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LAMENESS IN PIGS: A REVIEW

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Contents

1. Abstract

Lameness is a frequently encountered clinical manifestation of different locomotory disorders affecting all age groups of modern pig herds. It results in decreased animal welfare, health and economic losses to pork producers (Anil et al. 2005, Heinonen et al. 2013). Lameness is a multifactorial condition with infectious, nutritional, housing, managemental and/or genetic components. It is of great importance to detect the lame pigs, diagnose the cause of lameness, treat them efficiently and move them to sick pen whenever needed. In addition, culling should be considered without delay to prevent unnecessary suffering of the animals (Heinonen et al. 2006). The final aim is to prevent lameness efficiently. This chapter elaborates current knowledge about lameness in pigs, with emphasis on the etiology, diagnosis, treatment and prevention.

2. Anatomy and physiology of pigs, short introduction

Lameness can be defined as a deviation from normal gait or posture while a normal degree of consciousness is displayed (Wells 1984, Straw et al. 1999, Beusker 2007). The severity vary greatly and lameness can manifest as an asymmetrical limb movement, an altered or shortened stride, a reduced ability or inability to bear weight and even total recumbency (Straw et al. 1999, Maas 2009). Lameness may develop when the functional integration of the nervous system, bones, muscles, cartilages, tendons, joints and ligaments is disturbed. Disorders can be located in the limbs as well as in the trunk of the animal, and may include painful lesions as well as mechanical defects (Wells 1984, Smith 1988, Beusker 2007). A proper understanding of lameness requires knowledge of the gait of healthy pigs and insight into the anatomy and physiology of the locomotory system.

The walking gait of a pig is characterized by a symmetrical four-beat gait with an alternating two- and three-limb support phase and even strides. Pigs walk mainly in a diagonal sequence in which the hind foot touches down slightly later than the contralateral fore foot. Fore limbs have a longer stance time

and a lower swing/stance time ratio than hind limbs. Yet, stride elevation is higher in hind limbs (Thorup et al. 2007, von Wachenfelt et al. 2008). While standing, pigs show a merely straight back and stand squarely on all four legs. The body weight of a standing pig is not evenly distributed between and within feet: feet at the fore limbs carry the highest load, i.e. 57% of the total body weight (Pluym et al. 2013a). Within feet, lateral claws carry 78% of the body weight mostly by the heel bulb followed by the junction between the heel bulb and the abaxial wall. Within the medial claw, the tip of the toe is the most loaded area (Webb 1984).

The pig's body contains about 216 bones (Figure 2.1.). Bones are not static, but highly metabolically active during skeletal growth as well as during bone modelling and remodelling, the latter occurring throughout life.

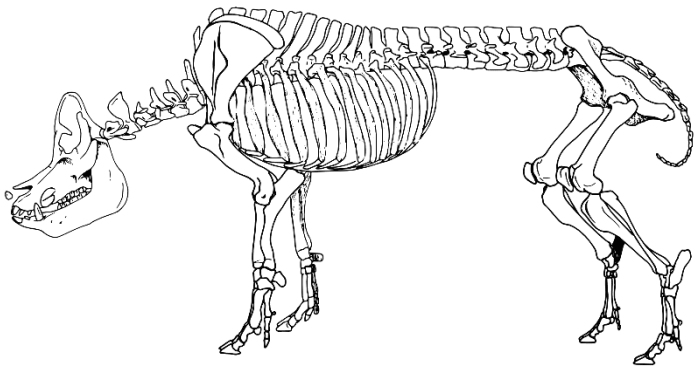

Figure 2.1: Lateral view on the skeleton of a pig. (Source: Prof. dr. Pieter Cornillie, Morphology lab, Ghent University)

The bones of skeletally immature pigs contain some anatomical components in addition to the ones found in bones of skeletally mature pigs, mainly: the specialized form of cartilage known as growth cartilage, and its blood supply (Carlson et al. 1991, Ytrehus et al. 2004b, 2007). The bones of skeletally immature pigs are also engaged in an additional physiological process of growth by endochondral

ossification, defined as growth of a cartilage scaffold that is progressively replaced by bone. The fact that skeletally immature pigs differ slightly in anatomy and physiology from mature pigs means that they are susceptible to different diseases and may respond differently to disease agents. This is reflected by the variation in etiology and pathogenesis of locomotory diseases as described in the following chapters.

Specialized growth cartilage is present at two sites of either end of long bones: the sub-articular, epiphyseal growth cartilage and the metaphyseal growth plate, or physis (Carlson et al. 1991). The physis is predominantly responsible for growth in terms of increase in bone length, whereas the epiphyseal growth cartilage is responsible for shaping the articulating bone ends. Both physeal (Hill et al. 1985) and epiphyseal growth cartilage (Carlson et al. 1991, Ytrehus et al. 2004c, 2007) contain a temporary blood supply that runs within so-called cartilage canals. In epiphyseal growth cartilage, the blood supply is organized as anatomical end arteries (Ytrehus et al. 2004c, 2007), whereas in the physis, organization varies with age (Hill et al. 1985, Wormstrand et al. 2021). In both cases, the cartilage canals are regularly spaced and separated by extra-cellular matrix, rendering chondrocytes vulnerable in case of failure because there is limited potential for collateral supply (Carlson et al. 1991). As the pig approaches skeletal maturity, the blood supply regresses through physiological chondrification and incorporation into the advancing ossification front (Ytrehus et al. 2004c). Studies in horses have shown that regression follows an age-dependent pattern for different joints and regions (Carlson et al. 1991, Olstad et al. 2008b). Proliferation of chondrocytes slows down to such an extent that all growth cartilage eventually becomes replaced by bone, at which point the growth plates close and skeletal maturity is reached. Closure time depends on the plates and bones considered. One source (Zeder et al. 2015) states that all the major growth plates are closed by 60 months/5 years.

The process of bone modelling occurs in response to physiological influences and mechanical forces. The structure of bones is gradually adjusted to the forces they encounter through bone formation and resorption. During bone modelling, formation and resorption are not tightly coupled. The homeostatic

process of bone remodeling throughout life aims to renew bone in order to maintain bone quality and mineral homeostatis. Bone remodeling is a cyclical process with four components: activation of (pre)osteoclasts, resorption of bone, transition from bone resorption to formation and formation of bone mediated by osteoblasts (Clarke 2008).

Joints create the connection between bones, transmit the mechanical load between the bones they connect and allow movement. They are the most loaded parts of the locomotory system (Done et al. 2012). A joint consists of articular cartilage that covers the bones it connects. Joint cartilage includes a layer of articular cartilage, beneath which, in growing animals, is the subarticular (epiphyseal) growth cartilage. The joint capsule is a membrane that surrounds the entire circumference of the end of the bones involved in the joint. The synovial membrane that secretes the synovial fluid covers the inner surface of the joint capsule. Synovial fluid acts as a lubricant and normally appears as clear to straw-coloured viscous fluid. Joints have been suggested to be highly susceptible to infection after local trauma or in case of septicaemia (Done et al. 2012).

The pig is an even-toed ungulate with four digits on every limb, two principal ones (III, medial and IV, lateral) and two accessory ones (dewclaws, II and V). In contrast to cattle, the accessory digits have a full complement of phalanges and they are attached to the distal row of the carpal and tarsal bones (Sack 1982). Dewclaws have been regarded as non-wearing accessory digits, which have no load-carrying capacity, or only some on soft underground (Sack 1982). Papadopoulos et al. (2021), however suggested a positive association between the dewclaws and the principal claws: dewclaw length is approximately 2 cm shorter than the dorsal length of the main claws. They defined the dorsal claw length as the linear distance along the dorsal wall from just below the coronary band to the weight-bearing border (Figure 2.2).

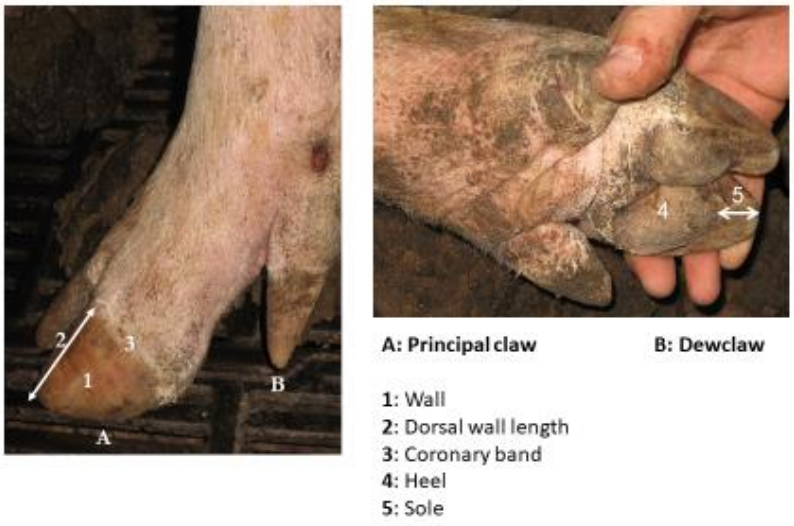

Figure 2.2: Lateral and palmar view on the principal claws (A) and dewclaws (B) of the pig with different distinguishable regions. (Photo: L. Pluym)

The pig's claw (Figure 2.2.) consists of a horn capsule covering the skeleton (distal phalanx, the distal part of the middle phalanx and the distal sesamoid bone) and soft tissue at the end of the limb (Sack 1982, Mülling 2000). Different regions can be distinguished: a hard wall, hard sole, soft heel bulb (with cushioning function) and a non-pigmented junction between the wall and sole, the so-called white line (Ossent 2010). The hoof capsule is in fact skin that is extensively modified. The epidermis is a strongly cornified epithelium and the dermis underneath is modified to dermal papillae or lamellae (only at the level of the wall) (Mülling 2000). Claw horn production is the result of proliferation, keratinization and cornification (programmed cell death) of the epidermal cells at the level of the claw (i.e. keratinocytes) (Tomlinson et al. 2004). In areas with dermal lamellae, the epidermis forms horn lamellae while tubular horn is formed at the level of the dermal papillae. Sufficient supply of nutrients, oxygen and hormones to the metabolic active keratinocytes within the avascular epidermis essentially depends on the dense vascular system within the dermis and the process of diffusion within the epidermis (Mülling 2000,

Tomlinson et al. 2004). Claw horn strength is directly related to the dimensions of the tubules in the tubular horn. The wide dimensions of the tubules in the white line makes this site more susceptible for lesions and bacterial invasion (Tomlinson et al. 2004).

3. Clinical examination and diagnosis of lame pigs

Lame animals need to be examined for the need of treatments, for herd health evaluations or for research purposes (Figure 3.1). They can be found by observing their behaviour or by scoring their lameness visually or with methods that are more objective. After detection of the lame animal, it should be examined clinically to reach the diagnosis and enable the right treatment if needed. Lameness diagnostics may include also analysis of diagnostic samples.

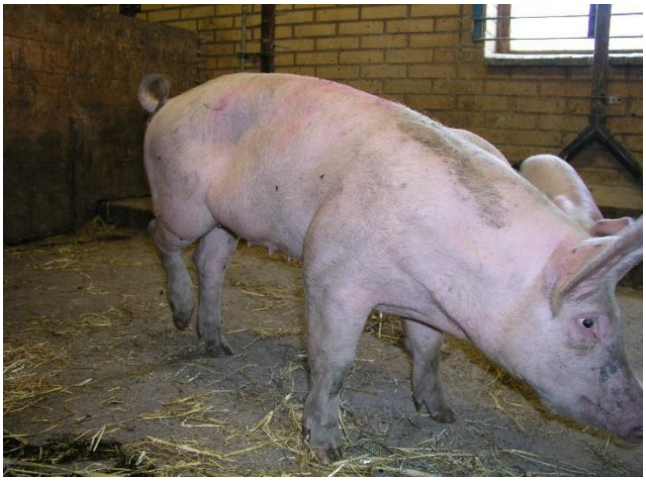

Figure 3.1. Lameness in a pig (Photo: Carl-Johan Ehlorsson)

3.1. Pig behaviour

Foot and skin lesions can contribute to lameness either by causing pain from the injury itself or by acting as an entrance for infections that may spread to joints through bacteraemia and thereby induce arthritis and pain (Gardner and Hird 1994, Zoric et al. 2004, 2016). The pig has sufficient cognitive and emotional capacity to experience negative affective states such as pain, and not just show reflexive avoidance behaviour in response to tissue damage (Herskin and Di Giminiani 2018). Lame pigs behave

differently from their healthy pen mates due to pain, general discomfort or sickness behavior (Heinonen et al. 2013). Lame sows have shorter latency time to lie down (Calderon-Diaz et al. 2014) and an uncontrolled lying-down behavior (Bonde et al. 2004) and they use longer time to stand up and are more hesitant during standing up (Gregoire et al. 2013) compared to non-lame sows. Jørgensen et al. (2000) reported that sows with claw lesions showed increased stepping of hind legs. Cargill and Buddle (2001) suggested that lame animals can be best detected by first chasing them up standing and then examining the first 20 pigs, which sit down. In a study by Ala-Kurikka et al. (2017), sows with non-resolved lameness stood and moved less, lied down more and were in contact with the wall more than their healthy controls. All of these behavioral changes are used in finding possible lame animals for individual clinical examination. Pigs have a short neck and stiff movements and that is why their lameness evaluation is challenging (Main et al. 2000). Mild lameness is often undetected, even though it would be very important to detect and treat the lame sows early (Alawneh et al. 2012). Possibly, in the future, remote monitoring technology applications will be developed to solve this problem.

3.2. Lameness scoring in pigs

The degree of lameness is usually evaluated visually by observing the gait of the pigs. Several different visual scoring systems have been used (reviewed by Nalon et al. 2013), but they have not always been found reliable because of high subjectivity and variable skills of the scorer (Main et al. 2000, D'Eath 2012). The scores usually include four to six levels (Main et al. 2000, Gevering et al. 2006, Karlen et al. 2007, Kilbride et al. 2009, Mustonen et al. 2011, D'Eath 2012, Gregoire et al. 2013), but even ten different (Dewey et al. 1993) or only three levels have been used (Welfare Quality 2009). Visual scoring requires substantial training (Petersen et al. 2004, Abell et al. 2014). However, it is often the only available, useful, quick and affordable way to quantify lameness (Nalon et al. 2013). If pigs are moved to walk on solid floor for lameness scoring, as they should, the ease and cheapness is lost.

More objective methods have been developed to assess the pig lameness (Table 3.1.), but they have been used so far mostly in research. Their use typically takes a lot of time due to complex installations,

motion captures and/or initial data processing (Nalon et al. 2013, Stavrakakis et al. 2015). Further validation is still needed for most automatic lameness systems before they can be used in pig herds (Ala-Kurikka 2020). The Sow Stance Information System based on force plates in a sow feeder (Pluym et al. 2013) has shown to be usable also in commercial setting (Briene et al. 2021).

Table 3.1. Selected objective methods in evaluation of lameness in pigs.

