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PORK INDUSTRY INFORMATION

Congenital defects as a general category occur in pigs across a broad percentage estimated between 0.11% and 4.96% (Straw et al., 2008). Inguinal and scrotal hernias are among the most common of these developmental defects of pigs. Umbilical and inguinal hernias have been reported by one source as occurring in 0.4% to 1.5% of all pigs (Keenlside, 2006). Various countries have reported prevalence for scrotal hernias with ranges from 0.22% to 5% (Ontario Canada, Germany, Netherlands and Thailand). Breed differences have been reported from 0.6%, 1.0% and 1.5% for Duroc, Landrace and Yorkshire breeds, respectively (Vogt and Ellersieck, 1990).

Inguinal hernia appears as a bulge over the external inguinal ring. Scrotal hernia appears when intestinal contents are located in the scrotum. Inguinal/scrotal hernias develop when there is an abnormally large and open vaginal orifice (Warwick, 1926) (comprised of internal and external inguinal rings) through the abdominal wall which allows the abdominal contents to be forced into the inguinal canal in response to increased intra-abdominal pressure at or after birth. An inguinal hernia might not be apparent at birth but can develop some weeks later.

Economic impact from hernia problems is evidenced through negative affects of poor growth, mortality rate and condemnation rates. The major economic consequence is death due to evisceration (expulsion of intestines or omentum) after castration, although intestinal strangulation may also occasionally cause death. The condition is of less significance where male pigs are not castrated and are slaughtered under 6 months of age so that boar odor or taint is not a problem.

Inguinal and scrotal hernia defects can be significantly affected by genetic (Edwards and Mulley, 1999; St. Jean and Anderson, 2006) and environmental (handling) factors. Studies of the genetic affects have been documented back to 1926 (Warwick). It is believed multiple genes are involved. Environmental or animal handling factors can create a predisposition to increased prevalence of inguinal hernias. This includes sudden severe abdominal pressure that may increase the incidence of ruptures. Improper castration procedures such as handling pigs aggressively, extremely long and deep incisions in castration or pulling the testicle cords or induction of farrowing (disrupts the closure of the vaginal orifice). Proper castration technique is important to eliminating hernia issues. Refer to Newsham Choice Genetics' guidelines for *Castration of Pigs*.

BRIEF REVIEW OF THE GENETICS OF SCROTAL (INGUINAL) HERNIA

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There is pressure on the swine genetic community to provide a remedy for reduction of the frequency of hernia problems. A great deal of effort is applied today to identify and describe genetic effects of this commercial problem. The problem of scrotal hernias is exasperated because the breeding and genetics community have yet to find an explanation that accounts for the changing frequency of this trait – there is considerable evidence to suggest that the probability of expressing a scrotal hernia has a heritable component but there is no single environmental factor identified that the genetic and breeding community can manipulate to change the frequency of expression. Selection has been aggressively practiced to reduce the frequency of any genes that may be associated with the trait; boar and gilt pigs are not retained to enter the nucleus population that come from a litter with a male pig expressing the trait. Obviously, the boar that does express the trait is also culled. These culling practices have had little apparent impact on the frequency of scrotal hernias over time.

There are three primary methods to determine if a trait has a genetic component; the first is to look at the differences between populations, the second is to look at the differences that exist within populations and associate variation that exists within a population by knowledge of the families, and the third is to practice selection on the trait and observe if the value changes. All three of these methods have been used to demonstrate that there is a genetic component that is influencing the expression of scrotal hernia.

In 1951, a study was published by WT Magee that demonstrated different frequencies of scrotal hernia occurrence in 13 different lines of pigs. The lines ranged from over 15% to 0.6% occurrence in boar pigs produced. In 1979 (Mikami and Fredeen), the percent incidence of scrotal hernia for Middle Yorkshire and Landrace showed that the two populations differed by 4.2%. Other papers exist in the literature showing differences in populations, which is a primary indicator of some type of genetic influence.

Variation within populations is often presented in the form of a heritability estimate. There are a number of published heritability estimates for the incidence of scrotal hernia. These estimates range from nearly zero to 0.84 (Magee, 1951; Mikami and Fredeen, 1979; Knap, 1986). An interesting result from the work presented by Magee (1951) is evidence of a “maternal” effect that is in the same magnitude as the additive genetic effect. This suggests that the sow may create an environment that is more or less favorable to the expression of scrotal hernias. There may be value in developing selection protocols that try to identify both sows with favorable maternal environments and individuals with lower probabilities for the expression of a scrotal hernia.

