholds used in various studies. A variety of other scoring systems for locomotory ability have been used in several other studies investigating gilt and sow lameness thus, influencing the reliability of comparing lameness levels between studies (Dewey *et al.*, 1993; Heinonen *et al.*, 2006; Karlen *et al.*, 2007; Anil *et al.*, 2009; Mustonen *et al.*, 2011; Pluym *et al.*, 2011; Grégoire *et al.*, 2013; Pluym *et al.*, 2013a; Willgert *et al.*, 2014). This study uses the Main *et al.* (2000) classification system similarly to KilBride *et al.*

(2009a) and Calderón Díaz *et al.* (2013) whereby an ordinal scale is used and a lameness threshold is set. Within the use of the Main *et al.* (2000) classification system there are different interpretations of the lameness threshold. KilBride *et al.* (2009a) considered a score of one and greater to be lame while in this study it was 2 or greater similar to Calderón Díaz *et al.* (2013).

Group housing was the main risk factor for lameness in gilts and sows as indicated in previous studies (Harris *et al.*, 2006; Chapinal *et al.*, 2010; Calderón Díaz *et al.*, 2014). In individual stalls, gilt and sow movement is restricted and the freedom of movement and interaction with their environmental and counterparts is limited (Gjein and Larssen, 1995; Pajor, 2002). Individual sows are also more vulnerable to stereotypic behaviours and shoulder ulcers (Gjein and Larssen, 1995; Vieuille-Thomas *et al.*, 1995; Zhou *et al.*, 2014). In group housed systems however while performance is not negatively affected, the associated social interactions make gilts and sows more prone to injury and aggression which are associated with increased lameness levels (Broom *et al.*, 1995; Gjein and Larssen, 1995; Bates *et al.*, 2003; Harris *et al.*, 2006). This increased risk of lameness in group housing systems may be exacerbated in intensive production systems due to the over reliance on slatted flooring and the lack of bedding due to a reluctance to use straw due to the liquid manure systems in use and the associated labour costs (Tuyttens, 2005; Scott *et al.*, 2006). Both slatted flooring and lack of bedding are risk factors for lameness (Heinonen *et al.*, 2006; Scott *et al.*, 2006; KilBride *et al.*, 2009a). A marginal increase in the risk of lameness when using one genetic company for maternal line genetics was observed in this study. The exact role in which this affects lameness cannot be established without a more detailed investigation into the relationship, however, previous studies have identified that aggression, limb soundness and osteochondrosis are heritable in pigs and are also associated with lameness (Lundeheim, 1987; Nakano *et al.*, 1987; Rothschild and Christian, 1988; Breuer *et al.*, 2005; Bench and Gonyou, 2006). No risk factors for lameness were identified for replacement gilts and very few in pregnant gilts and sows. This may be a result of a lack of variation in housing types making it difficult to identify risk factors.

The prevalence of limb swellings, wounds and scratches in replacement gilts, pregnant gilts and pregnant sows was not previously measured and wounds had only been measured in lactating sows previously (KilBride *et al.*, 2009a). We hypothesise that the reduced risk of the presence of limb swelling when replacement gilts were housed separately from terminal stock before 90kg may be a result of reduced agonistic behaviours within a single sex pen and also separation of replacement gilts from terminal stock prior to 50kg may indicate an increased awareness about pig health and welfare amongst farm management (Björklund and Boyle, 2006; Boyle and Björklund, 2007). This heightened awareness of the different requirements of the gilt to terminal stock may result in improved gilt care such as a high selection criteria and improved housing, stocking densities, nutrition and observation. In lactating sows the reduced risk of scratches on the limbs from week 1 post farrowing to week 4 post farrowing is due to healing over time within the farrowing crate as the confinement allows the scratches to heel and the risk for developing new ones is eliminated. The increased risk of scratches and wounds on the limbs in ESF group systems compared with long trough systems for group housed gilts may be the result of aggression on entry to the feeder as suggested by Anil *et al.* (2007). Alopecia at the flank region was most prevalent in pregnant sows. It is likely that this lesion worsens with age of the sow as body size increases the skin at the flank is constantly abrading each other resulting in alopecia of the flank.

Similar to weaner and finisher pigs (Chapter 3) calluses were the most prevalent limb lesions in replacement gilts, pregnant gilts, pregnant sows and lactating sows. Almost all gilts and sows had calluses present, similarly to levels reported by Harris *et al.* (2006) and higher than previously reported by KilBride *et al.* (2009a) (30-80%). We can hypothesise that the high prevalence is a result of the dominance of slatted concrete and absence of bedding, previously mentioned, as both have been identified as increasing the likelihood of calluses (KilBride *et al.*, 2009a). The association between calluses and alopecia is a result of their common aetiology, alopecia are either scars formed after a callus or other injury has healed and the hair has not yet reformed or when pen conditions lead to a low level damage to the

skin but not abrasive enough to result in a callus (Wechsler *et al.*, 2000; KilBride, 2008; Zaffino, 2012).

Both bursitis and capped hock prevalence was lower than in the only other previous study to date to determine the prevalence of these lesions in gilts and sows (32-37%) (KilBride *et al.*, 2009a). This difference may be a result of difference in recording methods between these studies; in this study, bursitis and capped hock was only recorded when an abnormal area was manipulated and a fluid filled sac was palpable otherwise this was recorded as a swelling, also several studies do not differentiate between bursitis and swellings. However in agreement with other studies, bursitis was most prevalent in the hind limbs (KilBride, 2008; KilBride *et al.*, 2009a).

This is the first cross-sectional study to determine the prevalence of overgrown dew claws, amputated dew claws, amputated toes, broken dew claws and broken toes in gilts and sows. The prevalence of overgrown toes is higher in this study than the only previous study to quantify the prevalence of this lesion in gilts and sows, however both dew claw overgrowth and uneven toes were combined in the previous study (KilBride, 2008). Anil *et al.* (2007) suggested increased body weight increase's susceptibility to claw lesions which may explain the higher incidence of overgrown dew claws and toes in this study in pregnant sows when compared to pregnant gilts. We can hypothesise the reduced risk of overgrown toes in pregnant gilts and sows group housed compared with individual stalls is due the increased growth to wear ratio in group housed systems than confined gestation stalls. The positive association between overgrown dew claws and overgrown toes, although at a low level of association, is similar to the findings of Anil *et al.* (2009), they hypothesised this association is due to an overall physiological defect in the growth of the claw. We believe the increased risk of broken dew claws in pregnant sows is due to the increased prevalence of overgrown dew claws as once the dew claw is elongated it is more vulnerable to damage from the slatted flooring and during aggression, supported by Jackson and Cockcroft (2007) and Pluym *et al.* (2011). The positive correlation between both overgrown dew claws and overgrown toes and

broken dew claws and broken toes goes to further strengthen our hypothesis that the elongation makes the claw more vulnerable to damage. Low prevalence of severe lesions such as dew and toe amputations may indicate a low occurrence of these injuries but they also may have been underestimated as these lesion types are the result of severe claw trauma and may result in a high level of pain for the animal and so may require isolation and treatment and therefore they would not have been sampled in this study. In some cases these lesions are also so severe that they result in the affected animal being culled/euthanized.

Not surprisingly there was a higher risk of body lesions (ear, shoulder, flank, hindquarter, anogenital and limb scratches) in group gestation housing systems when compared with individual gestation stalls as aggressive interactions between sows are not possible in the latter. In group housing systems only, pregnant gilts had a higher risk of aggression indicating injures than pregnant sows. We can hypothesise this is a result of the dominance hierarchy, young gilts are positioned lower down in the dominance hierarchy than multiparous sows and so receive less aggression induced injuries.

4.6 Conclusions

These findings have implications for replacement gilt, pregnant gilt, pregnant sow and lactating sow management. There is a high prevalence of lameness in replacement gilts, pregnant gilts and pregnant sows when compared to previously reported levels. The use of group housing systems is associated with increased levels of lameness levels and body lesions and a reduced incidence of overgrown weigh bearing claws. However due to the uniformity of pen environments identification of risk factors is difficult. Further research is required to determine the effect of a variety floor types and materials on lameness, limb lesions and claw lesions.

Chapter 5

The effect of feeding a diet formulated for developing gilts between 70kg and ~140 kg on lameness indicators and carcass traits

5.1 Abstract

This study investigated the effect of three dietary regimes for replacement gilts on lameness and carcass traits. Diets were: a diet specifically formulated for replacement gilts (diet 1, 14.04 MJ of DE/kg, 0.75% lysine), a finisher diet (diet 2, 13.54 MJ of DE/kg, 1.02% lysine) and a gestation sow diet (diet 3, 12.96 MJ of DE/kg, 0.69% lysine): the latter two diets are traditionally fed to replacement gilts. Thirty-six gilts were selected at d0 (70.8 kg ±0.78 SE, aged ~130d), housed individually and allocated at random to 1) DEV (restricted access diet 1, n = 12), 2) FIN (*ad-libitum* access diet 2, n = 12) or 3) GES (initially *ad-libitum* access diet 2, then restricted access diet 3 from d29, n = 12) treatments. All gilts were fed *ad-libitum* from d70-83. Locomotory ability (0 = normal to 5 = severely impaired) and limb lesions (0 = normal to 3 = severe) were scored weekly until d82. Hind-claw lesions and overgrowth were scored (0 = normal to 3 = severe) at d0, 40 and 82. Gilts were weighed at d0, 28, 70 and 82. Carcass traits were recorded at slaughter (d83) and one front leg was removed for dual-energy x-ray absorptiometry (DXA) to establish areal bone mineral density (aBMD). Joint surface lesions of the humeral condyle (HC; 1 = normal to 4 = severe, 5 = osteochondrosis dissecans) and anconeal process (AP; 1 = absent 2 = present) were scored. The percentage of lame animals (locomotion score ≥2) on ≥1 occasion were: DEV = 0% (0/11), FIN = 72% (8/11) and GES = 75% (9/12) ($P<0.01$). Fewer DEV gilts had humeral condyle lesions than FIN and GES gilts: DEV = 64% (7/11), FIN = 100% (11/11) and GES = 100% (12/12) ($P<0.01$). DEV gilts had lower scores for humeral condyle lesions (median; IQR: 2; 2) than GES (4; 1) and FIN gilts (4; 3) ($P = 0.05$). DEV gilts weighed less (132.3 kg ±2.05)

than FIN (142.2 ±2.05) gilts at d70 (P<0.05). DEV gilts had a lower ADFI than FIN and GES gilts over the trial period (P<0.05). DEV gilts had lower ADG than FIN (P<0.05) and GES (P<0.001) gilts at d0-28, lower energy intake than FIN and GES gilts from d0-28 (P<0.001) and higher energy intake than FIN gilts at d71-82. The DEV diet formulated for replacement gilts and fed restrictively from 70kg to 2 weeks before target service at ~140kg improved locomotory ability, claw evenness and humeral condyle joint lesions compared with traditional diets. It is likely these improvements were related to slower growth rate.

5.2 Introduction

Lameness is a major cause of poor welfare and premature culling, particularly in gilts and young sows (Dewey *et al.*, 1993; Boyle *et al.*, 1998; Anil *et al.*, 2009), with between 15 and 20% of gilts and first and second parity sows are culled due to lameness (D'Allaire *et al.*, 1987; Lucia *et al.*, 2000). The high levels of lameness in gilts and sows observed in Chapter 4 further support this and additionally highlight the requirement of novel strategies to address lameness as no environmental risk factors have been identified.

It is common practice to feed diets formulated for finisher pigs to replacement gilts through development until service or to switch from a finisher diet to a gestating sow diet at the end of the finishing period (Boyd *et al.*, 2002). Such regimes are not likely to meet the nutritional requirements of developing gilts. A gestating sow diet is formulated for an animal that has completed growth and key requirements are maintenance of appropriate body composition and meeting the amino acid demand (Harper *et al.*, 2002). In contrast, finisher diets are formulated to maximise lean tissue growth (Harper *et al.*, 2002). Diets for developing gilts should aim to prepare them for maximum lifetime performance by satisfying the nutritional requirements for reproductive performance and bone, joint and claw health and integrity (Sørensen *et al.*, 1993; Levis *et al.*, 1997; Gill and Taylor, 1999; Knauer *et al.*, 2012). Previous studies found that high growth rates in gilts are associated with premature culling for lameness (Sørensen *et al.*, 1993; Gill and Taylor, 1999) while optimum

dietary supplementation with Ca, P, Zn, Cu and Mg improve claw and leg health (Ferket *et al.*, 2009; Anil, 2011). Several studies have examined the use of gilt nutritional strategies on future reproductive performance (Sørensen *et al.*, 1993; Levis *et al.*, 1997; Gill and Taylor, 1999; Klindt *et al.*, 1999; Miller *et al.*, 2011; Knauer *et al.*, 2012), while other studies have examined the effect of feed intake on joint health (Carlson *et al.*, 1988; van Grevenhof *et al.*, 2011). No studies to date however have examined the potential benefits of restricted gilt developer diets on limb health and ultimately the potential increased longevity as a consequence of reduced limb-related culling.

It is hypothesised that feeding gilts with restricted access to a specially formulated developer diet during development could lead to improvements in limb health which would reduce premature culling for lameness and improve farm profitability. Therefore the aims of this study were to investigate the effect of restricted feeding of a developer diet (DEV) to replacement gilts from 70kg to 2 weeks before target service at ~140kg compared with either restricted feeding of a gestating sow diet (GES) from 100kg or *ad-libitum* provision of a finisher (FIN) diet from 70kg to 2 weeks before target service at ~140kg on indicators of lameness and carcass characteristics.

5.3 Methods

5.3.1 Care and use of animals

The research farm (Pig Development Department, Teagasc, Moorepark, Fermoy, Co. Cork Ireland) where the experimental work was carried out was compliant with Statutory Instrument number 311 of 2010 European communities (Welfare of Farmed Animals) Regulations 2000. Licensing under the European Communities (Amendment of Cruelty to Animals Act. 1876) Regulations (2002) was not required as no invasive procedures were employed in the study. This trial was conducted over an 83d period between April and June 2012.

Gilts (Large White x Landrace) were weaned at 28 days of age into single sex pens with 14 pigs per pen. Gilts were provided with *ad-libitum* access to dry pelleted feed until 84 days of age after which they were liquid fed a finisher diet (13.7 MJ of DE/kg, 0.4% lysine) three times per day until selection for the experiment (~116 days of age). Eighteen gilts were selected at 62.5kg (±0.55) and transferred to individual pens where they were allowed to acclimatise to the novel environment and feeding arrangement until they reached 70.8 kg (±0.78) (~130d). Gilts were housed in two identical rooms, each with 18 individual pens. Pen dimensions were 1.81 × 0.9 m and each pen was fully slatted with concrete slats (75 mm solid width, 20 mm slots). Air temperature was maintained at 20 - 22°C and ventilation was provided by a cross flow system (Stienen PCS 8200; Stienen BV). All pigs were fed manually from a stainless steel (300 mm width) trough (O'Donovan Engineering, Coachford, Co. Cork, Ireland) and had continual access to water provided by a nipple in bowl drinker (BALP, Charleville-Mezieres, Cedex, France). Rubber pipes suspended from a chain were provided as environmental enrichment.

A health assessment was carried out at selection (d-14) and post acclimatisation (d0) to ensure that none of the gilts were lame, injured, or sick. Thereafter, pigs were inspected twice daily and sick or injured animals were treated immediately; all veterinary treatments were recorded. Two gilts were removed from the trial due to morbidity (DEV = 1 [refusal to eat], FIN = 1 [infection]) and their data were excluded from the analysis. All gilts were slaughtered at d83 (~ 140kg aged ~212d), which corresponds to the approximate target weight for 1st service of replacement gilts.

5.3.2 Diet formulations

During the acclimatisation period gilts were provided with *ad-libitum* access to a finisher diet (13.54 MJ of DE/kg, 1.02% lysine). On d0 (i.e. when gilts weighed 70.8 kg; ±0.78), gilts were blocked on weight and lameness score and randomly assigned to the dietary regimens as shown in Table 5.1. Treatment DEV involved restricted feeding (2.25 kg/d) of diet 1 from d0-70 followed by *ad-libitum* access to diet 1 (Table 5.2) until d83 to mimic 'flushing' (Table 5.1). Diet 1 was a gilt developer diet

with a high energy to lysine ratio (14.04 MJ of DE/kg, 0.75% lysine), high Ca and P levels and in addition to the normal vitamin and mineral premix included a dietary supplement containing zinc, copper and manganese (Table 5.2). Treatment FIN involved *ad-libitum* access to diet 2 (13.54 MJ of DE/kg, 1.02% lysine) for the entire 83 d period (Table 5.1). Diet 2 was a standard finisher diet (Table 5.2). Treatment GES provided *ad-libitum* access to diet 2 (12.96 MJ of DE/kg, 0.69% lysine) until d28, followed by restricted feeding of diet 3 at 2.25kg/d until d70 followed by *ad-libitum* access to diet 3 until d82 (~ 140kg, aged 212d) to mimic the practice of flushing (Table 5.1). Diet 3 was a standard gestating sow diet (Table 5.2). All feed was provided in dry pelleted form (3mm diameter). Diet 1 and 3 provided Ca and digestible P that met NRC (2012) recommendations for growing gilts, Diet 2 was below the NRC (2012) recommendations for growing gilts. All diets were formulated in the on-site mill at the research farm (Pig Development Department, Teagasc, Moorepark, Fermoy, Co. Cork Ireland) by an experienced nutritionist.

Table 5.1. Dietary regimes for individually housed replacement gilts in three treatments fed to from 70 to 140kg.

Weight range (kg)	Dietary treatment		
	Developer	Finisher	Gestating sow
70 – 100	Diet 1 (2.25 kg/d)	Diet 2 (*ad-libitum*)	Diet 2 (*ad-libitum*)
100 - 130	Diet 1 (2.25 kg/d)	Diet 2 (*ad-libitum*)	*Diet* 3 (2.25kg/d)
130 - 140	Diet 1 (*ad-libitum*)	Diet 2 (*ad-libitum*)	Diet 3 (*ad-libitum*)

Table 5.2. Composition of the experimental diets (on an air-dry basis, %, as fed) for individually housed replacement gilts.

Item	Diet 1	Diet 2	Diet 3
Ingredients			
Barley	81.20	50.00	89.74
Wheat	0.00	34.87	0.00
Soybean	10.31	12.00	7.00
Soya oil	6.00	1.00	1.00
Lysine HCl	0.10	0.40	0.10
DL-Methionine	0.00	0.10	0.00
L-Threonine	0.00	0.12	0.00
Premix 1[a]	0.00	0.10	0.00
Premix 2[b]	0.15	0.00	0.15
Phytase	0.01	0.01	0.01
Salt feed grade	0.40	0.30	0.40
Di-Calcium phosphate	0.65	0.00	0.50
Limestone flour	1.10	1.10	1.10
Availa Sow® [c]	0.09	0.00	0.00
Chemical composition			
Dry matter	87.50	86.90	87.20
Crude protein	12.60	14.70	12.30
Crude Fibre	3.80	3.00	3.50
Total oil	9.26	3.54	3.62
Ash	3.70	3.70	3.60
Lysine	0.75	1.02	0.69
Threonine	0.48	0.64	0.45
Methionine	0.22	0.33	0.21
Methionine + Cystine	0.48	0.63	0.47
Tryptophan	0.17	0.19	0.16
Calcium [d]	0.76	0.61	0.69
Phosphorous [d]	0.49	0.37	0.46
Digestible phosphorus [d]	0.33	0.24	0.32
Digestible energy (MJ of DE/kg) [d]	14.04	13.54	12.96

[a] Premix provided per kilogram of complete diet 1 and 3: Cu, 15 mg; Fe, 70 mg; Mn, 62 mg; Zn,80 mg; I, 1 mg; Se, 0.4 mg; vitamin A, 10,000 IU; vitamin D3, 1,000 IU; vitamin E, 100 IU; vitamin K, 2.0 mg; vitamin B_{12}, 15 µg; riboflavin, 5 mg; nicotinic acid, 12 mg; pantothenic acid, 10 mg; choline chlorine, 500 mg; Biotin, 200 µg; Folic acid, 5 mg vitamin B_1,2 mg and vitamin B_6,3 mg.

[b] Premix provided per kilogram of complete diet 2: Cu, 15 mg; Fe, 24 mg; Mn, 31 mg; Zn, 80 mg; I, 0.5 mg; Se, 0.4mg; vitamin A, 2,000 IU; vitamin D3, 500 IU; vitamin E, 40 IU; vitamin K, 4 mg; vitamin B_{12}, 15 µg; riboflavin, 2 mg; nicotinic acid, 12 mg; pantothenic acid, 10 mg; vitamin B_1, 2 mg; and vitamin B_6,3 mg

[c] Availa Sow® provided per kilogram of complete Diet, Zn, 50 mg; Mn, 20 mg; Cu,10 mg.

[d] Calculated from standard book values for ingredients.

5.3.3 Measurements

Feed intake was measured weekly. Locomotory ability and limb lesions were scored weekly. Gilts were weighed on d0, 29, 70 and 82 of the experiment. Claw lesions were scored on d0, 42 and 82. Gilts were slaughtered on d83. Multiple recording sheets were used (Appendix 5).

5.3.3.1 Locomotory ability

Locomotory ability was assessed using the gait and standing posture aspects of the protocol described by Main *et al.* (2000) and as described in Chapter 3. Gilts were removed from their pen and walked on a fully slatted concrete corridor for a minimum of 6 m.

5.3.3.2 Limb lesions

All four limbs were examined. Lesions were categorised and scored as per KilBride *et al.* (2009a) and as described in Chapter 3. Due to lack of variability in limb lesion scores lesions were later defined as present (1) or absent (0) and reassigned a score based on severity to yield a total lesion score using a method adapted from Boyle *et al.* (2000) and originally described by de Koning (1985); score 1 = scratch, alopecia, callus, score 2 = swelling and score 3 = wound, abscess, bursitis and capped hock.

5.3.3.3 Claw lesions and uneven claw size

Claw inspections were carried out by raising gilts 0.75 m using a hydraulic chute (FeetFirst Sow Chute; Zinpro Performance Minerals, Eden Prairie, Minnesota, USA). Only the hind feet were examined. Lesions on the sole and heel of the medial and lateral toes and the dew claws of each foot were inspected and scored. The following lesions were examined: heel overgrowth, heel erosion, heel-sole separation, white line separation, dew claw crack, toe crack (horizontal and vertical), dew claw overgrowth and toe overgrowth. The scoring system used was a modified version of the FeetFirst™ claw lesion scoring guide (Zinpro Corporation) as described by Calderón Díaz *et al.* (2013). Lesions were scored from 0 (no lesion) to 3

(severe lesion) based on severity. Overgrown weight bearings claws and dew claws were scored from 0 (not uneven) to 3 (severely uneven) based on severity (Appendix 3).

5.3.3.4 Feed intake and growth performance

Gilts were weighed on d0, 29, 70 and 82 of the experiment and average daily gain (ADG) was calculated. Average daily feed Intake (ADFI) was calculated in grams per day. Gain/feed was calculated as ADG divided by daily feed intake in grams per kilogram. Energy intake (MJ DE/kg/day) was calculated by multiplying the predicted DE content of the diet (MJ DE/kg) by the average daily feed intake (kg/day) of the gilts.

