Swedish University of Agricultural Sciences
Faculty of Veterinary Medicine and Animal Science

Prevalence of congenital defects in Swedish Hampshire, Landrace and Yorkshire pig breeds and opinions on their prevalence in Swedish commercial herds

Petra Mattsson

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Förekomst av medfödda missbildningar hos grisraserna hampshire, lantras och yorkshire och åsikter om förekomsten av missbildningar i svenska bruksbesättningar

Petra Mattsson

Supervisor:
Nils Lundeheim, SLU, Department of Animal Breeding and Genetics

Examiner:
Lotta Rydhmer, SLU, Department of Animal Breeding and Genetics

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Table of content

1. SAMMANFATTNING

Navelbråck, pungbråck, kryptorchism, knipta ryggar, fläkta grisar, sluten ändtarm, tvekönthet och skakande grisar är missbildningar som förekommer bland grisar. Syftet med denna studie är att beskriva och skatta förekomsten av dessa missbildningar samt att skatta arvbarheten för pungbråck och kryptorchism i renrasig svensk hampshire, lantras och yorkshire. Information och åsikter gällande missbildningar i bruksbesättningar har även samlats in.

En låg andel missbildade grisar är viktigt både ur ett djurvälfärdsperspektiv och ur ett ekonomiskt perspektiv. Noggrann registrering av förekomsten av defekta grisar i kullarna har en avgörande roll för att kunna selektera mot defekter. Kopplat till detta är även frågan om defekter nedärvs.

Under sommaren och hösten 2008 analyserades data från Nordic Genetics databas med registrerade defekter under perioden januari 2000 till oktober 2007. Data från 15 renrasiga besättningar, med minst 400 producerade renrasiga kullar under perioden, med totalt 19 308 kullar inkluderades i analysen. Effekter av ras, besättning, säsong, kullnummer och kullstorlek analyserades statistiskt. Galtarnas effekt på förekomsten av defekter, som far eller morfar i kullarna, studerades.

Resultatet visar att den registrerade förekomsten av defekter varierade mellan de renrasiga besättningarna. Medelförekomsten av kullar med minst en defekt gris var 7,0 % för hampshirekullarna, 12,0% för lantraskullarna och 16,8 % för yorkshirekullarna. Kryptorchism var den vanligaste defekten i hampshire (4,3 %) och yorkshire (8,3 %), pungbråck var vanligast i lantras (6,5 %). Det fanns signifikanta effekter av ras och besättning och signifikanta samspel mellan ras och år för förekomsten av pungbråck och kryptorchism. Förekomsten av pungbråck och kryptorchism varierade beroende på kullnummer och i vilken månad kullen var född. När kullstorleken ökade, ökade även andelen kullar med defekta grisar. För lantras- och yorkshiregaltar fanns en positiv korrelation mellan förekomsten av kryptorchism i kullen när galten varit far och morfar, 0,4 för lantrasgaltar och 0,3 för yorkshiregaltar. Hos yorkshiregaltar fanns en positiv korrelation även för pungbråck, 0,3. De skattade arvbarheterna var generellt sett låga (<0,1).

17 bruksbesättningar med erkänt hög kunskapsnivå besöktes och lantbrukarna intervjuades under augusti och september 2008. Intervjuerna från bruksbesättningarna sammanställdes och förekomsten av defekter hos de levande födda grisarna beräknades. I besättningarna uppgav man att man såg de allra flesta defekta grisarna vid grisning, tandslipning och kastrering. Grisar med navelbråck och knipta ryggar sågs vid senare ålder. Fläkta grisar beräknades vara den vanligast förekommande defekten (4 % av alla levande födda grisar) näst följt av navelbråck (2 % av alla tillväxtgrisar). Navelbråck beskrevs som den mest besvärliga defekten beroende på försämrad tillväxt, problem att leverera till slaktgrisuppfödare eller slakteri och framförallt på grund av försämrad djurvälfärd för dessa grisar. I bruksbesättningarna upplevde man att förekomsten av navelbråck hade ökat de senaste två åren. Defekter i navelregionen är komplexa och behöver studeras mer.

I Norge har avelsorganisationen Norsvin inkluderat information om navelbråck, pungbråck och kryptorchism i avelsprogrammet för norsk lantras och duroc. Den stora variationen i förekomsten av defekter i de svenska renrasiga besättningarna visar på att det troligen finns skillnader i noggrannhet vid registreringen av dessa defekter. Om fler besättningar registrerade förekomsten av defekter kunde de viktigaste defekterna inkluderas i avelsprogrammet som hälsoegenskaper. Inför framtiden finns hoppet att selektion mot missbildningar kan ske med hjälp av genetiska markörer.

2. ABSTRACT

This study concerns congenital defects in pigs; umbilical and scrotal hernia, cryptorchidism, kyphosis, splay legs, anal atresia, intersexuality and congenital tremor. The aim was to describe and estimate the prevalence of these defects and estimate the heritability for the most frequent defects, scrotal hernia and cryptorchidism in purebred Swedish Hampshire, Landrace and Yorkshire populations. Furthermore, information and opinions from herdsmen in commercial herds was collected and analysed. A low proportion of congenital defect pigs is of importance both from animal welfare and economic point of view. The accuracy of registration of the presence of defects in the litters has a decisive role for the ability to select against these defects. Linked with that is also the question to what degree these defects have a hereditary background.

During summer and autumn 2008, data from Nordic Genetics' database of recorded defects for purebred litters born in the period January 2000 to October 2007 was analysed. Data from 15 purebred herds, 5 from each breed, with at least 400 purebred litters, with in total 19 308 litters were included in the analysis. Two statistical models were applied to the data. One included fixed effects of breed, herd, season and parity number and the other was expanded with the effect of litter size. The effect of the boars on the prevalence of scrotal hernia and cryptorchidism, as fathers or maternal grandfathers of the litters, was analysed.

The results show a variation of mean prevalence of congenital defects in purebred litters. The mean prevalence of any defect in litters was 7.0% in Hampshire, 12.0% in Landrace and 16.8% in Yorkshire. Cryptorchidism was the most common defect in Hampshire (4.3%) and Yorkshire (8.3%), and in Landrace (6.5%) scrotal hernia was most common. Significant effects of breed and herd and significant interactions between breed and year was found for scrotal hernia and cryptorchidism. Prevalence of scrotal hernia and cryptorchidism varied with parity number and month of birth. Prevalence increased with increasing litter size.

For Landrace and Yorkshire boars, positive correlations were found between the prevalence of cryptorchidism in the litters when the boar was used as father and maternal grandfather, 0.4 for Landrace and 0.3 for Yorkshire. For Yorkshire there were positive correlations also for scrotal hernia, 0.3. Estimated heritabilities were generally low (<0.1).

17 commercial herds with known high level of skill were visited and the herdsmen were interviewed during August and September 2008. The data were summarized and estimations of prevalence of defects among live born piglets were done. The commercial herds did all report that they see most of the defect piglets during farrowing, at teeth grinding and castration. Umbilical hernias and pigs with kyphosis were found later in life. Most common defect was splay legs, with a prevalence of 4% of all piglets born alive. Umbilical hernia was found in about 2% of all growers. Umbilical hernias were pointed out to be a troublesome defect due to decreased growth, problems to deliver pigs with the defect and reduced animal welfare. An increase of umbilical hernias had been seen during the last two years. Defects in the umbilical region are complex and need to be investigated more.

In Norway defects have been included as health traits in the breeding program for Norwegian Landrace and Duroc. The large variation of prevalence of defects in the analysed Swedish purebred herds, within breeds, indicate a variation in accuracy of recording the defects. If more herds did record the prevalence of defects, the most important defects could be included in the breeding program. For the future, it might be possible to select against defects using genetic markers.

3. INTRODUCTION

A low proportion of congenital defects among piglets is a general aim in pig production, both from animal welfare and economic point of view. Some malformed piglets die before birth, but most are born alive. The question for the herdsmen is whether these piglets should be euthanized immediately after birth, if they should be raised without any measures, or if some correction measures should be done.