A selection study was conducted by Warwick (1926) where boars with a scrotal hernia were bred to sows with littermates that expressed a scrotal hernia and the number of individuals that expressed a scrotal hernia was observed. Warwick describes a rapid increase in observed frequency of scrotal hernias during the study. In only three generations of selection, the frequency of males produced expressing scrotal hernias increased from under 2% to over 40%.

It would appear there is little doubt that scrotal hernias have, to some degree, a genetic component. Newsham Choice Genetics assumes that the underlying genetic mechanism influencing hernias is that of a quantitative threshold trait with incomplete penetrance. Or, more simply, the trait expresses itself in a binary fashion, the boar pig either expresses a hernia or it does not. When it expresses the trait, we assume that it has exceeded some threshold level. We cannot differentiate the degree of severity that the scrotal hernia expresses itself – it is either normal (no observed scrotal hernia) or it is abnormal (expression of a scrotal hernia). The incomplete penetrance suggests that there may be environmental factors that influence the expression, so that two individuals with the exact same genetic value may or may not express the trait depending on the environment in which they are born. This is how populations raised in different environments can have different average expression values.

Newsham Choice Genetics estimates a breeding value for this trait. Individuals with higher breeding value estimates are excluded from the breeding population. This, along with not allowing siblings from a litter with abnormalities and the culling of the sow, places a considerable degree of selection pressure against the increased frequency of the trait in monitored populations.

The limitation of using BLUP estimates for the EBV of scrotal hernia is that it has a variety of statistical problems that appear to reduce the accuracy of estimating the true value for any individual. A new technology available to geneticists to improve the accuracy of selection is the use of Marker Assisted Selection (MAS). If identification of genetic markers can be associated with influencing the expression of scrotal hernias, breeding values that include both markers and pedigree information may assist in reducing the frequency in which scrotal hernias are expressed.

There have been a number of studies conducted that attempt to identify markers that may be associated with the expression of scrotal hernias. The marker work has looked at both broad genome scans (Ding et al., 2009) and candidate gene expression (Du et al., 2009). Both methods are yielding information that will eventually be useful in identifying markers that will improve the accuracy with which breeding values are estimated. These studies have identified informative regions on Chromosome 2 of the pig that will eventually assist in predicting the breeding value for scrotal hernia. As studies continue, additional markers will be identified and included in the MAS BLUP estimation procedures.

It is evident that there is a genetic component that influences the expression of scrotal hernias; it can be shown that populations differ, that families within a population vary in their expression of the trait, and that the trait can be selected for and the frequency of occurrence increased. Concurrent with the work that was started in the early 1920's to study scrotal hernias, a variety of genetic mechanisms have been postulated that explain the control of the trait. Early studies suggested a double recessive genetic model where two independent genes work together to determine the expression of the phenotype. This model was proposed in 1926 by Warwick, but recently studies using both genome wide scan techniques and candidate gene analysis have failed to identify major genes that would account for a major component of variation in the trait.

The first recorded reference to scrotal hernias was in 1847. A number of papers and texts published prior to Warwick's selection study published in 1926 suggest that scrotal hernias are a heritable trait. Warwick makes the following recommendations when working with a population with a higher incidence of hernias than is acceptable:

Never use a boar for breeding purposes which is or ever has been afflicted with inguinal hernia.

Discard any normal boar which has sired one or more herniated pigs.

Do not select breeding-stock from sows which have produced one or more inguinally herniated pigs.

Do not retain for breeding purposes littermates to inguinally herniated boars.

Elimination of all of the progeny of boars which have been known to sire inguinally herniated pigs is also advised.

Each of these recommendations is based on the logic of placing selection pressure against the trait and reducing the genes that influence the expression of scrotal hernias regardless of the genetic model that is thought to control the incidence of this trait.

Selection to reduce the frequency of scrotal hernias has been practiced to some degree in all breeding stock since the early 1920s. The following table shows the frequency of this trait as reported by different authors over time and around the world. It does not appear that the severity of the problem has increased or decreased.