5.3.3.5 Slaughter and carcass traits

Pigs were fasted for 15 hours prior to slaughter. On the d83 (day of slaughter), gilts were mixed and transported 90 km. Gilts were slaughtered by exsanguination post CO^2 stunning. After evisceration, carcass traits were measured by slaughtering facility staff. Muscle depth and backfat thickness were measured using a Hennessy grading probe (Hennessy and Chong, Auckland, New Zealand) 60 mm from the edge of the split back, at the level of the third and fourth last rib. Lean content was calculated using the following formula (Department of Agriculture and Food, Ireland, 2001): Estimated lean meat content (%) = 60.30 – 0.847x + 0.147y, where x = backfat depth (mm); y = muscle depth (mm). Carcass weight (cold) was estimated as the weight of the hot eviscerated carcass (minus the tongue, bristles, genital organs, kidneys, flare fat and diaphragm) 45 minutes post-slaughter x 0.98. Dressing out (%) was calculated as: (carcass weight/live weight prior to slaughter) x 100. The front right limb was dissected dorsal to the elbow joint, identified by a tag and frozen within 2 hours at -20°C for DXA scanning and elbow joint surface cartilage scoring at a later date.

5.3.3.6 Areal bone mineral density (aBMD)

The front right limb was DXA scanned to determine aBMD (g/cm^2) by a Hologic QDR-4500 Elite bone densitometer, using the "left forearm" option and analysed using Apex software version 2.3.1.

5.3.3.7 Joint surface lesions

The front right limb was dissected at the elbow joint to expose the humeral condyle (HC) and anconeal process (AP) surfaces as these are the most commonly and severely affected joint surfaces in pigs (Jørgensen, 1995). Furthermore, as lesions tend to be bilaterally symmetrical in the limbs only one limb was examined. Abnormalities of the articular surface of the HC and AP were examined and scored (Grondalen, 1974; Jørgensen, 1995; Ytrehus *et al.*, 2007; Kirk *et al.*, 2008; Jensen and Toft, 2009) (Appendix 4). Joint lesions were scored on the HC from 1 (normal) to 4 (severe abnormality) and a score of 5 was reserved for cases of osteochondrosis dissecans (OCD) only and as 1 (present) or 2 (absent) on the AP as per Jørgensen *et al.* (1995), Christensen *et al.* (2010) and Busch and Wachmann (2011) (Appendix 4).

5.3.4 Laboratory analysis of diets

Representative samples of each diet were taken at 3 times during the study (d0, d40 and 82). Samples were ground using a laboratory hammer mill (Christy and Norris, Scunthorpe U.K.) through a 2 mm screen. Proximate and amino acid analyses were carried out by a commercial laboratory (Sciantec Analytical Services Ltd., Cawood, UK).

5.3.5 Data management

Data were entered into a Microsoft Access 2003 database by AQ. All data were checked for outliers (none were discovered), and impossible values were checked against the raw data.

5.3.6 Statistical analysis

Data were analysed in SAS V9.3 (Statistical Institute Inc., Cary, North Carolina), using the individual gilt as the experimental unit. Differences between the treatments in scores for locomotory ability, limb lesions, claw lesions and joint cartilage lesions were investigated using the Kruskal–Wallis test (Proc NPAR1WAY). Pair-wise comparisons were then carried out if the Kruskal–Wallis test produced a significant result using the Wilcoxon Rank test. Fisher's exact test was used to analyse differences between treatments for the presence or absence of lameness, claw lesions, uneven claw size, joint surface lesions and OCD (joint lesion score 5). A gilt was categorised as being not lame (score ≤1) or lame (score ≥2).

Carcass characteristics, aBMD, and growth performance parameters were analysed using one way ANOVA (Proc MIXED) and body weight using repeated measures one way ANOVA (Proc MIXED). The model included fixed effects of treatment and batch, and period where appropriate. Least squares means were determined and *P*-values were adjusted for multiple comparisons using the Tukey-Kramer adjustment. Pearson correlations between variables were analysed (Proc CORR). Non normal data (ADFI d0-28 and ADFI d29-70) were analysed using the Kruskal Wallis test.

5.4 Results

5.4.1 Locomotory ability

The percentage of animals with locomotion score ≥2 (i.e. lameness) on at least one occasion during the experiment were DEV = 0% (0/11), FIN = 72% (8/11) and G = 75% (9/12) (Table 5.3). The incidence of lameness increased over time from d29-56 ($P<0.05$) and d57-82 ($P<0.05$) for treatments FIN and GES (Table 5.3).

Table 5.3. Number and % of individually housed replacement gilts on three dietary treatments with locomotion score ≥2, claw and limb lesions during the experimental period.

Variables	DEV		FIN		GES		P-value
n	11		11		12		
	n	%	n	%	n	%	
Lame (≥2 locomotion score)							
d0	0	0.0	0	0.0	0	0.0	1
d0-28	0	0.0	1	9.1	1	8.3	1
d29-56	0	0.0	4	36.4	6	50	0.02
d57-82	0	0.0	6	54.6	5	41.7	0.01
d0-82	0	0.0	8	72.7	9	75.0	<0.001
Claw lesion, present							
d0	6	54.6	6	54.6	6	50.0	1
d40	5	45.5	8	72.7	10	83.3	0.18
d82	9	81.8	11	100	11	91.7	0.51
Claw uneven, present							
d0	10	90.9	11	100	11	91.7	1
d40	9	81.8	11	100	12	100	0.2
d82	3	27.3	11	100	12	100	<0.001
Joint surface lesions, present							
HC	7	63.6	11	100	12	100	0.01
AP	5	45.5	8	72.7	7	58.3	0.47
OCD present or absent							
HC	3	27.3	4	36.4	3	25.0	0.89

5.4.2 Limb lesions

There was no effect of treatment on total limb lesion score and there was a 100% prevalence of limb lesions in all gilts. Medians and IQR are reported in Table 5.4. No correlation was found between the locomotory ability and limb lesion scores.

Table 5.4. Median and inter quartile range (IQR) total limb lesion score for three dietary treatments for individually housed replacement gilts.

	DEV		FIN		GES	
	Median	IQR	Median	IQR	Median	IQR
d0	8	1	7	2.5	7.5	1.5
d0-28	6	5	5	6	4	4
d29-56	7	6	4	5.25	4	3
d57-82	7.5	5.25	7.5	6	5	4

5.4.3 Claw lesions and uneven claw size

DEV gilts had a lower occurrence of overgrown toes at d82 than FIN and GES gilts ($P<0.001$) (Table 5.3). There was no significant effect of treatment on claw lesion prevalence or score. DEV gilts had a numerically lower score for overgrown toes at d40 (2; 1 [median; IQR]) and d82 (0; 0.5) compared with FIN and GES gilts (2; 0) and lower scores than FIN gilts at d82 (2; 0) ($P = 0.2$). No correlation was found between the locomotion and claw lesion scores or between locomotion scores and uneven claw size.

5.4.4 Feed intake and growth performance

The effect of dietary treatment on pig growth is presented in Table 5.5. Gilts were weighed at d0 (70.8 kg; ±0.78), d29 (101.3 kg; ±1.22), d70 (137.4 kg; ±1.61) and d82 (145.0kg ±1.45). The DEV gilts weighed less than FIN ($P<0.05$) at d70 and had a lower ADFI than FIN and GES gilts from d0-28 ($P<0.001$) and d29-70 ($P<0.05$). DEV ($P<0.05$) and GES ($P<0.001$) gilts had a higher ADFI than FIN gilts from d71-82 ($P<0.05$). Overall DEV gilts had a lower ADFI than FIN and GES gilts over the trial period ($P<0.05$). DEV gilts had a lower ADG than FIN ($P<0.05$) and GES ($P<0.001$) gilts

from d0-28 and GES gilts had a lower ADG than FIN from d29-70 ($P<0.05$). Overall DEV gilts had a lower ADG than FIN gilts over the trial period ($P<0.05$). DEV gilts had lower energy intake than FIN and GES gilts from d0-28 ($P<0.001$) and higher energy intake than FIN gilts at d71-82. Overall DEV gilts had a lower energy intake than FIN gilts over the trial period ($P = 0.05$).

Table 5.5. Effect of dietary regime on performance indicators (LSM[1] ±SEM) of individually housed replacement gilts from 70 to 140kg.

	Treatment							Treatment comparison		
	DEV	SEM	FIN	SEM	GES	SEM	T	DEV v. FIN	DEV v. GES	FIN v. GES
n	11		11		12		P-value			
Weight, kg										
d0	71.5	1.18	70.4	1.22	69.9	1.14	0.58	1.00	1.00	1.00
d29	96.1	1.82	102.9	1.88	104.7	1.76	0.07	0.29	0.20	1.00
d70	132.5	2.83	142.9	2.03	136.9	2.74	0.10	<0.05	0.06	0.91
d82	142.5	2.57	149.5	2.66	143.5	2.49	0.38	0.78	0.99	0.99
ADFI[2], g/d										
d0 to 28[4]	2271.2	10.92	3435.6	96.11	3122.1	73.93	<0.001	<0.001	<0.001	<0.05
d29 to 70[4]	2250.0	0.00	2369.6	93.97	2231.7	7.80	<0.05	<0.05	<0.05	<0.05
d71 to 82	3708.7	157.90	3033.5	157.90	3989.0	148.10	<0.001	<0.05	0.41	<0.001
d0 to 82	2492.7	60.11	2798.0	60.11	2818.0	56.38	<0.05	<0.05	<0.05	0.97
ADG[3], g/d										
d0 to 28	804.9	53.74	1126.8	53.74	1198.9	50.41	<0.001	<0.05	<0.001	0.60
d29 to 70	883.4	49.18	965.7	49.18	793.5	46.13	0.06	0.48	0.39	<0.05
d71 to 82	771.7	150.46	451.2	150.46	570.5	141.13	0.34	0.31	0.60	0.83
d0 to 82	838.5	26.92	941.4	26.92	900.2	25.25	<0.05	<0.05	0.24	0.52
Gain/feed, g/kg										
d0 to 28	354.0	18.87	331.7	18.87	382.8	17.70	0.17	0.69	0.52	0.14
d29 to 70	107.8	1.30	118.0	7.30	102.9	6.84	0.33	0.59	0.88	0.31
d71 to 82	205.0	43.13	152.1	43.13	140.0	40.46	0.53	0.67	0.53	0.98
d0 to 82	336.6	8.33	337.8	8.33	319.3	7.81	0.21	0.99	0.31	0.26
Energy Intake, MJ										
d0 to 28	31.9	0.15	46.5	1.30	40.5	0.96	<0.001	<0.001	<0.001	<0.05
d29 to 70	31.6	0.00	32.1	1.27	28.9	0.10	<0.001	0.24	<0.001	<0.05
d71 to 82	52.1	2.15	41.1	2.15	51.7	2.01	<0.001	<0.05	0.99	<0.05
d0 to 82	35.0	0.81	37.9	0.81	36.5	76.20	0.06	0.05	0.37	0.45

[1]LSM = least squares mean. [2]ADFI= average daily feed intake. [3]ADG= average daily gain.

[2]Mean ± standard error presented for this value only, all other measures are least squares means are presented.

Table 5.6. Effect of dietary regime on carcass traits (LSM[1] ±SEM) at 140kg for individually housed replacement gilts.

	Treatment							Treatment comparison		
	DEV	SEM	FIN	SEM	GES	SEM	T	DEV v. FIN	DEV v. GES	FIN v. GES
n	11		11		12					P-value
Carcass weight, kg	111.7	2.27	117.2	2.27	114.3	2.13	0.25	0.23	0.68	0.63
Lean meat, %	56.0	0.76	54.1	0.76	55.8	0.71	0.16	0.20	0.99	0.23
Muscle, mm	63.0	1.68	62.8	1.68	64.9	1.58	0.62	0.99	0.70	0.66
Fat, mm	16.1	1.02	18.3	1.02	16.6	0.96	0.29	0.29	0.93	0.45
Dressing out, %	78.4	0.29	79.2	0.29	78.8	0.28	0.16	0.13	0.58	0.54

[1]LSM = least squares mean.

5.4.5 Carcass traits

There was no effect of treatment on the carcass characteristics; carcass weight, back fat depth, muscle depth, lean meat percentage, and dressing out percentage (Table 5.6).

5.4.6 Areal bone mineral density

There was no effect of treatment on aBMD. DEV gilts had a numerically higher bone mineral density (1.04 g/cm^2; ±0.02) than FIN (1.01 g/cm^2; ±0.02) and GES gilts (0.99 g/cm^2; ±0.01). No correlation was found between locomotion scores and aBMD.

5.4.7 Joint lesions

The percentage of gilts which had any lesion in the joint cartilage of the humeral condyle were DEV = 64% (7/11), FIN = 100% (11/11) and GES = 100% (12/12) (Table 5.3). The percentage of gilts affected by OCD was DEV = 27% (3/11), FIN = 36% (4/11) and GES = 25% (3/12) respectively. DEV gilts had lower joint lesion scores on the humeral condyle (Median 2; IQR 2) than GES (4; 1) and FIN gilts (4; 3) (P=0.051). There was no difference between treatments in the presence of lesions on the anconeal process; DEV = 45% (5/11), FIN = 73% (8/11) and GES = 58% (7/12). Humeral condyle and anconeal process scores were significantly correlated (P<0.05). There was a tendency for a positive correlation between the scores for lesions on the humeral condyle and locomotion scores (P = 0.08).

5.5 Discussion

This study is the first to examine the effect of a novel diet, specifically formulated for replacement gilts, on indicators related to lameness. Improvements in limb and claw health alone would improve gilt/sow welfare and may also reduce culling for lameness. This dietary regime reduced lameness, uneven claws and the severity of joint surface lesions in the elbow joint.

While none of the DEV gilts were scored lame during the experiment over 70% of the gilts on the other two treatments were scored as lame at least once during the study. Overall lameness levels were much higher than those previously reported. Heinonen *et al.* (2006) reported a prevalence of lameness in replacement gilts of 9.9%, KilBride *et al.* (2009a) observed abnormal gate in 18.9% of replacement gilts housed on partially slatted flooring and Chapter 4 of this thesis reported a lameness prevalence of 39% for replacement gilts. These studies however reported point prevalence using a large sample size while this study reports cumulative incidence. Previous studies have found associations between osteochondrosis, infectious arthritis and physical injuries, such as; claw lesions, joint lesions, muscle damage, tendon damage and bone fractures (Jensen *et al.*, 2007; Jensen and Toft, 2009). In the current study, however, whilst locomotory score was lower in DEV gilts there was no treatment effect on the severity of claw or limb lesions or on levels of bone mineral density suggesting that these were not associated with lameness in this study. The gilts in the current study had a low prevalence of severe limb and claw lesions and 100% prevalence of mild limb and claw lesions. Hence these lesions may not have been severe enough to influence locomotory ability or were too unvarying to associate with lameness in the small number of gilts in the current study (Gjein and Larssen, 1994; Anil *et al.*, 2007). In contrast to Calderón Díaz *et al.* (2013) there was no association between lameness and mild claw lesions.

In the current study there was a slight tendency for reduced locomotory ability (i.e. lameness) to be associated with increasing severity of joint lesions. However, while osteochondrosis has previously been identified as a contributor to leg

weakness/lameness in pigs the majority of studies supporting this link are based on sows culled for lameness (D'Allaire *et al.*, 1987; Kirk *et al.*, 2005; Engblom *et al.*, 2007; Engblom *et al.*, 2008; Jensen *et al.*, 2010). Other studies found no association between osteochondrosis and lameness/leg weakness (Brennan and Aherne, 1986; Jørgensen, 1995; Jørgensen *et al.*, 1995; Stern *et al.*, 1995; Arnbjerg, 2007). Hence the relationship is poorly understood (Dewey *et al.*, 1993; Heinonen *et al.*, 2006). For example, the extent of severity of abnormalities to the joint surface cartilage in the elbow joint required to alter pig locomotion and result in clinical lameness is not well established. Furthermore, osteochondrosis tends to occur bilaterally in the elbow joint, this could make it difficult to reduce weight bearing simultaneously in both front limbs while walking (Grondalen, 1974; Jørgensen *et al.*, 1995; Ytrehus *et al.*, 2007; Kirk *et al.*, 2008; Jensen and Toft, 2009). This would thereby render traditional lameness scoring systems based on reduced weight bearing in the affected limb less useful for the detection of osteochondrosis. Lameness detection methods involving pressure pads or the use of kinematics may prove more useful in establishing the link between joint lesions and lameness (de Koning *et al.*, 2012; Stavrakakis *et al.*, 2014). Stiff movements and out turned fore legs are associated with lesions of the cartilage in the elbow joint (Jorgensen and Andersen, 2000; Jørgensen and Nielsen, 2005; Kirk *et al.*, 2008; Jensen and Toft, 2009). Earlier identification of locomotion impairment relating to osteochondrosis through a more sensitive recording system could allow gilts exhibiting these traits to be excluded from the gilt pool (de Koning *et al.*, 2012).

The current study found no effect of dietary regime on limb or claw lesions. This was despite the gilt developer diet being highly fortified with an additional source of zinc, copper and manganese which are essential for skin and claw health (Socha *et al.*, 2002; Tomlinson *et al.*, 2004; Tomlinson *et al.*, 2008). However, the prevalence of severe limb and claw lesions was very low in this study likely because the animals were not housed in groups where social interactions on concrete slatted floors increase the likelihood of incurring injuries (Gjein and Larssen, 1994; Pluym *et al.*, 2011). Hence, potential benefits of supplementing gilt developer diets with trace minerals in terms of improved limb, skin and claw health would likely be

better tested under commercial group housed settings (Anil *et al.*, 2005). Nonetheless, DEV gilts had more even sized claws after d40. It is important to consider that the magnitude of the difference between the size of the medial and lateral claws was very small in this study. In addition, its biological importance is not known as Grégoire *et al.* (2013) reported that 54-60% of sows had uneven toes in the hind legs. Mild unevenness in the toes may even be considered normal (Penny *et al.*, 1963; Dewey *et al.*, 1993). It is when the difference between the toes and dew claws extend beyond a mild deviation, which it did not in this study, that it has the potential to hinder locomotion and make the claws more vulnerable to damage (Pluym *et al.*, 2011). The point at which the difference between the lateral and medial claw becomes abnormal resulting in detrimental effects needs to be elucidated.

The difference in body weight at d70 (prior to the flushing period) between the treatments, when FIN gilts were heavier than DEV gilts was an expected consequence of restricted feeding of the developer diet which was associated with reduced energy intake in DEV gilts in conjunction with a high energy to lysine ratio of diet 1 in comparison to diet 2 (Wilson and Osbourn, 1960; Varley *et al.*, 2011). The higher daily weight gain in DEV than in FIN gilts during the flushing period was probably due to compensatory growth once *ad-libitum* access to feed was restored (Wilson and Osbourn, 1960; Klindt *et al.*, 2001; Mitchell, 2007; Varley *et al.*, 2011). The lack of treatment effect on body weight or carcass characteristics post flushing (i.e. slaughter which occurred at approximate time of service) illustrates that restricting feed during development did not affect target weight for gilts at first service.

Ca and digestible P levels fed to DEV and GES gilts met the NRC (2012) recommendations while the FIN feeding regime was below the NRC (2012) recommendations for growing gilts. Ca and P are essential elements for bone development and maintenance as well as playing a role in metabolic and biochemical functioning (Underwood, 1999; Varley *et al.*, 2011). P and Ca levels also improve pig performance and bone mineralization (Jendza *et al.*, 2005; Brana *et al.*,

2006; Varley *et al.*, 2011). Increased bone mineralization ensures higher Ca and P availability from the skeletal reservoir during manure systems in use and the associated labour costs without negatively affecting bone mineral density or bone strength (Nimmo *et al.*, 1981) and therefore potentially improves sow longevity. However, there were no significant treatment differences in areal bone mineral density (aBMD) in the present study. The lack of a benefit of supplementation with Ca and P to aBMD may be because bone formation was largely complete by the time the treatments were applied. It is thought that dietary restriction of Ca and P in the first 12 weeks of life can result in impaired bone mineralisation during the reproductive cycle (Mahan, 1982). Therefore Ca and P supplementation above that normally provided in finisher or dry sow diets may be required prior to 70kg in order to influence aBMD (Tanck *et al.*, 2001; Varley *et al.*, 2011). A further consideration is the increase in Ca and P demand for milk production and an increase in dietary Ca and P supply from gilt development throughout gestation and into lactation may prove beneficial during these phases (Mahan, 1990; Marchant Forde and Broom, 1996; Almeida *et al.*, 2000). Future studies should evaluate the potential benefits of Ca and P supplementation prior to first service and during gestation on bone mineral density of gilts for multiple parities.

Osteochondrosis has been identified as an economic and welfare concern in commercial pig farming due to reduced productivity and associated pain respectively (van Grevenhof *et al.*, 2011). Beneficial effects of DEV treatment on joint lesions of the humeral condyle were observed in this study. Furthermore, and in accordance with (Nakano *et al.*, 1979), the prevalence of lesions in both FIN and GES gilts was high but just significantly lower in DEV gilts. It is likely that these findings were related to the fact that gilts in the DEV treatment were restrictively fed and fed a diet (1) which was also formulated to slow down growth by reducing lean tissue growth rate, therefore DEV gilts had a lower initial growth rates than FIN and GES gilts (Carlson *et al.*, 1988; van Grevenhof *et al.*, 2011). Busch and Wachmann (2011) reported a 20% increase in the risk of joint lesion occurrence for every 100g increase in ADG during the weaner and finisher period. It is thought that slower rates of growth reduce loading pressure on the joints which is particularly

important in developing animals (Nakano and Aherne, 1988; Carlson *et al.*, 1991; Ytrehus *et al.*, 2004).

This study illustrates the beneficial effects of feeding a gilt developer diet on lameness and joint lesions of the elbow joints in replacement gilts compared with the two most commonly practiced feeding regimes. These benefits could potentially lead to improvements in sow lifetime reproductive performance and longevity. Indeed on most farms where replacement gilts are produced they are housed and managed as finishing stock until the approximate age at which they would be slaughtered. This further precludes the feeding of diets specifically aimed at improving gilt development and welfare. This study would indicate that segregating gilts from finisher stock at least at 70 kg and applying a specifically formulated developer diet can improve gilt limb health

5.6 Conclusion

In conclusion, limit feeding a diet specifically formulated for developing gilts from 70kg to 2 weeks before target service at ~140kg (~212d) resulted in reduced occurrence of lameness and less severe surface lesions in the elbow joint and increased uniformity of medial and lateral claw size on a foot. Further work is required to establish whether these improvements could translate to improved gilt/sow welfare and increased sow longevity and productivity within the breeding herd.

Chapter 6

The effect of *ad-libitum* feeding a diet formulated for developing gilts between 65kg and ~140 kg on lameness indicators, carcass traits and behaviour

6.1 Abstract

This study investigated the effects of three dietary regimes for replacement gilts on lameness indicators, behaviour and carcass traits. Diets were: a diet specifically formulated for replacement gilts (diet 1, 14.04 MJ of DE/kg, 0.75% lysine), a finisher diet (diet 2, 13.54 MJ of DE/kg, 1.02% lysine) and a gestation sow diet (diet 3, 12.96 MJ of DE/kg, 0.69% lysine); the latter two are traditionally fed to replacement gilts. One hundred and eighty Large White x Landrace gilts were selected at d0 (64.18 kg, ±0.20), housed in 18 pens in groups of 10 and allocated at random with *ad-libitum* access to one of the following diets: 1) DEV (diet 1, n = 6 pens), 2) FIN (diet2, n = 6 pens) or 3) GES from 100kg (diet 3, n = 6 pens) treatments. The DEV diet was supplemented with Zn, Mn, and Cu and had higher Ca and P concentration and increased energy to lysine ratio when compared with the other two diets. Locomotory ability (0= normal to 5 = severely impaired), limb lesions (0 = normal to 3 = severe) and body lesions (0=no lesions to 6= >1 extensive lesion) were scored weekly until slaughter at d84 (145.00 kg; ±1.45). Hind claw lesions and claw evenness were scored (0 = normal to 3 = severe) at d0, 42 and 83. Gilts were weighed at d0, 29 and 83. Carcass traits were recorded at slaughter. The front right leg was removed at slaughter (d84) for dual-energy x-ray absorptiometry (DXA) to establish areal bone mineral density (aBMD). Joint surface lesions of the humeral condyle (HC; 1 = normal to 4 =severe, 5 = osteochondritis dissecans) and anconeal process (AP; 1 = lesions absent 2 = lesions present) were scored. FIN gilts (OR 1.72; CI 1.19, 2.48) and GES gilts (OR 1.73; CI 1.20, 2.49) had an increased risk of abnormal locomotion (score ≥1) in comparison to DEV gilts. FIN gilts had an

increased risk of being lame (score ≥2) in comparison to DEV gilts (OR 3.07; CI 1.23, 7.53). DEV gilts had significantly higher aBMD than FIN gilts (P<0.05), no differences were found between GES and other treatments. In all gilts there was an increased risk of uneven sized toes in week 5-8 (OR 0.10; CI 0.04, 0.30) and 9-12 (OR 0.09; CI 0.03, 0.26) when compared to the initial inspection at day 0. There was also an increased risk of heel overgrowth at inspection 2 (d42) (OR 14.30; CI 6.20, 32.97) and 3 (d83) (OR 31.82; CI 12.50, 81.01) compared with d0. There was no effect of treatment on limb lesions, claw lesions, uneven claw size, gilt behaviour, body weight or carcass traits. In conclusion, a diet specifically formulated for replacement gilts and fed *ad-libitum* led to an improvement in locomotory ability and aBMD.