Method	Parameters	Age group	Reference
Force plates	Weight distribution of each foot	sows	Sun et al. (2011), Pluym et al. (2013b), Karriker et al. (2013), Mohling et al. (2014), Conte et al. (2015), Briene et al. (2021)
Pressure algometry	Mechanical nociceptive threshold in kg of force	sows	Tapper et al. (2013)
Pressure mat	Weight distribution, asymmetry	sows	Meijer et al. (2014)
Gait analysis walkway	Stride time, stride length, maximum pressure, stance time	sows	Mohling et al. (2014), Karriker et al. (2013)
Kinematics, motion capture	Vertical head displacement, asymmetric stride, phase timing, walking speed, stance time, swing time, foot height, stride length, angle and amplitude of joint	sows, pre-breeding gilts, growing pigs	Stavrakakis et al. (2015), Stavrakakis et al. (2014), Gregoire et al. (2013), Conte et al. (2015)
Positioning and acceleration, ear sensors in a group of sows	Path length walked, number of areas visited, variance of accelerations	sows	Traulsen et al. (2016)
Accelerometers	Time standing / lying, stepping behaviour (number of steps/min)	sows	Gregoire et al. (2013), Scheel et al. (2017)
Footprint (image) analysis	Stride lenth, contralateral distance, ipsilateral distance, diagonal distance	sows	Gregoire et al. (2013)
Infrared thermography	Thermal images, temperature	sows	Amezcua et al. (2014)

3.3. Clinical examination of lame pigs

It is not easy to examine lame pigs: their large joints are covered with muscles, they do not like to be touched and they are not used to be tied and walked with humans in a controlled manner. Clinicians examining other animal species, pet pigs or pigs for research can use diagnostic methods that are not available in swine practice, such as radiography, ultrasound imaging and nerve blocks. In addition, the value of an individual pig is usually so low that clinical samples such as joint fluid for bacterial culture or blood for hematological analysis are seldom used in confirming the diagnosis of a single pig. Diagnosis is mostly based on visual inspection, palpation and measuring of rectal temperature.

For proper diagnosis, a through clinical examination and collection of anamnestic information is needed: for example age, morbidity, mortality, environment, length of lameness period, response to earlier treatments, feeding, appetite, vaccinations and herd history. Depending on the case, all age categories of pigs on the farm may need to be examined, not just those with the most severe signs. The clinical examination includes first the inspection of the pig in detail: posture, symmetry, appearance, body condition, cleanliness and skin wounds. High rectal temperature may reveal infections. Through examination and palpation of the legs, hoofs and joints are used in finding swellings, heat, pain or any abnormalities, and in helping to localize the affected area. It is important to investigate, if the locomotory disorder is acute or chronic and if more than one leg is affected.

Observation of the claws can be performed while the sow is lying down (e.g. in the farrowing crate). If the sow is standing, her lame legs should be lifted up and claws examined, which is difficult in adult animals. It is easy to lift a piglet (Figure 3.2) but two to four persons are needed to lift feet of adult pigs for proper examination, see figures 3.3 and 3.4. One person holds the sow standing still with a snout snare. One person is needed to handle a front foot, but the sow needs help with balance when her hind foot is lifted. Two people hold a broomstick under the hind leg in the groin area while they lift the leg, and then a third person can inspect the hind claws of an adult pig. In addition, technical devices exist to facilitate claw scoring such as the FeetFirst© chute from Zinpro Corporation and the Mobile

Claw Scoring Device as developed by van Riet et al. (2019). The latter is based on sliding the device under a standing sow and taking video images for further analysis. Future will show, if this device will be used in practice.

Figure 3.2. Joint swelling and lameness are the most prominent clinical signs of infectious arthritis in a piglet. (Photo: Magdalena Jacobson)

Figure 3.3 Two persons are needed in through examination of front feet. (Photo: Sylvia Persson)

Figure 3.4 When a hind foot of an adult pig is examined, one person is needed to hold the animal and two to lift the leg. (Photo: Sylvia Persson)

3.4. Assessment of pig claws

This part concentrates mostly on the procedures performed in sows, but the knowledge can be utilized also for other age groups. Assessment of the claws is an important part of clinical examination of lame sows to reach a proper diagnosis. Although not all claw injuries are associated with lameness, they may indicate an underlying problem and may lead to lameness, reduced reproductive performance and early culling. Therefore, claw lesion assessment can also be done to monitor the occurrence of claw lesions on the farm.

Diagnosis can be impaired by the fact the severity of the lesions is not always commensurate with the impact on the sow welfare. Internal damage can be more severe than expected based on the visual assessment (Kilbride et al. 2009). Contrary, severe claw lesions in appearance may be painful only for a short time or not at all, while they persist for a long time, since it takes time for new horn to grow (Calderón Díaz et al. 2013, Calderón Díaz et al. 2014). An ideal method would allow the detection of claw injuries before they become relevant, i.e. when they may become a risk of developing lameness, reduced feed intake and impaired reproductive performance. Unfortunately, such a detection method does not exist at present and this threshold has not been defined for claw lesions in sows.

Mapping of claw injuries and scoring of their severity is mainly done visually with either a numerical rating scale (NRS) or a visual analogue scale (VAS). Various NRS use different numbers of ordered categories and VAS score scores claw lesions on a continuous scale allowing observers to record more subtle changes (Quinn et al. 2007, Nalon et al. 2014). In practice, a VAS is a long horizontal line anchored by the minimum and maximum score at each end. Severity of a claw lesion type is determined by measuring the distance from 0 mm. A modified scale in which the thresholds of a numerical rating scale are indicated on the continuous scale, a so-called tagged VAS (tVAS), which can help observers make more consistent choices (Nalon et al. 2014, van Riet et al. 2018).

The scorer, the scale used, the scoring conditions and claw cleanliness influence the outcome of scoring. Training and experience of the scorer, the type and severity of the claw lesion, the scoring condition and scoring scale influence inter- and intra-observer reliability (D'Eath 2012, Nalon et al. 2014, van Riet et al. 2020). Van Riet et al. (2020) compared sow chute and the Mobile Claw Scoring Device and concluded that both conditions cannot be used interchangeably and the choice will depend on the possibilities on-farm. Claw cleanliness clearly affects the outcome of claw lesion scoring. If claws are soiled with manure, visibility of lesions is reduced (van Riet et al. 2020). This may lead to detection of the injuries at severe stage when the animals are already lame and treatment is unrewarding. Detailed claw scoring in live sows is difficult to be performed accurately and objectively and therefore some researchers have used post-mortem examination of culled sows in their claw lesion prevalence studies (See later in this chapter, epidemiology of claw lesions).

Claw conformation can be assessed by means of different claw dimensions using a digital calliper (van Riet et al. 2018). Jørgensen (2000) reported an association between claw lesions and buck-kneed forelegs, upright pasterns, steep hock joints, turn out of hind legs, standing under position on hind legs, stiff movements, swaying hindquarters, goose-stepping hind legs, tendency to slip and lameness. However, most of these clinical signs have been associated with other leg disorders as well (such as osteochondrosis and arthrosis) and therefore have no specific diagnostic value for claw lesion diagnosis.

3.5. Sampling of lame pigs

Many pathological conditions can be diagnosed only in necropsy (Nalon et al. 2013). A conclusive diagnosis of lameness can be made through necropsy including histopathological and microbiological examination of representative samples of lame, untreated pigs. If a pig herd suffers from therapy failure, pigs should be sent to necropsy to establish the etiological diagnosis including antimicrobial resistance investigation. In practice, this is easy for piglets, because of their small size. Some studies have collected culled sows for pathological-anatomical diagnosis and found detailed causes of lameness (Dewey et al. 1993, Kirk et al. 2005, Sanz et al. 2007, Engblom et al. 2008, Ala-Kurikka et al. 2019). These studies do not include information about the clinical signs of the culled animals, but they describe the common pathological-anatomical diagnosis. However, one should bear in mind that the study animals are not always randomly selected and therefore the results may be partly misleading. Actually, the combination of clinical and postmortem examinations is a very good method for diagnosing the cause of lameness in a pig (Dewey et al. 1993, Ala-Kurikka 2020). However, the combination is not often used in scientific literature possibly due to practical constraints. Dewey et al. (1993) recommended that herd lameness problems can be diagnosed by performing postmortem examinations on a representative sample of sows culled for lameness during 6-12 months. However, this approach is time-consuming and expensive, and autolysis should be prevented. The microbial cause of lameness in piglets varies, and laboratory diagnostic methods are therefore valuable (Zoric et al. 2008, 2009).

If arthritis is suspected, aseptically obtained joint fluid can be used in detection of pathogens, antimicrobial resistance and antimicrobial resistance genes (Oh et al. 2020). This can be done on the living animal under anaesthesia (Sack 1982) but, for the welfare of the animal and for practical reasons, it is usually done during necropsy (Gomes Neto et al. 2012). In both cases, a sterile technique is critical. If performed during necropsy, it has been recommended to obtain the sample from the medial side of the joint as this area is less covered by muscle tissue. After the skin is removed, an incision with a sterile scalpel blade is made through the joint capsule following the outline of the bones. By applying the leverage, the joint is exposed and samples can be collected (Torrison 2012). In addition, sterilization of the tissue before incision by the use of *e.g.* a small propane flame has been suggested to reduce bacterial contamination (Gomes Neto et al. 2012).

In case of arthrosis, usually necropsy is needed until a specific diagnosis can be made. If the synovial fluid is not turbid or fetid and surface cartilage lesions (e.g. fibrillation, tears, erosion, ulcers) are present, the condition is likely to be osteochondrosis/arthrosis (Rowles 2001).

Reference values for synovial fluid (Canning et al. 2018) and blood hematological analyses (e.g. Klem et al. 2010) have been established, but they are used only in research or for valuable individuals. Acute phase proteins concentrations (Heinonen et al. 2006) have been found to be higher in lame than in non-lame animals, but this method is too unspecific to be used in detailed lameness diagnosis. In growing and finishing phases some infectious agents are associated with lameness occurrence. Collection of oral fluids with soft rope has been used in detection of *Mycoplasma hyorhinis* and *Mycoplasma hyosynoviae* connected with lameness in a group of growing pigs (Pillman et al. 2019). The same pathogens were detected also in the tonsillae of the piglets later developing lameness (Roos et al. 2019).

4. Lameness in suckling piglets and weaned pigs

Lameness in piglets and growing pigs cause ill-thrift and contribute to losses in form of dead pigs, decreased growth, and increased use of antimicrobials and labour. Arthritis is the most common cause for individual antimicrobial treatment in piglets (Zoric et al. 2003, Zoric 2008).

4.1. Predisposing factors, prevalence and effects of lameness in piglets and weaned pigs

Lameness in suckling piglets causes significant problems in farrowing units. Abrasions, wounds and necrosis in the skin or on claws (Figures 4.1 and 4.2) and accessory digits are very common in newborn piglets (Dewey 2006). Risk factors relate to housing conditions especially floor type, nutrition and genetics (Baxter et al. 2011, Le et al. 2015). In addition to that, the passive immunity obtained from colostrum is very important to suckling piglets (Oliviero et al. 2019). Skin lesions in piglets are presumably mainly a result of contact with the floor, especially during suckling (Mouttotou and Green 1999, Westin and Algers 2006, Zoric et al. 2008, 2009). The total time spent suckling at three to six days of age, and the total time spent lying near the sow at

three and four days of age, are positively correlated to development of skin abrasions (Mouttotou and Green 1999).

Figure 4.1. Abrasions, wounds and necrosis in the skin of a suckling piglet. (Photo: Mate Zoric)

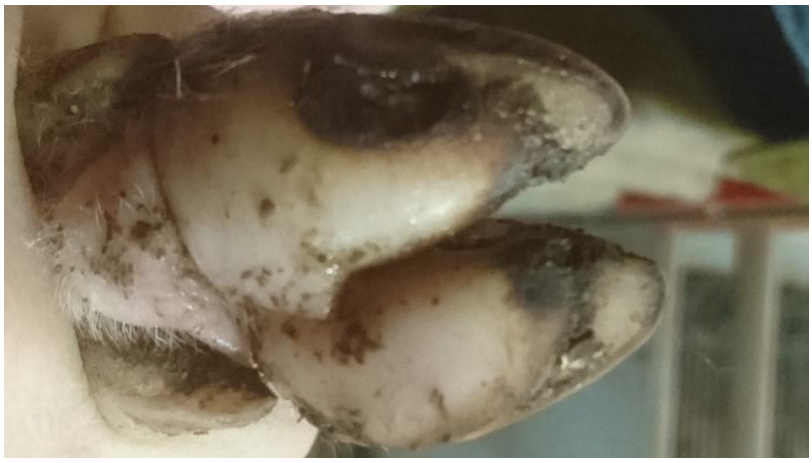

Figure 4.2. Abrasions, wounds and necrosis on the claws of a suckling piglet. (Photo: Marie Sjölund)

Zoric et al. (2003) studied piglets up to nine weeks of age in a research station. Out of 9,411 piglets born alive, 9.8% were treated for lameness. Around 75% of the lameness cases were observed in piglets less than three weeks of age and the incidence risk for lameness decreased from 2.7% during the first week of life to 0.3%

after weaning. Litters with 12 or more piglets had a higher incidence of lameness, but no difference between the sexes was seen.

The housing conditions in farrowing houses present a dilemma because the needs of the sow differ from those of the piglets (Roach 1981). Concrete floor can be rough and abrasive, thereby contributing to removal of horn from hooves and skin from especially the limbs in newly born piglet, which may cause acute lameness (Barnett et al. 2001). Piglets prefer floors with low abrasive properties (Clark 1983, Nilsson 1988, Lewis et al. 2005). Still, floor abrasiveness is a major cause of superficial injury in piglets in farrowing pens (Clark 1985, Lium et al. 2002). In contrast, the sow requires access to an abrasive surface in order to prevent hoof overgrowth. Attempts to provide a non-abrasive surface for piglets have resulted in floors that were too slippery for sows and gilts, while abrasions and sole bruising in piglets decreased (Barnett et al. 2001). In some other studies, doubling the amount of chopped straw prevented the development of skin lesions and sole bruising to some extent (Zoric et al. 2009, 2016).

Zoric et al. (2017) analysed arthritis and growth of pigs with documented pedigree from birth to slaughter in a farm with a floor adapted especially to piglets. Altogether 9,569 piglets were alive at 9 weeks of age and 208 (2.2%) of these had been treated for arthritis. Of these 208 pigs, three (1.4%) were treated again during the fattening period, compared to 0.3% of those pigs remaining healthy. At slaughter, 1.0% of the lame pigs and 0.3% of the healthy ones were recorded to have arthritis in meat inspection. The mean birth weight (1.5 kg) did not differ between the groups, but the weight at 5 weeks, 9 weeks and the carcass weight at slaughter were 1.3, 2.4 and 2.3 kg lower, respectively, in pigs having been treated for lameness during suckling ($P<0.001$) (Table 4.1). Piglets treated for arthritis grew slower than healthy pigs. In addition, the results indicated a greater risk to become lame during the fattening period if treated for lameness during suckling (OR=4.7). However, the results also suggested that the negative impact of arthritis can be reduced by proper floor surfaces, since the incidence of pre-weaning arthritis was lower than the national average (2% vs 10%, OR=0.2).

Table 4.1. Mean weight of piglets treated for lameness during suckling compared to non-lame piglets (Zoric et al. 2017). All weights are presented as live weights, except at slaughter the weight is the carcass weight.