AUTHOR	YEAR	BREED	INCIDENCE
WARWICK	1926	Jersey Duroc	1.68%
BERGE	1941	Large White and Norwegian Landrace	1.99%
MAGEE	1951	Various	5.10%
MIKAMI AND FREDEEN	1979	Yorkshire	6.60%
		Landrace	2.40%
KNAP	1986	Dutch Landrace	2.00%
		Dutch Yorkshire	2.56%
VOGT AND ELLERSIECK	1990	Duroc	0.60%
		Landrace	1.00%
		Yorkshire	1.50%

SUMMARY

The incidence of scrotal hernias has a genetic component that influences the expression of the trait.

Genetic models proposed to describe the inheritance have not been effective in optimizing selection practices that should effectively reduce the incidence of the trait.

The trait has been a problem since the scientific community started looking at the genetic component. It continues to be problematic and, in spite of efforts to select against it, will continue to be a commercial production issue.

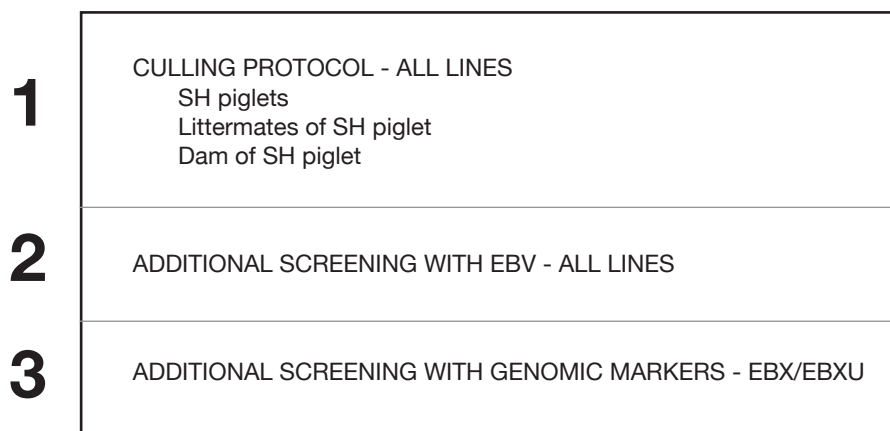
The industry traditionally places blame on the boar as the primary cause of increasing the incidence of scrotal hernias. This is contrary to most genetic models and the breeding and genetics industry knows that the dam is contributing both 50% of the offspring's genes and a maternal environment that may also influence the expression of the trait.

NEWSHAM CHOICE GENETICS - REDUCING GENETIC RISK FOR SCROTAL HERNIA

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Research by Newsham Choice Genetics and others has confirmed that incidence of scrotal hernia in pigs has a significant genetic component. Figure 1 depicts our strategy and procedures for reducing genetic risk.

FIGURE 1



- 1. At the base of this approach in all lines is a process of rigorous culling of immediate families within the Genetic Nucleus in which a scrotal hernia occurs. When a scrotal hernia piglet is observed in any line, that piglet, its full sibs and its dam are culled from the breeding herd. Culling of sires of affected piglets is not included in this base protocol because the sires have already left the herd due to rapid turnover of generations.**
- 2. We also utilize Estimated Breeding Values (EBV's) for scrotal hernia risk, which include incidence information not only from immediate families but from throughout the entire line pedigree. EBV's are generated for all individuals in all Genetic Nucleus herds within Newsham Choice Genetics. Thresholds for these EBV's are used for additional screening for high-risk animals.**
- 3. In addition to the base culling and use of EBV's for scrotal hernia applied in all lines, we use genomic markers for scrotal hernia in EBX to improve the accuracy with which we select against risk for scrotal hernia.**

Just as in commercial systems, the exact incidences observed in our Genetic Nucleus herds depend on the time period and farms characterized, but typical observed incidence is less than 2% of male piglets in lines based on Duroc, Landrace or Large White and slightly more than 2% of male piglets in Pietrain-based lines. Consequently, we have focused our genomics studies of scrotal hernia in our EBX population which was originally derived from the Pietrain breed. In 2004 we reported the results of a scan of the EBX genome (Du et al., 2004a,b) in which locations were mapped on 2 chromosomes that are associated with incidence of scrotal hernia. More recently, we have teamed with Iowa State University to discover specific genes within these and other genomic locations that are strongly associated with incidence of scrotal hernia (Du et al., 2009; Zhao et al., 2009).

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