6.2 Introduction

Restricted feeding of a developer diet specifically designed for replacement gilts fed to individually housed replacement gilts from 65kg resulted in reduced levels of lameness and osteochondrosis (Chapter 5) and is recommended to maximise reproductive performance (Sørensen *et al.*, 1993; Levis *et al.*, 1997; Gill and Taylor, 1999; Klindt *et al.*, 1999; Knauer *et al.*, 2012). However restricted feeding and individual housing of gilts is not common practice at farm level and would require substantial investment in feeding systems and infrastructure. The predominant method for feeding replacement gilts is largely *ad-libitum* access to feed. Replacement gilts are also predominantly group housed and, due to social interactions, injuries such as lameness and body lesions are more severe than when compared with individually housed pigs (Calderón Díaz *et al.*, 2013).

It is hypothesised that providing *ad-libitum* access to a gilt developer diet during gilt development will lead to improvements to limb health indicators which could reduce inactive behaviours and ultimately reduce premature culling for lameness and improve farm profitability. Therefore the aim of this study was to determine the effect of *ad-libitum* feeding of a developer diet (DEV) to replacement gilts from 65kg to service compared with *ad-libitum* feeding of a gestating sow diet (GES) from 100kg or *ad-libitum* feeding of a finisher (FIN) diet for developing gilts on indicators

of lameness and carcass traits. It is also hypothesised that provision of a gilt developer diet may result in increased active behaviours due to improved locomotory ability and limb health.

6.3 Methods

6.3.1 Care and use of animals

The research farm (Pig Development Department, Teagasc, Moorepark, Fermoy, Co. Cork, Ireland) where the experimental work was carried out was compliant with statutory Instrument number 311 of 2010 European communities (Welfare of Farmed Animals) Regulations 2000. Licensing under the European Communities (Amendment of Cruelty to Animals Act. 1876) Regulations (2002) was not required as no invasive procedures were conducted during the study. This trial was conducted between April and September 2012. Gilts were selected in 4 batches throughout this period, treatments were balanced per batch.

Gilts (Large White x Landrace) were weaned at 28 days of age (ds) into single sex pens with 14 pigs per pen. Gilts were provided with *ad-libitum* access to dry pelleted feed until ~84ds after which they were dry fed a finisher diet 3 times per day (13.7 MJ of DE/kg, 0.4% lysine) until selection for the experiment (~112d). Gilt pens were identified for selection based on appropriate age and weight (~112d 65kg), 3-4 pigs per pen were removed to make a group size of 10 pigs per pen; the excess pigs were removed from the pen based on a health inspection, whereby lower health pigs were removed. Gilt pens were selected at an average pig per pen weight of 64.18 kg (±0.20), each pen was allocated at random, using a random number generator, to an experimental treatment (DEV, FIN and GES). At selection four focal pigs per pen were selected for more detailed measures. The focal pigs were selected by determining the median four weights of the group, if a pig scored a higher lameness score than 1 at selection the pig was not used as a focal pig and the next pig closest to the median weight was selected.

Pen dimensions were 2.32m x 4.73m and each pen was fully slatted with concrete slats (80 mm solid width, 18 mm slots). Air temperature was maintained at 20 to 22°C and ventilation was provided by a cross flow system (Stienen PCS 8200; Stienen BV). All pigs were manually fed from a stainless steel trough and had continual access to water provided by a nipple in bowl drinker (BALP, Charleville-Mezieres, Cedex, France). Rubber pipes suspended from a chain were provided as environmental enrichment.

A health assessment was carried out twice daily to ensure that none of the gilts were injured or sick. Sick or injured animals were treated immediately; all veterinary treatments were recorded. If gilts required isolation for treatment, they did not return to the group to prevent disruption and were removed from the trial. All gilts were slaughtered at d84 (~140kg, approximate target weight for 1st service).

6.3.2 Diet formulations

On d0 (i.e. when gilts weighed 64.05 kg; ±0.58) 18 pens of gilts were randomly assigned to one of the dietary regimes, DEV (n = 60 pigs, 6 pens), FIN (n = 60 pigs, 6pens), and GES (n = 60 pigs, 6pens) as shown in Table 6.1. Treatment DEV involved *ad-libitum* access to diet 1 until d83 (Table 6.1 and 5.2). Diet 1 was a gilt developer diet with a high energy to lysine ratio (14.04 MJ of DE/kg, 0.75% lysine), high Ca and P levels and included a dietary supplement containing zinc, copper and manganese (Table 5.2). Treatment FIN involved *ad-libitum* access to diet 2 (13.54 MJ of DE/kg, 1.02% lysine) until d83 (Table 6.1). Diet 2 was a standard finisher diet (Table 5.2). Treatment GES provided *ad-libitum* access to diet 2 (12.96 MJ of DE/kg, 0.69% lysine) until d28, followed by *ad-libitum* access to diet 3 until d83 (~140kg) to mimic the practice of flushing (Table 6.1). Diet 3 was a standard gestating sow diet (Table 5.2). All feed was provided in dry pelleted form (3mm diameter). Diet 1 and 3 provided Ca and digestible P that met NRC (2012) recommendations for growing gilts, Diet 2 was below the NRC (2012) recommendations for growing gilts.

Table 6.1 Dietary regimes for group housed replacement gilts in three treatments from 65 to 140kg.

Weight range (kg)	Dietary treatment		
	DEV	FIN	GES
65 – 100	Diet 1 (*ad-libitum*)	Diet 2 (*ad-libitum*)	Diet 2 (*ad-libitum*)
100 - 140	Diet 1 (*ad-libitum*)	Diet 2 (*ad-libitum*)	Diet 3 (*ad-libitum*)

6.3.3 Measurements

Locomotory ability and limb lesions were scored weekly in focal pigs. All gilts were weighed at d0, 29 and 83 of the experiment. Claw lesions were scored, in focal pigs, on d0, 42 and 83. Behaviour of focal pigs was recorded over 24hrs on weeks 1, 3, 6, 9 and 12. For all gilts, carcass weight, backfat depth and muscle depth were recorded at slaughter (d84) and lean meat and dressing out percentages were calculated. The front right leg of each focal pig was removed at slaughter and frozen at -20°C. Dual-energy x-ray absorptiometry (DXA) analysis was used to measure areal bone mineral density (aBMD) and following this the limb was dissected to expose the joint surface lesions on the cartilage of the elbow joint for lesion scoring in all focal pigs. Multiple recording sheets were used (Appendix 5).

6.3.3.1 Locomotory ability

Locomotory ability was assessed using the gait and standing posture aspects of the protocol described by Main *et al.* (2000) and as in Chapter 5.

6.3.3.2 Limb lesions

Lesions were examined on all four legs. Lesions were categorised and scored as in KilBride *et al.* (2009a) and as described in Chapter 3.

6.3.3.3 Claw lesions and uneven claw size

Claw inspections were carried out as outlined in Chapter 5. Only the hind feet were examined. The scoring system used was a modified version of the FeetFirst™ claw lesion scoring guide (Zinpro Corporation) as described by Calderón Díaz *et al.* (2013).

6.3.3.4 Body lesions

Body lesions were examined as in O'Driscoll *et al.* (2013) and described in Chapter 4.

6.3.3.5 Behaviour

Behaviour of focal pigs was recorded for 24 hour period using an Ozone V2 DVR with P400 600TVL bodied cameras. Animal marker (Coyle's Animal Marker Spray, Coyle Vet. Products Ltd., Co. Galway, Ireland) was used to identify each focal pig with in each pen using both symbols and colours to aid identification. Recordings were watched back and analysed using instantaneous scan sampling at 10 minute intervals over the 24 hour period (Appendix 5). Four behaviours were recorded; standing, dog sitting, lying and feeding. Behaviours were then also classified into active (standing and feeding) and inactive (dog sitting, lying) behaviours.

6.3.3.6 Slaughter

Pigs were fasted for 14 hours prior to slaughter. On the d84 (day of slaughter), gilts were mixed and transported 90 km. Gilts were slaughtered by exsanguination post CO^2 stunning. After evisceration, muscle depth and back fat thickness were measured as in Chapter 5 by the slaughtering facility staff. Lean content and carcass weight (cold) were calculated as in Chapter 5. The front right limb was dissected dorsal to the elbow joint, identified by a tag and frozen within 2 hours for DXA scanning and elbow joint surface cartilage scoring at a later date.

6.3.3.7 Areal bone mineral density (aBMD)

The front right limb was DXA scanned to determine aBMD (g/cm^2) by a Hologic QDR-4500 Elite bone densitometer as per Chapter 5 and analysed using Apex software version 2.1.3 and 2.3.1. Two software versions were used due to a machine fault which resulted in the upgrading of the software mid-way through the trial, software version was included in analysis.

6.3.3.8 Joint surface lesions

The front right limb was dissected at the elbow joint to expose the humeral condyle (HC) and anconeal process (AP) as per Jørgensen *et al.* (1995), Christensen *et al.* (2010)and Busch and Wachmann (2011) and as described in Chapter 5.

6.3.4 Laboratory analysis of diets

Representative samples of each diet were taken as per Chapter 5. Samples were ground using a laboratory hammer mill (Christy and Norris, Scunthorpe U.K.) through a 2 mm screen. Proximate and amino acid analyses were carried out by a commercial laboratory (Sciantec Analytical Services Ltd., Cawood, UK).

6.3.5 Data management

Data were entered into a Microsoft Access 2003 database by AQ. All data were checked for outliers and impossible values were checked against the raw data.

6.3.6 Data analysis

Lameness, limb lesions, body lesions, joint lesions, claw lesions and uneven claw size data were analysed using MlwiN 2.27 (Rasbash *et al.*, 2012). Gilts were categorized as lame (score ≥2) or non-lame (≤1) and lesions (limb, body, claw and joint lesions) were classified as present or absent due to the lack of distribution of severity of lesions across the scoring systems. Multilevel mixed effects binary logistic regression was used to allow for repeated measures where by weeks were clustered within pig and pig was clustered within pen, therefore a three level random effect model was used. The model included fixed effects of treatment and batch, and period where appropriate. All continuous variables were transformed in to categorical variables and checked for linearity, if a linear association was found the variables were reverted back to a continuous variable, otherwise they were left as categorical variables. The following model was used:

$$\text{Logit}(p_{ijk}) = \beta_0 + \Sigma\beta x_{ijk} + \Sigma\beta x_{jk} + \Sigma\beta x_k + v_k + u_{jk}$$

p_{ij} = the proportion of the litter that were affected (score ≥1) with the lesion being investigated, Logit= logit link function, β0= constant, βx = vector of fixed effects varying at level 1 (ijk), level 2 (jk), or level 3 (k), i = period, j= pig (i.e. litter), K= pen, v_k= level 3 residual variance, u_{jk}= the level 2 residual variance.

Data for body weight, aBMD, and behaviour of the four focal pigs in each pen were averaged and checked for normality. Data for carcass traits for all pigs in each pen were averaged and checked for normality. Body weight, aBMD and carcass traits (carcass weight, muscle, fat and lean meat content) were analysed in SAS V9.3 (Statistical Institute Inc., Cary, North Carolina) using repeated measures one way ANOVA (Proc MIXED) for body weight and one way ANOVA (Proc MIXED) for aBMD and carcass traits, with treatment and batch included in the model and also period and weight at d0 in the analysis of body weight. Results are reported as least-square means ±standard error. Gilt behaviour data were non normal and were transformed by arcsine transformation and were analysed in SAS V9.3 (Statistical Institute Inc., Cary, North Carolina), using repeated measures one way ANOVA (Proc MIXED) Results are reported as least-square means ±standard error.

6.4 Results

6.4.1 Lameness

The percentage of animals with locomotion scores ≥2 (i.e. lame) on at least one occasion during the experiment were: DEV = 37.5% (9/24), FIN = 66.7% (16/24) and GES = 58.3% (14/24) (Appendix 6). FIN gilts (OR 1.72; CI 1.19, 2.48) and GES gilts (OR 1.73; CI 1.20, 2.49) had an increased risk of abnormal locomotion (score ≥1) in comparison to DEV gilts. FIN gilts had a significantly increased risk of being lame (score ≥2) in comparison to DEV gilts (OR 3.07; CI 1.23, 7.53). No association was found between lameness and OCD, limb lesions, body lesions, claw lesions, uneven claw size or bone mineral density.

6.4.2 Limb lesions and body lesions

There was no effect of treatment on limb lesions, however, gilts had an increased risk of capped hock and limb swellings in weeks 9 to 12 when compared to the initial inspection at d0 (Table 6.2). FIN gilts had an increased risk of lesions to the ear and GES gilts had an increased risk of lesions to the hindquarter than DEV gilts.

The risk of scratches to the limbs and body lesions to the ear, shoulder, flank and hindquarter were lower in weeks 1-5, and in weeks 5-8 for hindquarter only, when compared to the initial inspection at d0 (Table 6.3).

6.4.3 Claw lesions and uneven claw size

There was no effect of treatment on any of the claw lesions or on uneven claw size (Appendix 3). There was an increased risk of uneven toes in weeks 5-8 and 9-12 than when compared to the initial inspection at d0. There was also an increased risk of heel overgrowth at inspection 2 (d29) and 3 (d83) when compared to the initial inspection at d0 (Table 6.4).

6.4.4 Behaviour

There were no differences in the proportion of time spent in each four postures (standing, feeding, lying, dog-sitting), or in the time spent active and inactive per treatment (Table 6.5).

6.4.5 Body weight and carcass traits

There was no effect of treatment on body weight and the carcass traits; carcass weight, back fat depth, muscle depth and lean meat percentage (Table 6.6). Average daily gain per day was similar between treatments on an average per pig per pen basis (DEV = 915.69 ±29.81, FIN = 912.89 ±21.40, GES = 925.25 ±22.71). Calculated feed intake per day did not differ between treatment on an average per pig per pen basis (DEV 2775.04g ±130.86, FIN 2882.05g ±93.54, GES 2793.70 ±123.66).

6.4.6 Areal bone mineral density (aBMD)

DEV gilts had significantly higher aBMD than FIN gilts, no difference was found between GES and other treatments ($P<0.05$) (Table 6.6). There was an effect of software on aBMD however numbers of pigs per treatment were balanced per software version.

6.4.7 Joint surface lesions

The percentage of gilts which had any lesion in the joint cartilage of the humeral condyle were DEV = 100% (24/24), FIN = 96.8% (23/24) and GES = 100% (24/24) (Appendix 6). There was no effect of treatment on joint surface lesions scores of the humeral condyle. The percentage of gilts affected by OCD was DEV = 0% (0/24), FIN = 8.3% (2/24) and GES = 16.7% (4/24) respectively. The percentage of gilts which had any lesion in the joint cartilage of the anconeal process were DEV = 37.5% (9/24), FIN = 54.2% (13/24) and GES = 50.0% (12/24) (Table 6.7). There was no significant effect of treatment on joint surface lesions scores of the anconeal process.

Table 6.2. Multilevel binomial models of the risks associated with limb lesions; scratches, wounds, swellings, bursitis and capped hock for three dietary regimes for group housed replacement gilts from 65kg.

	Capped hock			Bursitis			Swelling			Wound			Scratch		
Intercept Coefficient	-2.8			-0.4			-0.6			-2.3			-0.5		
	OR	CI		OR	CI		OR	CI		OR	CI		OR	CI	
Treatment diet															
DEV															
FIN	1.13	0.41	3.09	0.85	0.45	1.59	1.29	0.60	2.77	0.96	0.53	1.73	0.85	0.60	1.21
GES	1.20	0.44	3.27	0.68	0.36	1.29	0.76	0.35	1.65	1.05	0.59	1.87	1.34	0.95	1.88
Period															
d0															
d1-28	0.24	0.05	1.25	0.91	0.53	1.55	1.15	0.59	2.25	0.84	0.30	2.40	0.32	0.18	0.55
d29 -56	2.00	0.57	6.94	0.72	0.42	1.23	1.34	0.69	2.57	1.05	0.38	2.93	0.77	0.46	1.31
d57-83	4.19	1.24	14.09	0.64	0.38	1.11	2.07	1.08	3.94	1.68	0.63	4.53	1.18	0.70	1.99
Batch	0.92	0.69	1.23	0.84	0.14	5.21	1.19	0.95	1.48	0.93	0.79	1.10	1.07	0.96	1.18
Random effects	Var	SD		Var	SE		Var	SE		Var	SE		Var	SE	
Pen	0.0	0.0		0.0	0.0		0.2	0.2		0.0	0.0		0.0	0.0	
Pig	1.9	0.5		0.88	0.2		0.6	0.2		0.0	0.0		0.0	0.0	

Table 6.3. Multilevel binomial models of the risks associated with body lesions to the ear, shoulder, flank, hindquarter, tail and vulva for three dietary regimes for group housed replacement gilts from 65kg.

	Ear			Shoulder			Flank			Hindquarter			Tail			Vulva		
Intercept Coefficient	-1.36			0.22			0.81			0.3			-0.84			0.74		
	OR	CI		OR	CI		OR	CI		OR	CI		OR	CI		OR	CI	
Treatment																		
DEV																		
FIN	1.56	1.01	2.41	0.74	0.46	1.17	0.95	0.67	1.34	1.27	0.83	1.95	1.09	0.70	1.68	0.69	0.32	1.49
GES	1.43	0.92	2.22	1.00	0.63	1.59	1.16	0.82	1.65	1.67	1.09	2.57	0.96	0.62	1.50	0.94	0.46	1.93
Period																		
d0																		
d1-28	0.28	0.15	0.53	0.32	0.18	0.58	0.47	0.27	0.80	0.39	0.23	0.67	0.29	0.15	0.53	0.16	0.03	1.00
d29-56	0.65	0.34	1.22	0.71	0.40	1.27	0.76	0.44	1.30	0.79	0.46	1.35	0.55	0.33	0.91	1.35	0.38	4.85
d57-83	0.86	0.46	1.64	1.24	0.00	46.18	1.49	0.86	2.59	1.34	0.78	2.32	0.85	0.48	1.50	2.10	0.61	7.28
Batch	0.97	0.85	1.10	0.92	0.80	1.05	1.05	0.95	1.17	0.87	0.77	0.98	1.00	0.88	1.14	1.01	0.81	1.25
Random	Var	SE		Var	SE		Var	SE		Var	SE		Var	SE		Var	SE	
Pen	0.06	0.05		0.60	0.06		0.00	0.00		0.60	0.05		0.00	0.00		0.00	0.00	
Pig	0.00	0.00		0.07	0.08		0.33	0.06		0.00	0.00		0.00	0.00		0.00	0.00	

Table 6.4. Multilevel binomial models of the risks associated with uneven toes and the claw lesions; heel overgrowth and heel-sole separation for three dietary regimes for group housed replacement gilts from 65kg.

	Uneven toes			Heel overgrowth			Heel-sole separation		
Intercept Coefficient	1.48			-1.95			-0.41		
	OR	CI		OR	CI		OR	CI	
Treatment									
DEV									
FIN	1.37	0.44	4.26	1.10	0.39	3.08	2.08	0.74	5.83
GES	2.58	0.81	8.26	1.34	0.48	3.77	1.89	0.67	5.32
Inspection									
1									
2	0.10	0.04	0.30	14.30	6.20	32.97	0.72	0.28	1.85
3	0.09	0.03	0.26	31.82	12.50	81.01	1.10	0.46	2.65
Batch	1.17	0.84	1.63	1.04	0.77	1.40	0.73	0.55	1.95
Random effects	Var	SE		Var	SE		Var	SE	
Pen	0.48	0.35		0.07	0.30		0.00	0.00	
Pig	0.24	0.42		0.85	0.60		0.53	0.52	

Table 6.5. Effect of three dietary regimes for group housed replacement gilts on behaviour (LSM[1] ±SEM) from 65 to 140kg.

	Treatment				T	DEV v. FIN	DEV v. GES	FIN v. GES
Behaviour	DEV	FIN	GES	SEM	P-value	P-value	P-value	P-value
Lying	88.4	87.5	87.7	0.01	0.47	0.24	0.38	0.76
Dog	1.7	1.5	1.7	0.01	0.85	0.61	0.90	0.63
Standing	5.3	6.2	5.5	0.01	0.32	0.15	0.68	0.29
Feeding	3.5	3.7	3.9	0.01	0.67	0.64	0.38	0.67
Active	9.1	10.3	9.8	0.01	0.26	0.11	0.31	0.53
Inactive	90.6	89.5	90.0	0.01	0.28	0.12	0.33	0.52

[1]LSM = least squares mean.

Table 6.6. Effect of three dietary regime for group housed replacement gilts on aBMD, body weight and carcass traits (LSM[1] ±SEM) from 65 to 140kg.

	DEV	FIN	GES	SEM	T	DEV v. FIN	DEV v. GES	FIN v. GES
aBMD	0.95	0.87	0.93	0.025	0.13	0.05	0.51	0.16
Weight, kg								
Day 0	63.8	64.9	64.1	2.24		0.72	0.91	0.81
Day 29	96.7	98.8	99.3	2.24		0.52	0.41	0.86
Day 83	138.9	143.69	141.2	2.24		0.14	0.46	0.44
Carcass weight, kg	108.0	111.6	108.8	2.25	0.51	0.28	0.82	0.39
Lean meat, %	59.4	60.9	60.9	1.62	0.77	0.53	0.54	0.99
Muscle, mm	56.2	57.1	54.9	1.29	0.50	0.63	0.49	0.25
Fat, mm	16.7	16.9	17.0	0.87	0.97	0.88	0.80	0.91

[1]LSM = least squares mean. SEM

6.5 Discussion

This study is the first to examine the effect of *ad-libitum* feeding a novel gilt developer diet, specifically formulated for replacement gilts, on limb and claw health in group housed gilts. Any improvement in limb and claw health will ultimately improve gilt/sow welfare prior to service and may potentially reduce culling for limb/claw related problems. This novel gilt diet reduced lameness, and increased aBMD.

High levels of lameness were reported in the current study in FIN gilts (66.7%) and GES gilts (58.3%) when compared with DEV gilts (37.5%), similarly to results in Chapter 5. In this instance, however, lameness incidence in DEV gilts was higher than those reported in Chapter 5. This higher levels of lameness may be a result of a larger sample size as well as the effect of group housing on lameness levels as a result of social interaction. The lameness levels for all three feeding regimes are also higher than those previously reported in replacement gilts, 9.9% in Heinonen *et al.* (2006) and 18.9% abnormal gait prevalence in KilBride *et al.* (2009a) on partially slatted flooring. However, as previously mentioned in Chapter 5, these studies report point prevalence while this study reports cumulative incidence.