Age	Weight of non-lame piglets, kg	Weight of piglets treated for arthritis during suckling, kg	p-value
Birth	1.5	1.5	n.s.
5 weeks	11.5	10.2	<0.001
9 weeks	28.9	26.5	<0.001
At slaughter	83.1	80.8	<0.001

Lameness in suckling piglets is often associated with skin lesions, often bilateral and most commonly observed as abrasions over the carpal joints. Such lesions are present already on day three, they increase in magnitude until day ten and thereafter decline. Hocks, face, abdomen and tails are affected in a similar way, but at lower magnitudes and these lesions usually vanish on day 17 (Zoric et al. 2008, 2009, 2010). Foot and skin lesions can contribute to lameness in two ways, either due to pain induced by the injury itself or by acting as an entrance for infections that spread to joints through bacteraemia and thereby induce arthritis and pain (Gardner and Hird 1994, Zoric et al. 2004, 2016).

Infectious arthritis affecting single joints has been most commonly observed in piglets less than three weeks old. Zoric and Wallgren (2014) analysed the localization of affected joints in lame piglets until five weeks of age. In total, 6.1% out of 6,780 liveborn piglets were diagnosed with lameness and 91% of these diagnoses took place during the first 3 weeks of life. At 5 and 9 weeks of age, healthy piglets were 1.3 and 1.8 kg heavier than piglets recorded having been lame. In lame animals, one, two and three clinically affected joints were observed in 91.5%, 7.5% and 1% of piglets, respectively. The distribution of affected joints is shown in Figures 4.3 and 4.4. In intensive pig production, the weight of the weaned piglet has a significant influence on lifetime performance (Declerck et al. 2016, Zoric et al. 2017).

During the first week of life, the piglets spend most of their time lying in the nest, and it is obvious that their immediate environment during this period plays a primary role in the appearance and development of leg injuries. Although most commonly observed in back hoof joints, followed by elbows, front hoof joints and

hocks, arthritis was fairly evenly distributed between joints (Zoric and Wallgren 2014). This suggests a septicemic spread of the pathogens associated with lameness.

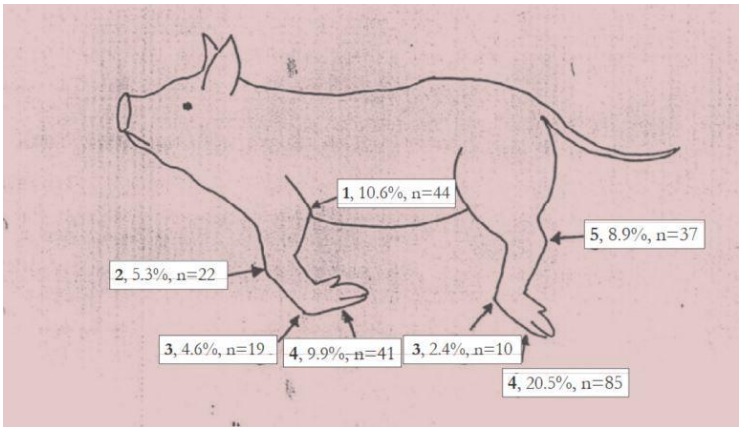

Figure 4.3. The prevalence of joints affected with infectious arthritis on the left side of the piglets (Zoric and Wallgren 2014).

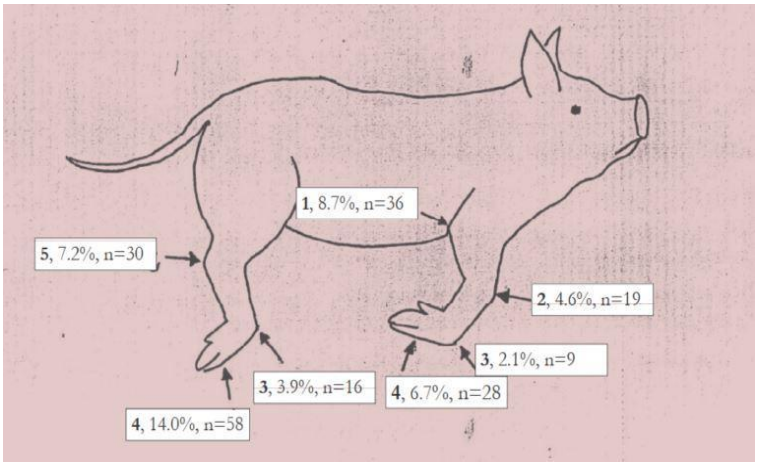

Figure 4.4. The prevalence of joints affected with infectious arthritis on the right side of the piglets (Zoric and Wallgren 2014).

4.2. Infectious causes of lameness in piglets and weaned pigs

Piglets can be infected with different pathogens, either originating from the sow and/or the environment. They may enter the bloodstream via skin wounds, the navel or the tonsils (Gottschalk and Segura 2019). In

routine diagnosis, septicemic polyarthritis can rarely be distinguished from joint infections caused by scrub and hoof injuries (Zoric 2008, Zoric et al. 2009). In piglets, usually hemolytic streptococci dominate, but also opportunistic bacteria such as staphylococci, *Trueperella pyogenes* and *Escherichia coli* are frequently diagnosed. Among weaned piglets, arthritis is more typically associated with microbes such as *Glaesserella parasuis*, *Streptococcus suis*, *Erysipelothrix rhusiopathiae*, *Mycoplasma hyorhinis* or/and *Mycoplasma hyosynoviae* (Dewey 2006, Done et al. 2012). However, the latter pathogen mainly affects growing pigs, usually during the fattening pig period.

4.2.1. Streptococcal infections

Several different streptococcal species can cause infections in pigs. The streptococci domination of sucking piglets suggests the sow to be a significant source of pathogen load to the piglets (Zoric et al. 2008, 2009). The association between arthritis and streptococci belonging to groups C and L and arthritis declines as the pigs age. Several streptococcal species, even the potentially pathogenic ones can be found also in clinically healthy piglets.

Beta-hemolytic streptococci, such as *Streptococcus dysgalactiae* subspecies *equisimilis*, are considered as important pathogens causing infections in young piglets. Streptococci are common in sow secretions and they enter into the piglets through their umbilicus, tonsils or skin wounds. Usually pigs between 1-3 weeks of age are affected. The organisms settle in joints and cause arthritis or polyarthritis and affected animals become lame and their joints become swollen. In addition, they may suffer from inappetence, fever and apathy. At necropsy, in early cases one can see swollen and hyperemic synovial membranes, periarticular edema and turbid synovial fluid. At later stages, also necrotic articular cartilage can be seen and fibrosis and abscesses in periarticular tissue (Gottschalk and Segura 2019).

Streptococcus suis (*S. suis*) is an important pathogen worldwide, being also an emerging zoonotic agent (Gottschalk and Segura 2019). *Streptococcus suis* is a facultative anaerobic gram-positive α-hemolytic coccus.

It can cause systemic diseases in newborn and, more commonly, weaned pigs, resulting in septicemia, meningitis, endocarditis and arthritis. Disease outbreaks are generally related to predisposing factors such as stress and high animal density (Touil et al. 1988, Gottschalk and Segura 2019, Yongkiettrakul et al. 2019). Different *S. suis* strains can be isolated in the same herd (Vela et al. 2003). Even though a large proportion of weaned pigs carry different *S. suis* strains, only few carry virulent strains and usually a single strain causes most disease in a herd (Marois et al. 2007). The disease is confirmed by culturing and identifying the pathogen in tissues of the affected animals. Serotyping *S. suis* isolates helps to indicate, if the disease is caused by the most common serotype or if the serotype causing diseases in a herd has changed. Serotype 2 is considered to be the predominant and most virulent serotype in many countries, but also other serotypes occur commonly (Gottschalk and Segura 2019). The pathogen has a coat of capsular polysaccharide to protect it against the immune system of the host, but this virulence factor is not the only one (Segura et al. 2017). It is difficult to control through vaccination, even though both autogenous and commercial vaccines have been used with inconsistent results (Segura 2015).

4.2.2. *Glaesserella parasuis*

Glässer´s disease is caused by *Glaesserella parasuis* (*G. parasuis*), which is considered to belong also to normal respiratory microbiota of pigs. The bacterium is found in pig herds all over the world, even in herds with good health, but there is usually a balance between infection pressure and immunity. The piglets are colonised soon after birth from their dams (Aragon et al. 2019). Even though several different strains can be found in a herd at the same time, usually one strain is associated with an outbreak of the disease (Rafiee et al. 2000). The immunity of the piglets, other diseases present in the herd, stress conditions (e.g. transport), virulence of the strain involved and genetic resistance have an effect on the severity of the disease (Aragon et al. 2019). This pathogen can act as a primary or secondary pathogen. The disease is seen mainly in pigs of four to eight weeks old. The bacterium can cause peracute and acute lameness, depression, high fever, abdominal breathing, warm swollen joints, unwillingness to stand or move, tremors, paralysis and sudden

death (Aragon et al. 2019). Lesions include fibrinous and fibrinopurulent polyserositis, polyarthritis and meningitis. *G. parasuis* can be detected in the lesions of affected tissues by IHC, in situ hybridization or PCR (Aragon et al. 2019). Pigs recovering from the acute phase may become neglected and develop lameness. Vaccines prevent Glässer´s disease and can be used until a balance between infection and immunity occurs, or in non-immune herds when selling animals to herds where *G. parasuis* occurs (Aragon et al. 2019, Evira 2018).

4.2.3. Mycoplasma infections

Mycoplasma hyorhinis (*M. hyorhinis*) is found in swine populations worldwide also as a commensal in the upper respiratory tract of pigs. It usually affects pigs younger than 10 weeks of age (Heinritzi, 2006, Pieters and Maes 2019) and is often associated with the decline of passive immunity (Roos and Spear 1973) and the purchase of breeding animals (Done et al. 2012). Older pigs in the herd or dams transmit the pathogen to the piglets. The process during the infection or the virulence factors are not well known. Symptoms may include moderately hot and swollen joints, polyserositis, abdominal breathing and unwillingness to move. After about two weeks, clinical signs subside in most animals. In an experimental study, mortality was not found to be high (Gimenez-Lirola et al. 2019). In cases where *M. hyorhinis* causes arthritis, swollen joints and lameness may persist for months. At necropsy, the synovia is swollen and hyperemic. Infection in the joint can lead to adhesions, capsular fibrosis and articular erosions (Pieters and Maes 2019). *M. hyorhinis* arthritis are usually confirmed by culture and / or PCR of swabs collected from joint cavities (Pieters and Maes 2019). Diagnosis of the pathogen can be reached also by detecting the characteristic microscopic lesions in synovial and serosal membranes (mono- and polymorphonuclear cells) (Gomes neto et al. 2012) and by using in situ hybridisation. Measures to control the infections caused by *M. hyorhinis* are difficult to implement, because there is lack of understanding about the epidemiology and strain virulence of this highly prevalent pathogen. Autogenous vaccines against *M. hyorhinis* may be of help (Pieters and Maes 2019).

The other mycoplasma causing arthritis, *Mycoplasma hyosynoviae*, is more common in the finishing stage and it is discussed in detail later in the chapter.

4.2.4. *Erysipelothrix rhusiopathiae*

Infections with *E. rhusiopathiae* in pigs can occur in an acute form characterized by high fever, rhomboid skin lesions and possible arthritis, and in a chronic form that is mainly associated with chronic arthritis (Opriessnig and Coutinho 2019). Because this pathogen causes more lameness problems in finishing pigs, it is discussed in that part of the chapter.

4.3. Treatment of lame piglets and weaned pigs

Most lameness treatments are given parenterally to piglets, because lameness usually affects single animals. Only a few studies exist about pharmacokinetics of different medicines in pigs. Due attention must be paid to the correct dosage of parenterally administered medicines in newborn piglets: overdoses and concentrated preparations must be avoided (Evira 2018). In terms of treatment time, the variables that affect the length of treatment have not been defined. Responses to different types of antimicrobial drug infections vary, and clinical experience is important in assessing response to treatment. For acute infections, improvement should be obvious within two days. If no response is seen at that time, both diagnosis and treatment should be reconsidered. Treatment of acute infections should be continued for at least two days after clinical resolution. In case of chronic infections, euthanasia of the piglet should be considered. Treatment with antimicrobials should be started as early as possible. Else, the infection may have developed into chronic state and treatment might be ineffective.

For suckling piglets and weaned pigs, the first treatment choice is usually benzylpenicillin, because many pathogens causing arthritis for young pigs are penicillin sensitive (Zoric et al. 2009, Sjölund et al. 2018). Tylosin or amoxicillin are possible alternatives. Trimethoprim-sulfa is effective against *Escherichia coli*

isolated in pigs with arthritis (Zoric et al. 2009). An antimicrobial sensitivity testing is recommended especially in case of failure in therapy. Beta-lactam antimicrobials are not effective against mycoplasmas and in these cases recommended antimicrobials include tylosin, tiamulin and lincomycin (Pieters and Maes 2019). High doses of antimicrobials should be administered parenterally as soon as clinical signs appear and all pigs in the affected group should be treated (Desrosiers et al. 1986).

Prompt treatment with antimicrobials is required to achieve a positive treatment effect in lame piglets due to arthritis (Dewey 2006). Treatment of inflammation and pain management is important, because of pig welfare. Different non steroidal anti-inflammatory drugs are available for use in pig, but specific indications are lacking (Schoos et al. 2019). Zoric et al. (2012) compared the clinical effects of a concurrent treatment of lame piglets with ketoprofen and with penicillin and ketoprofen. The occurrence of lameness of 6,780 piglets was registered from birth until five weeks of age, and the clinical efficacy of the treatments was assessed daily. Lameness was diagnosed in 6.1 % of the piglets, mostly (91%) during the first three weeks of life. Both treatment strategies improved the clinical status from day to day (Table 4.2.), and the clinical response did not differ between the two treatment groups. Non-lame piglets were 1.3 and 2.5 kg heavier than piglets treated for lameness at five and nine weeks of age, respectively. There was no difference between body weights of the piglets. However, the NSAID most likely improved the welfare of the lame piglets. It is notable that decreasing of the pain due to arthritis did not lead to injurious use of affected joints. The study showed also that about half of the lame piglets treated with penicillin only or with penicillin and ketoprofen were still clearly lame five days after initiating the treatment and that the lame piglets had reduced weight gain. Possibly the treatments were not sufficient or started too late.

Table 4.2. Number of pigs with daily clinical scores of lame piglets treated for lameness with penicillin for five days (n=208) or with penicillin for five days and ketoprofen with three days (n=207). Lameness and general condition were scored clinically as 0 (good), 1 (almost good), 2 (not good) or 3 (not good at all) (Zoric et al. 2012). ** p<0.01, *** p<0.001. Treatment group did not have an effect on the development of the score.