Swedish pig production can be divided in specialized production, farrow-to-finish production and sow pools. Farrow-to-finish production is characterized by raising fattening pigs at the farm where they are born. Specialized production is characterized by farms with sows raising growers and selling 25 kg's growers to specialized fattening pig producers. Specialized farms with contracts between piglet producing and fattening pig herds are considered to be external farrow-to-finish. In sow pools, pregnant sows are rented from a central unit to contracted satellite herds, and the sows are, after farrowing and weaning, sent back to the central unit for next reproduction cycle.

Most pig breeding programs have a hierarchical structure, and the selection is performed at the top of the pyramid. The accuracy of registration of the presence of defects in the litters has a decisive role for the ability to select against these defects. Linked with that is also the question to what degree these defects have a hereditary background. Purebred and multiplier herds contracted to the Swedish breeding company Nordic Genetics (NG) have to register defects which are observed in the litters, in purebred as well as in crossbred litters. Recorded congenital defects in the Swedish pig breeding are: umbilical and scrotal hernia, cryptorchidism, kyphosis/humpy back, splay legs, anal atresia, intersexuality, congenital trembling syndrome and miscellaneous.

The aims of this study were to:

- Review literature on congenital defects observed among piglets.
- Describe and estimate the prevalence and causes of variation of defects within the Swedish Hampshire, Landrace and Yorkshire breeds.
- Describe and analyze the variation between boars on the prevalence of the most common congenital defects, cryptorchidism and scrotal hernia in Swedish Hampshire, Landrace and Yorkshire.
- Estimate heritability for the most frequent congenital defects.
- Collect information and opinions about the prevalence of, and the herdsmen's attitude to, congenital defects in commercial pig production.

4. LITERATURE REVIEW

Prevalence of defects are presented in different ways in different studies. Some studies express the prevalence as number or percentage of total number born pigs or number born alive, others as percentage of litters with the defect. Cryptorchidism and scrotal hernia are defects found only in male pigs and the prevalence are in some studies expressed as percentage of all pigs born and in some studies as percentage of male pigs born.
Some defects included in this study are illustrated in fig 1.

Congenital Defects

Hernia

Most common hernias in pigs are hernias involving the abdomen (Warwick, 1928; Ingwersen, 1998). A protrusion of part of the intestines or any other internal organ through an opening of the body wall is defined as hernia. Hernias may be observed on the newborn piglet or in some cases the defect appears later in life. The mode of inheritance of hernias is not well known.

Umbilical hernia

During foetal life, the foetus is connected via the umbilical cord to the placenta. The umbilical cord breaks after birth and the umbilical ring normally closes. If the ring remains open or closes incompletely, a risk for umbilical hernia occurs (Warwick, 1928; Ingwersen, 1998). The size of the hernia depends on the size of the umbilical defect and the amount of internal organs that has passed through the umbilical ring. The diagnosis of hernia is complex. Ingwersen (1998) suggest that a genuine umbilical hernia is reducible. If the hernia is irreducible there is a potential for an umbilical abscess being present. In cattle and swine, umbilical hernias and umbilical abscesses are often seen together. In calves some success in reducing the prevalence of the defect has been made by bandaging the navel for 3-4 weeks after birth (Ingwersen, 1998).

In pigs, most umbilical hernias are diagnosed during week 7-14 in life (Searcy-Bernal & Gardner, 1994; Rutten-Ramos & Deen, 2006). Pigs with the defect have a lower daily weight gain compared to non affected pigs (Straw *et al*., 2008).

Inguinal and scrotal hernia

An inguinal hernia is defined by hernial content in the inguinal canal; scrotal hernia is defined as abdominal organs in *scrotum* (Warwick, 1926; Grindflek *et al*., 2006). Scrotal hernia is a congenital sex-linked defect and originates from a failed closure of the vaginal ring during *processus vaginalis*. Scrotal hernia can be unilateral or bilateral. Unilateral scrotal hernias on the left side are more common than on the right side (Warwick, 1926; Magee, 1951). Most scrotal hernias are diagnosed at the time of castration. The defect is often diagnosed by detecting an enlarged scrotum early in life (Ingwersen, 1998).

Cryptorchidism

Cryptorchidism is a congenital sex-linked defect (Rothschild *et al*., 1988) characterized by one or both testicles failure to descend into the scrotum from the abdomen via the inguinal canal. Lush and Jones (1930) reviewed the prevalence of cryptorchidism in different species. The defect is more common among swine and horses than in cattle, sheep and goats (Amann & Veeramachaneni, 2007). Cryptorchid testicles show symptoms of heat degeneration (Lush & Jones, 1930) but males with the defect can be fertile (Sjaastad *et al*., 2003). Warwick (1926) found that unilateral cryptorchidism on the left side is most common in pigs.

Kyphosis/Humpy back

Pigs with kyphosis are often referred to as “humpy-back”, “kinky back” or “dipped shoulder” (Holl *et al*., 2008). The defect originates from a deformation in the hemi vertebrae (Nielsen *et al*., 2005). Among pigs, the defect is in most cases diagnosed at the age of 3-16 weeks (Holl *et al*., 2008; Straw *et al*., 2008). Bradley (2005) scored 1000 live purebred and hybrid Swedish sows for back conformation and concludes that the prevalence of kyphosis among sows is low.

Splay legs

Congenital splay legs are also referred to as congenital myofibrillar hypoplasia (Jirmanová, 1982). The defect is a result of muscular weakness due to a developmental disorder of skeletal muscles, most common in the hind limbs. The defect will limit the newborn piglet’s ability for suckling, increase the risk to be crushed by the sow (Sellier *et al*., 1998) and increase piglet mortality during the suckling period (Holl & Jonsson, 2005). The occurrence of the defect is considered to be an effect of the sow. Short gestation length increases the frequency of piglets born with splay legs (Van der Heyde *et al*., 1988; Sellier *et al*., 1998). There is a variation in gestation length between breeds, 114.1-115.5 days of gestation (Omtvedt *et al*., 1965; Van der Heyde *et al*., 1988; Sellier *et al*., 1998). Holl & Johnson (2005) found that incidence of splay legs was 226% higher in male piglets, compared to female piglets. They also found that low birth weight and larger litters did increase the prevalence of the defect. Also, the floor in the farrowing pen was of importance for the occurrence of splay legs.

Anal atresia

Anal atresia is caused by abnormal development of the hindgut and occurs when the cloacae membrane at the anal outlet fails to break through (Norrish & Rennie, 1968). The defect is also seen in ruminants and humans. Anal atresia in pigs and humans are somewhat similar (Cassini *et al*., 2004) and when studying if there is a genetic background in humans, genomes from pigs have been used. Anal atresia is fatal to male pigs (Norrish & Rennie, 1968). In female piglets the rectum can form a rectovaginal fistula and permit defecation via the vulva.

Intersexuality

Intersexuality occurs when there is an abnormal development of the reproductive system and it can be phenotypicaly expressed in different ways (Halina *et al*., 1984). Defect pigs are considered to be either true hermaphrodites or pseudo-hermaphrodites. In both these cases, the animal has two X-chromosomes, i.e. is a ‘female’.A true hermaphrodite have active female and active male gonads and in some cases both female and male external genitalia (Halina *et al*., 1984 & Pailhoux *et al*., 1997). A pseudo-hermaphrodite is most commonly characterized by a female phenotype with male internal and external genitals in different degree of development and function.

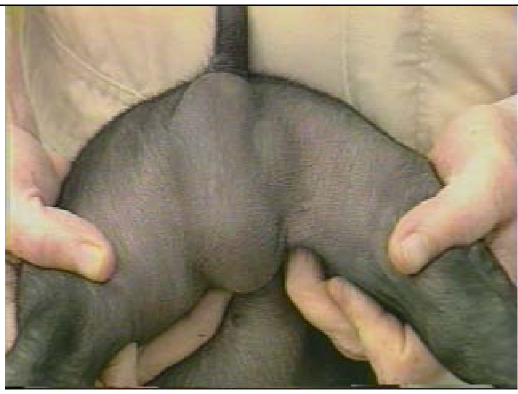
Congenital tremor syndrome

Tremor is characterized by rhythmic tremor associated with myelin deficiency (Patterson, 1973). There are different causes to the defect, originating form a genetic background, placental environmental factors or viral infections of the sow resulting in an unsuccessful myelination of the spinal cord (Patterson, 1976; Stevenson, 2001). Mortality among piglets born alive with the defect may be as high as 50% due to their inability to suckle (Stevenson, 2001). Affected piglets may recover within four weeks of age and some can be raised to slaughter still trembling (Lindström & Lundeheim, 1986).