As previously mentioned the previous studies have linked lameness and osteochondrosis, infectious arthritis and physical injury, such as; claw lesions, joint lesions, muscle damage, tendon damage and bone fractures (Jensen *et al.*, 2007; Jensen and Toft, 2009). However, locomotory ability and lameness were not however associated with claw lesions, limb lesions, joint lesions and bone mineral density as measure of limb health in this study.

Beneficial effects of an *ad-libitum* provision of a gilt developer diet in DEV gilts on aBMD were observed. No such effect was reported in Chapter 5 when the developer diet was restrictively fed to DEV gilts, but feed restriction has previously been linked with aBMD decline (Weremko *et al.*, 2013). DEV and GES gilts were provided with Ca and digestible P by their feeding regime that met the NRC (2012)

recommendations for replacement gilts while the FIN gilts feeding regime was below the NRC (2012) Ca and digestible P recommendations for gilts. This difference is most likely due to the *ad-libitum* access of the developer diet as feed intake of the developer diet was higher than in Chapter 5.

In the current study no beneficial effects of *ad-libitum* feeding a gilt developer diet (diet 1) on joint lesions were observed, unlike Chapter 5, whereby the gilt developer treatment was restrictively/limit fed to DEV gilts, this differential is most likely the result of lower initial growth rates when gilts were restrictively fed a developer diet, which was not the case when *ad-libitum access* was provided (Carlson *et al.*, 1988; van Grevenhof *et al.*, 2011). Busch and Wachmann (2011) reported a 20% increase in the risk of joint lesion occurrence for every 100g increase in ADG during the weaner and finisher period. Reducing growth rate reduces the loading pressure on the joints which is particularly important in developing animals (Nakano and Aherne, 1988; Carlson *et al.*, 1991; Ytrehus *et al.*, 2004). Therefore implementation of an *ad-libitum* feeding regime of a developer diet eliminated the beneficial effect of the diet on joint lesions when restricted fed.

In chapter 5, there was no effect of dietary regime on limb and claw lesions. This was despite the gilt developer diet (diet 1) being highly fortified with an additional source of Zinc, Copper and Manganese. The overall prevalence of severe limb and claw lesions was low in this study compared to what is seen in other group housed pigs (Pluym *et al.*, 2011). The length of provision of the treatments whereby the developer diet which was supplemented with chelated organic zinc, copper and manganese and elevated calcium and phosphorous may not have been sufficient to alter claw lesion prevalence as other studies have reported supplementation of minerals for 6 and 12 months in order to influence claw lesions may be required therefore the potential benefits of gilt nutrition on claw health may be evident in the breeding herd as a result (Brooks *et al.*, 1977; Pötzsch *et al.*, 2003).

No evidence of behavioural alterations was observed despite DEV gilts having significantly improved locomotory ability than FIN and GES gilts. This study

hypothesized that lameness alterations as a result of gilt feeding regime would alter postural and time at inactive and active behaviors based on the findings in broiler chickens of Weeks *et al.* (2000) and Kestin *et al.* (1992) and previous work in sows (Bonde *et al.*, 2004; Velarde and Geers, 2007; Valros *et al.*, 2009; Calderón Díaz and Boyle, 2014). Anil *et al.* (2009) also suggested that lameness affects the expression of a pig natural behaviour. However, in the current study, only mild lameness was observed. Therefore the discomfort and pain of mild lameness may not be above the threshold of lameness inflicted pain to result in behavioural alterations.

6.6 Conclusion

In conclusion, *ad-libitum* feeding a diet specifically formulated for developing gilts from 65kg resulted in reduced lameness and increased aBMD but unlike restricted feeding no effect was found on claw or joint lesions. *Ad-libitum* feeding a gilt developer diet did not alter postural behaviours. These improvements could translate to improved gilt/sow welfare and increased sow longevity and productivity within the breeding herd.

Chapter 7

General discussion

7.1 Introduction

In this Chapter key findings of the thesis are outlined, and the implications of these findings for stakeholders in the pig industry examined. The limitations of the work are highlighted as well as areas warranting further investigation. The overarching aim of the study was to determine the prevalence and risk factors for lameness, limb and claw lesions as well as to investigate the effects of gilt nutrition on indicators of limb health.

The cross-sectional survey (Chapters 2, 3 and 4) is the first to examine the prevalence and risk factors for lameness, limb and claw lesions in pigs of all stages of production on commercial farms in Ireland. It was the first outside the UK, and the largest cross-sectional study to date of indoor intensive housing systems. Twenty four percent of Irish pig farms distributed across representative herd sizes and geographic locations were sampled, which was a larger sample size of intensive indoor pig production systems than that of Kilbride (2008). Hence this study was more representative of European pig farming outside of the UK, where outdoor management of pigs is rare. The inclusion of individual as well as group housing systems for pregnant gilts and sows provides findings of international relevance because sows are still kept in stalls in many countries. In addition, a larger proportion of lactating sows was sampled on each farm (Chapter 4) compared with KilBride (2008), which increased the statistical power for this group.

In relation to gilt nutrition (Chapters 5 and 6), previous studies have investigated effects on future reproductive performance (Sørensen *et al.*, 1993; Levis *et al.*, 1997; Gill and Taylor, 1999; Klindt *et al.*, 1999; Miller *et al.*, 2011; Knauer *et al.*, 2012). However, no studies to date have examined the potential benefits of

restricted diets on limb health, which could ultimately improve sow longevity as a consequence of reduced limb-related culling and performance.

7.2 Key findings, implications and further work

7.2.1 Lameness

One of the key findings of this thesis is the high prevalence of lameness in finishers, gilts and sows in Irish production systems (Chapters 3 and 4). These levels of lameness are detrimental to the pig production industry due to their effects on productivity and welfare, and the serious ethical concerns arising as a consequence (Dewey *et al.*, 1993; Anil *et al.*, 2002;2005; Kirk *et al.*, 2005; Jensen *et al.*, 2007; Mustonen *et al.*, 2011; Pluym *et al.*, 2011). The prevalence of lameness in finishers (32%) (Chapter 3), replacement gilts (39%), pregnant gilts (41%) and sows (42%) (Chapter 4), is considerably higher than that previously reported in other studies: finishers (2-20%) replacement gilts (11%), and sows (5-17%) (Heinonen *et al.*, 2006; Petersen *et al.*, 2008; KilBride *et al.*, 2009a; Pluym *et al.*, 2011; Ellingson *et al.*, 2012; Pluym *et al.*, 2013a; Willgert *et al.*, 2014). However, this may be partially related to different scoring systems used for locomotory ability, which have different thresholds for lameness. The low threshold for lameness in the scoring system used in this thesis was applied because even slight alterations to gait and posture may affect a pigs biological functioning, such as the ability to compete for resources like feed and water (Heinonen *et al.*, 2013). This study defined lameness as any deviation from normal locomotion beyond stiffness of movement, and was similar to that used in previous experiments (Mustonen *et al.*, 2011; Calderón Díaz *et al.*, 2013). Contrarily other studies have considered lameness to be when more overt evidence of pain within a limb was displayed (e.g. limping) (Pluym *et al.*, 2011; Temple *et al.*, 2011; Temple *et al.*, 2013). However, the prevalence of lameness in this study remains higher than the only previous study to date to quantify lameness in these groups (KilBride *et al.*, 2009a), despite the fact a lower threshold for lameness was used in the previous study.

The width of voids between the slats in finisher pens was the only environmental factor which influenced lameness (Chapter 3). This lends support to EC Council Directive 2008/120/EC which states that slat voids for finishers should be no wider than 18mm. Voids which are too wide do not provide suitable support for the foot, thus making it more susceptible to injury and increasing uneven weight bearing of the foot pad, which may result in altered gait and potential lameness (Baxter, 1984; Mouttotou *et al.*, 1999a; Straw *et al.*, 2006). Additionally Chapter 3 highlights that the average slat void of the farms sampled in this study (20mm) is in excess of the EC Council Directive 2008/120/EC minimum requirements highlighting a concern over large scale non-compliance. It is worth considering however, the background of the development of these minimum requirements are unknown and appear not to be based on the scientific literature as there is a dearth of information on the topic. Future work into the effect of void and slat width on limb health in all age categories of pigs may provide valuable information on the suitability of the current requirements.

The only management procedure measured that influenced lameness in finishers was frequency of pen washing (≥4 times per year), which reduced lameness. This association is likely the result of a reduced pathogen level in the environment, as dirty pens have previously been associated with infected claw lesions which could result in lameness (Heinonen *et al.*, 2006; Cook and Nordlund, 2009). With regard to replacement gilts, the only management related risk factor was that there was a reduced risk of limb swellings when gilts were separated from finishing stock before 90kg (i.e. prior to sale of terminal stock). The reason for this association is unclear and requires further research. It may be a result of reduced antagonistic behaviours in single sex pens, (Björklund and Boyle, 2006; Boyle and Björklund, 2007) or perhaps an awareness of the benefits of housing gilts separately during development reflects a heightened awareness of, and better gilt management.

Chapter 4 demonstrated the high prevalence (39%) of lameness in replacement gilts inspected in this study. This suggests that for most Irish pig producers the selection of lame gits as replacement breeding stock is unavoidable, as approximately 90% of

the replacement gilt pool is generally selected for service. This high prevalence is supported by a recent study conducted in Ireland by Calderón Díaz (2013); in that experiment 39% of replacement gilts were lame on entry to the breeding herd. Compromised limb health at entry to the herd makes a sow more susceptible to early removal, reduced litter size and a reduced number of litters per sow (Dewey *et al.*, 1993; Grandjot, 2007; Anil *et al.*, 2008;2009; Wilson *et al.*, 2009; Pluym *et al.*, 2011). In this survey replacement gilts were consistently housed in suboptimal pens for limb health, according to previous research (Gjein and Larssen, 1994; Mouttotou *et al.*, 1999a; Scott *et al.*, 2006; KilBride *et al.*, 2009a). In general, gilts were housed in largely fully slatted or partially slatted flooring without bedding, similar to terminal line stock which have a considerably shorter life span. The use of more comfortable floor surfaces such as rubber (Calderon et al., 2014) or bedding may prove beneficial to herd performance, despite the associated costs or increased labour (Kroneman *et al.*, 1993; Andersen and Bøe, 1999; Tuyttens, 2005; KilBride, 2008).

Lameness levels were substantially higher in the group housed systems (48%) compared to gestation stall systems (30%). The higher level of lameness associated with group housing systems in comparison with the use of gestation stalls is likely an indirect result of aggression between sows on slatted and unbedded floor types, which were common on the farms included in this study i.e. fully or partially slatted flooring with no provision of bedding. Such underfoot conditions are a major risk factor for lameness (Gjein and Larssen, 1994; Mouttotou *et al.*, 1999a; Scott *et al.*, 2006; KilBride *et al.*, 2009a). Hence, this study supports the prediction that a substantial increase in sow lameness will have occurred as a result of the transition to group housing in response to the EC Directive 2008/120/EC, particularly in Ireland and other countries where such flooring predominates. This information is extremely valuable for countries currently considering group housing options such as the USA, Australia and Canada, and may be valuable to guide future legislative decisions. Additionally, this study highlights that identification of alternative flooring systems to improve limb health suitable for intensive production systems is required, as currently the predominant flooring systems are an ethical concern.

The finding that few management factors were associated with lameness may be due to the limitations of assessing such an association with a questionnaire, in conjunction with the limitations of obtaining information on management parameters with a single visit to each farm. For example, there was no opportunity to observe management routines which may influence limb health, such as observing pigs being moved by stock persons, sows being mixed or feeding events. It was only possible to capture information at an individual time point, and not possible to accurately record details of all management practices within the farm questionnaire. Future work quantifying lameness, claw and limb injuries prior to and post influential management events (e.g. transfers, mixing and feeding) may reveal more information about the association between lameness and other limb injuries. Identification of influential management parameters would provide valuable information on improved farm practices to reduce the current limb health issue.

The nutritional studies (Chapters 5 and 6) were conducted in response to growing concerns regarding high levels of lameness in replacement gilts (Boyle *et al.*, 2010) and culling due to lameness of young sows (D'Allaire *et al.*, 1987; Lucia *et al.*, 2000; Stalder *et al.*, 2000). The need for investigation of such strategies was supported by the findings of Chapter 4, where high levels of lameness in gilts were confirmed. Both these Chapters provide evidence that feeding a diet specifically formulated for developing gilts reduces lameness levels when compared to the two most commonly practiced feeding regimes, feeding a diet formulated for finisher pigs to replacement gilts through development until service or to switch from a finisher diet to a gestating sow diet at the end of the finishing period (Boyd *et al.*, 2002). Lameness levels in gilts on the developer diet were higher when these animals were kept in groups (Chapter 6), rather than when individually housed (Chapter 5). This result supports the survey findings reported in Chapter 4, whereby group housing appeared to partially dilute the beneficial effect of the dietary regime on lameness indicators. Restrictively feeding a gilt developer diet during early development also reduced joint lesion prevalence. This was likely due to lower initial growth rates

compared with *ad-libitum* feeding throughout development (Carlson *et al.*, 1988; van Grevenhof *et al.*, 2011). This finding was not replicated when *ad-libitum* access to the same diet was provided in Chapter 6. Additionally, an increase areal bone mineral density when gilts were ad-libitum (Chapter 6) rather than restricted (Chapter 7) fed a gilt developer diet was likely due to a higher feed intake of a Ca and P fortified diet.

The lack of association between lameness and the limb health indicators investigated in Chapters 3 and 4 highlights a requirement for further lameness indicators to be identified, so that further factors contributing to lameness can be identified. The limitations of osteochondrosis detection through the use of visual scoring methods was also highlighted, as mild, moderate and severe joint lesions were not reflected in locomotory ability in this study. As joint lesions have previously been associated with stiff movements and out turned fore legs, (Jorgensen and Andersen, 2000; Jørgensen and Nielsen, 2005; Kirk *et al.*, 2008; Jensen and Toft, 2009; de Koning *et al.*, 2012). The identification of non-invasive methods of osteochondrosis detection could provide commercially valuable information regarding its development. Such methods could include the monitoring of more subtle changes in gait through the use of kinematics, footprint analysis and weight distribution analysis (Nalon *et al.*, 2013). Future work could examine in more detail the beneficial effects of a gilt diet designed to specifically improve gilt limb health, its effectiveness in commercial conditions, and whether improvements persist over multiple parities. Quantification of longevity and production benefits will also have significant commercial value.

7.2.2 Limb and foot lesions

Chapter 2 provides the first information on the prevalence of coronary band damage and associated risk factors in piglets. There was a high prevalence of damage to the coronary band in piglets of less than one week of age (19%), which then decreased with increasing age. This lesion is of high biological importance as it compromises immediate piglet welfare as a result of pain and potential for

infection, which in turn could have longer term welfare and productivity implications (KilBride *et al.*, 2009b). Additionally, in piglets, weaners, finishers, gilts and sows (Chapters 2, 3, and 4), a similarly influential lesion, limb and foot swellings, was quantified. Swellings have the potential to reduce pig welfare as they are the result of an inflammatory response, which is associated with pain and reduced performance. Thus prevalence should be kept to a minimum (Johnson, 1997; Reichlin, 1999; Lucia *et al.*, 2000; Mülling and Greenough, 2006; Anil *et al.*, 2009; Wilson *et al.*, 2009; Ossent, 2010; Wilson *et al.*, 2010; Wilson and Ward, 2012).

Floor type, particularly floor material, influenced the prevalence of both limb and foot lesions. In relation to piglets it seems the development of certain lesions types such as skin abrasions appears to be unavoidable with unbedded indoor systems. The use of oval slatted plastic floors in the piglet area of the farrowing pen however, were associated with a lower risk of sole bruising, probably as a result of improved support for the foot pad. Metal slats on the other hand (Gregory and Grandin, 2007) were associated with an increased risk of coronary band damage, sole erosion and limb swellings, probably due to their abrasive properties. In Chapter 2 the presence of swellings in piglets was associated with lesions that allow for the entry for pathogens, thus causing infection resulting in an inflammatory response (Penny *et al.*, 1971; Mouttotou and Green, 1999b; Knura-Deszczka *et al.*, 2002; Straw *et al.*, 2006; KilBride *et al.*, 2009b). It is therefore important to reduce the prevalence of lesions which penetrate the epidermis, such as sole erosion and coronary band damage, in an effort to reduce limb infection. The results from Chapter 2 suggest that avoiding the use of metal slats in both the piglet and sow areas of the lactation pen should be recommended to reduce lesions associated with infection and the most potential for reduced welfare. Previous research indicates that use of alternative flooring material to metal, such as the addition of bedding or rubber mats, may prove beneficial to limb health (Gravås, 1979; Furniss *et al.*, 1986; Mouttotou *et al.*, 1999c; KilBride *et al.*, 2009b).

Limb and foot lesions in weaner and finisher pigs may also be affected by the pen floor type (Chapter 3). Concrete slats were associated with a higher risk of certain limb lesions, scratches, wounds and alopecia in weaners. This is likely related to the high abrasiveness of concrete flooring compared with plastic; indeed concrete has been previously associated with other lesions such as callus, bursitis and capped hock (Cagienard *et al.*, 2005; Gillman *et al.*, 2008; KilBride *et al.*, 2008). The use of partially slatted flooring in finishers as opposed to fully slatted flooring was associated with an increased risk of alopecia, scratches and wounds to the limbs. It is hypothesised this may be related to the use of solid areas to create functionally distinct lying zones in pens away from desirable resources (feeders, drinkers, environmental enrichment) to prevent interrupted lying behaviour making pigs less likely to be interrupted (stood upon or encourage aggression) (Boyle *et al.*, 2012; Levis *et al.*, 2013).

Changing floor materials to those more favourable to good animal welfare involves significant financial investment, which would then require a reciprocal financial saving as a result of improved productivity/longevity and reduced mortality to be justifiable from a producers perspective. As the link between limb and foot lesions and productivity, particularly in terminal line stock, is poorly understood, research into this area is required in order to allow for a cost benefit analysis. Additionally, results from this study could guide future legislative decisions regarding minimum flooring standards for pigs, for example in relation to flooring material, whereby the use of floor types hazardous for pig heath could be prohibited, or the use of bedding of some form made a requirement.

Limited environmental risk factors for reducing lameness, limb and claw lesions in weaners, finishers, gilts and sows were identified (Chapters 3 and 4). This may be a reflection of the intensive housing systems operated in Ireland; slatted flooring predominates and bedding is not provided, and both these factors are linked to reduced lameness and limb and claw lesions (Gravås, 1979; Furniss *et al.*, 1986; Mouttotou *et al.*, 1999c; Lewis *et al.*, 2005; KilBride *et al.*, 2009b; Zoric *et al.*, 2009). This results in a high uniformity of pen types throughout Ireland which hinders risk

factor identification and also limits the ability of these farms to reduce the prevalence of these injuries, without significant change to the existing infrastructure.

Factors influencing the development and healing of lesions throughout a pig's life are still unclear. A cohort study following piglets over time could examine the development and progression of limb lesions and lameness from birth to slaughter for terminal line stock, and birth through several parities for maternal line stock. This may reveal if lesions remain the same, become more severe, or heal over time, and how the physical environment affects recovery.

7.3 Limitations of the research

The cross-sectional study carried out (Chapters 2, 3 and 4) sampled 24% of Irish pig farms distributed across representative herd sizes and geographic locations. The farms that were used in this study were sourced from a database of farmers who opted to be clients of the Teagasc advisory service. The results collected may thus be biased towards herds that are more production and health focused than the average, as they are using advice from pig development specialists on a regular basis. It is thus possible that the findings presented in this thesis underestimate the national prevalence of the lesions examined. A limitation of a cross-sectional study design (Chapter 2, 3 and 4) is the difficulty associated with determining cause and effect. This study attempted to overcome this limitation through the sampling of multiple age groups per category, to identify trends with age and trends with environment over time (dose effect), and indeed some trends were observed.

A low level of severe lameness and claw lesions (i.e. amputated toes and dewclaws) was observed in finishers, gilts and sows (Chapters 3 and 4). While it is possible that these severe injuries have a low prevalence, it is more likely that these injuries were underestimated because severely injured or lame pigs (i.e. ≥ score 3, in this study) are removed from the pen for treatment, or in extreme cases culled. As this is a cross-sectional study, only taking a snap shot of lameness on each farm on a single

visit, it is difficult to obtain a representative sample of the range of lameness on farm. A potential way to overcome this may be to include “hospital pens” in future studies.

In relation to Chapters 5 and 6 whereby gilt developer diets were provided as alternative feeding regime, several factors (energy and lysine ratio and Ca, P, Zn, Mg, Cu content) varied from the two most commonly fed regimes. This makes it difficult to attribute the beneficial aspects of the diet to a specific component. Future work could alter aspects of the developer diet formulation to examine if the potential for further beneficial effect on lameness, joint lesions and aBMD.

Chapter 8

Overall conclusions

The results from the cross-sectional survey and trials conducted for this thesis provide valuable information in relation to lameness, limb and claw lesion prevalence and their risk factors as well as the potential limb health benefits of feeding a specifically designed gilt developer diet. It can be concluded that:

- The prevalence of severe foot and limb lesions in commercial farms in Ireland is high and is a substantial welfare concern.
- Severe lesions in piglets, including sole erosion, coronary band damage and swellings, could be reduced by avoiding the use of metal flooring in both the piglet and sow areas of the farrowing pen.
- Very high levels of lameness in finisher pigs were observed and were influenced by both a slat void width of greater than 20 mm and pen cleanliness (cleaned more than 4 times per year).
- A high lameness prevalence was observed in pregnant gilts and sows, particularly those that were group housed. However, owing to the lack of variation between systems in which such animals were kept, no other environmental or management risk factors were identified.
- The high prevalence of lameness in replacement gilts is a substantial welfare and economic concern due to their value and important role as the future breeding herd.
- Feeding a diet specifically formulated for developing replacement gilts reduced lameness levels when compared to the two most commonly practiced feeding regimes for developing gilts.
- Restrictive feeding a gilt developer diet during gilt development reduced joint lesion prevalence and claw unevenness, while no such joint lesion and claw benefits were observed when *ad-libitum* feeding during development,

however an increase in areal bone mineral density was observed in the latter situation.

These conclusions provide valuable information for key decision making groups and practically in relation to farm decisions (management, pen design and nutrition) for pig producers. This consequently provides the potential to improve limb health throughout the development stage and ultimately lead to improved welfare.

Bibliography

Almeida, F., Kirkwood, R., Aherne, F. and Foxcroft, G. (2000). Consequences of different patterns of feed intake during the estrous cycle in gilts on subsequent fertility. *Journal of Animal Science* **78**, 1556-1563.

Ammann, P. and Rizzoli, R. (2003). Bone strength and its determinants. *Osteoporosis International* **14**, 13-18.

Andersen, I. and Bøe, K. (1999). Straw bedding or concrete floor for loose-housed pregnant sows: consequences for aggression, production and physical health. *Acta Agriculturae Scandinavica, Section A-Animal Science* **49**, 190-195.

Anil, S. S. (2011). Epidemiology of lameness in breeding female pigs. In *PhD dissertation*, p. 113. University of Minnesota.

Anil, S. S., Anil, L. and Deen, J. (2002). Challenges of pain assessment in domestic animals. *Journal of the American Veterinary Medical Association* **220**, 313-319.

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. *Journal of the American Veterinary Medical Association* **226**, 956-961.

Anil, S. S., Anil, L. and Deen, J. (2008). Analysis of periparturient risk factors affecting sow longevity in breeding herds. *Canadian Journal of Animal Science* **88**, 381-389.

Anil, S. S., Anil, L. and Deen, J. (2009). Effect of lameness on sow longevity. *Journal of the American Veterinary Medical Association* **235**, 734-738.