	Treated with penicillin			Treated with penicillin and ketoprofen		
	Clinical score		Difference between days,	Clinical score		Difference between days,
	0-1	2-3	p-value	0-1	2-3	p-value
Day 1	0	52-156	-	0	49-158	-
Day 2	0-7	79-114	$^{D1}/_{D2}$ **	2-8	71-120	$^{D1}/_{D2}$ **
Day 3	2-32	85-77	$^{D2}/_{D3}$ ***	4-36	85-77	$^{D2}/_{D3}$ ***
Day 4	12-51	73-53	$^{D3}/_{D4}$ ***	15-53	76-49	$^{D3}/_{D4}$ ***
Day 5	34-58	66-36	$^{D4}/_{D5}$ **	38-63	57-40	$^{D4}/_{D5}$ **

4.6. Prevention of lameness in piglets and weaned pigs

Lameness in suckling piglets and weaned pigs can be prevented most of all by improving environmental conditions. In addition, good passive immunity obtained by ingesting sufficient amount of colostrum is of utmost importance for the piglets (Oliviero et al. 2019). Adding peat on the floor for the first three weeks after farrowing and taping the front knees of the piglets may be of help. Proper maintenance of the floors is a relevant measure. A successful concept to reduce abrasions, sole bruisings and lameness is to increase the amount of chopped straw during the first days of life and again after weaning. In addition, during the first week of life, the lying nest of the piglets should be warm enough (32-33 °C). A proper hygiene programme is important and it requires all in- all out production in farrowing and weaning rooms. In continuous production,

rooms cannot be properly cleaned, disinfected and dried. It is also important to avoid regrouping of pigs to reduce fighting and aggression to minimize the stress and injuries (Coutellier et al. 2007).

5. Lameness in finishing pigs and breeding pigs

Lameness is a frequent health and welfare problem in finishing pigs. It causes also great economic losses. Lameness has accounted for 11-15 % of the recorded clinical signs in finishing pigs being the second most common clinical sign in Danish finishing herds (Christensen et al. 1994, Petersen et al. 2008). Similarly, arthritis together with locomotory problems was the second most frequent disease treated in Finnish finishing pigs (Heinonen et al. 2001) and the main reasons for antimicrobial treatments of finishing pigs were musculoskeletal diseases (Stygar et al. 2020). However, lameness is just a clinical sign and its etiology may differ considerably.

Lameness in finishing and breeding age pigs can be caused by infectious agents (bacteria or viruses) or by non-infectious causes (trauma, fractures, muscle disorders, osteochondrosis, and degenerative joint diseases). In many cases of lameness in single pigs, the exact diagnosis remains unknown, because individual pigs have low monetary value and the expenses for microbial investigations or other diagnostic procedures are too high. Therefore, usually clinical diagnosis alone is used when the choice of therapy and prophylaxis is determined. Only when several finishing pigs become affected, necropsies or additional diagnostic investigations are performed to reach the diagnosis.

5.1. Predisposing factors for lameness in finishing pigs

Plenty of studies have reported risk factors for lameness of sows, with emphasis on the association between claw health and either nutrition or flooring (Pluym et al. 2013b). However, much less has been published about risk factors of lameness in finishing pigs. The lameness prevalences vary in different housing systems,

but the associations are not always easy to interpret because of the many factors having an effect at the same time. Some of the studies about finishing pigs focused on joint lesions in the slaughterhouse or on farm whereas others focused on lameness in live pigs, which makes it difficult to make proper comparisons between the studies.

The most important predisposing factor for lameness in finishing pigs and especially for claw lesions is poor housing. Housing conditions can be very variable and therefore the needed sample size of different housing conditions becomes large and difficult to study. In addition, different characteristics of the housing likely have an interacting effect, which further complicates the analysis and the interpretation of the results.

Usually soft bedding has been considered to be beneficial for the pig welfare. However, the research results have not always been consistent. For example, de Koning et al. (2014) did not find an age-dependent effect of floor type on the prevalence of osteochondrosis in gilts reared on different floor surfaces. However, they found that gilts kept on floor with wood shavings had greater odds to be affected with severe osteochondrosis lesions compared with those kept on partly slatted floor (twisted metal slats and epoxy coated concrete). In other studies, fully slatted floors were found to be a risk factor for lameness in finishing pigs compared to deep bedding (Kilbride et al. 2009b, Scott et al. 2006) or to solid concrete floor with sparse bedding (Kilbride et al. 2009b).

Especially the floor surface should not be too slippery or too rough and the in-between spaces of the slats should be suitable for the age of the pigs (Mouttotou et al. 1999, EU legislative 2008, Kilbride et al. 2009). The front legs carry higher load and have been observed to slip more both forward and backward when the pig walks (Applegate et al. 1988). This may have an effect on the development of lesions in front feet. Wet floors may have an effect on slipping and softening the sole increasing the risk of erosions especially when the surface is rough. Soft floors prevent the hoof from wearing, but increase the risk of claw overgrowth which may cause lameness. However, finishing pigs are not usually affected by claw overgrowth, because they are slaughtered at an young age.

In a study investigating claws of finishing pigs in the slaughterhouse, pigs kept on bedded, partially slatted or totally slatted floors had different kinds of claw lesions (Mouttotou et al. 1999). No ideal floor surface was found for pigs, because the development of claw lesions in finishing pigs is complex and multifactorial. Another group found that finishing pigs raised on straw bedding had more severe toe erosions, whereas the ones raised on fully slatted floors had more severe sole and heel erosions (Scott et al. 2006). Heinonen et al. (2007) investigated factors associated with arthritis detected at slaughter and recorded information about the housing conditions, management, feeding and hygiene practices, health and disease prevention in the farrowing, weaning and finishing units of integrated pig herds. The herds with high clinical sign scores including all disease signs of lactating sows, piglets less than one week old and finishing pigs in the herds had greater chance of being a case herd with high arthritis prevalence in slaughterhouse. The case herds were generally larger, aimed at higher efficiency and tended to have housing and management practices such as use of slatted floors and handling of manure as slurry. In addition, the case herds were more likely to wash and disinfect the pens and they invested more in hospital pens.

The results have not always been easy to interpret. For example Krieter et al. (2004) found that growing-finishing pigs kept on bedded multi-surface system with outdoor run had higher risk of lameness than pigs kept on fully slatted pens. The authors discussed that the outdoor area may have caused the lameness. However, this nicely brings out the fact that different studies are quite difficult to compare because of the very different housing conditions, and the major differences in the study design.

Greater leg lesions scores (wounds or swellings on legs) and lameness scores were found in finishing pigs housed in large groups (108 pigs) compared with the ones in small groups (18 pigs) in a study of Street and Gonyou (2008). They found also an interaction of group size and space allowance for lameness. Pigs were more susceptible for lameness in large groups with restricted space allowance. In large groups, pigs might fight more especially if their area to move is restricted. Their unconfirmed hypothesis was, however, that pigs in large groups had more space for moving around and therefore were more likely to get their claws caught in slats or run into other pigs and get hurt.

5.2. Infectious causes of lameness of finishing pigs

The most common infectious lameness diagnosis in finishing pigs is arthritis caused by different pathogens, the two most important ones for this age group being *E. rhusiopathiae* and *M. hyosynoviae*. Other possible pathogens are presented here shortly, and in more detail in the part discussing lameness in younger pigs.

5.2.1. *Erysipelotrix rhusiopathiae*

E. rhusiopathiae is ubiquitous and the domestic pig is an important reservoir (Opriessnig and Coutinho 2019). The presence of neuraminidase and hyaluronidase has been suggested to be the virulence factors of this organism (Wang et al. 2010). The virulence of *E. rhusiopathiae* strains is known to vary considerably. *E. rhusiopathiae* can be found in many animal species and in water, soil and slurry. About 30-50 % of clinically healthy pigs harbor the organism in their tonsils and other lymphoid tissues. Carriers and acutely infected pigs excrete the organism in the urine, feces, saliva and nasal mucus (Opriessnig and Coutinho 2019). The organism can remain viable for 12 days in direct sunlight and for months in carcasses (Reboli and Farrar 1989).

In acute infections, cutaneous lesions and septicemia are the most prominent findings; however, the infection may pass also subclinically. Later, chronic proliferative lesions may develop in the joints of some affected pigs resulting in lameness about three weeks after the initial outbreak (Opriessnig and Coutinho 2019). Affected animals show varying degrees of lameness, joint stiffness and inappetence and their hocks, stifles or carpal joints can be enlarged. The joint swellings are firm and not warm. Morbidity and mortality vary in herds due to different immune status of the pigs and the occurrence of other infections in the herd (Opriessnig and Coutinho 2019).

Chronic lesions of erysipelas include chronic arthritis involving several joints. The synovial membrane proliferates and the capsule is often hyperemic. In addition, proliferation and erosion of the articular cartilage leading to fibrosis, ankyloses and spondylitis can be seen (Opriessnig and Coutinho 2019). Isolation of the organism from tissues or fluids from affected animals gives a definite laboratory diagnosis. However, in

chronic cases, it may be difficult to isolate the organism (Friis et al. 1992). A variety of tests can be used to diagnose *E. rhusiopathiae*: direct isolation, enrichment, fluorescent antibody assay, immunohistochemistry, PCR and serology (Opriessnig and Coutinho 2019).

Protection against *E. rhusiopathiae* is achieved through regular vaccinations of all sows in a herd. Because of short-lasting immunity, vaccinations need to be repeated every six months. Vaccinations are effective in prevention of the acute form, but they may not be as effective in the prevention of the chronic form. Vaccination of pigs to prevent erysipelas in fattening pigs is rare.

5.2.2. *Mycoplasma hyosynoviae*

M. hyosynoviae causes non-purulent arthritis in finishing pigs and young breeding animals older than 10 weeks. Outbreaks with morbidity rates between 10 % and 50 % have been reported (Kobish and Friis 1996). Suckling piglets are usually protected against *M. hyosynoviae* arthritis by passive immunity obtained from the sow (Lauritsen et al. 2017) and most of the pigs are infected after weaning or during finishing stage (Hagedorn-Olsen et al. 1999b, Geudeke et al. 2016). Okholm-Nielsen et al. (2005) studied the epidemiology of the disease and found that the most critical period for spread of *M. hyosynoviae* infection in the herds is the age between 4 and 12 weeks. In addition, they concluded that other herd or host factors than virulence or antibody levels are more likely to be the triggers of clinical arthritis due to *M. hyosynoviae*. Affected pigs probably remain infected (Okholm Nielsen et al. 2005). The pathogen resides in the tonsils, nasal cavity and airways of the pigs and occasionally invades systemically (Friis et al. 1991, Hagedorn-Olsen et al., 1999b, Okholm Nielsen 2000). The clinical outcome at pen level has not been associated with detection of *M. hyosynoviae* in oral fluids or tonsils of the finishing pigs (Pillman et al. 2019). The organism reaches the synovia of the joints through blood stream and causes edema in the synovial membrane and synovial villi and the amount of synovial fluid increases (Hagedorn-Olsen et al. 1999a). A bacteremia of about one week precedes the joint infection (Hagedorn-Olsen et al. 1999b). Acute signs are usually observed 7-10 days after an experimental inoculation (Gomes Neto et al. 2012). According to Gomes Neto et al. (2016) this took 1-15

days depending on the route of inoculation. The diseased pigs may become severely lame showing a dog-sitting position for several hours, but also only mild lameness can be seen (Okholm Nielsen 2000). The stifle and elbow joints are primarily affected. The pigs may have a slight rise in body temperature. The affected animal usually recovers and the condition does not become chronic (Okholm Nielsen 2000).

Gross pathological changes are highly suggestive of infection of *M. hyosynoviae* (Hagedorn-Olsen et al. 1999a), but must be confirmed by showing the presence of the mycoplasma. *M. hyosynoviae* should be suspected when synovial membranes are dark, proliferative or hyperemic and the synovial is brown or yellow and serofibrinous or serosanguinous (Gomes Neto et al. 2012). Okholm Nielsen et al. (2001) studied arthritis in growing-finishing pigs in nine Danish herds 1-98 days before slaughter. They obtained synovial fluid from joints of lame and non-lame pigs. *M. hyosynoviae* was the only pathogen isolated in 20% of the lame pigs and in 8% of the non-lame pigs. They showed that *M. hyosynoviae* infection can be present also in pig joints without lameness. Some of the affected pigs (5%) needed to be euthanized due to lameness, but others recovered after treatment and were slaughtered. The agent is often present in the joints at low concentrations (Kobisch and Friis 1996). Culturing is not always successful and the diagnosis is often reached by clinical signs and absence of other pathogens causing arthritis or by PCR tests (Friis et al. 1992, Gomes Neto et al. 2015). No commercial vaccine is available to prevent mycoplasmal arthritis (Pieters and Maes 2019).

5.2.3. *Streptococcus suis and Glaesserella parasuis*

S. suis infections are known to occur worldwide as epidemic outbreaks of meningitis, septicemia and arthritis. This pathogen has a predilection for pigs in the preweaning and immediate postweaning periods, but also finishing pigs can be affected (Williamson 2018). *G. parasuis* is another bacterium affecting mostly young pigs in endemically infected herds, but having a possibility to cause infections also in finishing pigs. Both of these pathogens have been discussed in more detail in the section of lameness in suckling piglets and weaned pigs.

5.2.4. Other bacteria

Many other, opportunistic bacteria cause septicemia after entering the body of the pig often through skin wounds or other routes and localize in joints, meninges, serosa surfaces, lungs and other organs (Madson et al. 2019). In finishing pigs, tail biting is a common risk factor for joint infection. Pathogens such as *Actinobacillus pleuropneumoniae*, *Trueperella pyogenes*, *Brucella suis*, *E. coli*, *M. hyorhinis*, *Salmonella* spp. and pyogenic *Streptococcus* spp. may cause arthritis in growing pigs (Okholm Nielsen 2000). Affected animals are lame and they may have fever and inappetence. In addition, they may have swollen joints, inflamed synovial membranes, fibrinous periarthritis and the joint fluid can be purulent. In addition, due to the septic nature of the condition, abscesses can be found in these animals. Usually most often affected joints are the hock, stifle, carpus, elbow and hip (Madson et al. 2019). Joint swellings are sometime difficult to be recognized clinically, because some of these joints are largely covered by muscle layers.

It is difficult to find scientific articles about abovementioned pathogens taken from joints of live animals. Old studies report pathogens found in affected joints in slaughterhouse material, which have revealed *E. rhusiopathiae*, streptococci, staphylococci and mycoplasmas in arthritic joints, but the proportions present vary considerably. Hariharan et al. (1992) could isolate the following bacteria in the synovial fluid of 153 finishing pigs with swollen joints and enlarged accompanying lymph nodes: *E. rhusiopathiae* (45%), *S. suis* (16%), *Trueperella pyogenes* (10%), *Mycoplasma* spp. (7%), *Staphylococcus* spp. (7%), *Streptococcus* spp. (6%) and organisms of uncertain significance (7%). They did not isolate any bacteria in the joints of 80 healthy control animals. Turner et al. (1991) found that 6.1% of joints of 192 carcasses of finishing pigs had arthritis and they isolated eit her *Staphylococcus aureus* or *Streptococcus* spp. from these joints. Buttenschon et al. (1995) studied non-purulent arthritis in slaughter pigs. They found that more than 70% of the joint samples were sterile. *E. rhusiopathiae* was isolated in 9% and *M. hyosynoviae* in 12-14% of the pigs, and the following pathogens from a few animals: *Staphylococcus* sp., haemolytic *Streptococcus* sp. and haemolytic coliforms.

5.2.5. Infectious contagious viral diseases

Also contagious viral infections should be considered in the differential diagnoses of lameness. These diseases include Foot and Mouth Disease (picornavirus), Seneca-virus (picornavirus), Vesicular Exanthema (calicivirus), Swine Vesicular Disease (enterovirus) and Vesicular Stomatitis (rhabdovirus) (Ramirez 2019). Even though these infections are rare, they may affect the whole herd, whenever suspected. They cause similar clinical signs, the lesions are indistinguishable from each other and therefore the laboratory diagnosis is always needed. These diseases usually cause high fever in the beginning of the disease and then formation of vesicles in and around the mouth and/or feet. Affected pigs are reluctant to stand and walk and they lose their appetite. The details of these diseases are not included in this chapter.