Miscellaneous congenital abnormalities

Seldom occurring congenital abnormalities observed in pigs are: head and body defects, congenital heart defects, absence or deformity of tail and *Epitheliogenesis imperfecta*, which is a congenital defect where skin is missing at parts of the body at the time of birth ((Mulley & Edwards, 1977;Thaller *et al.*, 1996; Holyoake et al., 2006; Benoit-Biancamano *et al.*, 2006).

Umbilical hernia (M.T. See)



Scrotal hernia (M.T. See)

Cryptorchidism (M.T. See)

Kyphosis (B. Straw)

Splay leg (M.T. See)

Anal atresia (M.T. See)

Intersexuality (M.T. See)

Figure 1. Pictures of different defects occurring in pigs, with permission from Professor M. T. See, Department of Animal Science, North Carolina State University and Professor Barbara Straw, Department of Large Animal Clinical Science, Michigan State University.

Prevalence of defect pigs within different pig populations

There is a variation of reported overall prevalence of defect pigs in different populations, 2.0-2.5% see table 1a. The reported prevalence of individual defects differs between breeds and populations see table 1b. Kyphosis, splay legs and congenital tremor were the defects with highest reported prevalence in the reviewed literature.

Table 1a.Prevalence of defect piglets, percentage piglets born defected as well as percentage of litters with at least one recorded defect in different pig populations

Number of litters and breed	Country	Overall prevalence of defects in offspring		Reference
		Defect piglets (%)	Litters with defect piglets (%)	
1 443 litters from 2800 (L*Y)-sows	Australia	2.3	17.4	(Mulley & Edwards, 1984)
6596 litters from 194 L-boars 9996 litters from 294 Y-boars	Holland	2.3 2.5	2.4-12.0 1.6-11.0	(Knap, 1986)
9370 litters from Y-sows	Sweden	2.5	20.5	(Andersson & Rydhmer, 1991)
8604 litters from 266 L-boars 21933 litters from 675 Pietran boars	Germany	2.0 2.1	not specified	(Thaller *et al*., 1996)

Table 1b. Reported prevalence (% of all pigs born alive) and estimated heritability (h^2) of defects in different pig populations

Defect	Prevalence	h^2	References
Umbilical hernia	0.3 – 1.5	0.06-0.08	(Warwick, 1926; Andersson & Rydhmer, 1991; Searcy-Bernal *et al*., 1994; Rutten-Ramos & Deen, 2006; Ranberg , 2007)
Scrotal hernia	0.4-2.6	0.03-0.15	(Warwick, 1926; Magee, 1951; Knap, 1986; Andersson & Rydhmer, 1991; Thaller *et al*., 1996; Grindflek *et al*., 2006; Ranberg , 2007;)
Cryptorchidism	0.16-0.7	0.02- 0.7	(Knap, 1986; Rothschild et al., 1988; Andersson & Rydhmer, 1991; Thaller *et al*., 1996; Ranberg , 2007;)
Kyphosis	1.5-23.3	0.2-0.3	(Bradley, 2005; Nielsen et al., 2005; Holl & Johnson, 2008;)
Splay legs	0.2-4.9	0.01-0.18	(Andersson & Rydhmer, 1991; Thaller *et al*., 1996; Sellier *et al.*, 1998 ; Holl & Johnson, 2005)
Anal atresia	0.1-1.0	0.11-0.35	(Knap, 1986; Andersson & Rydhmer, 1991; Thaller et al., 1996)
Intersexuality	0.03-0.89	0.001 – 0.5	(Knap, 1986; Andersson & Rydhmer, 1991; Thaller *et al*., 1996)
Congenital tremor	0.13-11	0.2-0.4	(Lindström & Lundeheim, 1984; Andersson & Rydhmer, 1991; Thaller *et al*., 1996)

Factors that influence prevalence

Factors presented in literature with effect on prevalence of defect piglets are breed, herd, season, parity number, litter size and sex. In the reviewed literature, a variation between populations and breeds for the overall frequency of prevalence of defects is shown (table 1a).

Season

Defects with seasonal variation reported in literature are splay legs and congenital tremor (Sellier *et al*., 1998; Van der Heyde *et al.*, 2005; Lindström & Lundeheim, 1984). Van der Heyde *et al*. (2005) show a higher frequency of splay legs among piglets born in May, August and November. An increased prevalence of congenital trembling was seen in piglets born in autumn (Lindström & Lundeheim, 1984).

Parity number & Litter Size

Litters with any defect present and number of defect piglets in litters tend to increase with parity number (Mulley & Edwards, 1984). Litter size increase with increased parity number (up to fifth parity) and as result, the probability to find litters with defect piglets increase.

Sex

Besides cryptorchidism and scrotal hernia, sex is of importance for the prevalence of splay legs. Holl & Johnson (2005) found that the incidence of splay legs was 226% higher in male piglets. Kyphosis was found more frequently in castrated males than gilts in a study done by Straw *et al*. (2009).

Correlation between cryptorchidism and scrotal hernia

Failure during *processus vaginalis* leads to a positive correlation of the prevalence of cryptorchidism with the prevalence of scrotal hernia (Knap, 1986; Grindflek et al. 2006). Genetic correlations between prevalence of cryptorchidism and scrotal hernia in litters have been estimated to 0.2-0.7 (Mikami & Fredeen, 1979; Knap, 1986).

Genetic background

Congenital defects in pigs have a genetic background. The reviewed literature on estimated heritabilities show that most estimates are low, see table 1b. Inheritance of scrotal hernia has recently been studied by Grindflek et al. (2006). Significant QTLs were found for this defect and six different haplotypes were found in different frequency in herniated and healthy pigs. One haplotype on *Sus Scrofa* chromosome 5 (SSC5) was found in herniated pigs with four time higher frequency than in non-herniated pigs. Cryptorchidism in male mice is suggested to depend on mutations in Insulin-like 3/Relaxin Like Factor gene (Zimmerman *et al*., 1999; Tomboc *et al*., 2000). Genini *et al*. (2004) studied possible genetic markers for congenital defects of fore and hind limbs in pigs and found significant genetic linkage between eight marker loci in one allele located on SSC5. Hori *et al*. (2001) have suggested that one locus at SSC15 is involved in the genetic background of anal atresia in pigs.

Consideration of defects in breeding programs

The Swedish-Finnish breeding company Nordic Genetics has outlined regulations on defects in nucleus herds (Kihlberg, 2009). In case of scrotal hernia and intersexuality in purebred litters, no female or male pigs from these litters can be used for breeding. In case of cryptorchidism in purebred litters, it is today accepted to sell purebred gilts from these litters to multiplier herds. The recommendation for splay legs, anal atresia, kyphosis and tremor is not to use the defect animals in further breeding.

The Norwegian Pig Breeders Association (Norsvin) has initiated a project “Breeding for better health” (Norsvin, 2009a). Scrotal and umbilical hernia and cryptorchidism are since 2007 included as health traits in the breeding goal for Norwegian Landrace and Norwegian Duroc. For these two breeds, 3% of the selection is, in both breeds, represented by umbilical hernias, scrotal hernia and cryptorchidism together (Norsvin, 2009b).

In Denmark, the pig breeding company Danavl has regulations for breeding in nucleus herds when defects are present in litters. In case of congenital tremor, cryptorchidism and scrotal hernia no pigs with the defect should be selected for breeding (Danske Svineproducenter, 2009).

5. OWN STUDY

Data from nucleus herds

Data used in the analysis were collected from Nordic Genetics (NG) database. This database includes information on all Swedish nucleus herds, as well as on all multiplier herds with contract with NG. The database includes information on all litters, including registration of number of piglets with congenital defects in each litter. The recorded defects were: umbilical hernia, scrotal hernia, cryptorchidism, kyphosis/humpy back, splay legs, anal atresia, intersexuality, congenital tremor and ‘miscellaneous’.