Anil, S. S., Anil, L., Deen, J., Baidoo, S. K. and Walker, R. D. (2007). Factors associated with claw lesions in gestating sows. *Journal of Swine Health and Production* **15**, 78-83.

Arey, D. and Edwards, S. (1998). Factors influencing aggression between sows after mixing and the consequences for welfare and production. *Livestock Production Science* **56**, 61-70.

Arey, D. S. and Brooke, P. (2006). Animal welfare aspects of good agricultural practice: pig production. p. 82. Compassion in World Farming Trust.

Arnbjerg, J. (2007). Effect of a low-growth rate on the frequency of osteochondrosis in Danish Landrace pigs. *Archives of Animal Breeding* **50**, 105-111.

Barnett, J., Hemsworth, P., Cronin, G., Jongman, E. and Hutson, G. (2000). A review of the welfare issues for sows and piglets in relation to housing. *Crop and Pasture Science* **52**, 1-28.

Bates, R. O., Edwards, D. B. and Korthals, R. L. (2003). Sow performance when housed either in groups with electronic sow feeders or stalls. *Livestock Production Science* **79**, 29-35.

Baumann, G. and Bilkei, G. (2002). Effect of vaccinating sows and their piglets on the development of Glasser's disease induced by a virulent strain of Haemophilus parasuis serovar 5. *The Veterinary Record* **151**, 18-21.

Baxter, S. (1984). *Intensive Pig Production: Environmental Management and Design*. p. 588: Granada Publishing Ltd, London.

Bench, C. and Gonyou, H. (2006). Effect of environmental enrichment at two stages of development on belly nosing in piglets weaned at fourteen days. *Journal of Animal Science* **84**, 3397-3403.

Björklund, L. and Boyle, L. (2006). Effects of finishing boars on mixed and single sex groups and split marketing on pig welfare. *Acta Veterinaria Scandinavica* **48**, 2.

Bonde, M., Rousing, T., Badsberg, J. H. and Sørensen, J. T. (2004). Associations between lying-down behaviour problems and body condition, limb disorders and skin lesions of lactating sows housed in farrowing crates in commercial sow herds. *Livestock Production Science* **87**, 179-187.

Boon, C. and Wray, C. (1989). Building design in relation to the control of diseases of intensively housed livestock. *Journal of Agricultural Engineering Research* **43**, 149-161.

BordBia (2014). Factsheet on the Irish Agriculture and Food & Drink Sector. Bordbia.

Boyd, R. D., Castro, G. C. and Cabrera, R. A. (2002). Nutrition and management of the sow to maximize lifetime productivity. *Advances in Pork Production* **13**, 47-59.

Boyle, L. and Björklund, L. (2007). Effects of fattening boars in mixed or single sex groups and split marketing on pig welfare. *Animal Welfare* **16**, 259-262.

Boyle, L., Bullo, E. and Nolan, T. C. (2010). Lameness and limb lesions in replacement gilts on a commercial farm. *Advances in Animal Biosciences* **1**, 198-198.

Boyle, L., Carroll, C., McCutcheon, M., Clarke, S., McKeon, M., Lawlor, P., Ryan, T., Fitzgerald, T., Quinn, A., Calderón Díaz, J. A. and Lemos Teixeira, D. (2012). Towards January 2013. Updates, implications and options for group housing pregnant sows. p. 75. Teagasc, Pig Development Department. Moorepark, Fermoy, Co. Cork, Ireland.

Boyle, L., Leonard, F., Lynch, B. and Brophy, P. (1998). Sow culling patterns and sow welfare. *Irish Veterinary Journal* **51**, 354-357.

Boyle, L., Leonard, F., Lynch, P. and Brophy, P. (1999). Prevalence and severity of skin lesions in sows housed individually during the production cycle. *Irish Veterinary Journal* **52**, 601-605.

Boyle, L., Leonard, F., Lynch, P. and Brophy, P. (2000). Influence of housing system during gestation on the behaviour and welfare of gilts in farrowing crates. *Animal Science* **71**, 561-570.

Boyle, L., Leonard, F., Lynch, P. and Brophy, P. (2002). Effect of gestation housing on behaviour and skin lesions of sows in farrowing crates. *Applied Animal Behaviour Science* **76**, 119-134.

BPEX (2004). An industry update on farrowing systems. In *British Pig Executive, Milton Keynes, U.K.*, p. 30.

Bradley, C. L. (2010). Evaluating the impact of dietary inorganic or organic trace mineral supplementation on gilt development and sow reproduction, lameness, and longevity. In *PhD Dissertation*, p. 398. University of Arkansas.

Brana, D., Ellis, M., Castaneda, E., Sands, J. and Baker, D. (2006). Effect of a novel phytase on growth performance, bone ash, and mineral digestibility in nursery and grower-finisher pigs. *Journal of Animal Science* **84**, 1839-1849.

Brennan, J. and Aherne, F. (1986). Effect of dietary calcium and phosphorus levels on performance, bone bending moment and the severity of osteochondrosis and lameness in boars and gilts slaughtered at 100 or 130 kg body weight. *Canadian Journal of Animal Science* **66**, 777-790.

Brennan, J. and Aherne, F. (1987). Effect of floor type on the severity of foot lesions and osteochondrosis in swine. *Canadian Journal of Animal Science* **67**, 517-523.

Breuer, K., Sutcliffe, M. E. M., Mercer, J. T., Rance, K. A., O'Connell, N. E., Sneddon, I. A. and Edwards, S. A. (2005). Heritability of clinical tail-biting and its relation to performance traits. *Livestock Production Science* **93**, 87-94.

Brooks, P., Smith, D. and Irwin, V. (1977). Biotin-supplementation of diets; the incidence of foot lesions, and the reproductive performance of sows. *The Veterinary Record* **101**, 46-50.

Broom, D., Mendl, M. and Zanella, A. (1995). A comparison of the welfare of sows in different housing conditions. *Animal Science* **61**, 369-385.

Bryant, K., Knight, J. and Kornegay, E. (1980). Supplemental Biotin for Swine. II. Foot Lesions for Developing Gilts. In *Research Report*, pp. 136-144.

Busch, M. E. and Wachmann, H. (2011). Osteochondrosis of the elbow joint in finishing pigs from three herds: associations among different types of joint changes and between osteochondrosis and growth rate. *Veterinary Journal* **188**, 197-203.

Cagienard, A., Regula, G. and Danuser, J. (2005). The impact of different housing systems on health and welfare of grower and finisher pigs in Switzerland. *Preventive Veterinary Medicine* **68**, 49-61.

Calderón Díaz, J. A. (2013). Effect of Rubber Covered Slats on the Behaviour, Health and Welfare of Group Housed Sows. In *PhD thesis*, p. 225. University College Dublin.

Calderón Díaz, J. A. and Boyle, L. A. (2014). Effect of rubber slat mats on the behaviour and welfare of group housed pregnant sows. *Applied Animal Behaviour Science* **151**, 13-23.

Calderón Díaz, J. A., Fahey, A. and Boyle, L. (2014). Effects of gestation housing system and floor type during lactation on locomotory ability; body, limb, and claw lesions; and lying-down behavior of lactating sows. *Journal of Animal Science* **92**, 1675-1685.

Calderón Díaz, J. A., Fahey, A., Kilbride, A. L., Green, L. E. and Boyle, L. (2013). Longitudinal study of the effect of rubber slat mats on locomotory ability, body, limb and claw lesions, and dirtiness of group housed sows. *Journal of Animal Science* **91**, 3940-3954.

Campbell, J. R., Greenough, P. R. and Petrie, L. (2000). The effects of dietary biotin supplementation on vertical fissures of the claw wall in beef cattle. *The Canadian Veterinary Journal* **41**, 690-694.

Carlson, C., Hilley, H., Meuten, D., Hagan, J. and Moser, R. (1988). Effect of reduced growth rate on the prevalence and severity of osteochondrosis in gilts. *American Journal of Veterinary Research* **49**, 396-402.

Carlson, C., Meuten, D. and Richardson, D. (1991). Ischemic necrosis of cartilage in spontaneous and experimental lesions of osteochondrosis. *Journal of Orthopaedic Research* **9**, 317-329.

Carvalho, V. C. d., de Alencar Nääs, I., Mollo Neto, M. and Souza, S. R. L. d. (2009). Measurement of pig claw pressure distribution. *Biosystems Engineering* **103**, 357-363.

Chapinal, N., Ruiz de la Torre, J., Cerisuelo, A., Gasa, J., Baucells, M., Coma, J., Vidal, A. and Manteca, X. (2010). Evaluation of welfare and productivity in pregnant sows kept in stalls or in 2 different group housing systems. *Journal of Veterinary Behavior: Clinical Applications and Research* **5**, 82-93.

Christensen, J., Ellegaard, B., Kirkegaard Petersen, B., Willeberg, P. and Mousing, J. (1994). Pig health and production surveillance in Denmark: sampling design, data recording, and measures of disease frequency. *Preventive Veterinary Medicine* **20**, 47-61.

Christensen, O. F., Busch, M. E., Gregersen, V. R., Lund, M. S., Nielsen, B., Vingborg, R. K. and Bendixen, C. (2010). Quantitative trait loci analysis of osteochondrosis traits in the elbow joint of pigs. *Animal* **4**, 417-24.

Cook, N. B. and Nordlund, K. V. (2009). The influence of the environment on dairy cow behavior, claw health and herd lameness dynamics. *The Veterinary Journal* **179**, 360-369.

Crenshaw, T., Schneider, D., Carlson, C., Parker, J., Sonderman, J., Ward, T. and Wilson, M. (2013). Tissue mineral concentrations and osteochondrosis lesions in profilic sows across parities 0 through 7. *Journal of Animal Science*, 2012-5252.

CSO (2014). Pig Survey. p. 1: CSO statistical release.

D'Allaire, S., Stein, T. and Leman, A. (1987). Culling patterns in selected Minnesota swine breeding herds. *Canadian Journal of Veterinary Research* **51**, 506.

de Koning, D., van Grevenhof, E., Laurenssen, B., Ducro, B., Heuven, H., de Groot, P., Hazeleger, W. and Kemp, B. (2012). Associations between osteochondrosis and conformation and locomotive characteristics in pigs. *Journal of Animal Science* **90**, 4752-4763.

de Koning, D., van Grevenhof, E., Laurenssen, B., van Weeren, P., Hazeleger, W. and Kemp, B. (2014). The influence of floor type before and after 10 weeks of age on osteochondrosis in growing gilts. *Journal of Animal Science* **92**, 3338-3347.

de Koning, R. (1985). On the well-being of dry sows. In *PhD dissertation*. University of Utrecht, Utrecht, the Netherlands.

Deen, J., Anil, S. and Anil, D. (2007). Claw lesions as a predictor of lameness in breeding sows. In *Book of abstracts of 58th annual meeting of the European Association for Animal production, Dublin, Ireland August*, pp. 26-29.

Dewey, C. E., Friendship, R. M. and Wilson, M. R. (1993). Clinical and postmortem examination of sows culled for lameness. *The Canadian Veterinary Journal* **34**, 555.

Donabédian, M., Fleurance, G., ROBERT, C., Lepage, O., Leger, S., Ricard, A. and MARTIN-ROSSET, W. (2006). Effect of fast vs. moderate growth rate related to nutrient intake on developmental orthopaedic disease in the horse. *Animal Research* **55**, 471-486.

EFSA (2007). Animal health and welfare aspects of different housing and husbandry systems for adult breeding boars, pregnant, farrowing sows and unweaned piglets. *The EFSA Journal* **572**, 1-13.

Ekkel, E. D., Spoolder, H. A., Hulsegge, I. and Hopster, H. (2003). Lying characteristics as determinants for space requirements in pigs. *Applied Animal Behaviour Science* **80**, 19-30.

Ellingson, J. S., Karrikerb, L. A., Borgmannc, M. H. and A.C., B. (2012). Finishing Lameness – What Do We Know? , p. 8: SMEC Search and Summary of Current Evidence.

Elmore, M. R. P., Garner, J. P., Johnson, A. K., Richert, B. T. and Pajor, E. A. (2010). A flooring comparison: The impact of rubber mats on the health, behavior, and welfare of group-housed sows at breeding. *Applied Animal Behaviour Science* **123**, 7-15.

Engblom, L., Eliasson-Selling, L., Lundeheim, N., Belák, K., Andersson, K. and Dalin, A.-M. (2008). Post mortem findings in sows and gilts euthanised or found dead in a large Swedish herd. *Acta Veterinaria Scandinavica* **50**, 25-34.

Engblom, L., Lundeheim, N., Dalin, A.-M. and Andersson, K. (2007). Sow removal in Swedish commercial herds. *Livestock Science* **106**, 76-86.

European Comission (2013). Prospects for Agricultural Markets and Income in the EU 2013-2023 p. 138.

Ferket, P., Oviedo-Rondón, E., Mente, P., Bohórquez, D., Santos, A., Grimes, J., Richards, J., Dibner, J. and Felts, V. (2009). Organic trace minerals and 25-hydroxycholecalciferol affect performance characteristics, leg abnormalities, and biomechanical properties of leg bones of turkeys. *Poultry Science* **88**, 118-131.

Fritschen, R. (1979). Housing and its effect on feet and leg problems. *Pig Veterinary Society* **4**, 95-98.

Furniss, S., Edwards, S., Lightfoot, A. and Spechter, H. (1986). The effect of floor type in farrowing pens on pig injury. I. Leg and teat damage of suckling piglets. *British Veterinary Journal* **142**, 434-440.

Geverink, N. A., Engel, B., Lambooij, E. and Wiegant, V. M. (1996). Observations on behaviour and skin damage of slaughter pigs and treatment during lairage. *Applied Animal Behaviour Science* **50**, 1-13.

Gill, B. and Taylor, L. (1999). The nutritional management of gilts to enhance lifetime productivity: Second progress report on the Stotfold gilt trial–Body composition and first litter performance. *Society of Feed Technologists, Coventry, UK*.

Gillman, C. E., Kilbride, A. L., Ossent, P. and Green, L. E. (2008). A cross-sectional study of the prevalence and associated risk factors for bursitis in weaner, grower and finisher pigs from 93 commercial farms in England. *Preventive Veterinary Medicine* **83**, 308-22.

Gillman, C. E., KilBride, A. L., Ossent, P. and Green, L. E. (2009). A cross-sectional study of the prevalence of foot lesions in post-weaning pigs and risks associated with floor type on commercial farms in England. *Preventive Veterinary Medicine* **91**, 146-52.

Gjein, H. and Larssen, R. (1994). Housing of pregnant sows in loose and confined systems-a field study. 2. Claw lesions: morphology, prevalence, location and relation to age. *Acta Veterinaria Scandinavica* **36**, 433-442.

Gjein, H. and Larssen, R. (1995). Housing of pregnant sows in loose and confined systems-a field study 1. Vulva and body lesions, culling reasons and production results. *Acta Veterinaria Scandinavica* **36**, 185.

Goedegebuure, S., Häni, H., Van der Valk, P. and Van der Wal, P. (1980). Osteochondrosis in six breeds of slaughter pigs: I. A morphological investigation of the status of osteochondrosis in relation to breed and level of feeding. *Veterinary Quarterly* **2**, 28-41.

Goodband, R., Hines, R., Nelssen, J., Kropf, D. and Schricker, B. (1993). Porcine somatotropin and dietary lysine influence bone mineralization and mechanical properties of bones in finishing swine. *Journal of Animal Science* **71**, 673-678.

Grandjot, G. (2007). Claw problems cost money. *SUS—Schweinezucht und Schweinemast*, 28-31.

Gravås, L. (1979). Behavioural and physical effects of flooring on piglets and sows. *Applied Animal Ethology* **5**, 333-345.

Grégoire, J., Bergeron, R., D'Allaire, S., Meunier-Salaün, M.-C. and Devillers, N. (2013). Assessment of lameness in sows using gait, footprints, postural behaviour and foot lesion analysis. *Animal* **7**, 1163-1173.

Gregory, N. G. and Grandin, T. (2007). *Animal welfare and meat production*. CABI, p. 400.

Grondalen, T. (1974). Osteochondrosis i and arthrosis in pigs. III. A comparison of the incidence in young animals of the Norwegian Landrace and Yorkshire breeds. *Acta Veterinaria Scandinavica* **15**, 43-52.

Grondalen, T. and Vangen, O. (1974). Osteochondrosis and arthrosis in pigs. V. A comparison of the incidence in three different lines of the Norwegian Landrace breed. *Acta Veterinaria Scandinavica* **15**, 61-79.

Hacker, R., Ogilvie, J., Morrison, W. and Kains, F. (1994). Factors affecting excretory behavior of pigs. *Journal of Animal Science* **72**, 1455-1460.

Hall, D., Cromwell, G. and Stahly, T. (1991). Effects of dietary calcium, phosphorus, calcium: phosphorus ratio and vitamin K on performance, bone strength and blood clotting status of pigs. *Journal of Animal Science* **69**, 646-655.

Harper, A., Coffey, R., Hollis, G., Mahan, D. and Radcliffe, J. (2002). Swine Diets. In: Pork Industry Handbook. p. 23. Purdue University, West Lafayette, IN.

Harris, M. J., Pajor, E. A., Sorrells, A. D., Eicher, S. D., Richert, B. T. and Marchant-Forde, J. N. (2006). Effects of stall or small group gestation housing on the production, health and behaviour of gilts. *Livestock Science* **102**, 171-179.

Hedges, J., Blowey, R., Packington, A., O'Callaghan, C. and Green, L. (2001). A longitudinal field trial of the effect of biotin on lameness in dairy cows. *Journal of Dairy Science* **84**, 1969-1975.

Heinonen, M., Oravainen, J., Orro, T., Seppä-Lassila, L., Ala-Kurikka, E., Virolainen, J., Tast, A. and Peltoniemi, O. (2006). Lameness and fertility of sows and gilts in

randomly selected loose-housed herds in Finland. *The Veterinary Record* **159**, 383-387.

Heinonen, M., Peltoniemi, O. and Valros, A. (2013). Impact of lameness and claw lesions in sows on welfare, health and production. *Livestock Science* **156**, 2-9.

Hill, M., Kincaid, S. and Visco, D. (1990). Use of histochemical techniques in the characterisation of osteochondroses affecting pigs. *The Veterinary Record* **127**, 29-37.

Jackson, P. G. and Cockcroft, P. D. (2007). *Handbook of pig medicine*. Elsevier Health Sciences, p. 296.

Jendza, J., Dilger, R., Adedokun, S., Sands, J. and Adeola, O. (2005). Escherichia coli phytase improves growth performance of starter, grower, and finisher pigs fed phosphorus-deficient diets. *Journal of Animal Science* **83**, 1882-1889.

Jensen, K. H., Bertelsen, D., Pedersen, A. R., Nielsen, N. and Vestergaard, K. (2000). Management factors affecting activity and aggression in dynamic group housing systems with electronic sow feeding: a field trial. *Animal Science* **71**, 535-546.

Jensen, T. and Toft, N. (2009). Causes of and predisposing risk factors for leg disorders in growing-finishing pigs. *CAB Reviews: Perspectives in Agriculture, Veterinary Science, Nutrition and Natural Resources* **4**, 1-8.

Jensen, T. B., Baadsgaard, N. P., Houe, H., Toft, N. and Østergaard, S. (2007). The effect of lameness treatments and treatments for other health disorders on the weight gain and feed conversion in boars at a Danish test station. *Livestock Science* **112**, 34-42.

Jensen, T. B., Bonde, M. K., Kongsted, A. G., Toft, N. and Sorensen, J. T. (2010). The interrelationships between clinical signs and their effect on involuntary culling among pregnant sows in group-housing systems. *Animal* **4**, 1922-8.

Jensen, T. B., Kristensen, H. H. and Toft, N. (2012). Quantifying the impact of lameness on welfare and profitability of finisher pigs using expert opinions. *Livestock Science* **149**, 209-214.

Johansen, M., Alban, L., Kjærsgård, H. D. and Bækbo, P. (2004). Factors associated with suckling piglet average daily gain. *Preventive Veterinary Medicine* **63**, 91-102.

Johnson, A. K. and Marchant-Forde, J. N. (2009). Welfare of pigs in the farrowing environment. In *The Welfare of Pigs*, pp. 141-188. Springer.

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

Jørgensen, B. (1995). Effect of different energy and protein levels on leg weakness and osteochondrosis in pigs. *Livestock Production Science* **41**, 171-181.

Jørgensen, B. (2002). Influence of floor type and stocking density on leg weakness, osteochondrosis, and claw disorders in slaughter pigs. *Animal Science* **77**, 439-449.

Jorgensen, B. and Andersen, S. (2000). Genetic parameters for osteochondrosis in Danish Landrace and Yorkshire boars and correlations with leg weakness and production traits. *Animal Science* **71**, 427-434.

Jørgensen, B., Arnbjerg, J. and Aaslyng, M. (1995). Pathological and radiological investigations on osteochondrosis in pigs, associated with leg weakness. *Journal of Veterinary Medicine Series A* **42**, 489-504.

Jørgensen, B. and Nielsen, B. (2005). Genetic parameters for osteochondrosis traits in elbow joints of crossbred pigs and relationships with production traits. *Animal Science* **81**, 319-324.

Jørgensen, B. and Sørensen, M. T. (1998). Different rearing intensities of gilts::: II. Effects on subsequent leg weakness and longevity. *Livestock Production Science* **54**, 167-171.

Karlen, G. A., Hemsworth, P. H., Gonyou, H. W., Fabrega, E., David Strom, A. and Smits, R. J. (2007). The welfare of gestating sows in conventional stalls and large groups on deep litter. *Applied Animal Behaviour Science* **105**, 87-101.

Kestin, S., Gordon, S., Su, G. and Sørensen, P. (2001). Relationships in broiler chickens between lameness, liveweight, growth rate and age. *Veterinary Record* **148**, 195-197.

Kestin, S., Knowles, T., Tinch, A. and Gregory, N. (1992). Prevalence of leg weakness in broiler chickens and its relationship with genotype. *The Veterinary Record* **131**, 190-194.

KilBride, A. L. (2008). An epidemiological study of foot, limb and body lesions and lameness in pigs. In *PhD Dissertation, School of Life Sciences*, p. 283. University of Warwick.

KilBride, A. L., Gillman, C. E. and Green, L. E. (2009a). A cross-sectional study of the prevalence of lameness in finishing pigs, gilts and pregnant sows and associations with limb lesions and floor types on commercial farms in England. *Animal Welfare* **18**, 215-224.

KilBride, A. L., Gillman, C. E., Ossent, P. and Green, L. E. (2008). A cross-sectional study of the prevalence and associated risk factors for capped hock and the associations with bursitis in weaner, grower and finisher pigs from 93 commercial farms in England. *Preventive Veterinary Medicine* **83**, 272-284.

KilBride, A. L., Gillman, C. E., Ossent, P. and Green, L. E. (2009b). A cross sectional study of prevalence, risk factors, population attributable fractions and pathology for foot and limb lesions in preweaning piglets on commercial farms in England. *BMC Veterinary Research* **5**, 31-42.

Kilbride, A. L., Mendl, M., Statham, P., Held, S., Harris, M., Cooper, S. and Green, L. E. (2012). A cohort study of preweaning piglet mortality and farrowing accommodation on 112 commercial pig farms in England. *Preventive Veterinary Medicine* **104**, 281-91.

Kirk, R. K., Jørgensen, B. and Jensen, H. E. (2008). The impact of elbow and knee joint lesions on abnormal gait and posture of sows. *Acta Veterinaria Scandinavica* **50**, 5-12.

Kirk, R. K., Svensmark, B., Ellegaard, L. and Jensen, H. (2005). Locomotive disorders associated with sow mortality in Danish pig herds. *Journal of Veterinary Medicine Series A* **52**, 423-428.

Klindt, J., Yen, J. and Christenson, R. (1999). Effect of prepubertal feeding regimen on reproductive development of gilts. *Journal of Animal Science* **77**, 1968-1976.