5.3. Non-infectious lameness in finishing pigs

5.3.1. Osteochondrosis in finishing pigs

Osteochondrosis (OC) is defined as a focal disturbance (delay) in endochondral ossification (Reiland 1978a). Osteochondrosis can occur in sub-articular, epiphyseal growth cartilage and metaphyseal growth plates or physes (see Anatomy section of this chapter) in both the appendicular and axial skeleton, but this section will focus on articular OC in limbs.

OC occurs as a consequence of failure of the temporary, end-arterial blood supply to growth cartilage (Carlson at al. 1991, Ytrehus et al. 2004a) (Figure 5.1). Vascular failure leads to ischemic necrosis of chondrocytes at intermediate depth of growth cartilage, outside diffusion distance from alternative sources, known as osteochondrosis latens or an infarct (Carlson et al. 1991, Ytrehus et al. 2004) (Figure 5.1). With time, the ossification front advances to surround the infarct, at which stage it causes the focal delay in endochondral ossification that is the definition of osteochondrosis, also referred to as osteochondrosis manifesta (Ytrehus et al. 2004b) (Figures 5.2 and 5.3).

Figure 5.1. In osteochondrosis, failure of the temporary, end-arterial blood supply (white, barium-filled vessels) to growth cartilage leads to ischemic necrosis at intermediate depth of growth cartilage (dashed circles). Reprinted with permission (Olstad et al. 2015).

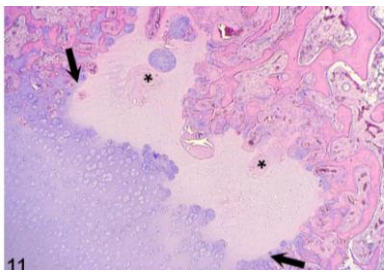

Figure 5.2. With time, the ossification front advances to surround the failed vessels (asterisks) and area of chondronecrosis (between arrows), which then causes a delay in endochondral ossification. Reprinted with permission (Olstad et al. 2015).

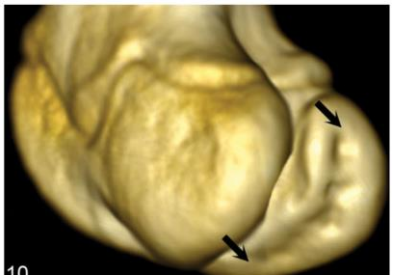

Figure 5.3. The delay in ossification is detectable in CT scans, typically as multi-lobulated or “stair-step” defects (between arrows) reflecting the configuration of the failed, branching blood vessels as seen in Figure 5.1. (between arrows. Three-dimensional rendering of bone density only; cartilage and soft tissues not shown. Reprinted with permission (Olstad et al. 2015).

OC lesions can resolve spontaneously before joint-specific age thresholds (Olstad 2014), or progress to pathologic fracture known as osteochondrosis dissecans (OCD), pseudo- or true subchondral bone cysts (Olstad et al. 2014). Several factors have been implicated in the pathogenesis of OC such as heritability, rapid growth, over-feeding or lack of certain micronutrients in the diet, exercise and trauma (Ytrehus et al. 2007, Olstad et al. 2015). These factors seem capable of modifying the outcome of the disease process. Whether they are capable of causing the failure of blood supply that triggers the pathogenesis in the first place, i.e., whether they are true etiological factors remains to be confirmed (Olstad et al. 2015). The best-documented etiological factors are failure of vessels at the point where they traverse junctions between growth cartilage and other tissues (Ytrehus et al. 2004c, Olstad et al. 2008c), without any concurrent traumatic or other physical disruption of surrounding cartilage or bone (Finnøy et al. 2017, Olstad et al. 2008a), and occlusion of cartilage canal vessels by septic thrombi following bacteremia (Denecke et al. 1986, Wormstrand et al. 2018).

When using techniques capable of detecting early lesions, the prevalence of OC can easily be 100% Aasmundstad et al. 2013). OC is therefore probably present to some extent in any herd, but the condition does not necessarily cause clinical signs. Osteochondrosis latens and manifesta lesions are generally believed to represent subclinical stages, whereas OCD and cysts that communicate with the joint are considered as clinical stages of the disease. When OC does cause clinical signs, articular OC has been associated with joint effusion and various manifestations of pain (Reiland 1978b).

OC is a condition that occurs multi-focally at predilection sites and in the majority of cases, it affects more than one joint simultaneously (Olstad et al. 2014a). Thus, whether an individual pig shows lameness depends on the overall distribution and severity of OC in that particular pig. Pigs with symmetrical lesions may not show overt lameness on one limb, or lameness may change from one limb to the other depending on which joint feels worst on the day. It is a useful diagnostic aid to examine posture, as affected pigs will often stand on their toes or rocked back on their heels, with their fore- and/or hind limbs tucked under (Figure 5.4), presumably to offload OC lesions in weight-bearing regions of the elbow and stifle joints (Reiland 1978b).

The lameness associated with OC was originally referred to as “leg weakness”, and a proportion of pigs with OC will prefer to adopt a dog-sitting position, or lie down altogether. If further diagnostic work-up is indicated, live pigs can be examined using ultrasound or radiography in the field, or computed tomography (CT; Figure 5.5) (Olstad et al. 2014b) or magnetic resonance imaging (MRI) (Toth et al. 2013) in hospital, screening or research settings. More commonly, definitive diagnosis is made on post-mortem examination (Reiland 1978a), through macroscopic inspection of intact joints and slabs sawed through multiple bones from slaughtered pigs. At post-mortem examination, it is also possible to collect and process slabs for histological evaluation, where standard morphological and gram-staining enables definitive differentiation between aseptic and septic vascular failure (Wormstrand et al. 2018).

Figure 5.4. Pigs affected with osteochondrosis may stand with their forelegs tucked under presumably to offload lesions in weight-bearing regions of the elbow joints. (Photo: Mari Heinonen).

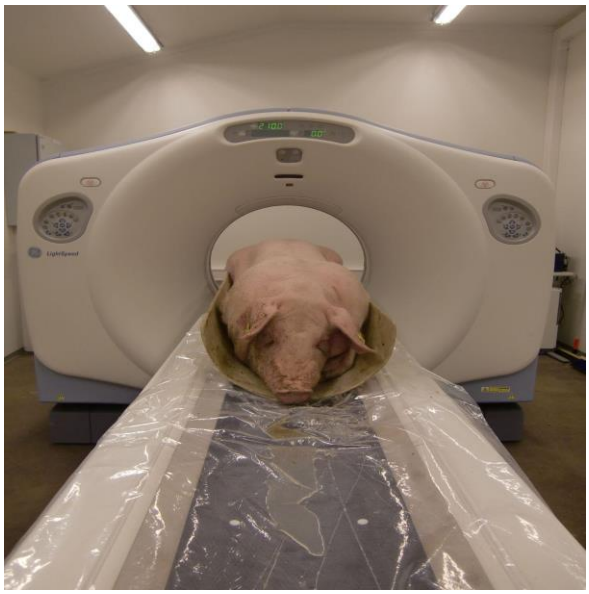

Figure 5.5. Entire live pigs can be scanned under sedation using computed tomography. Their joints are examined for screening and research purposes. (Photo: Courtesy of Norsvin SA)

Pigs with subclinical OC lesions do not require any treatment. Pigs that show only mild signs can be monitored in the hope that their lesions will resolve (Olstad et al. 2014a), and may be put on restricted feeding in a sick pen to encourage resolution, if feasible. For pigs showing marked signs like overt lameness, dog-sitting or prolonged recumbency, treatment is mandatory. In a commercial setting, treatment of individual pigs may not be financially viable, necessitating premature culling of the pig instead. One of the main reasons why OC must be diagnosed and controlled, even if in most cases it causes only mild or no clinical signs, is that it is associated with significant losses due to premature culling. OC can also progress to irreversible, debilitating osteoarthritis in older pigs (Grøndalen 1974, Reiland 1978b), meaning the condition is important for the durability of breeding sows (Heinonen et al. 2006, Jensen et al. 2010).

5.3.2. Trauma in finishing pigs

Various traumas can cause acute, sporadic lameness due to bone fractures and muscle or tendon ruptures. In case of frequent prevalence, a thorough investigation should be carried out to find risk factors related to

diet, flooring, housing, mechanical sources of trauma, transport and handling (Madson et al. 2019). Fractures in slaughterhouse leading to hemorrhage may increase in prevalence in case of disbalances in diets, especially concerning mineral composition. Accidental electrocution or outdoor lightning strike may result in fractures in several animals at the same time affecting mostly lumbosacral and thoracic vertebrae, femur, humerus, neck of scapula or pelvis (Van Alstine and Widmer 2003).

Pig claws are especially sensitive to trauma and therefore the prevalence of claw lesions is common in finishing pigs. Mouttotou et al. (1997, 1999) published a prevalence of 93.8% of finishing pigs having at least one lesion (heel corrugation, white line lesion, wall separation, false sand crack, wall penetration, overgrown hooves, unequal claw size) in their slaughterhouse studies. They found that lateral digits and hind legs were more frequently affected than medial digits and front legs. The two most commonly observed lesions were sole erosion and white line lesions.

The etiology of different claw lesions has mostly been investigated in sows and less in the finishing pigs. Mouttotou et al. (1999) reported that floor type had an effect on claw lesions. Finishing pigs housed on bedded floors had less sole erosions, heel erosions and heel flaps and more white line lesions, false sand cracks, wall separations and toe erosions than pigs kept on bare solid concrete floors. Partially slatted floors were associated with an increased prevalence of heel erosions, heel flaps, white line lesions, wall separations and false sand cracks, and totally slatted floors were associated with an increased prevalence of sole erosions and heel flaps.

In a study of Okholm Nielsen et al. (2001), claw lesions were found in 63.2% of growing-finishing pigs. Most of the lesions (48.3% of the animals) were cracks in the soft heel and 14.9% of the pigs had deep cracks in the wall, the white line or the soft heel.

Claw and soft heel lesions may cause lameness and especially if they get infected, the lesions may become very painful (Gjein and Larsen 1995). The association between lameness and different kinds of claw lesions cannot always be found. In sows, lesions such as cracks in the heel or white line have not correlated with

lameness of the same leg (Kroneman et al. 1993). It is therefore also possible that not all lesions cause lameness in finishing pigs.

5.3.3. Muscle disorders in finishing pigs

Primary myopathies are rare nowadays, because porcine stress syndrome (PSS) leading to PSE meat (pale, soft exudative) has mostly been mitigated (Madson et al. 2019). This stress syndrome causes sudden death of pigs who have a hereditary predisposition, which is triggered by stress such as transport or excitement. Back muscle necrosis is considered to be one type of the syndrome and it is found mostly in pigs heavier than 50 kg. Affected pigs have swollen, warm and painful back muscles and difficulties in moving. Muscle enzyme CPK (creatine phosphokinase) concentration is high. The back muscle is dark and hemorrhagic when cutted open (Madson et al. 2019).

Nutritional myopathies include vitamin E and selenium deficiency leading to generalized skeletal muscle myopathy but more commonly to mulberry heart disease or hepatosis dietetica. The problem may arise when diets low in selenium / vitamin E are fed to growing pigs or if factors in diets such as mycotoxins destroy vitamin E in the diet. Pigs are not very susceptible to nutritional muscular dystrophy, but in case it is seen, usually a pig of 50-60 kg is ataxic, walks stiffly, is weak, depressed and anorexic. The condition may lead to paralysis and death. The muscle is edematous with white streaks (Madson et al. 2019).

5.4. Treatment of lameness in finishing pigs

Neither controlled trials nor evidence-based guidelines can be found about treatment of lameness in sows (Ala-Kurikka 2020) and the same applies to finishing and breeding age pigs. In case of lameness suspected to be of nutritional or environmental origin, the first action is to correct the diet or environment. In these cases, the affected animals cannot totally be cured, but the condition will not appear in new, younger groups of pigs.

The decisions of treatment mostly rely on clinical experience and the difficulty in reaching a proper diagnosis complicates the situation. Therefore, the treatment of single lame finishing pigs is usually symptomatic aiming to relieve the symptoms without targeting the underlying cause of the disease. Usually the treatment includes antimicrobials, if any kind of bacterial infection is suspected and pain and inflammation mitigation. The third part of the treatment is supportive care of the affected animal (Table 5.1.).

Table 5.1. Main items in the treatment of lame pigs

Cause of lameness	Medical treatment	Other treatments	Supportive care	Other considerations
Nutritional		Correction of the diet	Use of proper sick pen, if needed to ensure that the lame pig can eat, drink and rest without problems	In severe cases, consider euthanasia
Environmental, managemental		Improvement of environment and/or management		
Infective, bacterial	Antimicrobials, NSAIDs			
Infective, viral	NSAIDs			
Non-infective	NSAIDs			

NSAIDs: non-steroidal anti-inflammatory drugs

It is very important to try to reach a good clinical diagnosis. If the condition causing lameness in the pig is detected late and the disease is already in severe or chronic stage, the pig needs to be culled rather than treated. In addition, economic considerations, long withdrawal times of some medications, or lack of effective treatment of the affected individual may lead to euthanasia rather than medication.

The microbes causing lameness should be treated with antimicrobials ideally based on culture and antimicrobial sensitivity testing. Sampling of individual lame pigs is seldom performed and therefore the selection of the drug is usually done based on general knowledge and experience. In herds with frequent problems of infectious lameness, it is recommended to take samples from affected animals in early stages of the disease to obtain information about the pathogens causing the lameness and their antimicrobial sensitivity. Penicillin and other beta-lactam antimicrobials are usually considered as drugs of choice. They are

active against most of the pathogens causing arthritis in finishing pigs as discussed earlier in this chapter. Of these pathogens, only Mycoplasmas are resistant to beta-lactam antimicrobials and some studies have shown the efficacy of macrolides, lincosamides or tetracyclines in their treatment (Burch and Goodwin 1984, Hannan et al. 1997ab, Karriker et al. 2019). The duration of treatment in pigs has not been adequately defined in pigs. For acute infections, response to treatment is usually observed within two days and in case no response is observed, diagnosis and treatment should be reconsidered (Karriker et al. 2019). However, very often the common practice is to treat the pigs only 1-7 days (Ala-Kurikka 2020).

Because scientific literature about treatment of infectious arthritis in finishing pigs is lacking, we need to refer to other animal species. In cattle, successful therapy of infectious arthritis has included early administration of appropriate antimicrobial agent and mechanical removal of deleterious material from the joint and continuation of the therapy long enough to complete elimination of the infectious organisms (Trent and Plumb 1991). If debilitating damage has occurred, therapy should stop the progression of infection and allow pain-free function of the limb. In pigs, any kind of joint lavage is extremely difficult to be performed under farm conditions and in addition to this, very expensive. Horses with septic arthritis are recommended to be treated systemically 10-14 days and the affected joints lavaged (Haerdi-Landerer et al. 2010). In humans, several weeks of antimicrobial treatment is recommended together with drainage of the affected joint (Smith et al. 2006). The antimicrobial medication given to pigs may be too short to resolve the problem, but on the other hand, long treatment duration is usually not economically possible or discouraged because of antimicrobial resistance. However, clinical experience has shown that also these short treatments for lame pigs can be effective, especially if the treatment is initiated early enough. More research is definitely needed about the different aspects of antimicrobial treatment of infectious lameness is pigs.