Data were handled and statistically analysed, using the SAS software (Version 9; SAS Inst. Inc., Cary, NC). The statistical analyses were based on data on purebred Hampshire, Landrace and Yorkshire litters, born in the period January 2000 until October 2007. Further restrictions were imposed: only herds with at least 400 purebred litters in the period remained for analysis. Among these herds the 5 nucleus herds of each breed, with the highest average proportion of litters with recorded defects, were kept for statistical analysis. After these restrictions, data on 19 308 purebred litters remained: 5942 Hampshire litters, 6197 Landrace litters and 7169 Yorkshire litters.

The statistical analyses included, besides calculation of frequencies and means, also analysis of variance of the binary trait 'defect present or not in the litters' (using PROC GLIMMIX). Two statistical models were applied to the data:

1) including the fixed effects of breed, herd nested within breed, birth year (2 years combination), birth month (2 months combinations), parity number, 1, 2, 3+ and the interaction between breed and birth year, breed and birth month, breed and parity number, parity number and birth month
2) the model above, expanded with the effect of litter size nested within breed:

   for Hampshire: 5 to 15+ total born piglets in the litters

   for Landrace and Yorkshire: 7 to 18+ total born piglets in the litters

Cryptorchidism and scrotal hernia were recorded in all herds studied and considered to be the most common defects. The estimations of heritability (h^2) for presence of cryptorchidism and scrotal hernia were performed using PROC MIXED for each breed. The statistical model included year and parity number as fixed effects and father and maternal grandfather as random effects. The estimated variance components for father, maternal grandfather and error were used to calculate the heritability.

For each father and maternal grandfather (with more than 20 litters in the data) the average prevalence of defects in litters was calculated. The correlation between mean prevalence of cryptorchidism and scrotal hernia in litters when the boar was a father and when the boar was a maternal grandfather was estimated, using Spearman’s Correlation.

Levels of significance are presented conventionally:

ns	(not significant): $p>0.05$
*	$0.01<p\leq0.05$
**	$0.001<p\leq0.01$
***	$p\leq0.001$

Data from commercial herds

With assistance from Maria Malmström, LRF Konsult AB and Barbro Mattsson, Svenska Pig, a number of commercial farrow-to-finish and specialized piglets producers were identified. The herds were known to belong to producers with high level of skill and interest in the production. In total, 25 herds were contacted by a letter and later contacted per telephone and asked to participate in an interview focusing on their opinion on the magnitude of defects among the piglets in their herds. The interviews were done during late August and early September 2008. Because of the time of year, with a late harvest, only 17 herdsmen were interviewed. The main purpose of this interview was to collect data and opinions whether congenital defects was regarded to be a real problem in commercial production. Interviews with owner and/or herd staff were done according to a form, Appendix 1. The collected information was summarized in Microsoft Excel.

Since all herd owners were not willing to share their production data some information used in the evaluations were average values from Swedish commercial herds in 2007, obtained from the herd monitoring software program “PigWin”. All herds did share their number of sows and average number of weaned piglets per sow. For all herds, mortality was estimated both during the growing phase as well as during the fattening phase.

The estimation of the prevalence of defects was based on number of piglets born alive per year, irrespective of sex. For umbilical hernia, in most cases observed after weaning, the estimation was expressed as proportion of piglets per group of growers.

6. RESULTS

Prevalence of defects in nucleus herds

Data on 15 Swedish nucleus herds and 19 308 purebred litters born in the studied period were included in the analyses, five Hampshire herds with total 5942 litters, five Landrace herds with total 6197 litters and five Yorkshire herds with total 7169 litters.

Mean proportion of litters with at least one defect registered in the litter was 7.0% in Hampshire, 12.0% in Landrace and 16.8% in Yorkshire. The average prevalence of litters with each defect is shown in table 2. Cryptorchidism and scrotal hernia were recorded in all herds. Cryptorchidism was the most common defect in Hampshire, present in 4.3% of all litters. Scrotal hernia was the most common defect in both Landrace and Yorkshire litters, present in 6.5 and 9.1% of the litters respectively. Noteworthy is that cryptorchidism was registered at a very low level (0.5% of all litters) in Landrace compared to 8.3% of all litters in Yorkshire. In Hampshire, kyphosis was seldom reported (only one herd), followed by intersexuality and congenital tremor with registrations in only two herds. Anal atresia was less frequently registered in Landrace (only one herd). In Yorkshire anal atresia and splay legs was recorded in only two herds.

Table 2. Mean proportion (%) of litters with recorded defects within breed, and range between herds for five Hampshire, five Landrace and five Yorkshire nucleus herds

	Breed		
	Hampshire	Landrace	Yorkshire
	n=5942	n=6197	n=7169
Defect			
Umbilical hernia			
Herd mean (%)	0.30	1.4	1.7
Herd range (%)	0 – 0.8	0 – 3.6	0 – 4.0
Herds with no finding	2	1	1
Scrotal hernia			
Herd mean (%)	0.9	6.5	9.1
Herd range (%)	0.2 – 1.5	2.9 – 8.8	2.1 – 4.3
Herds with no finding	0	0	0
Cryptorchidism			
Herd mean (%)	4.3	0.5	8.3
Herd range (%)	1.7 – 7.2	0.2 – 1.1	5.8 – 11.0
Herds with no finding	0	0	0
Kyphosis			
Herd means (%)	0.2	1.0	0.6
Herd range (%)	0 – 1.0	0 – 3.1	0 – 1.0
Herds with no finding	4	2	1
Splay legs			
Herd mean (%)	0.5	1.5	0.5
Herd range (%)	0 – 2.3	0 – 4.3	0 – 2.3
Herds with no finding	2	2	3
Anal atresia			
Herd mean (%)	0.2	0.05	0.04
Herd range (%)	0 – 0.4	0 – 0.2	0 – 0.1
Herds with no finding	1	4	3
Intersexuality			
Herd mean (%)	0.05	0.5	0.3
Herd range (%)	0 – 0.2	0.2 – 1.5	0.09 – 0.8
Herds with no finding	3	0	0
Tremor			
Herd mean (%)	0.1	1.0	2.7
Herd range (%)	0 – 0.6	0 – 3.1	0 – 8.0
Herds with no finding	3	2	1
Miscellaneous			
Herd mean (%)	0.7	0.05	0.9
Herd range (%)	0.07 – 2.3	0 – 0.2	0 – 2.4
Herds with no finding	0	3	2
Any defect			
Herd mean (%)	**7.0**	**12.0**	**16.8**
Herd range (%)	**5.5 – 8.6**	**9.3 – 16.6**	**11.9 – 23.2**
Herds with no finding	**0**	**0**	**0**

The average number of defect piglets in litters with the defect recorded is shown in table 3. The highest average number of defect piglets in the litters was found for splay legs (L), congenital tremor (Y) and intersexuality (H).

Table 3. Average number of piglets in purebred litters with respective defect recorded

	Breed		
	Hampshire	Landrace	Yorkshire
Defect			
Umbilical hernia	1.0	1.1	1.1
Scrotal hernia	1.2	1.3	1.1
Cryptorchidism	1.2	1.0	1.2
Kyphosis	1.4	1.2	1.4
Splay legs	1.3	2.8	1.1
Anal atresia	1.3	1.0	1.0
Intersexuality	1.5	1.1	1.2
Congenital tremor	1.3	1.2	1.7
Miscellaneous	1.1	1.0	1.2
Mean (No.)	**1.2**	**1.3**	**1.2**

Causes of variation

Herd within breed had significant effect on all but one of the analysed variables. For the effects of the other factors included in the statistical model: year, season and parity number, only few significances were found. The levels of significance are shown in table 4a. There were significant effects of breed for cryptorchidism and scrotal hernia, and the effect of year was significant for scrotal hernia.

Table 4a. Levels of significance for the effects included in the statistical model

Variable	Breed	Herd (Breed)	Year (2 years)	Month (2 months)	Parity number (1^{st}, 2^{nd}, 3^{rd}+)
Umbilical hernia	Ns	***	ns	ns	ns
Scrotal hernia	***	***	*	ns	ns
Cryptorchidism	***	***	ns	ns	ns
Kyphosis	Ns	**	ns	ns	ns
Splay legs	Ns	***	ns	ns	ns
Anal atresia	Ns	ns	ns	ns	ns
Intersexuality	Ns	***	ns	ns	ns
Tremor	Ns	***	ns	ns	ns
Miscellaneous	Ns	***	ns	ns	ns

There were significant interactions between breed and year for cryptorchidism, scrotal hernia, splay legs and kyphosis, table 4b. There were also significant influences of the interaction between season and parity number for prevalence of cryptorchidism, scrotal hernia and congenital tremor.