Klindt, J., Yen, J. T. and Christenson, R. (2001). Effect of prepubertal feeding regimen on reproductive development and performance of gilts through the first pregnancy. *Journal of Animal Science* **79**, 787-795.

Knauer, M., Stalder, K. J., Karriker, L., McKean, J. D., Serenius, T., Baas, T. J., Johnson, C. D. and Layman, L. L. (2007). Characterization of US Cull Sows. *Animal Industry Report* **653**, 65.

Knauer, M. T., Cassady, J. P., Newcom, D. W. and See, M. T. (2012). Gilt development traits associated with genetic line, diet and fertility. *Livestock Science* **148**, 159-167.

Knura-Deszczka, S., Lipperheide, C., Petersen, B., Jobert, J., Berthelot-Herault, F., Kobisch, M. and Madec, F. (2002). Plasma haptoglobin concentration in swine after challenge with Streptococcus suis. *Journal of Veterinary Medicine, Series B* **49**, 240-244.

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

Leeb, B., Leeb, C., Troxler, J. and Schuh, M. (2001). Skin lesions and callosities in group-housed pregnant sows: animal-related welfare indicators. *Acta Agriculturae Scandinavica* **51**, 82-87.

Levis, D. G., Consultancy, L. W. S. and Connor, L. (2013). Group Housing Systems: Choices and Designs. *National Pork Board. Des Moines, IA*.

Levis, D. G., Leibbrandt, V. D. and Rozeboom, D. W. (1997). Development of gilts and boars for efficient reproduction. In *Pork Industry Hanbook*, p. 8. Indiana: Purdue University.

Lewis, E., Boyle, L., O'Doherty, J., Brophy, P. and Lynch, P. B. (2005). The effect of floor type in farrowing crates on piglet welfare. *Irish Journal of Agricultural and Food Research* **44**, 69-81.

Lorenz-Depiereux, B., Guido, V. E., Johnson, K. R., Zheng, Q. Y., Gagnon, L. H., Bauschatz, J. D., Davisson, M. T., Washburn, L. L., Donahue, L. R. and Strom, T. M. (2004). New intragenic deletions in the Phex gene clarify X-linked hypophosphatemia-related abnormalities in mice. *Mammalian Genome* **15**, 151-161.

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

Luescher, U., Friendship, R. and McKeown, D. (1990). Evaluation of methods to reduce fighting among regrouped gilts. *Canadian Journal of Animal Science* **70**, 363-370.

Lundeheim, N. (1987). Genetic analysis of osteochondrosis and leg weakness in the Swedish pig progeny testing scheme. *Acta Agriculturae Scandinavica* **37**, 159-173.

Mahan, D. (1982). Dietary calcium and phosphorus levels for weanling swine. *Journal of Animal Science* **54**, 559-564.

Mahan, D. (1990). Mineral nutrition of the sow: a review. *Journal of Animal Science* **68**, 573-582.

Main, D., Clegg, J., Spatz, A. and Green, L. (2000). Repeatability of a lameness scoring system for finishing pigs. *Veterinary Record* **147**, 574-576.

Marchant Forde, J. and Broom, D. (1996). Effects of dry sow housing conditions on muscle weight and bone strength. *Animal Science* **62**, 105-113.

Marchant Forde, J. N. (2008). *The welfare of pigs*. Springer, p. 349.

Marchant Forde, J. N., Rudd, A. R. and Broom, D. M. (1997). The effects of housing on heart rate of gestating sows during specific behaviours. *Applied Animal Behaviour Science* **55**, 67-78.

McKee, C. and Dumelow, J. (1995). A review of the factors involved in developing effective non-slip floors for pigs. *Journal of Agricultural Engineering Research* **60**, 35-42.

Meese, G. and Ewbank, R. (1973). The establishment and nature of the dominance hierarchy in the domesticated pig. *Animal Behaviour* **21**, 326-334.

Miller, P., Moreno, R. and Johnson, R. (2011). Effects of restricting energy during the gilt developmental period on growth and reproduction of lines differing in lean growth rate: responses in feed intake, growth, and age at puberty. *Journal of Animal Science* **89**, 342-354.

Mills, D., Marchant-Forde, J., McGreevy, P., Morton, D., Nicol, C., Phillips, C., Sandoe, P. and Swaisgood, R. (2010). *The encyclopedia of applied animal behaviour and welfare*. CABI, Wallingford, U.K., p. 656.

Mitchell, A. (2007). Impact of research with cattle, pigs, and sheep on nutritional concepts: body composition and growth. *The Journal of nutrition* **137**, 711-714.

Mitchell, A., Conway, J. and Scholz, A. (1995). Incremental changes in total and regional body composition of growing pigs measured by dual-energy x-ray absorptiometry. *Growth, Development and Aging* **60**, 95-105.

Mitchell, A., Scholz, A., Pursel, V. and Evock-Clover, C. (1998). Composition analysis of pork carcasses by dual-energy X-ray absorptiometry. *Journal of Animal Science* **76**, 2104-2114.

Mohammadina, A. (2008). The role of trace minerals in bovine claw horn quality and lameness *Iranian Journal of Veterinary Surgery*, 133-140.

Mouttotou, N. and Green, L. (1999a). Foot and limb lesions in growing pigs. *The Pig Journal* **43**, 54-71.

Mouttotou, N. and Green, L. (1999b). Incidence of foot and skin lesions in nursing piglets and their association with behavioural activities. *The Veterinary Record* **145**, 160-165.

Mouttotou, N., Hatchell, F. and Green, L. (1998). Adventitious bursitis of the hock in finishing pigs: prevalence, distribution and association with floor type and foot lesions. *The Veterinary Record* **142**, 109-114.

Mouttotou, N., Hatchell, F. and Green, L. (1999a). Foot lesions in finishing pigs and their associations with the type of floor. *Veterinary Record* **144**, 629-632.

Mouttotou, N., Hatchell, F. and Green, L. (1999b). Prevalence and risk factors associated with adventitious bursitis in live growing and finishing pigs in south-west England. *Preventive Veterinary Medicine* **39**, 39-52.

Mouttotou, N., Hatchell, F. and Green, L. (1999c). The prevalence and risk factors associated with forelimb skin abrasions and sole bruising in preweaning piglets. *Preventive Veterinary Medicine* **39**, 231-245.

Mouttotou, N., Hatchell, F. and Green, L. (1999d). Prevalence of foot and limb lesions in 528 weaned pigs from 15 units in south-west England. *Veterinary Record* **144**, 449-451.

Mouttotou, N., Hatchell, F., Lundervold, M. and Green, L. (1997). Prevalence and distribution of foot lesions in finishing pigs in south-west England. *The Veterinary Record* **141**, 115-120.

Mujuni, B., McFarlane, J., Curtis, S. and Taylor, I. (1985). Effect of group size on social interactions in newly weaned sows. *Journal of Animal Science* **63**, 164.

Mülling, C. K. and Greenough, P. R. (2006). Applied physiopathology of the foot. In *Proc. 24th World Buiatrics Congress. HF Navetat and HF Schelcher, ed. Nice, France*, pp. 103-117.

Mustonen, K., Ala-Kurikka, E., Orro, T., Peltoniemi, O., Raekallio, M., Vainio, O. and Heinonen, M. (2011). Oral ketoprofen is effective in the treatment of non-infectious lameness in sows. *Veterinary Journal* **190**, 55-9.

Nakano, T. and Aherne, F. (1988). Involvement of trauma in the pathogenesis of osteochondritis dissecans in swine. *Canadian Journal of Veterinary Research* **52**, 154.

Nakano, T., Aherne, F. and Thompson, J. (1979). Effects of feed restriction, sex and diethylstilbestrol on the occurrence of joint lesions with some histological and biochemical studies of the articular cartilage of growing-finishing swine. *Canadian Journal of Animal Science* **59**, 491-502.

Nakano, T., Brennan, J. and Aherne, F. (1987). Leg weakness and osteochondrosis in swine: a review. *Canadian Journal of Animal Science* **67**, 883-901.

Nalon, E., Conte, S., Maes, D., Tuyttens, F. A. M. and Devillers, N. (2013). Assessment of lameness and claw lesions in sows. *Livestock Science* **156**, 10-23.

Newton, G., Booram, C., Hale, O. and Mullinix Jr, B. (1980). Effect of four types of floor slats on certain feet characteristics and performance of swine. *Journal of Animal Science* **50**, 7-20.

Nielsen, D. H., McEvoy, F., Madsen, M., Jensen, J. and Svalastoga, E. (2007). Relationship between bone strength and dual-energy X-ray absorptiometry measurements in pigs. *Journal of Animal Science* **85**, 667-672.

Nielsen, D. H., McEvoy, F., Poulsen, H., Madsen, M., Buelund, L. E. and Svalastoga, E. (2004). Dual-energy X-ray absorptiometry of the pig: Protocol development and evaluation. *Meat Science* **68**, 235-241.

Nielsen, E., Nielsen, N. and Friis, N. (2001). Mycoplasma hyosynoviae Arthritis in Grower-Finisher Pigs. *Journal of Veterinary Medicine Series A* **48**, 475-486.

Nielsen, N., Bille, N., Larsen, J. and Svendsen, J. (1975). Preweaning mortality in pigs. 7. Polyarthritis. *Nordisk Veterinaermedicin* **27**, 529-543.

Nimmo, R., Peo Jr, E., Moser, B. and Lewis, A. (1981). Effect of level of dietary calcium-phosphorus during growth and gestation on performance, blood and bone parameters of swine. *Faculty Papers and Publications in Animal Science*, 656.

NRC (2012). Nutrient Requirements of Swine,. p. 400. National Academy Press, Washington, D.C.

O'Driscoll, K., O'Gorman, D. M., Taylor, S. and Boyle, L. A. (2013). The influence of a magnesium-rich marine extract on behaviour, salivary cortisol levels and skin lesions in growing pigs. *Animal* **7**, 1017-27.

Ossent, P. (2010). *An introduction to sow lameness, claw lesions and pathogenesis theories*. p. 44.

Pajor, E. (2002). Group housing of sows in small pens: Advantages, disagvantages and recent research. In *Symposium of swine housing and well-being*, pp. 37-44. U.S. Department of Agriculture: Des Moines, Iowa.

Papatsiros, V. G. (2012). The Splay Leg Syndrome in Piglets: A Review. *American Journal of Animal and Veterinary Sciences* **7**, 80-83.

Penny, R., Edwards, M. and Mulley, R. (1971). Clinical observations of necrosis of the skin of suckling piglets. *Australian Veterinary Journal* **47**, 529-537.

Penny, R., Osborne, A. and Wright, A. (1963). The causes and incidence of lameness in store and adult pigs. *Veterinary Record* **75**, 1225-1235.

Petersen, H. H., Nielsen, E., Hassing, A., Ersbøll, A. K. and Nielsen, J. P. (2008). Prevalence of clinical signs of disease in Danish finisher pigs. *The Veterinary Record* **162**, 377-382.

Phillips, P., Fraser, D. and Thompson, B. (1996). Sow preference for types of flooring in farrowing crates. *Canadian Journal of Animal Science* **76**, 485-489.

PigSys (2013). National Pig Herd Performance Report 2012. pp. 1-13. Teagasc Pig Development Department, Cork, Ireland.

Pluym, L., Van Nuffel, A., Dewulf, J., Cools, A., Vangroenweghe, F., Van Hoorebeke, S. and Maes, D. (2011). Prevalence and risk factors of claw lesions and lameness in pregnant sows in two types of group housing. *Veterinarni Medicina* **56**, 101-109.

Pluym, L., Van Nuffel, A. and Maes, D. (2013a). Treatment and prevention of lameness with special emphasis on claw disorders in group-housed sows. *Livestock Science* **156**, 36-43.

Pluym, L. M., Van Nuffel, A., Van Weyenberg, S. and Maes, D. (2013b). Prevalence of lameness and claw lesions during different stages in the reproductive cycle of sows and the impact on reproduction results. *Animal* **7**, 1174-81.

Pötzsch, C., Blowey, R., Packington, A. and Green, L. (2003). The impact of parity and duration of biotin supplementation on white line disease lameness in dairy cattle. *Journal of Dairy Science* **86**, 2577-2582.

Prunier, A., Heinonen, M. and Quesnel, H. (2010). High physiological demands in intensively raised pigs: impact on health and welfare. *Animal* **4**, 886-98.

Puls, R. (1994). *Mineral Levels in Animal Health: Diagnostic Data*. p. 356: Sherpa International, Clearbrook, BC, Canada.

Rasbash, J., Steele, F., Browne, W. and Goldstein, H. (2012). *A User's Guide to MLwiN V2.26*. p. 292: Centre for Multilevel Modelling, University of Bristol.

Rauw, W. (2007). Physiological consequences of selection for increased performance. In *17th Conference of the Association for the Advancement of Animal Breeding and Genetics*, pp. 240-247: Armidale, Austrailia.

Rauw, W., Kanis, E., Noordhuizen-Stassen, E. and Grommers, F. (1998). Undesirable side effects of selection for high production efficiency in farm animals: a review. *Livestock Production Science* **56**, 15-33.

Reichlin, S. (1999). Neuroendocrine consequences of systemic inflammation. *Military Strategies for Sustainment of Nutrition and Immune Function in the Field* **8**, 5724-5099.

Rothschild, M. and Christian, L. (1988). Genetic control of front-leg weakness in Duroc swine. I. Direct response to five generations of divergent selection. *Livestock Production Science* **19**, 459-471.

Rushen, J. (1984). Stereotyped behaviour, adjunctive drinking and the feeding periods of tethered sows. *Animal Behaviour* **32**, 1059-1067.

Ryan, W., Lynch, P. and O'Doherty, J. (2011). Effect of dietary phosphorus on the development of bone mineral density of pigs assessed using dual energy X-ray absorptiometry. *Livestock Science* **137**, 101-107.

Sather, A. and Fredeen, H. (1978). Effect of selection for lean growth rate upon feed utilization by the market hog. *Canadian Journal of Animal Science* **58**, 285-289.

Savage, C., McCarthy, R. and Jeffcott, L. (1993). Effects of dietary energy and protein on induction of dyschondroplasia in foals. *Equine Veterinary Journal* **25**, 74-79.

Savary, P., Gygax, L., Wechsler, B. and Hauser, R. (2009). Effect of a synthetic plate in the lying area on lying behaviour, degree of fouling and skin lesions at the leg joints of finishing pigs. *Applied Animal Behaviour Science* **118**, 20-27.

Scott, K., Chennells, D. J., Campbell, F. M., Hunt, B., Armstrong, D., Taylor, L., Gill, B. P. and Edwards, S. A. (2006). The welfare of finishing pigs in two contrasting housing systems: Fully-slatted versus straw-bedded accommodation. *Livestock Science* **103**, 104-115.

Sellier, P. and Ollivier, L. (1982). A Genetic Study of Splayleg in the Newborn Piglet. 1. Multifactorial Model with one Threshold. *Annales de Génétique et de Sélection Animale* **14**, 77-92.

Shearer, J., Bray, D., Umphrey, J. and Elliott, J. (1996). Factors influencing the incidence of lameness in dairy cattle. In *Annual FLorida Dairy Production Conference*, pp. 40-51.

Simonsen, H. (1993). PST treatment and leg disorders in growing swine: a welfare hazard? *Livestock Production Science* **36**, 67-70.

Smith, B. (1988). Lameness in pigs associated with foot and limb disorders. *In Practice* **10**, 113-117.

Smith, W. and Mitchell, C. (1976). Observations on injuries to suckled pigs confined on perforated floors with special reference to expanded metal. *The Pig Journal* **1**, 91-104.

Socha, M. T., Johnson, B. and Schugel, L. M. (2002). Effect of Nutrition on claw health. In *The Society of Dairy Cattle Veterinarians, New Zealand Veterinary Association Conference*, pp. 73-91.

Sørensen, M., Jørgensen, B. and Danielsen, V. (1993). Different feeding intensity of young gilts: Effect of growth, milk yield, reproduction, leg soundness, and longevity. In *National Institute of Animal Science, Copenhagen, Denmark.*, p. 14.

Stalder, K., Long, T., Goodwin, R., Wyatt, R. and Halstead, J. (2000). Effect of gilt development diet on the reproductive performance of primiparous sows. *Journal of Animal Science* **78**, 1125-1131.

Stavrakakis, S., Guy, J., Warlow, O., Johnson, G. and Edwards, S. (2014). Walking kinematics of growing pigs associated with differences in musculoskeletal conformation, subjective gait score and osteochondrosis. *Livestock Science*.

Stein, T., Dijkhuizen, A., D'Allaire, S. and Morris, R. (1990). Sow culling and mortality in commercial swine breeding herds. *Preventive Veterinary Medicine* **9**, 85-94.

Stern, S., Lundeheim, N., Johansson, K. and Andersson, K. (1995). Osteochondrosis and leg weakness in pigs selected for lean tissue growth rate. *Livestock Production Science* **44**, 45-52.

Stokka, G. L., Smith, J. F., Dunham, J. R. and Van Anne, T. (1997). Lameness in dairy cattle. p. 7. Kansas State University: Agricultural Experiment Station.

Straw, B. E., Zimmerman, J., D'Allaire, S. and Taylor, D. (2006). *Diseases of swine 9 th edition*. Blackwell Publishing Professional, p. 1153.

Street, B. and Gonyou, H. (2008). Effects of housing finishing pigs in two group sizes and at two floor space allocations on production, health, behavior, and physiological variables. *Journal of Animal Science* **86**, 982-991.

Svendsen, J., Olsson, O. and Nilsson, C. (1979). The occurrence of leg injuries on piglets with the various treatment of the floor surface of the farrowing pen. *Nordisk Veterinaermedicin* **31**, 49-61.

Tanck, E., Homminga, J., Van Lenthe, G. and Huiskes, R. (2001). Increase in bone volume fraction precedes architectural adaptation in growing bone. *Bone* **28**, 650-654.

Temple, D., Dalmau, A., Ruiz de la Torre, J. L., Manteca, X. and Velarde, A. (2011). Application of the Welfare Quality® protocol to assess growing pigs kept under intensive conditions in Spain. *Journal of Veterinary Behavior: Clinical Applications and Research* **6**, 138-149.

Temple, D., Manteca, X., Dalmau, A. and Velarde, A. (2013). Assessment of test–retest reliability of animal-based measures on growing pig farms. *Livestock Science* **151**, 35-45.

Tomlinson, D., Mülling, C. and Fakler, T. (2004). Formation of Keratins in the Bovine Claw: Roles of Hormones, Minerals, and Vitamins in Functional Claw Integrity. *Journal of Dairy Science* **87**, 797-809.

Tomlinson, D., Socha, M. and DeFrain, J. (2008). Role of trace minerals in the immune system. In *Penn State Dairy Cattle Nutrition Workshop*, pp. 39-52: Grantville, PA.

Turner, S. P., Farnworth, M. J., White, I., Brotherstone, S., Mendl, M., Knap, P., Penny, P. and Lawrence, A. B. (2006). The accumulation of skin lesions and their use as a predictor of individual aggressiveness in pigs. *Applied Animal Behaviour Science* **96**, 245-259.

Tuyttens, F. A. M. (2005). The importance of straw for pig and cattle welfare: A review. *Applied Animal Behaviour Science* **92**, 261-282.

Tuyttens, F. A. M., Wouters, F., Struelens, E., Sonck, B. and Duchateau, L. (2008). Synthetic lying mats may improve lying comfort of gestating sows. *Applied Animal Behaviour Science* **114**, 76-85.

Underwood, E. J. (1999). *The mineral nutrition of livestock*. CAB International. Wallingford. UK, p. 614.

Valros, A., Ala-Kurikka, E., Mustonen, K., Kettunen, S., Ahonen, P., Vainio, O., Peltoniemi, O. and Heinonen, M. (2009). Behavioural changes associated with lameness in sows. In *International Society of Applied Ethology* p. 113: Cairns, Australia.

Van Amstel, S. (2010). Practical understanding of claw lesions. In *FeetFirst® Sow Lameness Symposium II*, pp. 41-48: Minneapolis, Minnesota, USA.

Van Amstel, S. (2011). Questions and answers around sow claw trimming. *Pig Progress* **27**, p12-13.

van Grevenhof, E. M., Ott, S., Hazeleger, W., van Weeren, P. R., Bijma, P. and Kemp, B. (2011). The effects of housing system and feeding level on the joint-specific prevalence of osteochondrosis in fattening pigs. *Livestock Science* **135**, 53-61.

van Riet, M. M. J., Millet, S., Aluwé, M. and Janssens, G. P. J. (2013). Impact of nutrition on lameness and claw health in sows. *Livestock Science* **156**, 24-35.

Varley, P. F., Sweeney, T., Ryan, M. T. and O'Doherty, J. V. (2011). The effect of phosphorus restriction during the weaner-grower phase on compensatory growth, serum osteocalcin and bone mineralization in gilts. *Livestock Science* **135**, 282-288.

Velarde, A. and Geers, R. (2007). *On farm monitoring of pig welfare*. Wageningen Academic Pub, p. 207.

Vieuille-Thomas, C., Le Pape, G. and Signoret, J. P. (1995). Stereotypies in pregnant sows: indications of influence of the housing system on the patterns expressed by the animals. *Applied Animal Behaviour Science* **44**, 19-27.

Ward, P. (1978). The splayleg syndrome in new-born pigs: a review. Part 1. *Veterinary Bulletin* **48**, 279-295.

Wechsler, B., Schaub, J., Friedli, K. and Hauser, R. (2000). Behaviour and leg injuries in dairy cows kept in cubicle systems with straw bedding or soft lying mats. *Applied Animal Behaviour Science* **69**, 189-197.

Weeks, C., Danbury, T., Davies, H., Hunt, P. and Kestin, S. (2000). The behaviour of broiler chickens and its modification by lameness. *Applied Animal Behaviour Science* **67**, 111-125.

Welfare Quality Consortium (2009). *Welfare Quality® assessment protocol for pigs (sows and piglets, growing and finishing pigs)*. p. 122.

Weremko, D., Skiba, G., Raj, S. and Fandrejewski, H. (2013). The effects of feed and protein restriction between 90 and 118 days of age on performance, bone growth and mineralization of pigs reared to 168 days of age. *Animal Feed Science and Technology* **182**, 53-60.

Westin, R., Holmgren, N., Hultgren, J. and Algers, B. (2014). Large quantities of straw at farrowing prevents bruising and increases weight gain in piglets. *Preventive Veterinary Medicine* **115**, 181-190.

Willgert, K. J., Brewster, V., Wright, A. J. and Nevel, A. (2014). Risk factors of lameness in sows in England. *Preventive Veterinary Medicine* **113**, 268-72.

Wilson, M. and Ward, T. (2012). Impact of lameness on the productive potential of the sow. In *12th London Swine Conference Proceedings. A time for change, London, Ontario, Canada, 28-29 March 2012.*, pp. 27-33. London Swine Conference.

Wilson, M. E., Ward, T. and Rapp, C. (2010). Effects of Lameness on Sow Reproduction: Nutritional Impacts. In *FeetFirst® Sow Lameness Symposium II*: Minneapolis, Minnesota, USA.

Wilson, M. E., Ward, T. L. and Rapp, C. (2009). Lameness hurts sow reproduction. In *Feet First Symposium* pp. 64-74: Minneapolis, MN.

Wilson, P. and Osbourn, D. (1960). Compensatory growth after undernutrition in mammals and birds. *Biological Reviews* **35**, 324-361.

Yazdi, M., Lundeheim, N., Rydhmer, L., Ringmar-Cederberg, E. and Johansson, K. (2000). Survival of Swedish Landrace and Yorkshire sows in relation to osteochondrosis: a genetic study. *Animal Science* **71**, 1-9.

Ytrehus, B., Carlson, C. S. and Ekman, S. (2007). Etiology and pathogenesis of osteochondrosis. *Veterinary Pathology* **44**, 429-48.