Currently pain mitigation is becoming more important in different disease conditions also in pigs both in the attitudes of veterinarians and farmers (Ison and Rutherford 2014). The use of three NSAIDs have been reviewed in connection to treating lameness in sows and/or finishing pigs: flunixin-meglumine, ketoprofen and meloxicam (Schoos et al. 2019). In most of the studies, the use of NSAID has resulted in reduction of

lameness. In one report, clear lameness due to osteochondrosis of a boar was alleviated with NSAID (Oomah 2008). However, potentially pain is not treated because of difficulties in pain recognition (Flecknell 2008). In addition, as long as we do not make proper diagnosis early enough, the treatment is likely to be applied in the chronic phase of the condition (Schoos et al. 2019). We lack lameness studies, where different NSAIDs would have been used for finishing pigs with and without antimicrobial agents.

In horses, treatment of osteochondrosis would initially be conservative by restricting diet and exercise whilst waiting until the age thresholds for spontaneous resolution have passed, after which time surgical removal of any persistent OCD fragments cures the problem. It is probable that small OC lesions may go undetected and resolve in pig populations all the time. In the case of clinical lesions, signs might be either very mild not necessitating any treatment, or else treatment might not be economically justified, resulting in (premature) culling or slaughtering of the animal. It is more a question of deciding whether signs are so mild that pigs may live on, or must be sent to slaughter, rather than surgery. Thus, prevention is more important than cure.

The third cornerstone of treatments is the proper supportive care of the affected animal, which is sometimes neglected (Ala-Kurikka 2020). Each herd should have enough proper sick pens, in which lame finishing pigs can be moved. In these pens, the pigs should have a possibility to eat, drink and rest without problems.

5.5. Prevention of lameness in finishing pigs

Because the most important predisposing factor for lameness in finishing pigs is poor housing and management, their correction is the key to prevent lameness. More details are given in the chapter discussing the predisposing factors. In addition, proper nutrition and healthcare including vaccination programs whenever available for different pathogens are needed in lameness prevention.

The aim of prevention of osteochondrosis-type problems is to reduce the prevalence of that portion of OC that is due to heritably predisposed vascular failure (Olstad et al. 2015), through genetic selection. In pigs, generation of breeding values still includes an element of phenotypic scoring (Grøndalen 1974, Reiland

1978a). Historically, prospective breeding boars received a phenotypic OC score inferred from scoring of slaughtered half-siblings, and pedigree analyses. Since 2008 in Norway and more recently in Canada, prospective breeding boars have been sedated to undergo whole-body CT-scanning (Figure 5.5), enabling phenotyping for OC and other traits in the still-alive boar itself. At present, OC is scored manually in CT scans at medial and lateral sites in the elbow and stifle (Aasmundstad et al. 2013), but manual scoring is likely to be superseded by automated diagnosis via artificial intelligence (Kongsro et al. 2017), enabling scoring of more sites and joints per pig. Breeding values are then generated by coupling the phenotypic scoring with genetic information from pedigree analyses and single nucleotide polymorphism (SNP) chip analyses of the genome (Grindflek et al. 2014), and collectively, these techniques constitute powerful tools for maintaining and improving pig health and welfare.

6. Lameness in sows

Since 2013, group-housing of gestating sows has been obligatory in all member states of the European Union. Although the ban on confinement of sows was decided on welfare grounds, group-housing of pregnant sows also presented welfare-related shortcomings including an increased risk for lameness development (Anil et al. 2005, Calderón Díaz et al. 2014).

The list of differential diagnoses of lameness in sows is extensive. All components of the musculoskeletal system can be involved. However, in several studies joint disorders (arthritis and osteochondrosis/arthrosis) and claw lesions were diagnosed to be the principal causes of lameness and were associated with culling or euthanasia in sows (Dewey et al. 1993, Kirk et al. 2005, Heinonen et al. 2006, Sanz et al. 2007, Engblom et al. 2008).

6.1. Arthritis in sows

6.1.1. Aetiology and diagnosis of arthritis in sows

Infectious arthritis is commonly associated with bacteria. These may access the synovial membrane through the bloodstream, direct entrance into the joint after trauma or an expansion of a nearby infection (e.g. infected claw lesions) (Rowles 2001, Crenshaw 2006, Done et al. 2012). Bacteria isolated from arthritic joints in pigs include *Erysipelothrix rhusiopathiae*, *Streptococcus suis*, *Glaesserella parasuis*, *Actinobacillus suis*, *Trueperella pyogenes*, *Staphylococcus* species, *Salmonella* Choleraesuis, *Mycoplasma hyorhinis* and *Mycoplasma hyosynoviae* (Rowles 2001, Crenshaw 2006, Gomes Neto et al. 2012). *Trueperella pyogenes* has been found to be the most common bacterium in arthritis of sows, followed by haemolytic *Streptococcus* spp. and *Staphylococcus aureus* (Kirk et al. 2005).

The clinical signs of arthritis are an acute refusal to put weight on the leg, a swelling of the joint that is warm and painful when palpated, as well as fever and anorexia (Hill et al. 1986). Although clinical examination may indicate arthritis, specific diagnosis usually requires post-mortem examination and laboratory analyses including histopathology and bacterial culture.

6.1.3. Epidemiology and impact of arthritis on sow welfare and production

Arthritis may be a cause of severe lameness in sows. In a Canadian study, 22% (11/50) of the lame sows were diagnosed with arthritis after post-mortem examination, with arthritis being the second most important cause of lameness. Heinonen et al. (2006) diagnosed arthritis clinically in 8.8% (5/57) of the lame sows included in their study. In addition to the impact on welfare, arthritis can also lower herd performance and financial return. Arthritis has been reported to be the most important reason for euthanasia of sows (Kirk et al. 2005, Sanz et al. 2007, Engblom et al. 2008, Ala-Kurikka 2020) (Table 6.1.).

Table 6.1. The prevalence of primary diagnosis after post-mortem examination in euthanized sows/gilts related to the locomotory system. N= total number of euthanized sows/gilts examined.

		Osteo-chondrosis		Arthrosis		Fracture		Arthritis		Osteo-myelitis (vertebral column)		Osteo myelitis (other locations)		Abscess in spinal cord		Other *	
	N	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Kirk et al. 2005	172			15	8.7	27**	15.7	41	23.8	19	11	12	6.9			9	5.2
Sanz et al. 2007	49	3	6.1					18	36.7								
Engblom et al. 2008	79	13	16.4			10	12.6	35	44.3					7	8.8		
Ala-Kurikka 2020	38					4	10.5	6	15.8	2	5.3						

* claw lesions, rupture of ligament, etc.

** including 13 cases of epiphysiolysis

6.1.4. Treatment and prevention of arthritis in sows

Many infections are chronic by the time they are detected and therefore they respond poorly to treatment (Rowles 2001, Dewey 2006). Especially in case of severe lameness, euthanasia should considered as an alternative to treatment (Rowles 2001). Because infectious arthritis is mostly caused by bacteria, the use of antimicrobials at an early stage of the disease is recommended. If the causative bacterium has been isolated from the joints, the choice of antimicrobial can be based on the results of an antibiogram. If an antimicrobial therapy is chosen as the only intervention, even 7 days of systemic treatment will presumably not be sufficient (Ala-Kurikka 2020). Nevertheless, treatment courses to sows suffering from arthritis are often

shorter in practice. Besides an antimicrobial therapy, pain killers should be used for the welfare of the sow. Although the use of non-steroid anti-inflammatory drugs (NSAID) has been reported to be effective mainly in non-infectious locomotor disorders in pigs (Friton et al. 2003, Mustonen et al. 2010), their use may also be beneficial in case of infectious arthritis (Zoric et al. 2016). Wound management and joint lavage have been proposed as part of the treatment of infectious arthritis (Anderson and St Jean 2012). For valuable breeding stock animals, this can be considered but for most sows in commercial farms this is not financially viable.

6.2. Arthrosis in sows

6.2.1. Definition and diagnosis of arthrosis in sows

Arthrosis is a non-infectious degenerative joint disease. Secondary arthrosis, also called secondary degenerative joint disease, is a progression of OC, which in turn is a local disturbance of endochondral ossification and the most common disorder found in articular growth cartilage (Nakano and Aherne 1993, Palmer 1993). Secondary arthrosis develops in a late stage of OC and is characterized by erosion of articular cartilage with exposure and excessive mineralization of subchondral bone, calcification of the synovial membrane and formation of osteophytes (Crenshaw 2006). Non-OC-related arthrosis is called primary arthrosis (Palmer 1993). It is a degenerative joint disease, in which the articular cartilage surface is first affected. Primary arthrosis is characterized by fibrillation, surface tears and erosion of articular cartilage covering unaffected or eburnated subchondral bone (Nakano and Aherne 1993, Palmer 1993).

Diagnosis of non-inflammatory degenerative joint disorders is difficult, especially in a living sow (Heinonen et al. 2006). The bilaterally symmetrical presence of lesions can induce lameness shifting between limbs (Rowles 2001). Gait and posture variables have been found to reflect joint lesions. Turned out hind legs and a stiff movement in rear legs have been associated with osteochondrotic and arthrotic lesions in knee joints while forelegs that are turned out and a stiff movement of the front and rear legs are highly indicative for lesions in the elbow joint (Jørgensen 2000, Kirk et al. 2008). Nevertheless, in most cases post-mortem examination will be required to find the cartilage lesions.

6.2.2. Epidemiology of arthrosis in sows

Studies focusing on arthrosis in sows are rare and often do not distinguish between the two types of arthrosis. In addition, the terms OC and arthrosis are often used ambiguously. In literature, articular cartilage changes found in sows have been defined either as OC or arthrosis and in some cases both terms are combined. Also, the method of diagnosis has a great impact on the number of affected animals found in different studies. This makes it difficult to interpret prevalence figures.

Osteochondrotic lesions or arthrosis are quite prevalent post-mortem findings in sows (Table 6.2.). Both can be the primary reason for culling or euthanasia. In euthanized sows, arthrosis was found as a primary post-mortem diagnosis in 8% (Kirk et al. 2005) and OC in 14% (Engblom et al. 2008). Furthermore, they are an important secondary finding. Kirk et al. (2005) found arthrosis as a secondary diagnosis after postmortem examination in 88% of euthanized sows and 93% of sows that died.

Table 6.2. Prevalence (percentage) of osteochondrosis (OC) and arthrosis as primary or secondary diagnostic finding in culled/euthanized sows.

	Osteochondrosis	Arthrosis as primary diagnosis	Arthrosis as secondary diagnosis
Nakano and Aherne 1993[a]			100[*]
Dewey et al. 1993[b]	34[**]	12	8
Kirk et al. 2005[c]	49[***]	8	88
Sanz et al. 2007[d]	3		
Engblom et al. 2008[e]	13,5		

[a] 19 sows from one farm culled for impaired reproductive performance
[b] 50 sows from ten farms culled for lameness
[c] 172 sows from ten farms, reason for euthanasia not reported
[d] 107 sows from one farm euthanized for several reasons
[e] 96 sows from one herd euthanized for several reasons
[*] It was not reported whether arthrosis was the primary cause or only a secondary finding

** Primary cause for culling due to lameness. In 8% of all investigated lame sows, OC was found as a secondary lesion. In 94% of all examined sows, microscopic lesions of OC were seen.
*** out of the 35 sows that were histologically examined

Arthrosis is frequently bilateral and symmetrical and it is found in sows most often in the elbow and stifle joint (Grøndalen 1974, Jørgensen 2000, Ytrehus et al. 2007, Kirk et al. 2008). Grøndalen (1974) reported a high incidence also in the lumbar region. The medial condyle of the humerus, the anconeal process of the ulna and the medial condyle of the femur are usually the most severely affected areas (Grøndalen 1974, Jørgensen 2000, Ytrehus et al. 2007, Kirk et al. 2008).

Severe lesions in the stifle joint have been reported to be more common in animals less than 1.5 years old. The stifle joint has been suggested to be more resistant to severe lesions once the most active growth period is over. In contrast, Grøndalen (1974) found a frequent, severe degree of arthrosis in the elbow joint in sows of different ages ranging from 9 months to 4 years. Kirk et al. (2008) reported that the elbow joint was more affected in sows compared to the stifle joint.

6.2.3. Impact of arthrosis on sow welfare and production

Osteochondrosis/arthrosis can be the primary cause of lameness and subsequently a reason for early culling and euthanasia. Dewey et al. (1993) reported arthrosis to be the primary cause in 12% and OC in 34% of the lame sows in their study. Heinonen et al. (2006) diagnosed clinically OC/arthrosis in 4.3% of lame sows and gilts. In a study by Engblom et al. (2008), 62% of the sows diagnosed with OC, showed clinical lameness. However, the high prevalence of OC/arthrosis as a secondary diagnosis (8% - 93%) at post-mortem examination of sows also indicates that the presence of lesions not always has a clinical relevance. Severe degree of arthrosis rarely occurs in the stifle joint of pigs more than 1.5 years old, whereas severe lesions in the elbow joint are found in all age groups up to 4 years. This may suggest that injuries in the knee might cause severe discomfort to the animal, and consequently result in early culling or euthanasia while injuries in the elbow joint might cause less discomfort (Grøndalen 1974).

6.2.4. Predisposing factors of arthrosis in sows

OC has been suggested to be a multifactorial disease in which rapid growth, heredity, anatomic characteristics, trauma, nutrition and a defect in vascular supply to the epiphysial cartilage may play a role (Ytrehus et al. 2007). Favored by intensive selection, pigs today grow to a body weight of over 100 kg within only 5 to 6 months. This rapid weight gain has been suggested to impair the soundness of legs and claws (Sack 1982, Kroneman et al. 1993). However, high growth rates may not play a direct role in the aetiology of OC (Ytrehus et al. 2007). The pathogenesis of OC in finishing pigs is discussed earlier in this chapter. Pathogenesis of primary arthrosis in sows is still not well understood. Nakano and Aherne (1993) suggested a limited exercise and consequent restricted joint motion as a risk factor. With the transition to group-housing in Europe, it could be assumed that primary arthrosis in sows is rather rare today.

6.2.5. Treatment and prevention of arthrosis in sows

Arthrosis is a degenerative joint disease most often not responsive to treatment (Rowles 2001). Treatments, such as surgery, applied to horses and humans are often neither practical nor financially justifiable for pigs. For those cases that are accompanied by pain and discomfort, a symptomatic treatment with NSAID drugs is advised and the lame animal should be moved to a proper sick pen to recover until sent to slaughter.

Prevention of secondary arthrosis in sows requires the prevention of OC development starting from skeletally immature pigs (see this chapter earlier, prevention in finishing pigs). Although rapid growth, heredity, conformation, trauma and nutritional deficiencies have been suggested to play a role, only heredity and conformation have been well supported by scientific literature (Ytrehus et al. 2007, Belkova and Rozkot 2022).

6.3. Claw lesions in sows

6.3.1. Epidemiology of claw lesions in sows

Claw lesions are highly prevalent in sows and may affect almost every sow (Table 6.3.). Different types of claw lesions can be distinguished with bruising/haemorrhage, heel erosions and overgrowth, cracks at the heel-sole junction, white line cracks, horizontal and vertical wall cracks and overgrown claws and dewclaws

being most commonly identified and investigated (Brooks et al. 1977, Anil et al. 2005, Engblom 2008, Ossent 2010).