Table 4b. Levels of significance for the interactions included in the statistical model

Variable	Breed*Year	Breed*Month	Breed*Parity number	Month*Parity number
Umbilical hernia	ns	ns	ns	ns
Scrotal hernia	**	ns	ns	**
Cryptorchidism	***	ns	ns	*
Kyphosis	**	ns	ns	ns
Splay legs	***	ns	ns	ns
Anal atresia	ns	ns	ns	ns
Intersex	ns	ns	ns	ns
Tremor	ns	ns	ns	*
Miscellaneous	ns	ns	ns	ns

The estimated least square means for prevalence of litters with cryptorchidism or scrotal hernia for the combination breed*year are illustrated in figure 2. Prevalence of cryptorchidism has increased over years in Yorkshire litters. The overall average prevalence of cryptorchidism in Landrace litters is low. Scrotal hernia did increase in Yorkshire litters from 2000-2001 to 2004-2005 and have decreased to 2006-2007. A decreased prevalence of scrotal hernia in Landrace is seen from 2004-2005 to 2006-2007. The prevalence of scrotal hernia in Hampshire litters is constantly low over years.

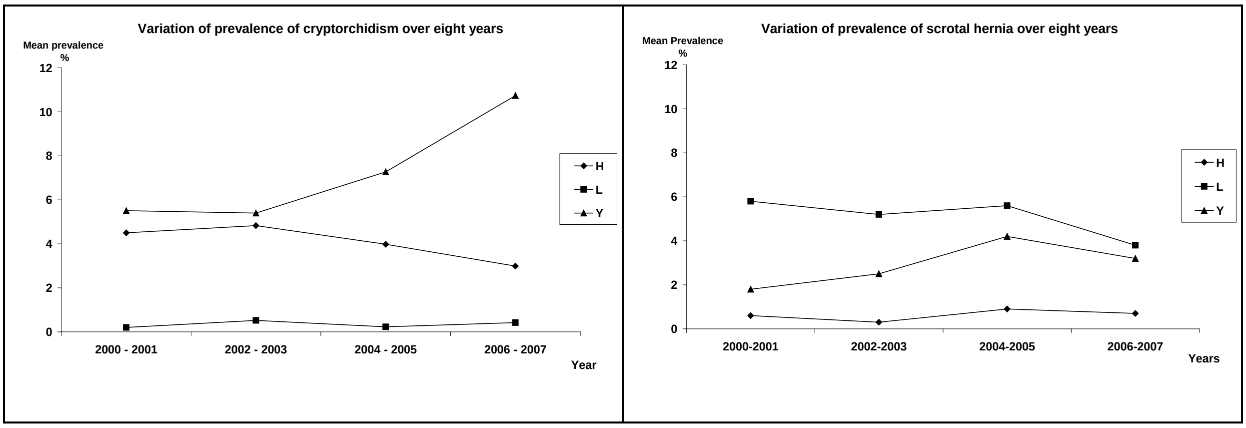


Figure 2. Variation in prevalence of litters with cryptorchidism (left) and scrotal hernia (right) in purebred litters over eight years.

Prevalence of cryptorchidism and scrotal hernia varies over season, figure 3. In first parity litters there was an increase in prevalence of both cryptorchidism and scrotal hernia during Mars-April and a decrease in May-June. Prevalence of cryptorchidism increased again whereas scrotal hernias continued to decrease. In litters from second parity sows, the prevalence of cryptorchidism and scrotal hernia decreased over months until July-August and cryptorchidism increased slightly during autumn, while scrotal hernia increased at a higher degree.

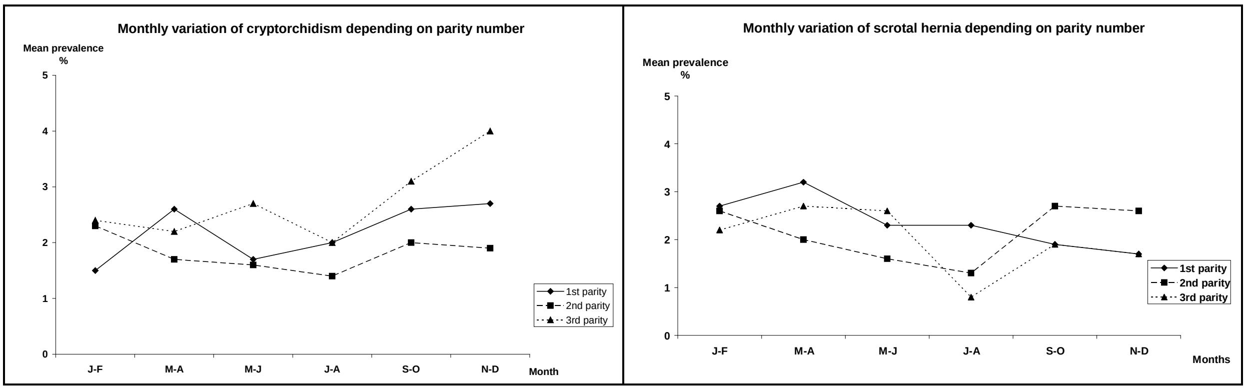


Figure 3. Prevalence of litters with cryptorchidism (left) and scrotal hernia (right) over season in relation to parity number in all included purebred Hampshire, Landrace and Yorkshire herds.

Proportion of litters with cryptorchidism present was highest for third parity sows. There was a small variation in prevalence of litters with cryptorchidism except from summer to late fall and a remarkable increase of registered cryptorchidism (from ~2.5% to 4%) until November-December. Scrotal hernias were remarkably lower in litters from 2nd and 3rd parity sows in July-August compared to the rest of the year. Noteworthy is that prevalence of both cryptorchidism and scrotal hernia decreased in second and third parity sows during July-August.

Figure 4 shows the effect of total born piglets in litters on the prevalence of litters with the defect. There was significant effect of the total number born piglets in litters on the prevalence of litters with cryptorchidism (*) and scrotal hernia (***). An increased litter size increased the prevalence of cryptorchidism in Hampshire and Yorkshire litters. Scrotal hernia increased with an increased number of piglets in both Landrace and Yorkshire litters.

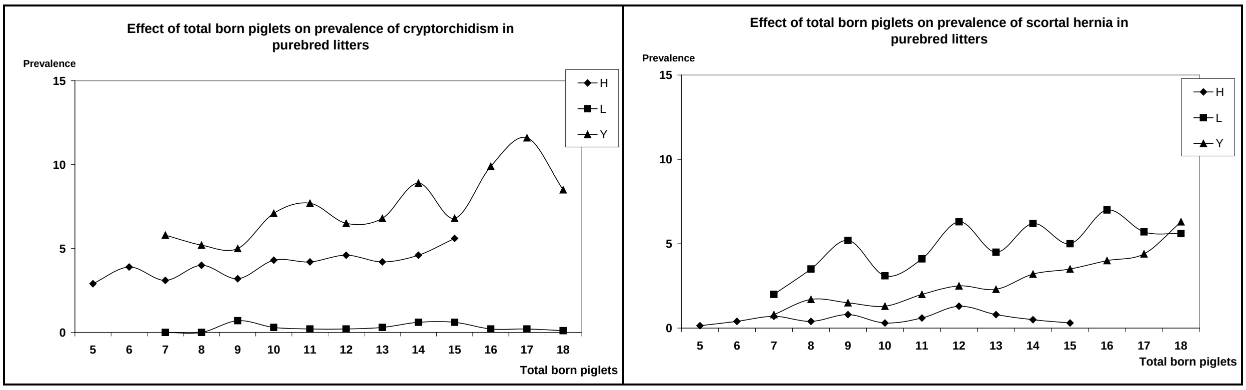


Figure 4. Prevalence of cryptorchidism (left) and scrotal hernia (right) in relation to litter size, within breed, Hampshire, Landrace and Yorkshire litters.