Ytrehus, B., Carlson, C. S., Lundeheim, N., Mathisen, L., Reinholt, F. P., Teige, J. and Ekman, S. (2004). Vascularisation and osteochondrosis of the epiphyseal growth cartilage of the distal femur in pigs--development with age, growth rate, weight and joint shape. *Bone* **34**, 454-65.

Zaffino, J. (2012). An evaluation of hock, knee, and neck injuries on dairy cattle in Canada. In *PhD Dissertation*, p. 111. The University of Guelph.

Zhou, Q., Sun, Q., Wang, G., Zhou, B., Lu, M., Marchant-Forde, J., Yang, X. and Zhao, R. (2014). Group housing during gestation affects the behaviour of sows and the physiological indices of offspring at weaning. *Animal*, 1162-1169.

Zoric, M. (2008). Lameness in piglets. p. 45. Acta Universitatis agriculturae Sueciae, Uppsala.

Zoric, M., Nilsson, E., Lundeheim, N. and Wallgren, P. (2009). Incidence of lameness and abrasions in piglets in identical farrowing pens with four different types of floor. *Acta Veterinaria Scandinavica* **51**, 23-31.

Zoric, M., Nilsson, E., Mattsson, S., Lundeheim, N. and Wallgren, P. (2008). Abrasions and lameness in piglets born in different farrowing systems with different types of floor. *Acta Veterinaria Scandinavica* **50**, 37-45.

Zoric, M., Sjölund, M., Persson, M., Nilsson, E., Lundeheim, N. and Wallgren, P. (2004). Lameness in piglets. Abrasions in nursing piglets and transfer of protection towards infections with Streptococci from sow to offspring. *Journal of Veterinary Medicine, Series B* **51**, 278-284.

Appendices

Appendix 1- Sample recording sheets for cross-sectional survey of limb health

Appendix 1.1 Sow Recording sheet

Sow observation sheet

Recorder (s)			Date		Farm name		
Building		Pen		Pig group		FCS	

---------- SOW 1 ----------

Sow ID			Parity		
Avg. no. piglets/litter			Avg. no piglets born alive/litter		
Lameness (0-5)		MOB (0-2)		BCS (0-5)	

Body Lesions (1-6) **Limb Lesions (0-3)** **Claw Lesions (√/X)**

				Front		Hind			Front		Hind	
	L	R		L	R	L	R		L	R	L	R
Ear			Scratch					Broken dew				
Shoulder			Wound					Broken main				
Middle			Alopecia					Amputation dew				
Hind Q			Alopecia Flank					Amputation main				
Anogenital			Abscess					Overgrown dew				
Tail			Swelling					Overgrown main				
Vulva			Callus									
			Bursitis									
			Capped hock									

---------- SOW 2 ----------

Sow ID			Parity		
Avg. no. piglets/litter			Avg. no piglets born alive/litter		
Lameness (0-5)		MOB (0-2)		BCS (0-5)	

Body Lesions (1-6) **Limb Lesions (0-3)** **Claw Lesions (√/X)**

				Front		Hind			Front		Hind	
	L	R		L	R	L	R		L	R	L	R
Ear			Scratch					Broken dew				
Shoulder			Wound					Broken main				
Middle			Alopecia					Amputation dew				
Hind Q			Alopecia Flank					Amputation main				
Anogenital			Abscess					Overgrown dew				
Tail			Swelling					Overgrown main				
Vulva			Callus									
			Bursitis									
			Capped hock									

Appendix 1.2 Piglet recording sheet

Preweaner observation sheet

Recorder (s)		Date		Farm name	
Pig group					

Group _____wks Piglet No. ___/___ Sow ID _________

Limb lesion's					Claw lesion's					
		Skin abrasion	Hairless area	Swollen joint			Sole bruising	Sole erosion	Coronary Band	Swelling
Front	L				Front	L				
	R					R				
Hind	L				Hind	L				
	R					R				

Group _____wks Piglet No. ___/___ Sow ID _________

Limb lesion's					Claw lesion's					
		Skin abrasion	Hairless area	Swollen joint			Sole bruising	Sole erosion	Coronary Band	Swelling
Front	L				Front	L				
	R					R				
Hind	L				Hind	L				
	R					R				

Group _____wks Piglet No. ___/___ Sow ID _________

Limb lesion's					Claw lesion's					
		Skin abrasion	Hairless area	Swollen joint			Sole bruising	Sole erosion	Coronary Band	Swelling
Front	L				Front	L				
	R					R				
Hind	L				Hind	L				
	R					R				

Group _____wks Piglet No. ___/___ Sow ID _________

Limb lesion's					Claw lesion's					
		Skin abrasion	Hairless area	Swollen joint			Sole bruising	Sole erosion	Coronary Band	Swelling
Front	L				Front	L				
	R					R				
Hind	L				Hind	L				
	R					R				

Group _____wks Piglet No. ___/___ Sow ID _________

Limb lesion's					Claw lesion's					
		Skin abrasion	Hairless area	Swollen joint			Sole bruising	Sole erosion	Coronary Band	Swelling
Front	L				Front	L				
	R					R				
Hind	L				Hind	L				
	R					R				

Appendix 1.3 Weaner recording sheet

Weaner observation sheet

Recorder (s)			Date		Farm name		
Building		Pen		No. Pigs/ group		Stage	

---------- **WEANER 1** ----------

ID			
MOB (0-2)		FCS (0-4)	

Limb Lesions (0-3)

	Front		Hind	
	L	R	L	R
Scratch				
Wound				
Alopecia				
Alopecia Flank				
Abscess				
Swelling				
Callus				
Bursitis				
Capped hock				

Claw Lesions (√/X)

	Front		Hind	
	L	R	L	R
Broken dew				
Broken main				
Amputation dew				
Amputation main				
Overgrown dew				
Overgrown main				

---------- **WEANER 2** ----------

ID			
MOB (0-2)		FCS (0-4)	

Limb Lesions (0-3)

	Front		Hind	
	L	R	L	R
Scratch				
Wound				
Alopecia				
Alopecia Flank				
Abscess				
Swelling				
Callus				
Bursitis				
Capped hock				

Claw Lesions (√/X)

	Front		Hind	
	L	R	L	R
Broken dew				
Broken main				
Amputation dew				
Amputation main				
Overgrown dew				
Overgrown main				

Appendix 1.4 Finisher recording sheet

Finisher observation sheet

Recorder (s)			Date		Farm name		
Building		Pen		No. pigs/group		Stage	

---------- **FINISHER 1** ----------

ID		Lameness (0-5)	
MOB (0-2)		FCS	

Limb Lesions (0-3) **Claw Lesions (√/X)**

	Front		Hind			Front		Hind	
	L	R	L	R		L	R	L	R
Scratch					Broken dew				
Wound					Broken main				
Alopecia					Amputation dew				
Alopecia Flank					Amputation main				
Abscess					Overgrown dew				
Swelling					Overgrown main				
Callus									
Bursitis									
Capped hock									

---------- **FINISHER 2** ----------

ID		Lameness (0-5)	
MOB (0-2)		FCS	

Limb Lesions (0-3) **Claw Lesions (√/X)**

	Front		Hind			Front		Hind	
	L	R	L	R		L	R	L	R
Scratch					Broken dew				
Wound					Broken main				
Alopecia					Amputation dew				
Alopecia Flank					Amputation main				
Abscess					Overgrown dew				
Swelling					Overgrown main				
Callus									
Bursitis									
Capped hock									

Appendix 1.5 Pen environment recording sheet

Pen observations

General data

Recorder (s)		Date		Farm name	
No/Pen			Pig group		

Wall composition

Material	Wall 1	Wall 2	Wall 3	Wall 4	*Wall structure*	Wall 1	Wall 2	Wall 3	Wall 4
Concrete					Fully solid				
Plastic					Part solid (+50%)				
Metal					Part solid (<50%)				
Wood									
Other									

Flooring

Type				**Slatted floor dimensions**		Location:		Location:	
Solid		Concrete	Plastic	Total slatted area (m)	Length				
					Width				
		Metal	Other	Width Inter void area (cm)					
Fully Slatted		Concrete slats	Plastic coated slats	Void width (cm)					
		Metal slats	Other	Void length (cm)					
Partially slatted	Solid area	Concrete	Slat edge	Void shape (cm)		Rect.	Other	Rect.	Other
		Metal	Other	Slat profile (cm)		Flat	Curve	Flat	Curve
	Slatted area	Concrete slats	Plastic coated slats	Slat edge profile (cm)		Angular	Curve	Angular	Curve
		Metal slats	other	Slat surface (cm)		Smooth	Text.	Smooth	Text.

Profile

Floor profile		Ground level NOT uniform throughout pen	Ground level uniform throughout pen
If NOT uniform	Slope(s) present	Y	N
	Step(s) Present	Y	N

Feeders & Drinkers

Feeder type	Number of feeders (no. of spaces)	Functional	Automatic/ manual
Hopper		Y / N	A / M
Wet/dry single		Y / N	A / M
Wet/dry double		Y / N	A / M
Wet long trough		Y / N	A / M
Wet feed probe feeder		Y / N	A / M
ESF		Y / N	A / M
Other _____		Y / N	A / M
Drinker type	Number of drinkers (no. of spaces)	Functional	Automatic/ manual
Nipple		Y / N	A / M
Bite		Y / N	A / M
Bowl (palp)		Y / N	A / M
Bowl (driko)		Y / N	A / M
Other _____		Y / N	A / M
		Y / N	A / M
		Y / N	A / M

Other

Environmental Enrichment device(s) present?	Y / N	If yes:	Type : Number:
Are there any other items/ equipment of note present (e.g. heat pads)			

Pen sketch (include location of; slatted.solid flooring, steps, slopes, position of; walls, gates, feeders, drinkers etc)

NOTES _____

Appendix 1.6 On farm questionnaire

Lameness In-Farm Questionnaire

I would like to thank you for agreeing to take part in this survey, your inclusion in this study is important to establish the level of lameness across Irish pig farms and potential risk factors. The questions I will be asking you refer to the past 12 months on this unit unless otherwise stated in the question

Date:	Farm Name:	Farm Contact No.:	Farm E-mail contact:
Farm Address:		People present:	
Manager Name:		Teagasc advisor (if any):	

General information and performance records

1. What type of unit is this?	Integrated	Breeder	Finisher
2. What is the current number of breeding sows?			
3. What was the average number of pigs born alive per sow, in the last year?			
4. What was the average number of stillborn pigs per sow, in the last year?			
5. What was the number of pigs finished per sow in the last year?			
6. What was the sow culling rate in 2010?			
7. What was the average parity at removal?	Actual from herd records ___	Estimate _____	
8. What was the average parity in the herd?	1, 2, 3, 4, 5, 6, 7, 8+		
9. What were the top 3 reasons (in order) for culling sows in the last 12 months? 1 _____ 2_____ 3_____			

10. What housing system was used for gilts in the last 12 months?	A) Individual (stall) housing (Q12) C) Both (Q11)	B) Loose/group housing (Q11)
11. If a loose housing system - what system was it:	ESF	Welfare/free access stalls
	Dump feeders	Finisher style/long trough pens
	Trickle feeders	other
12. What housing system was used for sows in the last 12 months?	A) Individual (stall) housing (Q16) C) Both(Q13)	B) Loose/group housing (Q13)
13. If a loose housing system - what system was it:	ESF	Welfare/free access stalls
	Dump feeders	Finisher style/long trough pens
	Trickle feeders	other
14. If loose housing -Was any other loose housing system other than those mentioned used on the farm that is now no longer used?	Yes (Q15)	No
15. If yes - what system and why was it stopped?		
16. If individual housing, - when is the expected date of transition to group loosing housing?	Month _____	Year _____
17. If individual housing, when, - what system are you considering	ESF	Welfare/free access stalls
	Dump feeders	Finisher style/long trough pens
	Trickle feeders	other

General unit breeding & genetics

I'm now going to ask you a few general questions on breeding and genetics in the unit over the last 12months.

18. What percentage of the herd is purebred?		
19. What percentage of the herd are F1's?		
20. What percentage of the herd criss-cross?		
21. Did you purchase AI for finisher stock in the last 12 months?	Yes (Q22)	No (Q29)

If yes i.e. purchase semen for AI			
22. What company did you purchase your AI for finisher stock, over the past 12 months?	Hermitage		PIC
	PIA		Other _____
23. What line of AI was used from that company for finisher stock, in the past 12 months?	Hermitage		PIC
	PIA		Other _____
24. Have you used AI from other companies over the past 5 years for finisher stock?	Yes (Q25)		No (Q27)
25. If yes please state the name of the company, the period over which they were used and the lines of AI used?	Hermitage		PIC
	PIA		Other _____
26. If yes please state the period over which they were used and the lines of AI used?	Month _____		Year _____
27. How many doses of AI are used per service?	Single	Double	Triple
28. How many doses of AI are used each week?			
29. How many boars do you have?			
30. Did you purchase boars?	Yes (Q31)		No (Q32)
31. If yes, where did you purchase the boars from?	Hermitage		PIC
	PIA		Other _____

Gilt selection & management up to farrowing

I'm now going to ask you a few general questions about the selection and management of gilts on this unit up to farrowing over the past 12 months.

Gilt selection		
32. Did you feed a gilt developer diet?	Yes (Q33)	No (Q35)
33. If yes, at what age and weight did feeding of the developer diet commence?	Age:_____ (R/E)	Weight: :_____ kg (R/E)
34. If yes, at what age and weight did feeding of the developer diet stop?	Age:_____ (R/E)	Weight: :_____ kg (R/E)
35. Do you breed your own replacement gilts?	Yes (Q36)	No (Q49)
Yes i.e. breed own replacements		
36. What company did you purchase your AI from, for replacement gilts, over the past 12 months?	Hermitage	PIC
	PIA	Other _____
37. What line of AI was used from that company, for replacement gilts, in the past 12 months?		
38. Were the replacement gilts bred from selected sows or finisher stock?	Specific sows (Q39)	General herd (Q43)
Specific sows		
39. Were these selected gilts housed separately from finishers, from weaning onwards?	Yes (Q 41)	No (Q 40)
40. If no, at what age and/or weight were they removed to be managed separately from the finishers?	Age:_____ (R/E)	Weight: :_____ kg (R/E)
41. What factors were considered to be important when selecting grandparent stock and AI for replacement gilts?		

42. What percentage of females from a litter were selected to be used as replacement gilts?		
General herd		
43. At what age and/or weight were they selected managed separately from the finishers?	Age:_____ (R/E)	Weight: :_____ kg (R/E)
44. What factors were considered to be important when selecting replacement gilts from the finishers?		
45. Was the Flooring for selected gilts different to that used for the finishers? If yes how?		
46. Did you manage replacement gilts and finishers differently in the last 12 months (housing, tail docking)?	Yes (Q47)	No (Q48)
47. If yes, how did it differ?		
The next few questions relate to how gilts are managed after selection		
48. On selection how were gilts housed?	Stalled	Grouped
49. What was the group number?		
50. Was there mixing and/or remixing at any stage?	Yes (Q51)	No(Q60)
51. If Yes, how many times?		
No i.e. do not breed own replacements		
52. Where do you purchase your replacement gilts from?		
53. What were the genetics lines for these gilts?		
54. How frequently do you purchase replacement gilts, based on the last 12 months?		

55. How many replacement gilts are purchased at a time, based on the last 12 months?		
The next few questions relate to how gilts are managed after arrival to the unit		
56. On selection how were gilts housed?	Stalled	Grouped
57. What was the group number?		
58. Was there mixing and/or remixing at any stage?	Yes (Q59)	No (Q60)
59. If Yes, how many times?		

Gilt service				
60. At what age and/or weight were gilts served?		Age:_____ (R/E)	Weight: :_____ kg (R/E)	
61. Were gilts moved to the service/boar house or kept in the gilt pens around the time of service?		Service house (Q56)	Gilt pens (Q57)	
62. If service house, how long before service were gilts moved to the service house?				
63. How were gilts housed in the service/boar house before service?		Grouped (Q58)	Stalled	
64. If in groups, describe the pens in relation to:	Group size:			
	Flooring:			
	Feeding arrangement:			
65. If housed in groups at service was the boar allowed into the home pen for stimulation/mating?			Yes	No
66. What type of service was used?		Natural service (Q61)	AI (Q62)	Both (Q62)
67. If gilts were naturally mated in a specialised mating area, describe it.	Size			
	Shape			
	Flooring			
	Other			
68. At what heat were gilts at 1st service (1, 2 or 3)?				
69. How long after service were gilts transferred to the dry sow house?				
70. How many (if any) times were gilts mixed between selection/arrival at the unit and service?				

71. If mixed, when were gilts mixed between selection/arrival at the unit and service?
72. Was there any difference in management practices, to what has been described above, when dealing with gilts which repeated?

Gilt management in dry sow house				
73. What housing system was used for pregnant gilts?		Individual (stall) housing	Loose/group housing	Both
74. If loose housed, are gilts:	Stalled initially and loose for remainder of pregnancy		Loose for entire pregnancy	
75. If initially stalled, how many days for?				
76. How were gilts grouped when they enter the loose housing system?		Service date	Size	
		Parity	Other	
77. What was the number of gilts per group?				
If an ESF loose housing system for gilts was in operation:				
78. Describe how/where were gilts trained				
79. Were gilts introduced to the Main/resident group individually or allowed to form sub-groups?		Individually (Q81)	Sub-groups (Q80)	
80. If sub-groups, what was the size of the sub-groups				

Sow management up to farrowing

I'm now going to ask you a few general questions about the management of sows on this unit up to farrowing over the past 12 months.

Sow service				
81. How many days after weaning were sows served?				
82. How are sows housed in the service/boar house				
83. How were sows housed in the service house during service?	Grouped (Q84)	Stalled (Q87)		
84. If in groups what was the group size?				
85. If in groups was the flooring type:	Solid	Slatted		
86. If housed in groups at service was the boar allowed into the home pen for stimulation/mating?			Yes	No
87. What type of service was used (if different from breeding of own replacement gilts)?	Natural service (Q88)		AI (Q89)	Both (Q88)
88. If sows were naturally mated in a specialised mating area, describe it.	Size			
	Shape			
	Flooring			
	Other			
89. How long after service were sows transferred to the dry sow house?				
90. Was there any different in management practices, to what has been described above, when dealing with sows which have repeated?				

Sow management in dry sow house			
91. What housing system was used for pregnant sows?	Individual (stall) housing (Q98)	Loose/group housing (Q92)	Both (Q92)
92. If loose housing are sows:	Stalled initially and loose for remainder of pregnancy (Q93)	Loose for entire pregnancy (Q94)	
93. If initially stalled, how many days for?			
94. How were sows grouped when they enter the loose housing system?		Service date Size Parity Other _____	
95. What was the number of sows per group?			
If an ESF loose housing system for sows was in operation:			
96. Describe how/where were sows trained			
97. Were sows introduced to the main/resident group individually or allowed to form sub-groups?		Individually	Sub-groups

Gilt and sow management practices in the farrowing house

I'm now going to ask you a few questions about gilt and sow management in the farrowing house over the past 12 months.

98. How many days before farrowing were sows moved to the farrowing house?			
99. Were sows washed prior to transfer to the farrowing house?	Yes	No	
100. Did you induce farrowings?	Yes	No	Sometimes
101. What age and weight were piglets at weaning?	Age:_____(R/E)	Weight:_____ kg (R/E)	
102. Did you foster piglets?	Yes (Q103)	No (Q104)	
103. Why did you foster? (do not read list out)	Sow morbidity	Sow mortality	Even out litter sizes
	Large litter sizes	Issues with savaging or crushing	Other _____

1st & 2nd stage & finisher pens

I'm now going to ask you a few questions about the management of weaners and finishers over the past 12 months.

104. Did you use 1 or 2 stage weaner accommodation?	1 stage	2 stage	
105. Did you mix litters at 1st stage	Yes	No	
106. Were pigs sorted by sex at 1st stage?	Yes	No	
107. Were pigs sorted by size at 1st stage?	Yes	No	
108. At what age/weight did pigs enter the 2nd stage pens?	Age:_____(R/E)	Weight:_____ kg (R/E)	
109. Did you remix between 1st and 2nd stage?	Yes	No	
110. If remixed, were pigs sorted by sex at 2nd stage?	Yes	No	
111. If remixed, were pigs sorted by size at 2nd stage?	Yes	No	
112. How many weeks after weaning have you found there to be a higher mortality rate?			
113. At what age/weight did pigs enter the finisher pens?	Age _____(R/E)	Weight_____(R/E)	
114. Were they remixed at this stage?			
115. Were pigs sorted by sex?	Yes	No	
116. Were pigs sorted by size?	Yes	No	
117. Has an all in - all out system been in operation over the past 12 months?	Yes	No	
118. What was the average live weight at slaughter?			
119. What was the average age at slaughter?			

Diet

120. What type of feed was used in the unit?		Maiden gilt	Pregnant gilt	Dry Sow	Lactating Sow	Piglet (starter)	Piglet (link)	Weaner	Finisher (1)	Finisher (2)
	Wet									
	Dry									
121. Did you limit, ad lib or scale feed the following groups?		Maiden gilt	Pregnant gilt	Dry Sow	Lactating Sow	Piglet (starter)	Piglet (link)	Weaner	Finisher (1)	Finisher (2)
	Limit									
	Ad lib									
	Scale									

122. What feed was used throughout the unit?	Purchased (Q123)	Home compound (Q124)	Both (Q124)
123. If purchased, what company supplied the feed?			

124.		Gilt developer	Dry Sow	Lactating Sow	Piglet (starter)	Piglet (link)	Weaner	Finisher (1)	Finisher (2)
Diet Composition:	Energy (MJDE)								
	Lysine								
	Phosphorous (total)								
	Calcium								
	Fibre								
	Zinc oxide								
	Phytase	Y/N	Y/N	Y/N			Y/N	Y/N	Y/N

125. Was any medication administered via the feed on this unit?		Yes (Q126)	No (Q127)
126. If yes:	What medication:	Water or food?	
	What was it treating:		
	What group(s):		

Basic hygiene practices

I'm now going to ask you a few general questions about the basic hygiene practices practised the past 12 months.

127. How often were the pens washed/power cleaned? (in units of months or weeks)	Dry sow house		Farrowing rooms	
	Maiden gilt pens		Service house	
	Weaner pens		1st stage house	
	Finisher pens		2nd stage house:	
	Replacement gilt holding area			

128. What steps did your washing routine involve: (please indicate in order the steps routinely undertaken)	Pre-soak ___	Power wash ___
	Dry 1 ___	Disinfect ___
	Dry 2 ___	Other _____

Farm health

I'm now going to ask you a few general questions about farm health on the unit over the past 12 months.

129. What did you vaccinate against on this unit? (If yes please indicate group in space provided)	Atrophic Rhinitis	Y	N	
	Clostridium	Y	N	
	E. coli	Y	N	
	Erysipelas	Y	N	
	Mycoplasma pneumonia	Y	N	
	Parvovirus	Y	N	
	PMWS (wasting)	Y	N	
	PRRS (blue ear)	Y	N	
	Other _____	Y	N	

130. If unsure of what was vaccinated against please state the vaccine brand name(s) (list available):

131. Were any of he following diseases present on this unit in the last 12 months?	Atrophic Rhinitis	Y	N	Aujeszky's disease	Y	N
	Clostridium	Y	N	E. coli	Y	N
	Erysipelas	Y	N	Greasy pig disease	Y	N
	Haemophilus pneumonia	Y	N	Ileitis	Y	N

	Meningitis Mycoplasma	Y	N	Pneumonia	Y	N
	Parvovirus	Y	N	PMWS (wasting)	Y	N
	PRRS (blue ear)	Y	N	Salmonella	Y	N
	Swine dysentery	Y	N	Swine influenza	Y	N
	Other _____					

132. Were all new piglets administered antibiotic at birth?	Yes	No

133. What antibiotics are currently in use on farm & reason for use?	Antibiotic	Reason

134. Was any medication administered through the water supply?	Yes (Q135)	No (Q136)

135. If yes:	What medication?	
	What was it treating?	
	What group(s)?	

136. What percentage of sows, weaners & finishers were treated for lameness in the last 12 months?	Sows__	Weaners___	Finishers__

137. In your opinion what was the main cause of lameness in:	Sows	
	Weaners	
	Finishers	
138. What steps were taken if a pig was considered to be lame? (note if there is there a difference in treatment type between groups)		
139. Did you take any action to prevent limb disorders? (e.g. dietary supplements, ventilation alterations)	Yes (Q140)	No (Q141)
140. If yes, please describe		
141. Are dedicated pens for isolation present in your unit?	Yes (Q142)	No (Q145)
142. If yes, how do they differ from regular housing facilities?		
143. Describe how these sick/isolation facilities were used in relation to lame pigs?		
144. Did pigs return to the same groups after these isolation areas?	Yes	No

Foot health

I'm now going to ask you a few general questions about foot health on the unit over the past 12 months.

145. Was claw trimming practiced in this unit?	Yes (Q146)	No (END)
146. If so, how frequently was claw trimming carried out?		
147. At what point is it claw trimming carried out (please circle correct answer)	a) As part of a routine b) If long claws are observed but locomotory ability is not affected c) If locomotory ability is affected by long claws d) If locomotion becomes difficult/impossible as a result of claw length	

END OF QUESTIONNAIRE, THANK YOU FOR YOUR TIME!!!

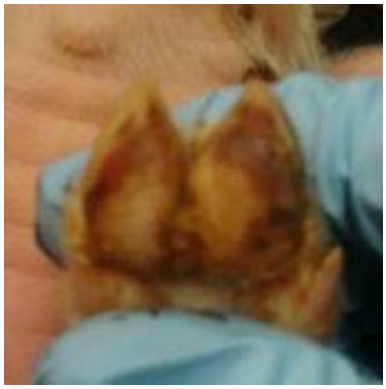
Appendix 2 - Definitions of limb, joint and claw lesions

Lesion	Description
Limb abrasion	Disruption to outer limb epidermis, with an open wound or healing scab (Mouttotou *et al.*, 1999; KilBride *et al.*, 2009).
Sole bruising	Dark red pigmentation of solar corium (Mouttotou and Green, 1999; KilBride *et al.*, 2009).
Sole erosion	Loss of sole tissue causing an irregular depression in solar corium (Mouttotou and Green, 1999; KilBride *et al.*, 2009).
Coronary band damage	Epidermis disruption at coronary band presenting as an open or healing wound.
Callus	Hyperkeratinosis of the epidermis resulting in a thickened area (Cagienard *et al.*, 2005; KilBride *et al.*, 2008).
Alopecia	Hairless patch on the limb, epidermis not disrupted, no scab (Mouttotou *et al.*, 1999; KilBride *et al.*, 2009).
Alopecia flank	Hairless patch on the flank, epidermis not disrupted, no scab (Mouttotou *et al.*, 1999; KilBride *et al.*, 2009).
Scratch	Superficial disruption to the epidermis.
Wound	Open wound or wound with scab.
Swelling	Any swelling to the limb or foot.
Bursitis	A fluid filled sac of the subcutaneous connective tissue on the limb (Mouttotou *et al.,* 1999a; Gillman *et al.*, 2008).
Capped hock	A fluid filled sac of the subcutaneous connective tissue at the hock (Mouttotou *et al.,* 1999a; Gillman *et al.*, 2008).
Overgrown toe	Lateral claw, medial claw or both elongated (Ossent, 2010).
Overgrown dew claw	One or both dew claws elongated (Ossent, 2010).
Broken toe	Partial removal of the medial or lateral claw (Ossent, 2010).
Broken dew claw	Partial removal of the dew claw (Ossent, 2010).
Amputated toe	Complete removal of the medial or lateral claw
Amputated dew claw	Complete removal of the dew claw.
Uneven claw size	Unequal medial and lateral claw.
Heel Overgrowth	Hyperkeratinisation of the heel (Ossent, 2010).
Heel Erosion	Partial removal of heel tissue (Ossent, 2010).
Heel-Sole separation	Separation at the junction of the heel and sole (Ossent, 2010).
White Line separation	Separation at the white line (joining of sole and hoof wall).
Horizontal Wall crack	Horizontal crack present in the medial or lateral claw wall (Ossent, 2010).
Vertical Wall crack	Vertical crack present in the medial or lateral claw wall (Ossent, 2010).
Dew claw crack	Vertical or horizontal crack in the dew claw(Calderón Díaz *et al.*, 2013).
Joint lesion	Irregularity or invagination of the cartilage (Busch and Wachmann, 2011).
Osteochondrosis dissecans	Cartilage has become separated from the underlying bone (Busch and Wachmann, 2011).

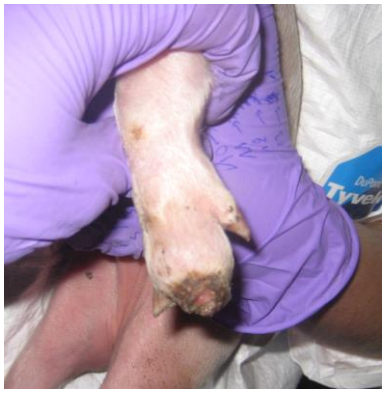
Appendix 3 – Sample photos of lesions

Appendix 3.1 Piglet Limb and foot lesions

Skin Abrasions



Sole bruising



Coronary band lesion and Foot swelling

Appendix 3.2 Limb Lesions

Alopecia flank

Callus and swelling

Abcess

Appendix 3.3 Sample claw lesions

Overgrown toes

Overgrown toe and dew claw and broken toe

Heel overgrowth

Heel/Sole separation

White line lesion

Amputated dew claw

Appendix 4 - Scoring systems

Appendix 4.1 Lameness scoring system (as adapted from Main *et al*.1999)

	0	1	2	3	4	5
Initial response to human	Bright, alert & responsive	Bright, alert & responsive	Bright, alert & responsive	Bright , less responsive	May be dull (only rises if strongly motivated)	Dull & unresponsive
Response after opening gate	Inquisitive, will tentatively leave pen	Inquisitive, will tentatively leave pen	Inquisitive, will tentatively leave pen	Often last to leave pen	Unwilling to leave familiar environment	No response
Behaviour of individual within group	Freely participates in group activity	Freely participates in group activity	May show mild apprehension to boisterous pigs	May show mild apprehension to boisterous pigs	Try's to remain separate from others within groups	Distressed by other pigs, unable to respond
Standing posture	Stands squarely on all four legs	Stands squarely on all four legs	Uneven posture	Uneven posture. Won't bear weight on affected limb	Affected limb elevated off floor	Won't stand unaided
Gait	Even strides: Caudal body sways slightly while walking. Can accelerate & change direction rapidly	Abnormal stride length. Movement no longer fluid: pig appears stiff**.** Can accelerate & change direction	Shortened stride. Lameness detected. Swagger of caudal body while walking. No hindrance in agility	Pig may not place affected limb on the floor while moving	Shortened stride. Min weight bearing on affected limb. Swagger of caudal body while walking.	Does not move

Appendix 4.2 Body lesion scoring system (as adapted from O'Driscoll *et al.* 2013)

Score	Description
0	**No** lesion in the area
1	1 small, superficial lesion
2	+ 1 small superficial lesion **or** 1 red lesion
3	+ 1 red lesion
4	1 deep red lesion
5	+1 deep red lesion **or** 1 big lesion
6	+ 1 big lesion

Score 1-1 small, superficial lesion

Score 2- + 1 small superficial lesion or 1 red lesion

Score 3 - + 1 red lesion

Score 4 - 1 deep red lesion

Score 5 - +1 deep red lesion or 1 big lesion

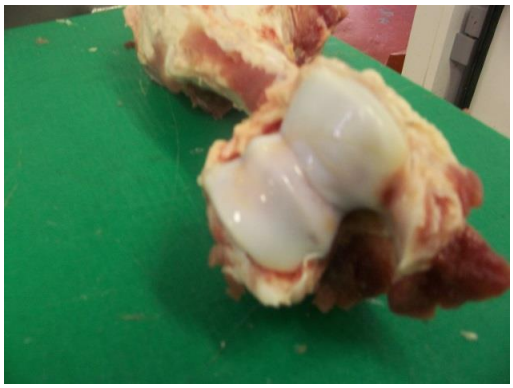
Appendix 4.3 Body condition scoring system (as per DEFRA guidelines for the condition scoring of pigs)

Score	Description
1	The sow is visually thin, with hips and backbone very prominent and no fat cover over hips and backbone
2	The hips and backbone are easily felt without any pressure on the palms
3	It takes firm pressure with the palm to feel the hip bones and backbone
4	It is impossible to feel the bones at all even with pressure on the palm of the hands
5	The sow is carrying so much fat that it is impossible to feel the hip bones and backbone even by pushing down with a single finger

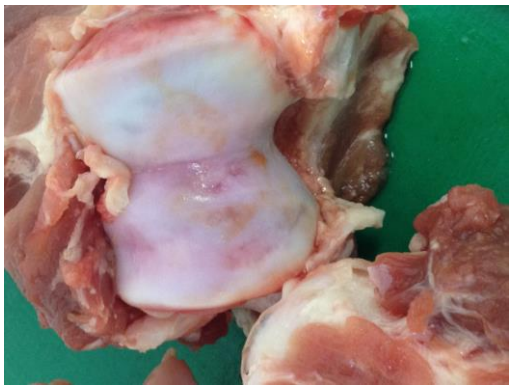
Appendix 4.4 Floor cleanliness scoring system (as adapted from Hacker *et al.* 1994)

Score	Description
0	Clean, dry, perhaps some meal. No excreta on the floor
1	Dry excreta on the floor
2	Excreta wet enough to dirty pigs but with no depth, in disconnected islands (<25%)
3	Excreta wet enough to dirty pigs but with no depth, in disconnected islands (26-50%)
4	Very wet excreta, often to a depth of 5mm, or excreta >50%

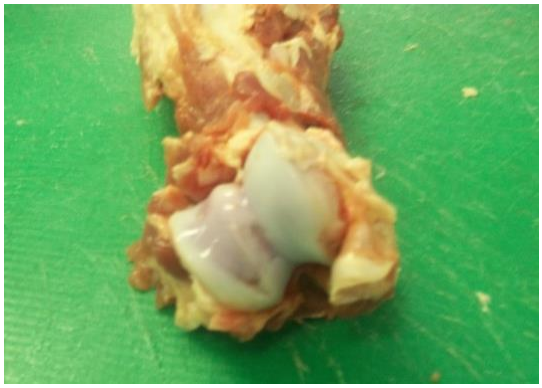
Appendix 4.5 Joint lesion scoring of the Humeral Condyle (as adapted in Jørgensen et al. 1995, Christensen et al. 2010 and Busch and Wachman, 2011)



Score 1- No irregularity or invagination of the cartilage

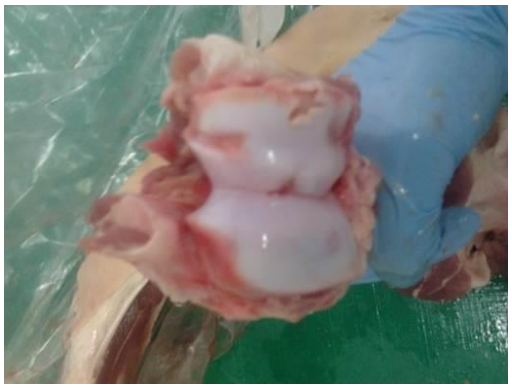
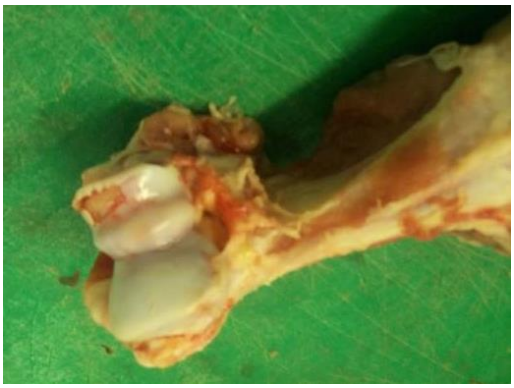


Score 2 – Mild irregularity or invagination of the cartilage

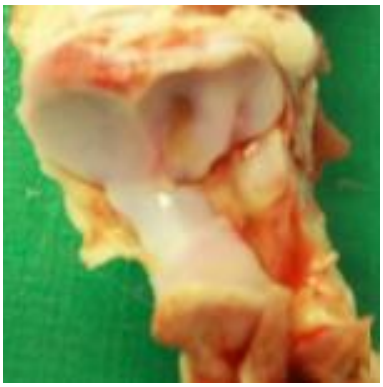
Score 3 – Moderate irregularity or invagination of the cartilage


Score 4 – Severe irregularity or invagination of the cartilage

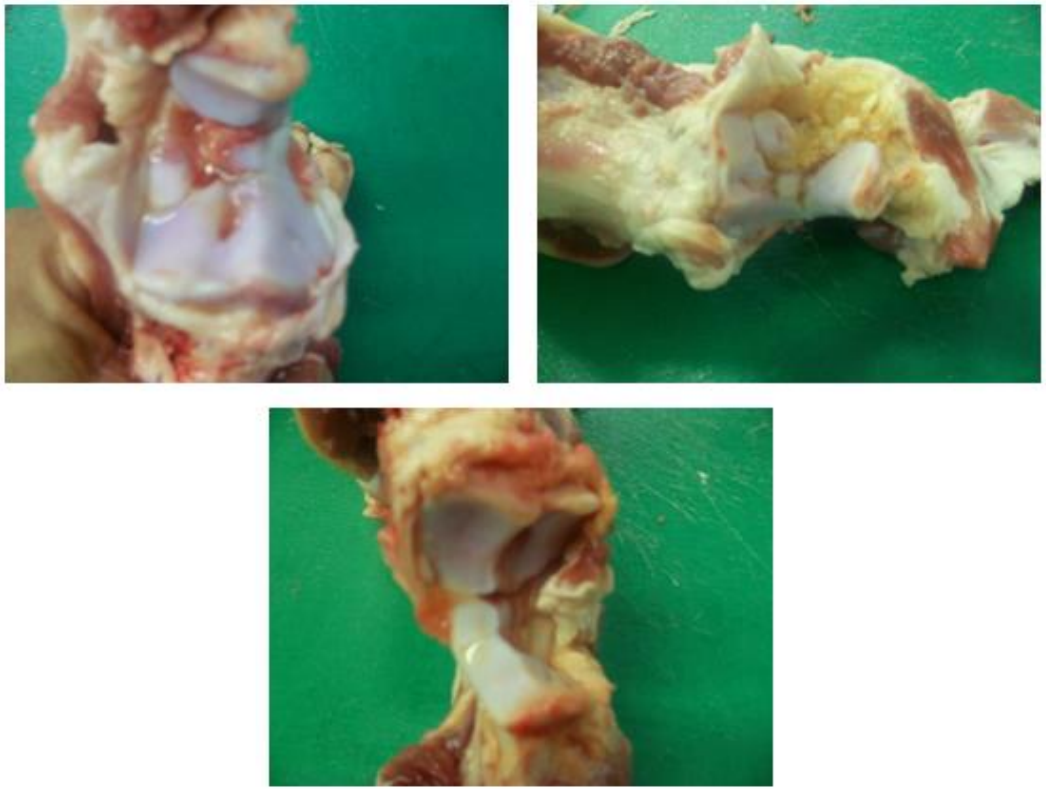



Score 5- Cartilage has become separated from the underlying and bone is exposed

Appendix 4.6 Joint lesion scoring of the anconeal process (as adapted (as in Jørgensen et al. 1995, Christensen et al. 2010 and Busch and Wachman, 2011)



Score 1 - No lesion



Score 2 - Lesion present

Appendix 5 - Recording sheets for gilt developer diet studies (Chapter 5 & 6)

Appendix 5.1 Weekly recording sheet

Recorder (s)		Date		Inspection period	

ID		Lameness	
Pen		MOB	

	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

ID		Lameness	
Pen		MOB	

	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

ID		Lameness	
Pen		MOB	

	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

ID		Lameness	
Pen		MOB	

	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

ID		Lameness	
Pen		MOB	

	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

ID		Lameness	
Pen		MOB	

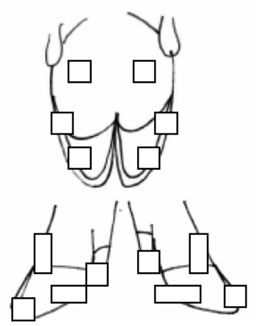
	L	R		Front		Hind	
				L	R	L	R
Ear			Scratch				
Shoulder			Wound				
Middle			Alopecia				
Hind Q			Alopecia Flank				
Anogenital			Abscess				
Tail			Swelling				
Vulva			Callus				
			Bursitis				
			Capped hock				

*Body lesions were not recorded for chapter 5

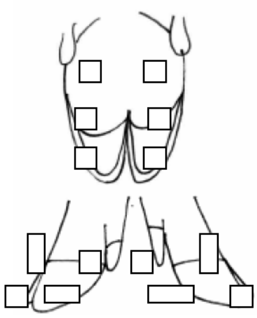
Appendix 5.2 Claw lesion recording sheet

Date__________

Pig ___ Pen___

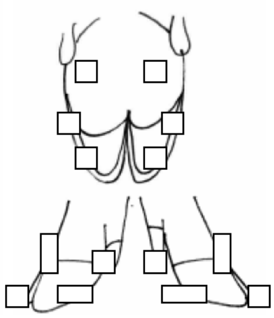


DCC____DCC____

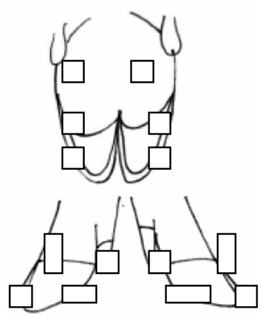


DCC____DCC____

Pig ___ Pen___

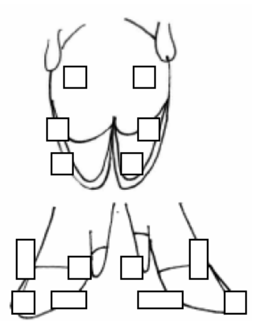


DCC____DCC____

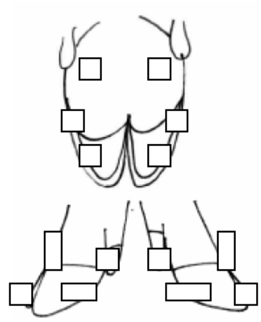


DCC____DCC____

Pig ___ Pen___

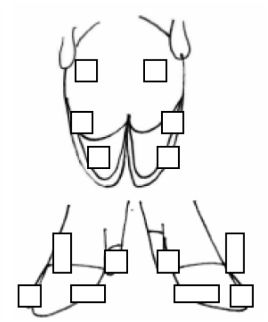


DCC____DCC____

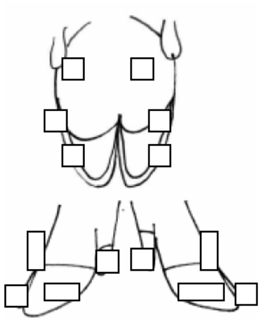


DCC____DCC____

Pig ___ Pen___

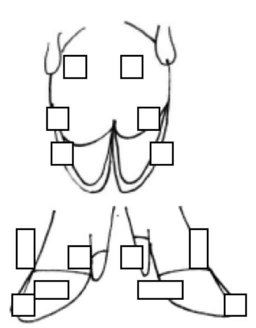


DCC____DCC____

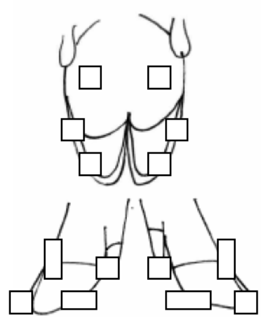


DCC____DCC____

Pig ___ Pen___

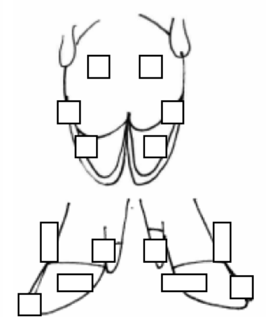


DCC____DCC____

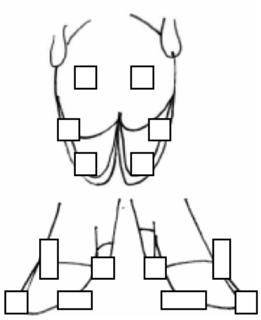


DCC____DCC____

Pig ___ Pen___



DCC____DCC____



DCC____DCC____

Appendix 5.3 Joint lesion scoring sheet

OCD Recording sheet EXP 710 & &711

Date _____ Trial No._____

Slap No	Trial ID	OCD Score HC	OCD Score AP	Description

Appendix 5.4 Areal bone mineral density recording sheet

BMD Recording sheet EXP 710 & &711

Date _____ Trial No._____

Slap No	Trial ID	Area (cm^2)	BMC (g)	BMD (g/cm^2)

Appendix 5.5 Scan sampling recording sheet

Scan sampling sheet EXP 711

			Pen___				Pen___				Pen___				Pen___				Pen___				Pen___			
		Focal ID	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Date	Time	Gilt ID																								

Appendix 6 -

Number and % of gilts on three dietary treatments with locomotory ability and joint surface lesion scores for three dietary regimes for replacement gilts from 65kg

Variables		DEV		FIN		GES	
n		24		24		24	
	Score	n	%	n	%	n	%
Locomotory ability							
d 0-29							
	0	2	8.3	1	4.2	1	4.2
	1	22	91.7	19	79.2	20	83.3
	2	0	0.0	2	8.3	3	12.5
	≥3	0	0.0	2	8.3	0	0.0
d 30-83							
	0	0	0.0	0	0.0	0	0.0
	1	15	62.5	8	33.3	11	45.8
	2	9	37.5	15	62.5	12	50.0
	≥3	0	0.0	1	4.2	1	4.2
d 0-83							
	0	0	0.0	0	0.0	0	0.0
	1	15	62.5	8	33.3	10	41.7
	2	9	37.5	13	54.2	13	54.2
	≥3	0	0.0	3	12.5	1	4.2
Joint surface lesions							
HC							
	1	0	0.0	1	4.2	0	0.0
	2	12	50.0	12	50.0	12	50.0
	3	8	33.3	7	29.2	6	25.0
	4	4	16.7	2	8.3	2	8.3
	5	0	0.0	2	8.3	4	16.7
AP							
	1	15	62.5	11	45.8	12	50.0
	2	9	37.5	13	54.2	12	50.0

Appendix 7 -

Number and % of gilts on three dietary treatments with uneven toes and the claw lesions; heel overgrowth and heel-sole separation for three dietary regimes for replacement gilts from 65kg

Variables		DEV		FIN		GES	
n		24		24		24	
		n	%	n	%	n	%
Uneven toe							
	d0	21	87.5	23	95.8	23	95.8
	d40	14	58.3	13	54.2	16	66.7
	d83	10	41.7	13	54.2	18	75.0
	d0-	23	95.8	24	100.0	23	95.8
Heel overgrowth							
	d0	5	20.8	3	12.5	4	16.7
	d40	17	70.8	18	75.0	18	75.0
	d83	19	79.2	21	87.5	22	91.7
	d0-	23	95.8	21	87.5	22	91.7
Heel sole separation							
	d0	4	16.7	4	16.7	5	20.8
	d40	2	8.3	4	16.7	4	16.7
	d83	2	8.3	7	29.2	5	20.8
	d0-	8	33.3	9	37.5	10	41.7