Table 6.3. The claw lesion prevalence/incidence (percentage) as found in different studies performed between 1993 and 2021, including information on the scoring method and time, housing conditions and sample size.

	Global	Claw region					Overgrown (dew) claws	Scoring in vivo (1) or post-mortem(2)	Reason for culling, euthanasia or dead	Scoring method	Housing during gestation	Time at scoring	Sample size
		Heel	Toe/sole	Heel-sole junction	Wall	White line							
Dewey et al. 1993	42[a]							2	Lameness	Gross examination	Not reported		50
Gjein and Larssen 1995b		50-66[b]	2-5	12-15	47-60	41-69		1		Hind feet, Visual, 5 point scale (1-5)	Group housing[c]		
Gjein and Larssen 1995b		30-40[d]	1-4	5-9	28-33	32-43		1		Hind feet, Visual, 5 point scale (1-5)	Confined		
Gjein and Larssen 1995b		77[e]	12	26	80	63		2		Hind feet, Visual, 5 point scale (1-5)	Group housing[c]		
Gjein and Larssen 1995b		53[f]	7	7	51	46		2		Hind feet, Visual, 5 point scale (1-5)	Confined		
Pluym et al. 2011	99	93			52		39	1		Hind feet, visual, 4 point scale (1-4)	Group-housing[g]	Early lactation	421
Kirk et al. 2005	5/>84[i]	74-84	75-77		44-49	63-65		2	Several	Scoring not reported (absence vs presence)	Both group and confined[h]		265
Knauer et al. 2007	86.4	32.9/67.5[j]		22.6/18.1[j]			3.5/21.1[j]	2	Several	Visual, 5 point scale and presence/absence	Not reported		3158

Anil et al. 2005, 2007	96.2[l]	86.4	45	66.3	88.6	60.9		1	Visual, 5 point scale (0-4)	Both group and confined[k]	After moving to farrowing crates	184
Kilbride 2008	70.7	6.5/17.5[m]	14.6/25.2[m]	14.1/33.3[m]	4/9[m]	0.3/5.0[m]	2.3/12.0[m]	1	Left hind feet, visual, 4 point scale (0-3)	Group housing[n]	In the farrowing crates	397 (gilts), 2005 (sows)
Calderón Díaz et al. 2013		24.8		8.6-11.3[o]	12.8-20.7[o]	9.8-10.7[o]	7.5-19.5[o]	1	Hind feet, Visual, sow chute, 4 point scale (0-3)	Group housing[p]	At insemination and between 50 and 70 days of gestation	133
Calderón Díaz et al. 2013		23.3 – 24[o]		32.3	16.5 – 23.3[o]	21.5 - 30.8[o]	16.5-29.7[o]	1	Hind feet, Visual, sow chute, 4 point scale (0-3)	Group housing[q]	At insemination and between 50 and 70 days of gestation	133
Pluym et al. 2011	99	93			52		38.5	1	Hind feet, visual, 4 point scale (1-4)	Group housing	In farrowing crate during lactation	421
Pluym et al. 2013		96		17	24	29	55	1	Hind feet, visual, 4 point scale (1-4)	Group housing	In farrowing crate during lactation	381
Sasaki et al. 2015	98.2	19.1 – 76.4[r]	0.9-4.5[r]	28.2-83.6[r]	1.8 - 3.6[r]	3.6-26.4[r]		1	Hind feet, visual, Five point scale (0-4)	Confined	In farrowing crate or gestation stall	110
Bos et al. 2016a	84.6/94.8[s]							1	Visual, sow chute, tagged analogue scale of 160mm	Group housing[t]	At d50 of gestation and in farrowing crate at the end	126

										of lactation	
Papadopoulos et al. 2021	62.5-96.5[u]	35.9-52.6[u]	21.9-87.7[u]	35.9-96.5[u]	31.2-82.5[u]	2	Several	Front feet, Visual, 3 point scale (0-2)	Group housing[v]		185
Papadopoulos et al. 2021	76.6-98.2[u]	60.9-83.6[u]	60.9-96.5[u]	59.4-100[u]	68.7-98.2[u]	2	Several	Hind feet, Visual, 3 point scale (0-2)	Group housing on fully slates concrete[v]		185

[a] diagnosed as « claw lesions » ; primary diagnosis in 20% of the lame sows, secondary diagnosis in 22% of the lame sows
[b] including heel cracks, erosion and bruising but excluding heel overgrowth (prevalence proportion of 30-51%)
[c] 15 herds on slatted floors, 3 herds with deep litter
[d] including heel cracks, erosion and bruising but excluding heel overgrowth (Prevalence proportion of 27-33%)
[e] including heel cracks, erosion and bruising but excluding heel overgrowth (Prevalence proportion of 45%)
[f] including heel cracks, erosion and bruising but excluding heel overgrowth (Prevalence proportion of 25%)
[g] Four herds with free-access stalls (stable groups) on partially slatted floors) ; 4 herds in dynamic groups with electronic sow feeder, one of these used deep straw bedding in the lying area).
[h] On 8 herds sows were loose housed, in two herds sows were tethered; in 5 herds deep bedding (straw) was used during gestation
[i] In 5% claw lesions were the primary diagnosis for euthanasia; claw lesions were found as a secondary diagnosis in at least 84% of all sows
[j] Frequency of claw lesions were scored for front and rear feet separately. In the table frequency is presented as front feet/rear feat.
[k] In this study 184 sows were included, 102 were housed in group pens with electronic sow feeders and others were housed in stalls during gestation
[l] In total 3.8% of all sows had no claw lesions, 9% of the sows in stalls had no claw lesions whereas there was no sow without claw lesions in group pens
[m] The percentage of each type of claw lesions is presented for gilts and sows separately: gilts/sows
[n] Group housing mainly on solid floors with deep bedding
[o] gilts were followed during two parities; variation in claw lesion prevalence is due to differences between both parities. The highest percentages are found during 2nd parity except for white line and wall lesions in sows housed on rubber slats.
[p] Group housing on slatted concrete floors
[q] Group housing on slatted rubber floors
[r] The lateral and medial claw of each hind limb was scored separately. The variation in prevalence is due to a difference in the percentage of claw lesions between the lateral and medial claws of the left and right hind limb. For all claw regions the lowest percentage was found on the medial claws.
[s] The prevalence of claw lesions was significantly higher at the end of lactation (compared to day 50 of gestation)
[t] Group housing with electronic sow feeder
[u] Claw lesion prevalence was measured on three different herds. Variation described is due to variation between herds.
[v] Group housing on fully slatted concrete

Interpreting the results of studies about the occurrence and severity of claw lesions should be done with caution, because the differing predisposing factors, the variety of scoring scales and the different time points of scoring, i.e. in vivo or post-mortem. Post-mortem examination enables more thorough assessment of the claws. However, sows are often euthanized or culled because of specific disorders or diseases, which may bias the results found on prevalence and severity of claw lesions.

6.3.2. Predisposing factors for claw lesions in sows

Claw lesions have been reported to be more prevalent and severe in group-housed compared to individually stalled sows (Gjein and Larssen 1995b Anil et al. 2005, Anil et al. 2007). The effect of housing differs according to the type of claw lesion considered. Calderón Díaz et al. (2014) found that group-housed sows had a reduced risk of white line lesions, horizontal wall cracks and dewclaw lesions but an increased risk of heel erosion and overgrowth. The degree and type of activity of the sow, particularly the ability to move around, the amount of aggressive interactions and the possibility to avoid confrontation can influence the type and severity of claw lesions. Fighting and aggressive behaviour at the time of mixing in order to establish a new hierarchical ranking (i.e. in case of dynamic groups) and at feeding time, especially in feeding systems associated with highly competitive situation at feeding, increase the risk for claw lesion development (Kroneman et al. 1993, Anil et al. 2005). Similarly, group-housing in pens with electronic sow feeders is associated with a higher proportion of claw lesions (Anil et al. 2007) and risk of leg injuries (Cador et al. 2014) compared to crate housing in which the crates can be accessed freely by the sows (Cador et al. 2014).

Knowledge about the predisposing factors for claw lesions in group-housed sows constitutes the basis for proper control and preventive measures. Development of claw lesions is determined by an interaction between internal (nutrition), external (housing conditions and management) and sow related factors (claw conformation, weight distribution, genetic line and parity). A disturbance in one or more of these factors may provoke trauma, inflammation and/or inferior horn quality, which can result in the development of claw lesions.

6.3.2.1. Claw conformation and weight distribution

Claw lesions are more prevalent and severe on the lateral compared to the medial claws, a difference that is more pronounced on the hind feet compared to the front feet (Gjein and Larssen 1995b, Anil et al. 2005, Anil et al. 2007, Knauer et al. 2007, Pluym et al. 2011, Lisgara et al. 2015b, Sasaki et al. 2015). Inequality in claw size may partly explain the difference in susceptibility to lesion development. Lateral claws are bigger compared to medial claws, possibly indicating that weight load, and as such mechanical impact of

locomotion, is higher on lateral claws of the rear feet (Kornegay et al. 1990, Gjein and Larssen 1995b, Bradley et al. 2009, van Amstel 2010, Sasaki et al. 2015, Papadopoulos et al. 2021). A total of 75% of the weight of the pig is born by the lateral claws (Webb 1984) and furthermore, claw size discrepancy increases as the sow ages (Bradley et al. 2009, Papadopoulos et al., 2021). The more pronounced the difference in claw size is, the more lesions are found in the area of the lateral claw (Kornegay et al. 1990).

In addition, abnormal claw and dewclaw growth may contribute to the susceptibility of claw lesion development. In case of overgrown dewclaws, an area of contact with the ground may appear and influence the distribution of the weight bearing force applied to the sow's feet, finally affecting its walking pattern (Papadopoulos et al. 2021). Overgrowth of the claws in general may influence the weight distribution pattern between and within claws. In addition, claw overgrowth has been associated with a decline in mechanical efficiency, expressed as Young's modulus, a measure of horn stiffness, which in turn has also been associated with an increased prevalence of wall lesions (Chalvatzi et al. 2021). Overgrown claws are associated with a greater lesion score for almost all types of claw lesions (Calderón Díaz et al. 2015, Sasaki et al. 2015, Papadopoulos et al. 2021). It also predisposes the sow to lameness development (Jørgensen 2000) and can even impair sow performance (Fitzgerald et al. 2012). Besides a genetic predisposition (Quintanilla et al. 2006, Fernàndez de sevilla et al. 2009a), claw overgrowth may naturally occur over time as horn growth may exceed the rate of wear, even if sows are housed on concrete flooring (Geyer 1979, Van Amstel 2010). As the sow ages, an increase in toe length and prevalence of claw overgrowth has been reported in several studies (Dewey et al. 1993, Bradley et al. 2009, Fernàndez de sevilla et al. 2009a, Pluym et al. 2011, Lisgara et al. 2015b).

6.3.2.2. Genetic lines

Sows and boars from specific genetic lines may be more prone to claw lesions. Kilbride (2008) reported that pigmented breeds such as Duroc, Pietrain and Hampshire are less likely to develop claw lesions, probably because of tougher claw horn. The prevalence of uneven hooves was significantly higher in Landrace boars compared to Yorkshire and Duroc ones according to a report of Jørgensen and Vestergaard (1990). In a study

of Fernàndez de Sevilla (2009a), abnormal claw horn growth tended to affect Landrace breed more severely than that of the Large White. The prevalence of abnormal claw horn growth at the end of the growing period was 36.5% and 28.2% for Landrace and Large White breeds, respectively. Landrace boars showed a higher likelihood of heel overgrowth and erosion, heel-sole cracks and horizontal wall cracks but had a lower likelihood of overgrown claws compared to Duroc boars in a study by Wang et al. (2018). In the same study, the probability of horizontal wall cracks, heel-sole cracks and dewclaw abnormalities was higher in Yorkshire than in Duroc Boars. Papadopoulos et al. (2021) investigated several claw and dewclaw length measurements and their associations with claw lesions among sows and found variation among the genetic lines studied (i.e. Pig Improvement Company (PIC), Danbred and Topigs). Mechanical efficiency, expressed as the Young's modulus, a measure of stiffness, varied as well among those breeds with the highest values found in Topigs, followed by Danbred and PIC, in decreasing order (Chalvatzi et al. 2021).

This variation among genetic lines should be interpreted with caution. Other factors may influence claw integrity and development of claw lesions, which may have differed between the above-mentioned studies.

6.3.2.3. Parity

The odds of having white line lesions were significantly higher in sows with less than five parities compared to the older ones (Anil et al. 2007). In the same study, white line lesions were also associated with lameness. The association between white line lesions and young sows might be because older sows with white line lesions had already been culled. Lisgara et al. (2015b) found heel lesions and overgrown claws and dewclaws to be more likely seen in sows with more than three parities. These sows have a high body weight, increasing the pressure on the claws per unit area (Anil et al. 2007, Knauer et al., 2007) and exhibit a higher claw size discrepancy, leading to an increase in weight bearing properties of the lateral claws. The highest load within the lateral claw found at the level of heel (Webb, 1984) might explain the increase in heel lesions as the sows age. A parity effect for claw measurements and claw overgrowth was reported earlier (Dewey et al. 1993, Bradley et al. 2009, Fernàndez de sevilla et al. 2009a, Pluym et al. 2011). Claw horn growth and wear in sows have been estimated to be 6.3 mm and 5.1 mm per month, respectively (Van Amstel, 2010). Although horn

growth rate has been suggested to diminish with age, the disbalance between horn growth and wear rate may indicate that claw and dewclaw overgrowth is to be expected with increasing age. In contrast, Anil et al. (2005) and Kilbride (2008) could not find any association between claw lesions and parity. As horn tissue in pigs grows faster than that of other animals, abnormal claw horn growth may develop faster but claw lesions, especially mild ones, may heal more quickly and therefore may not be carried over to subsequent parities (Geyer 1979). In addition, claw trimming may contribute to improved claw lesion score in successive parities (Pluym et al. 2013). Moreover, the different findings for the association between parity and claw lesion prevalence may be biased by the culling management in the herds included in the different studies.

6.3.2.4. Flooring

Three main floor types are used in indoor housing: solid, partially slatted and fully slatted floors. A different amount of bedding can be used on partially slatted and solid concrete floors.

With respect to the risk for claw lesion development, solid concrete floors with deep straw bedding were reported to be the best and solid floors without bedding the worst (Jørgensen 2003, Cador et al. 2014). Although floors covered with deep bedding seem to be preferred, claw lesion development is not entirely prevented (Jørgensen 2003, Cador et al. 2014). Deep bedding reduces the risk of overburdening the claw giving rise to less heel lesions (Kilbride 2008). Toe erosions, overgrown claws and heel-sole cracks, however, may be more prevalent on straw bedding, likely due to the lack of natural wearing (Kilbride 2008, Cador et al. 2014). Rubber mats can be used as an alternative to deep litter. The benefits in terms of claw lesion development are threefold. First of all, as on deep litter, the impact load on claws is lower as the area of contact between the claw and the floor on mats is larger and pressure distribution is improved (Carvalho et al. 2009). Secondly, the cushioning effect of rubber mats may improve blood circulation in the dermis of the claw, essential for claw horn integrity. Lastly, textured rubber mats may provide more traction and reduce slipping (Boyle et al. 2000). The latter, however, only applies if the mats are not soiled, otherwise the number of claw lesions may even increase (Calderón Díaz et al. 2013). Rubber mats may be associated with

insufficient wear of claw horn resulting in a higher prevalence of overgrown claws and heel-sole cracks (Calderón Díaz et al. 2013).