Correlation between prevalence of cryptorchidism and scrotal hernia

Cryptorchidism and scrotal hernia was recorded in all herds included in the analysis. For each father and maternal grandfather (with more than 20 litters in the data) the average prevalence of defects in litters was calculated. The correlation between mean prevalence of cryptorchidism and scrotal hernia in litters when the boar was the father or maternal grandfather was estimated. The average prevalence for each boar was calculated after the restriction of at least 20 litters as father and 20 litters as maternal grandfather during the period studied from each boar (43 Hampshire boars, 36 Landrace boars and 51 Yorkshire boars). The calculations were based on 543 Hampshire, 527 Landrace and 555 Yorkshire litters with the boar as a father and 587 Hampshire, 659 Landrace and 659 Yorkshire litters with the boar as a maternal grandfather. Correlations were estimated based on boar mean values. No significant correlations between the prevalence on boar level were found in Hampshire, see table 5. For both Landrace and Yorkshire, a positive correlation of prevalence of cryptorchidism between the boar as a father or maternal grandfather was found. For Yorkshire, the correlation between prevalence of scrotal hernia and cryptorchidism (when the boar was father of the litter) was positive.

Table 5. Spearman's correlation coefficients (significant, p≤ 0.05, in bold) between prevalence of cryptorchidism (CR) and scrotal hernia (SH) in litters with Hampshire, Landrace and Yorkshire boars as fathers (f) or maternal grandfathers (mgf)

	Hampshire			**Landrace**			**Yorkshire**		
	CR^{f}	SH^{f}	CR^{mgf}	CR^{f}	SH^{f}	CR^{mgf}	CR^{f}	SH^{f}	CR^{mgf}
SH^{f}	0.09			-0.2			**0.3***		
CR^{mgf}	-0.1	-0.2		**0.4***	-0.3		**0.3***	0.3	
SH^{mgf}	-0.2	0.1	-0.2	-0.1	-0.2	0.1	0.03	0.06	0.01

Heritability

Estimations of heritabilies are shown in table 6. Most estimates were below 0.1 but large breed differences were found.

Table 6. Estimated heritabilities for cryptorchidism and scrotal hernia within breed, based on information on boars as fathers or maternal grandfathers of the litters

	Hampshire		**Landrace**		**Yorkshire**	
	Father	Grandfather	Father	Grandfather	Father	Grandfather
Cryptorchidism	0.07	0.09	0.005	0.03	0.23	0.16
Scrotal hernia	0.09	0.03	0.20	0.18	0.02	0.012

Commercial herds

General on the herds

In total, 17 commercial herds were visited. Eight farms with only piglet production, three farms with partly farrow-to-finish production, four farms with complete farrow-to-finish production, one satellite herd and one farm with hybrid gilt production within a sow pool. The hybrid producing herd and the satellite herds are included in the farrow-to-finish production results. 15 farms used PigWin as a management tool. Two herds used PigWin to register all defect piglets found in the farrowing stable.The calculated mean production characteristics within production type are illustrated in table 7.

Table 7. Number of sows in the herds and average production volume/year of growing and fattening pigs in 17 interviewed commercial herds

Category of animals	**Piglet production n=8**	**Partly farrow-to-finish production n=3**	**Farrow-to-finish n=6**
Sows			
mean	504	723	327
range	300 - 1100	560 - 950	165 - 569
Growers			
mean	11 997	8147	
range	6790 – 26 071	2154 – 15 000	
Fattening pigs			
mean		9083	7489
range		7288 – 11 000	3997 – 13 665

All 17 herds used straw as bedding material in farrowing, growing and fattening stables. Six herds also used saw dust as a complementary bedding material during farrowing. All herds did clean the stables between batches, mechanically or with water, and all but one did use disinfection agents.

Castration routines were similar, all herds used scalpel and did disinfect the instruments after each piglet. In case of suspected scrotal hernia the treatment varied between herds. Ten herds did not castrate the piglets with indications of scrotal hernia. The other seven herds did castrate and stitch, tape or plaster the scrotum if there was a hernia present. 13 herds did castrate male pigs with a cryptorchid testis, cutting the visible testis. Three herds did call for veterinarian to castrate male pigs with suspected hernia or cryptorchidism.

15 herds used genetic material from Nordic Genetics (95% AI dozes) and one herd used genetic material from Avelspoolen. Eight herds did lease Hampshire boars from NG and eight herds used boars produced in the own herd. These boars were mainly used for stimulation of sows and gilts at oestrus and for remating.

Replacement gilts

Two herds bought replacement gilts at approximately 10 weeks of age, one herd bought gilts at the age of 5½ month and one herd bought purebred Yorkshire gilts at the age of 7 months. Twelve herds used two-breed rotational crossing for replacement. The strategies for raising gilts varied between herds. One herd raised gilts in a replacement stable from weaning. Ten herds raised gilts in the growing stable and thereafter moved them to a replacement stable. Three herds raised gilts in the fattening pig stable and moved gilts to replacement stable at the time for delivery of fattening pigs.

Criterias and routines for selection of gilts for breeding differed between herds. All 12 herds with rotational crossing had at least two selection occasions. Six herds did select and sort out non-fitting gilts a third time. The used criteria and number of selection stages are shown in table 8.

Table 8. Different criteria used at different stages of selection of replacement gilts, at different ages and weights, in 12 commercial herds using two breed rotational crossing

			Selection criteria						
No. of selection steps	Age/size	Number of herds	Teats	Legs	Hernia	Growth	Feet	Size	Other defect
1	1- 5 weeks	12	x	x	x		x	x	x
2	30 kg	8	x	x	x				
2	60 kg	1		x	x	x			
2	120 kg	1	x	x	x		x	x	
2	140 kg	2	x	x	x			x	
3	120 kg	1	x	x	x				
3	140 kg	5	x	x	x		x	x	x

The selection criteria "other defects" was at the first selection occasion depending on the prevalence of defects in the actual litter. If there were any defect in the litter supposed to be used for further breeding, five herds did not select any gilt from that litter. Two herds did register the defect and raised the gilts and did evaluate the gilt later in life.

Prevalence of defects

All herds reported that they noticed most of the defect pigs just after farrowing, teeth grinding and castration. Kyphosis was most commonly detected later in life in the farrowing and growing stable. In case of umbilical hernia all herds did detect pigs with hernia in the growing stable when the pigs weighed around 20 kg or during their 9th week of life. Three herds did control all piglets for defects before weaning. Ten herds did control growers the week before delivery, around week 10 of life, or when they were moved to the fattening pig stable.

For 15 of the farms the prevalence of defects were based on estimates made by the farmer and/or the personnel. For two herds the information of incidence of defects was collected from the herds' registrations in PigWin. Some herds did find the task to estimate the number of pigs with defects difficult. In table 9, each defect is presented with the actual number of herds with information. Only herds with estimated actual figures are included in the estimation of the overall prevalence of defects.

The estimated mean prevalence of defects (% of all piglets) in the 17 herds is shown in table 9. Most common defect was splay legs with a prevalence of 4.1%; umbilical hernia with an estimated prevalence of 1.8% and cryptorchidism 0.9%. Scrotal hernia was estimated to appear in 0.7% of all piglets. 0.2% of the piglets were estimated to have kyphosis and intersexes respectively. 0.1% of piglets were estimated to have congenital tremor or anal atresia. Miscellaneous defects were estimated to appear in 0.1% of all piglets.

Table 9. Number of commercial herds with records on prevalence of defects, together with estimated average prevalences

Defect	No. of herds	Average prevalence, %
Umbilical hernia	14	1.8
Scrotal hernia	14	0.7
Cryptorchidism	12	0.9
Kyphosis	6	0.2
Splay legs	9	4.1
Congenital tremor	4	0.1
Anal atresia	6	0.1
Intersexuality	10	0.2
Miscellaneous	7	0.1

Cryptorchidism

None of the visited herds did euthanize males with cryptorchidism. Six herds castrated and marked cryptorchid piglets at the time of castration, and checked the pigs later on for the remaining testis.

Splay legs

Splay legs are pointed out to be a major problem in four herds. The defect varied between batches and in one herd splay legs did only occur in litters from gilts. To prevent the defect three of the herds gave Tokosel®[1] in feed to all sows in late pregnancy and noticed a decrease in proportion of piglets born with splay legs as an effect. Two herds had noticed a decrease of splay legs prevalence when increasing the amount of straw, for bedding, during farrowing.