On slatted floors, the more limited contact area between the claw and the floor increase the pressure applied per unit area of the claw. Mainly the heel bulb will be affected as this region of the claw is subject to the highest load. Heel flap development is associated with bruising and haemorrhage in the soft heel. Kilbride (2008) reported an increased risk for heel flaps on slatted floors compared to solid floors with deep bedding. The impact of slatted floors on the risk for claw lesions (particularly wall cracks) may be higher for heavier sows as the pressure per unit area of the claw is greater (Anil et al. 2007, Knauer et al. 2007, Kilbride 2008, Calderón Díaz et al. 2014). The effect of slatted floors on claw lesion development has been found both during gestation and lactation (Calderón Díaz et al. 2014). Webb (1984) defined an equation, based on the pig's live weight, to derive the maximum safe void percentage, i.e. the ratio of area of holes per unit area of floor to unit area. For sows, this would suggest a maximum void percentage of 50%, above which a detrimental impact on the health of the heel region occurs, increasing the risk for heel overgrowth and erosions.

On solid floors, the pressure on the claws per unit area is lower and claws and dewclaws cannot get stuck and tear off. However, urine and faeces may stay on the solid floor making the floor more slippery, the claw horn weaker (Sobestiansky et al. 1999, Gregory 2004) and increase the risk for infections (Gjein and Larssen, 1995a).

Although claw lesion development in outdoor housed sows could be expected to be low, Kilbride (2008) reported a high prevalence (57%) of claw lesions in outdoor housed sows and gilts, which was similar to that of the sows housed indoors (68%). Wet conditions and stony paddocks were suggested as possible reasons for this finding.

The influence of the material on claw lesion development is not clear due to limited investigations. Calderón Díaz et al. (2014) reported a higher risk of heel overgrowth/erosion and heel-sole cracks when sows were housed in the farrowing crates on slatted steel compared to cast iron. The higher void ratios and the more abrasive properties of steel slats compared to cast iron were suggested as possible reasons for the difference.

An old study investigated the effect of steel, plastic, concrete and aluminium slats on the occurrence of wall cracks and foot pad lesions in finishing pigs and found out that the results could be extrapolated to breeding pigs. Pigs housed on concrete slats were demonstrated to have the best foot pad condition score (i.e. the percentage of the volar surface affected by lesions) but showed the highest prevalence of wall cracks (Newton et al. 1980).

Four main quality factors contribute to a floor's total injury potential with respect to claw lesion development: slipperiness, abrasiveness, hardness and roughness. Slippery floors and slipping has been associated with an increased risk of claw lesions (Kroneman et al. 1993, Kilbride 2008). The higher risk of hoof cracks on concrete slats compared to plastic or aluminium slats (Newton et al. 1980) and on steel slats compared to cast iron (Calderón Díaz et al. 2014) suggest wall cracks to be associated with abrasive floors (Calderón Díaz et al. 2014). Due to extensive wearing, the lateral wall horn becomes thinner and straightened and this mechanically worn wall horn might be more susceptible to splitting. A minimum of abrasiveness, however, is necessary to prevent hoof overgrowth. On soft surfaces, e.g. soil or rubber mats, the area of contact between the claw and the floor is higher, thus resulting in reducing overloading of the claws, which might in turn prevent claw lesion development (Carvalho et al. 2009). Rough concrete, worn or uneven slats and sharp protruding objects may also impair claw health. An increased risk of toe erosions has been associated with sharp slat edges and sharp protrusions at joints between surfaces on the farrowing pen floor (Kilbride 2008). De Belie (1997) found that concrete slats could already show degradation within five years giving rise to increased surface roughness, enlarged gaps and animal injuries.

Hygiene is without doubt of utmost importance. Dirty and wet floors can reduce claw horn strength making claws more vulnerable to damage (Sobestiansky et al. 1999, Gregory 2004). Toe erosions in lactating sows (Kilbride 2008) and heel overgrowth, considered as a chronic reaction, (Gjein and Larssen 1995b) have been associated with bad floor hygiene. The white line is the weakest part of the claw horn capsule (Tomlinson et al. 2004). Poor floor hygiene results in the sow to stand in faeces and urine, which may manifest damage to this vulnerable claw area (Cador et al. 2014, Calderón Díaz et al. 2014). Dirty and wet floors will also increase

the risk for claw infections. Sows with severe claw lesions seem to be more susceptible to claw infections compared to sows with minor claw lesions (Gjein and Larssen 1995a). If floors are covered with faeces and urine, the relative risk for claw lesions to be infected is quadrupled (Gjein and Larssen 1995a). Hygiene is also important when using deep litter or rubber mats. Damp straw, soiled with faeces and urine will soften the hooves and make them more prone to lesion development and infection. On rubber mats, poor hygiene will additionally increase slipperiness. On soiled mats, the number of claw lesions may even increase (Calderón Díaz et al. 2013).

6.3.2.5. Nutrition

Susceptibility to claw lesions also depends on claw horn strength, which is associated with multiple factors including genetics, housing conditions and nutrition (Torrison 2010). Functional integrity of claw horn highly depends on an appropriate supply of nutrients such as proteins, sulphur-containing amino acids (Cysteïne), hormones and, considered to be the main factors, vitamins (A, D, E and biotin) and minerals (Zn, Cu, Mn, Se, Ca, Cr, I, Mo) (Tomlinson et al. 2004, van Riet et al., 2013). Deficiencies may occur because of inadequate vascular supply or insufficient availability due to reduced dietary intake or low bioavailability. Late gestation and lactation are associated with a high nutritional demand, especially in high producing or young (growing) sows and gilts. Any reduction in nutrient supply therefore may result in compromised claw horn quality (Anil 2011, Pluym et al. 2011).

Bioavailability is especially of importance to trace minerals. It has been reported that bioavailability of organic trace minerals (i.e. a mineral joined with e.g. a protein or an amino acid) is higher compared to inorganic trace minerals, and this may be even increased if a combination of organic trace minerals is used in the diet (Anil 2011, van Riet et al. 2013). It has been shown that partly replacing inorganic by organic trace minerals (Zn, Cu, Mn) in the diet could decrease both claw lesion incidence and severity and more specifically severity of heel, sole (Lisgara et al. 2016) and wall lesions (Anil 2011, Lisgara et al., 2016). Yet, the effect was less pronounced on the rear claws and lateral heel bulb (Lisgara et al., 2016). Trace minerals do not seem to affect claw and dewclaw overgrowth (Lisgara et al. 2016). The extracellular matrix secreted in the end phase of the

horn production process is lipid rich and a major contributor to the mechanical strength of claw horn (Tomlinson et al. 2004). The impact of fatty acids, which can be influenced by the diet, on claw integrity in sows, however, remains unclear (van Riet et al. 2013).

Sows with a large body weight have an increased risk to develop especially heel lesions probably because the pressure per unit area is higher (Anil et al. 2007, Knauer et al. 2007). In contrast, Kilbride (2008) found a trend for an increased prevalence of claw lesions in sows with a low body condition score. Nutritional deficiencies may have increased the risk of claw lesions or the discomfort related to the claw lesions may have led to weight loss.

6.3.3. Impact of claw lesions on sow welfare and production

The prevalence of claw lesions and the extent to which they are associated with lameness and sow performance varies considerably in published literature including both sow and farm-specific parameters.

Although claw lesions have been considered an important cause of lameness in sows (Dewey et al. 1993, Heinonen et al. 2006), in most studies the association between lameness and claw lesions remains restricted to only a few types of claw lesions. Lameness has been associated with heel lesions (Cador et al. 2014), overgrown claws (Lisgara et al. 2015b), dewclaw lesions (Pluym et al. 2011, Cador et al. 2014, Lisgara et al. 2015b) and white line lesions (Anil et al. 2007, Lisgara et al. 2015b). Overgrown (dew)claws can get stuck between slats, crack or even tear off (Pluym et al. 2011, Cador et al. 2014) whereas severe white line lesions may be a predilection site for bacterial invasion especially on dirty wet floors (Cador et al. 2014, Calderón Díaz et al. 2014). Some studies have found no association between lameness and claw lesions (Gjein and Larssen 1995a, Calderón Díaz et al. 2014). Claw infections, on the other hand, are clearly considered a major cause and risk for lameness in sows (Dewey et al. 1993, Gjein and Larssen, 1995).

Claw lesions have also a detrimental effect on sow herd performance. They were one of the primary reasons for euthanasia or premature removal from the farm in several studies (Kirk et al. 2005, Engblom et al. 2008,

Pluym et al. 2013). Abnormal hoof growth has been shown to affect sow longevity in Duroc, Landrace and Large White sows (Fernàndez de Sevilla et al. 2008 and 2009b).

Combinations of claw lesions may negatively affect reproduction parameters such as the number of live-born piglets (Lisgara et al. 2015), weaned piglets (Fitzgerald et al. 2012, Lisgara et al. 2015), adjusted litter body weight at weaning (Fitzgerald et al. 2012), wean-to-first service interval (Lisgara et al. 2015) and increase the odds of having stillborn pigs (Pluym et al. 2013).

6.3.4. Treatment of claw lesions in sows

The first questions to be asked are whether the animal warrants treatment, whether it can be cured, or whether culling or euthanasia is preferred (Rowles, 2001).

The dermis is a well-drained tissue in the claw that contains various nerve fibres (Tomlinson et al. 2004). Severe lesions might induce pain when they affect the dermis or when they have been an entrance for infection and inflammation of the tissues have occurred. Treatment with a NSAID is needed, if observed claw lesions are suspected to be detrimental to the welfare of the sow because of physical reduced mobility, pain and general discomfort. Use of NSAID can be effective for pain mitigation in lame sows (Friton et al. 2003, Pairis Garcia et al. 2014, Conte et al. 2015).

Infected claw lesions are often detected in the chronic stage and prognosis is poor. Treatment is also challenging because of the difficulty in ensuring adequate local treatment. Ideally, infected lesions should be cleaned, debrided and disinfected followed by an antimicrobial (topical and general) and NSAID therapy (Martineau and Morvan 2010). Penicillin is commonly recommended (Rowles 2001). In non-responsive cases, amputation of the claw has been suggested (Anderson and St. Jean 2012), but in practice this procedure is seldom carried out.

Supplementing complexed trace minerals (Zn, Mn, Cu) has been reported to contribute to the heeling of claw lesions, especially for heel and wall lesions, among group housed sows (Anil 2011).

Claw trimming in case of digital overgrowth is necessary as overgrown claws have been associated with lameness (Jørgensen 2000), an increased prevalence of almost all types of claw lesions (Papadopoulos et al. 2021) and a negative impact on sow performance (Fitzgerald et al. 2012). Moreover, long dewclaws can be stuck in the slots of slatted floors and may be ripped off, which is very painful for the animal (Pluym et al. 2011, Cador et al. 2014). Claw trimming has also been reported as part of the treatment strategy of pododermatitis (Martineau and Morvan, 2010).

6.3.5. Prevention of claw lesions in sows

Because treatment of claw-related lameness is frequently unrewarding, there is merit in working towards prevention and management. Factors affecting the prevalence and severity of claw lesions and infections are related to claw conformation, housing, nutrition and management. They are the key to reduce claw lesions and related lameness.

Claw size discrepancy and claw overgrowth are both hereditary. Heritability values vary from 0.01 to 0.48 for uneven claws and from 0.24 to 0.38 for claw overgrowth, depending on breed and statistical method used (Quintanilla et al. 2006, Fan et al. 2009, Fernàndez de sevilla et al. 2009a). Given the low to moderate heritability values and the fact that conformation traits such as uneven toes are highly polygenic, genomic selection may benefit progress in conformation traits. Standardization in methods for phenotyping conformation traits, identifying genetic correlations with economically important traits and taking into account that conformation may evolve over a sow's reproductive life (Fernàndez de sevilla et al. 2008, Stock et al. 2018) are essential actions in achieving the right (genetic) strategy (Trenhaile Grannemann 2021).

It is impossible to define an ideal floor (Kroneman et al., 1993). Group housing on deep straw litter, appeared to be the best solution to reduce the incidence and severity of claw lesions (Jørgensen 2003, Cador et al., 2014) although toe erosion and claw overgrowth may still be frequent (Kilbride 2008). The use of a non-bedded dunging area may help to compensate the lack of natural wearing on the straw-bedded lying area. Use of a pitch of the solid floor towards the dunging area may help to drain urine. To keep the dunging area as dry and clean as possible, a slatted floor or scraper might be used (Altena et al. 2004). However,

maintaining a dry and clean floor is a challenge on solid floors. Even outdoor housing does not appear to be the ideal environment to minimize the risk of claw lesions. Wet conditions and stony paddocks may be risk factors in outdoor housing (Kilbride 2008).

Clean and dry floors moreover increase claw cleanliness, which improves (early) detection of claw lesions (van Riet et al. 2020) and consequently allows measures to be taken at an early stage preventing claw lesions from developing into more severe lesions, lameness and even premature culling.

Good-quality flooring, without protruding sharp objects, neither rough nor slippery and without crumbled slat edges and enlarged gap width, may help to reduce the development of claw lesions (Kroneman et al. 1993). In slatted floors, also the void ratio is important. For sows, a maximum void percentage of 50% could be suggested based on the equation of Webb (1984). A number of suggestions related to sledge design and maximum slot width have been made in studies conducted almost forty years ago, and we lack recent investigations.

The use of barriers may reduce aggression in group-housed sows (Arey and Edwards 1998). As the amount of aggressive interactions and the possibility to avoid confrontation, can influence the type and severity of claw lesions (Kroneman et al. 1993, Anil et al. 2005), the use of barriers and increasing lying comfort may help to prevent claw lesion development.

A sufficient dietary supply of bioavailable nutrients, especially during periods of high demand or low dietary intake such as late gestation and lactation, is an important element in the prevention of claw lesions and consequently sow lameness (van Riet et al. 2013). Supplementing complexed trace minerals (Zn, Mn, Cu) can contribute to both the prevention and the healing of claw lesions, especially for heel and wall lesions (Anil 2011).

Claw trimming is an essential part of the treatment and prevention of claw lesions and associated lameness. Claw trimming can improve locomotion by inducing more comfort in gait as well as stability of the sow by reducing the risk for slipping (Tinkle et al. 2017, Tinkle et al. 2020). It may also contribute to improved claw lesion score in successive parities (Pluym et al. 2013). Functional claw trimming is recommended (i.e.

trimming and shaping the full claw, balancing the sole, re-establishing even weight distribution within and between claws) although high labour inputs, training and special equipment are needed. If functional trimming is not feasible, trimming the dewclaws may be a valuable alternative. By reducing the length of dewclaws, interference of the heel contact by the dewclaws is avoided, making stride faster and reducing the chance of dewclaws being caught, torn or broken. Trimming too short and drawing blood should be avoided at all times. Trimming by only cutting the toe has not been recommend (Tinkle et al. 2020).

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