Umbilical and scrotal hernias

The size of the hernias and the ability to deliver these herniated pigs to fattening pig producers differ among farms. All but one piglet producing herd and one partly farrow-to-finish herd had an agreement with the fattening farm to deliver growers with defects, mainly scrotal and umbilical hernias. Some farms had an oral agreement; other farms had a written agreement. Alternatives in the agreements were to get less paid for defect pigs (price reduction) or the seller did deliver extra growers as compensation. Five farrow-to-finish farms raised the defect pigs in the fattening pig stable as long as the animals' welfare was maintained. Four fattening producers sorted out defect pigs when moving from growing to fattening stable and raised defect pigs in separate boxes.

Six herds pointed out umbilical hernia to be the most troublesome defect in the production, due to decreased growth, problems to deliver or raise the pigs to slaughter and animal welfare issues. Four of these herds estimated that the frequency of this defect had increased during the last two to three years. In case of scrotal hernia in a replacement litter, four herds did not take replacement gilts from the litter and these pigs were raised as commercial pigs and one herd did not take replacement gilts from the litter if scrotal or umbilical hernia was detected in the growing stable.

[1] Tokosel is a prescript mineral supply added to feed to prevent lack of vitamin E/Selenium in pigs, horses, cattle, sheep and poultry.

All 17 herds found umbilical hernias among pigs in the growing stable. All piglets with umbilical hernias at birth were euthanized. As for the reason and the background for umbilical hernia, 16 herdsmen believed in a genetic background, nine herdsmen did also suggest an environmental influence. The environmental influence was defined as poor hygiene in the farrowing pen and wearing of the umbilical in the farrowing box. Some farms had seen a decrease in prevalence of umbilical hernia after deleting saw dust as a complementary bedding material during farrowing. Three of the six farms that used saw dust as complementary bedding material at farrowing pointed out umbilical hernias as a problem. One herd had seen a decreased prevalence of umbilical hernias when bandaging the belly of all replacement gilts during first week of life. This bandage covered the umbilical area with a dressing that was taped.

Four producers had seen an increase in the prevalence of umbilical hernias during the last two years. Two producers said that the prevalence of the defect varied; some batches had as many as 5-10% pigs with umbilical hernias and some batches had few or none.

Miscellaneous

Other defects that occurred in the visited commercial herds were deformed legs and bodies in the newborn piglets, umbilical bleeding, deformed heads and snouts and tail necrosis. In one herd an increase of tail necrosis was observed during the autumn 2008. Two farrow-to-finish herds pointed out high prevalence of lameness of piglets day one after farrowing as a problem, suggesting it to be a health trait.

Three herdsmen informed that they had reported defects to the breeding organization at three different occasions:

- In one herd there had been very high prevalence of splay legs in one single batch (90 % of all piglets born alive).
- A second herd did find a relation between mortality in replacement litters in a specific farrowing batch and a specific boar as a father of these litters.
- The third herd did have batches following after each other with more than normal prevalence of several defects in litters with a father from specific breed.

All three herdsmen reported this to Nordic Genetics. None of them felt however that they did get any response or feedback. Four herdsmen asked for a better control of defects in nucleus herds, especially for umbilical and scrotal hernias.

7. DISCUSSION

Methods used in this study

In this study data from only 15 nucleus herds, five from each breed (H, L & Y), are included. There was a large variation in prevalence of recorded defects between herds. This implicates a variation in accuracy between herds in recording defects. It would have been interesting to include data from all nucleus herds, crossbred litters in nucleus herds and also multiplier herds and investigate if there is any heterosis effect on the prevalence of defect piglets. However, this requires that the recording of the defects is as accurate as possible in both purebred and multiplying herds.

As shown in table 2 there is a variation in number of herds with no finding of some defects. Cryptorchidism and scrotal hernia were registered in all 15 herds and this defect was used as a "model trait" to analyse the boar's effect as a father or maternal grandfather of the litters. Even though there were significant differences between breed and year for kyphosis, splay legs and tremor (table 4b) the prevalence did also differ markedly between herds within breed (table 2). The missing data, i.e. different accuracies of recording in some herds limits the accuracy to estimate the genetic background (heritability) of defects.

For the commercial herds, the main purpose was to collect information and opinions on prevalence of congenital defects in commercial pig production.

Defects in nucleus herds

The overall mean prevalence of "any defect" present in litters varies between breeds, table 2. There is also a range between herds within breeds. Yorkshire has the highest mean prevalence of defects present in litters (16.8) and also the largest variation between herds in recorded defects, 11.9-23.2. It might be explained by different accuracy to register defects in different herds, which is supported by Ranberg (2007) who points out the risk for incorrect registration.

Prevalence of defects

The prevalence of cryptorchidism is higher in Hampshire (4.3%) and Yorkshire (8.3%) than in Landrace (0.5%). As for the Yorkshire compared to Landrace this is supported by studies done by Knap (1986) and Ranberg (2007). Yorkshire has a higher mean proportion of litters with scrotal hernia (9.1%) compared to Hampshire (0.9%) and Landrace (6.5%). Knap (1986) however found the opposite: 12.0% in Yorkshire and 11.0% in Landrace.

Umbilical hernia, kyphosis, splay legs, anal atresia, intersexuality, tremor and miscellaneous defects were recorded in all breeds but not in all herds. In literature, umbilical hernia and kyphosis were often reported to be visible later in life and there might be that these defects are forgotten to be recorded when they can be clinically diagnosed. Anal atresia is lethal (Norrish & Rennie, 1968) in all cases in male pigs, and the low prevalence of the defect might be explained by the fact that the piglets die before controlled for defects. Prevalence of splay legs and tremor might be influenced by the sow, since the gestation length is of importance for the prevalence (Sellier *et al*., 1998; Stevenson, 2001). These defects might be affected by environmental factors in the uterus. The housing and management of pregnant sows in the herd can be of importance.

Nordic Genetic's restriction on replacement stock considering scrotal hernia and cryptorchidism can explain the decreased prevalence of the scrotal hernia over years since there is a reduction from 2004-2005 to 2006-2007. Cryptorchidism, as a contrast, has increased in Yorkshire from 2002-2003 to 2006-2007. This might be a result of a more accurate registration or a higher frequency of alleles causing the defect within the population.

Factors that effect prevalence

Very little information has been found in literature on different factors that might have an effect on the prevalence of defects, since all factors (e.g. effect of season and parity) are not studied in different studies. Concerning effect of season, one possibility is that the herdsmen are busy working out in the fields during spring and autumn and put less time in the herds. This might at least give some explanation to the decrease of cryptorchidism present in litters born in Mars-April and the decrease of scrotal hernia in July-August from second and third parity sows. In the opposite, in first parity sows, the prevalence of both cryptorchidism and scrotal hernia increases during May-April. Maybe the system for grouping the sows into batches and have farrowing during weekends has an effect on the registrations of defects. Many large herds have ordinary personnel during the weeks and since first parity sows might have a shorter gestation length (Sellier *et al*., 1998) compared to second and third parity sows, the litters from first parity sows are recorded more properly by the personnel. Mulley & Edwards (1984) found an increasing prevalence of defects with an increased parity number of the sow.

The effect of number born piglets in the litters has been studied by Mulley & Edwards (1984) suggesting an increasing number of defect piglets in larger litters. In this study the trend was the same, there is a higher probability to find at least one defect pig in the litters when the litters are larger.

Genetic background

Estimates of heritability for scrotal hernia and cryptorchidism were low (<0.2) which is in agreement with literature, see table 1b. The low estimates can be an effect of a low recording accuracy of defects generally in herds and that the trait was a binary trait, which gives lower estimates. In Yorkshire, the h^2 of cryptorchidism was estimated to be 0.23 (father) and 0.16 (maternal grandfather). In Landrace the h^2 of scrotal hernia was estimated to be 0.20 and 0.18. The differences between breeds implicates that there are different genetic backgrounds for the defects in different breeds. According to table 2, there is variation between herds' records of defects and this might reflect different attitudes on recording defects in different herds. This might give some explanation to the low estimates of heritability.

Defects in commercial herds

Prevalence

The estimated mean prevalence of defects in the visited herds are shown in table 9. Splay legs is estimated to be most common defect, with 4.1% of all pigs born followed by umbilical hernia with an estimated prevalence of 1.8%. The prevalence of cryptorchidism and scrotal hernia was estimated to 0.9% and 0.7% respectively.

Gestation length has an importance for the incidence of splay legs (Sellier *et al*., 1999). One reason for the high estimated prevalence of splay legs in commercial herds is that two herds reporting this to be very large problems occasionally, with as many as 90% of all born piglets affected. This might be caused by some environmental effect in these two herds, which we have not identified, more than a genetic defect.

Umbilical hernia

Umbilical hernia is pointed out to be an increasing problem in the visited commercial herds. Pigs with large umbilical hernia may suffer and in some cases they are found dead and the herdsmen will suffer from economic losses. In the interviews, it was pointed out that herniated pigs have a lower daily weight gain than not affected pigs, which is shown by Straw *et al.* (2009). Use of saw dust as a bedding material at farrowing can increase umbilical bleeding and negatively affect the healing of the umbilical ring after birth (Lambert Vet Supply, 2009). This is also confirmed by the interviews where some herds had excluded saw dust as a bedding material at farrowing and observed a decrease of umbilical hernias.

Defects in the umbilical region have a complex background. Any defect or protrusion at the umbilical region is often referred to as an umbilical hernia. The actual content of the umbilical sack is not easily determined on live pigs and umbilical hernias might be mixed up with umbilical abscesses. Herds with piglet production and partly farrow-to-finish production have in some cases problems to deliver growers with umbilical and scrotal hernia. It was common to make an agreement with the fattening pig producer regarding pigs with hernia present. This will make it easier for the piglet producer to empty the stable. Another option for piglet producing herds could be to euthanize herniated pigs when the defect is detected. Is it worth to raise a herniated piglet to a 30 kg's grower and then maybe not be able to sell the pig? Herds with farrow-to-finish production have the possibility to raise herniated pigs to slaughter within the herd.

Factors influencing presence of umbilical hernias need to be investigated more. How to handle pigs with this defect is one question to be answered. It would be of interest to know if umbilical abscesses will affect the weight gain to the same extent as an umbilical hernia. If there are differences, it would be interesting to find a model for examination and diagnosis on live pigs.

Boar taint

There is a risk for boar taint of the meat if the male pig not is castrated. Any pig found at slaughter with at least one testicle left, either in the scrotum or in the abdomen is tested for skatole level and the producer will have to pay for the test (Samuelsson, 2009). If the test result is under the set threshold, the pig will be paid as a normal fattening pig, 12 -13 SEK/kg (Swedish Meats, 2009). If the test is over the set threshold, the price for the pig will be in the same price level as a boar, 3 SEK/kg (Swedish Meats, 2009). In the visited herds there were different ways to handle pigs with scrotal hernia and cryptorchid pigs. It is interesting that only three herdsmen reported that it was worth the economic effort to call for veterinarian surgery for these pigs. The prevalence of entire male pigs with skatole levels over the set threshold is depending on several different factors and was in one study done by Lundström *et al*. (2003) 2.8%. According to Lindberg (2009) the cost for using veterinarian surgery on pigs with cryptorchidism and/or scrotal hernia is 300 SEK/pig. It could thus be of economic interest to call for veterinarian surgery at cryptorchid and herniated pigs to assure the income from the carcass.

Breeding at a herd level

Replacement gilts are selected and raised in many different ways and the time and criteria for selection differs among herds, table 7. All 12 herds that used two breed rotational crossing did include hernia as a criterion for selection. Five of these herds did not select any gilt from a litter with a defect present. This can be interpreted as a rather high level of skilfulness in selection of gilts. If more herds were informed of the importance of an accurate registration and correct selection criteria when using rotational crossing this might influence the prevalence of defects in commercial herds.

Three herds had contacted Nordic Genetics in three different matters concerning defects observed in their herds. None of the herdsmen felt that they had got any response or feed back. Such reports could have been opportunities for Nordic Genetics to provide all commercial producers with information about different defects and what can be done at a herd level.

Consider defects in breeding programmes

Genetic Markers

Genetic markers have been found for congenital defects in pigs. QTLs for scrotal hernia and defect fore and hind limbs have been found on SSC5 (Ranberg, 2007). It would be of interest to investigate the possibility to find QTLs for cryptorchidism in pigs, as done in mice (Zimmerman *et al*., 1999; Tomboc *et al*., 2000), since there was a positive correlation between scrotal hernia and cryptorchidism in the Yorkshire breed. If more QTLs could be found there is a possibility to check for these QTLs and evaluate purebred animals to exclude animals with the defect alleles from breeding.

Defects as health traits

In Norway, the pig breeding association Norsvin has included congenital defects as health traits in the breeding program. The most important defects (e.g. hernias, cryptorchidism and splay legs) in Swedish pig production, from an economic and animal welfare point of view, could be included in the breeding programmes in Nordic Genetics as well. A prerequisite for an effective selection would be to increase the accuracy of recording of defects in both nucleus and multiplier herds. Another possibility could be to use records from commercial level herds in the breeding evaluation. In that case, it would be necessary to leave good information on the pedigree of both the father and the mother of the litter. One difficulty is the risk for under-reporting of defects in herds. A better control of registration of defects at herd level could be a solution. The heritability estimates at the commercial herd level might be low, but there is a lot more information and thus a higher accuracy when analysing the data if these herds are included.

8. CONCLUSION

According to this study, cryptorchidism is the most common defect in Hampshire and Yorkshire litters. Scrotal hernia is the most common defect in Landrace litters. Significant effects on the prevalence of litters with defects present were found for breed, herd, year and month*parity number. A positive 'genetic correlation' between cryptorchidism and scrotal hernia was found in Yorkshire. Estimated heritabilites were generally low. Splay legs and umbilical hernia were estimated to be the most common defects in commercial herds. Prevalence of umbilical hernias have, according to the interviewed herdsmen, increased during the last two years. Defects can be considered as health traits in breeding programmes if more herds record the true prevalence of defects. Another possibility is to use genetic markers and analyse individuals that are interesting to use in breeding.

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11. APPENDIX

Herd enquiery form

Farm: ___________________________ Date: ____________________

General:

Herd description; (number of sows, production data (number piglets born alive, number weaned piglets) production system, group system, slats in different stables, bedding material, system for feeding, years in production, changes in production, e.t.c):

Routines for cleaning and disinfection between batches:

Number of employees:

Replacement gilts, type of system:

Breeding material/AI dozes/Boars (Nordic Genetics/Avelspoolen):

Slaughter organisation:

Prevalence of defects

Which defects are present, is there variation over year? How common are the defects? Umbilical hernia, scrotal hernia, cryptorchidism, splay legs, tremor, anal atresia, kyphosis, intersexuality, miscellaneous, etc.:

When are the defects first seen (Umbilical hernias, see down below):

Examination of piglets for defects, except daily control:

Control of causes of stillborn piglets/causes of death:

Control of growers/fattening pigs, except daily control, for example control a week before delivery:

Any marking of defect pigs:

Male piglets:

Catration method/Routines for castration. Any control of the umbilical region at castration:

Management of scrotal hernias/cryptorchid piglets at time of castration (culling/stitching/veterinarian surgery e.tc.):

Delivery

External agreement, any agreement on delivery of growers with defects:

How are defect growers raised to fattening pigs in specialized piglet producing farms, for example scrotal hernias, not castrated male pigs, e.t.c:

Any possibilies to raise growers that cannot be sold to fattening pig producers:

Umbilical hernia, when are they first seen, how do you handle pigs with umbilical hernia? What is your opinion about the background and reason to umbilical hernia?

Replacement gilts

Strategy for raising gilts? Housing/Management?

Selection and control of gilts, at what ages are the selections done? Most common defect present/ reasons for excluding gilts from further breeding?

Any change of replacement system during last years? If so, did this affect the prevalence of defects in your herd?

Miscellaneous:

Recording of defects, sow card, PigWin, control in different batches, e.t.c?

Any change of breeding material during last years (from Nordic Genetics to Avelspoolen or vice versa)? If yes, did you feel that this affected the prevalence of defects?

Any other comments or information that is important to point out: