Effects of porcine stress syndrome genotype on maternal traits in swine

by

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Studies were conducted to evaluate maternal performance differences between females with different porcine stress syndrome (PSS) genotypes. Two data sets were used to conduct this evaluation. The first involved 870 records from 333 females from a synthetic line of pigs selected for their PSS susceptibility. The second data set involved 3100 records from 841 purebred Landrace females. Only records from normal (NN) and carrier (Nn) dams were included.

The first study revealed that Nn females farrowed 0.91 and 0.69 more ($P<0.05$) live pigs than NN and positive (nn) females, respectively. No difference in litter birth weight of pigs born alive or proportion of pigs surviving from birth to transfer was observed between any dam PSS genotypes.

Normal females had 0.57 more ($P<0.05$) pigs at birth than did nn females. Normal sows raised 0.49 more ($P<0.05$) pigs to 21 d than did nn females. Normal sows produced 6.20 kg and 2.86 kg heavier ($P<0.05$) 21-d litter weights than did nn and Nn females, respectively. The proportion of pigs surviving from transfer to 21 d favored ($P<0.05$) NN dams by 13.1% and 9.3%, respectively, compared to nn and Nn dams. Normal females had 0.24 more pigs per litter at 42 d although the difference was not significant. Normal and Nn

dams produced ($P<0.05$) 8.22 kg and 5.16 kg, respectively, heavier litters at 42 d. A 7.69% and 4.48% survival rate to 42 d advantage ($P<0.05$) was found in litters of NN and Nn sows, respectively, when compared to nn sows. Normal and Nn dams had similar survival rates of their litters to 42 d.

The results from the study involving NN and Nn purebred Landrace dams revealed no significant differences for number born alive, number at 21 d, 21-d litter weight, survival rate to 21 d, and farrowing interval. This data set was also used to estimate genetic parameters for these traits. Heritabilities ranged from 0.04 to 0.06 for number born alive, from 0.21 to 0.32 for number at 21 d, and from 0.14 to 0.21 for 21-d litter weight depending on the method of adjusting for environmental factors. Heritabilities of 0.21 and 0.02 were found for survival rate to 21 d and farrowing interval, respectively.

GENERAL INTRODUCTION

Porcine stress syndrome (PSS) is an inherited neuromuscular disorder in pigs (Christian, 1972). The PSS condition is controlled by a defective recessive allele at a single genetic locus which results in three possible genotypes (normal, carrier, and positive). The PSS condition was first described by Topel et al. (1968), who noted physically stressed, susceptible pigs would collapse in a shock-like state and die. Much attention has been given to the PSS gene effects on muscle quality and performance of market hogs since its discovery. Increasing consumer and packer demand for lean meat has led to an increase in the use terminal sires with 1 or 2 copies of the PSS gene by producers of market hogs because of its perceived advantage in the production of lean, heavy muscled hogs. A large proportion of the homozygous recessive (nn) PSS positive animals and those heterozygous (Nn) animals carrying one copy of the PSS gene produce carcasses having inferior muscle quality (Murray et al., 1989; Christian et al., 1993). Debates have occurred in the popular press concerning the use of the PSS gene, particularly in terminal sire lines. The increased use of terminal sires having at least one copy of the PSS gene coupled with the fact that the majority of commercial swine producers still raise their own replacement breeding herd females, may result in an increase of the PSS gene frequency in the reproducing female population.

Sow productivity traits have a large impact on the profitability of commercial and seedstock swine producers (Mabry et al., 1990). Economically important sow productivity traits include: number of pigs

born alive, litter birth weight, number of pigs weaned, 21-d litter weight, and litters per sow per year (MacKay, 1993).

Few investigations have examined the effects of the PSS gene on maternal traits in swine. The majority of the investigations have focused on differences between stress susceptible (nn) and stress negative (NN or Nn) animals because of the difficulty in distinguishing between normal (NN) and carrier (Nn) animals based on phenotype alone. No consistent maternal productivity differences have been observed between stress susceptible and stress negative females. Those studies having reproductive performance results for NN, Nn, and nn dams as distinguishable groups are few and involve small numbers (Nystrom and Andersson, 1993) or were conducted when less precise methods were available to distinguish between NN and Nn females (Mabry, 1977, and Schnieder et al., 1980). Molecular biology advancements have resulted in the development of a simple and cost effective procedure to determine the PSS genotype of animals with accuracies approaching 100% (Fujii et al., 1991). Swine producers can submit blood samples from individual pigs to a licensed laboratory and have PSS genotype determined by the molecular method. Individual swine producers can then determine the appropriate use of the PSS gene to meet their breeding objectives.

The first objective of this study was to determine PSS gene effects on sow productivity in a PSS susceptible research herd. Reproductive performance data have been collected since 1975 in the composite stress-susceptible herd at Iowa State University. During this time a variety of methods have been used to determine the PSS genotype of the animals in this

born alive, litter birth weight, number of pigs weaned, 21-d litter weight, and litters per sow per year (MacKay, 1993).

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herd. The maternal performance data collected include: number of pigs born, number of pigs born alive, percentage of pigs surviving from birth to transfer, number of pigs alive at 21 d, 21-d litter weight, percentage of pigs surviving from transfer to 21 d, number of pigs alive at 42 d, 42-d litter weight, and percentage of litter surviving from transfer to 42 d.

The second objective of this study was to determine maternal performance differences among NN and Nn Landrace females. Registration numbers and PSS genotype (determined by the molecular test) of females were obtained from 9 purebred Landrace seedstock producers. Maternal performance data were obtained from the American Landrace Association. Maternal data were obtained for the following traits: number of pigs born alive, number of piglets alive at 21 d, 21-d litter weight, percentage of pigs surviving from transfer to 21 d, and farrowing interval.

The last objective was to examine the effects of different maternal-environmental adjustment factors on fixed effect and heritability estimates. Both previously mentioned data sets were used in this portion of the study. Current National Swine Improvement Federation adjustments and those adjustments reported by Brubaker et al. (1994) for parity, number after transfer, and age at which 21-d litter weight was taken were applied to both data sets. Separate estimates of PSS genotype effects and heritability estimates were calculated for each data set.

Dissertation Organization

This dissertation is presented as a general introduction, a general review of literature, three individual papers to be submitted to the Journal of Animal Science, and a general concluding summary. References

cited in the general introduction and the general review of literature follow the general summary section. All cited references follow the CBE Style Manual used by the Journal of Animal Science. Each paper consists of an abstract, introduction, materials and methods, results and discussion, and an implications section. References cited within each individual paper follow the implications section. Any additional tables or explanations of material follow the reference section of each individual paper in an appendix.

LITERATURE REVIEW

This literature review is divided into 4 sections. The first section will cover the porcine stress syndrome (PSS) in some detail. The remaining sections, genetic aspects of sow productivity, sow productivity environmental adjustment factors, and lastly, mixed model methodology and derivative-free restricted maximum likelihood procedures will be more briefly discussed. No attempt was made to cover every research article concerning each subject. An emphasis was placed on articles of historical value and an attempt was made to cover the development of knowledge concerning each subject across time.

Porcine Stress Syndrome

Porcine stress syndrome (PSS) is an inherited neuromuscular disorder found in swine. In an attempt to use pigs as a model for a similar condition in humans called malignant hyperthermia (MH), Hall (1967) reported that three pigs suddenly died when exposed to halothane and suxamethonium anesthesia. Topel et al. (1968) was the first to report that when stressed, some pigs would collapse in a shock-like state and die, thus the malady was given the name Porcine Stress Syndrome. Since these early reports, numerous reviews of PSS and its effects on various production and carcass traits have been published (Cassens et al., 1975; Harrison, 1979; Mitchell and Hefferon, 1982; Webb et al., 1982; Webb et al., 1985; Christian and Mabry, 1990; and Christian and Lundstrom, 1992).

Relation to human malignant hyperthermia

Similarities between malignant hyperthermia (MH), a rare inherited disorder in humans, and PSS have been noted. MH in humans is a condition occurring in surgical patients undergoing anesthesia. Most often the condition occurs if the anesthetic halothane and the muscle relaxant succinylcholine are used to anesthetize the patient (MacLennan and Phillips, 1992). Typical symptoms of MH in humans are very similar to those seen in PSS susceptible pigs. The malady can be avoided in humans by using alternative anesthetics and muscle relaxants (MacLennan and Phillips, 1992). MH affects an estimated 1 in 15,000 to 1 in 50,000 of the individuals undergoing anesthesia (MacLennan and Phillips, 1992). Distinct differences between PSS and MH include mode of inheritance and triggering mechanisms (Hefferon and Mitchell, 1985). MacLennan and Phillips (1992) suggested an association between MH and other human disorders including Barne's myopathy, central core disease, King-Denborough syndrome, and Duchene muscular dystrophy. The similarities between the two maladies have led to the use of similar physiological tests for the detection of PSS and MH (Heffron and Mitchell, 1985).

Triggering mechanisms and symptoms

Many physical stresses can result in the expression of PSS. Exercise, fighting, marketing, vaccination, castration, estrus, mating, parturition, and hot weather are all examples of stressors that can trigger PSS (Marple et al., 1985). It has also been noted that volatile anesthetics like halothane can bring about the onset of PSS (Christian, 1974; Webb and Jordon, 1978). Symptoms exhibited by a pig during a PSS

episode include: muscle and tail tremors, labored and irregular breathing, blanching and reddening of the skin, rapid rise in body temperature, acidosis, cyanosis, collapsing of the pig, muscle rigidity and eventual death (Christian and Lundstrom, 1992). Once PSS in pigs or MH in humans is triggered, the symptoms proceed rapidly. One of only a few of the known remedies for either condition is dantrolene sodium administered intravenously (Harrison, 1975; Kolb et al., 1982; Louis et al., 1990; MacLennan and Phillips, 1992). Dantrolene sodium is a muscle relaxant that inhibits calcium (Ca^{2+}) release from the sarcoplasmic reticulum (SR) of skeletal muscle cells (Kolb et al., 1982) but has no effect on cardiac or smooth muscle cells (Harrison, 1975). Loss of muscle rigor, a decrease in muscle temperature and termination of acidosis occur after prompt administration of dantrolene sodium (Harrison, 1975).

Mode of inheritance

Many modes of inheritance were proposed in early investigations of the PSS condition. Control of the PSS by a dominant gene at a single locus (Hall et al., 1972) or two separate loci (Britt et al., 1978; Carden et al., 1983) were proposed in early research trials. It is now clear that PSS is inherited in a single gene autosomal recessive manner with incomplete penetrance, as first proposed by Christian (1972) and later confirmed in several other studies (Minekema et al., 1976; Webb and Smith, 1976; Mabry, 1977; Mabry et al., 1981; O'Brien, 1986a; Louis et al., 1990). Molecular studies have also confirmed this mode of inheritance (Fujii et al., 1991).

There is speculation that a single mutation occurring in a single animal is the progenitor of the PSS condition (Fujii et al., 1991; O'Brien, 1995). By analyzing the co-segregation of 2 polymorphisms in five distinct breeds of PSS susceptible pigs Fujii et al. (1991) concluded that there was common ancestry for all of the animals across breeds. O'Brien (1995) suggests that this mutation occurred in Germany in the early twentieth century as first reports of meat unsuitable for the sausage industry were first described in 1914. It is also suspected that the newly formed recessive gene was the impetus for the development of the Pietrain breed in Belgian and the ancestors of the Poland China breed developed in the U.S. (Christian, personal communication). The recessive PSS gene has probably found its way into other breeds of the world through migration and not due to further mutations.

Chromosomal location

Susceptibility to PSS has been determined by reaction to the anesthetic halothane (Christian, 1974). This led to the gene responsible for PSS being named the HAL gene and the marker loci near the HAL gene designated as the HAL linkage group (Juneja et al., 1983; Honjy et al., 1985; Van Zeveren et al., 1988). This linkage group consists of several erythrocyte and isoenzyme marker loci for the HAL gene and includes: H blood group system (Rasmusen and Christian, 1976); S (A-D) blood group (Rasmusen, 1964; Rasmusen, 1972); phosphohexose isomerase (PHI) (Andersen, 1970); 6-phosphogluconate dehydrogenase (PGD) (Rasmusen et al., 1980); and post albumin-2 (PO2) (Juneja et al., 1983). Assignment of HAL and its linkage group was made to the long arm of chromosome 6 in the pig (Davies

et al., 1988 and Fujii et al., 1991) and later confirmed by Mariani et al. (1992). The exact order of HAL and the marker loci was found to be S, HAL, PHI, H, PO-2 and PGD (Vogeli, 1989). Additionally, Harbitz et al. (1990) placed the Ca^{2+} release channel gene, a candidate gene for the PSS, on the long arm of chromosome 6.

The mutation responsible for PSS is also known as HAL-1843 after the molecular discovery of the location of the defect within the genome of the pig (Fujii et al., 1991). Genotypic designation for stress resistant, stress carrier and stress positive are NN, Nn, and nn, respectively, and will be designated as such throughout the balance of the dissertation. Following the patent of the molecular test by the University of Toronto, the HAL-1843 designation was required of all animals tested by this method. Classification of stress resistant, stress carrier and stress positive under this system is non-mutant (nm), mono-mutant (mm) and di-mutant (dm), respectively, (O'Brien, 1995).

Frequency and gene frequency

The frequency of the nn genotype varies according to breed and country of origin. Webb and Jordon (1978) reported that 20% of Pietrain-Hampshire and 0.0% of the Duroc, Yorkshire, and Large White pigs exhibited positive reactions to halothane. A 8.5% halothane susceptibility was reported for 1495 Danish Landrace pigs (Jensen, 1979). Webb et al. (1982) summarized numerous studies and found that the Large White or Yorkshire breed had a very low or zero frequency of halothane positive (HP) pigs while 88% of the Belgium Pietrain pigs tested were HP. The Landrace breed had a range of HP pigs from 5% to 86% depending on country of origin (Webb

et al., 1982). Christian and Mabry (1990) also summarized several studies indicating that Dutch Pietrain were 100% HP while the Large White breed from several countries had 0% HP pigs. Kallweit (1985) reported a range in frequency of HP pigs from 0.0 to 100 percent. The Landrace breed frequency ranged from 1.1% to 100% HP depending on country of origin (Kallweit, 1985).

A report of being HP does not reveal the whole story. Pigs having 1 copy of the stress gene (n), as is the case with Nn animals, do not respond to halothane. The PSS gene frequency estimates are less common than reports of the number of pigs reacting to halothane. Hubbard et al. (1990) reported maximum likelihood estimates for 2554 Landrace and 2566 Yorkshire tested boars from Canada to be $0.10 \pm .02$ and $0.09 \pm .02$, respectively. Estimates of HAL gene frequency in 1664 Landrace and 1764 Large White pigs from 9 British nucleus herds were estimated as $0.33 \pm .03$ and $0.11 \pm .02$, respectively (Southwood et al., 1988). Houde et al. (1993) reported a HAL gene frequency of 0.03, 0.15, and 0.11 for Duroc, Landrace, and Yorkshire pigs in Canada. Goodwin (1994) found an overall gene frequency of .071 across 8 pure breeds in the purebred progeny test conducted for the National Barrow Show from 1991 to 1993. Estimates within breeds ranged from 0.00 for the Chester White breed to 0.419 for the Poland China breed, and frequency of the PSS gene was 0.063 in Landrace progeny (Goodwin, 1994). O'Brien (1995) published PSS gene frequencies by breed and by country of origin within breed. The Pietrain breed had the highest PSS gene frequency (0.50) of all breeds tested while the Chester White breed had the lowest breed frequency of the HAL gene (0.00) (O'Brien, 1995).

Canadian, U.S., and English estimates of HAL gene frequency in the Landrace breed were 0.18, 0.21 and 0.22, respectively (O'Brien, 1995).

Physiological basis

Early research noted that PSS was associated with abnormal acidosis and production of pale, soft, and exudative (PSE) skeletal muscle (Topel et al., 1968). It was also noted that a rapid rate of postmortem glycolysis occurred in the M. longissimus dorsi (M. ld) muscle of carcasses exhibiting PSE and resulted in rapid decline in pH of the M. ld (Topel et al., 1968).

For normal muscle contraction to occur, Ca^{2+} must be released through the SR. Relaxation of muscle and reduction of metabolic activity are controlled by a Ca^{2+} pump located in the SR (O'Brien and MacLennan, 1992). Thus, extreme muscle rigidity and excessive metabolic heat generated in PSS susceptible animals carries the connotation of defective Ca^{2+} regulation (O'Brien and MacLennan, 1992). PSS is triggered by volatile anesthetics such as halothane and by muscle relaxants such as suxamethanium chloride (Gronert, 1994). This results in increased muscle metabolism and muscular contractions which lead to increased production of lactic acid, carbon dioxide and heat production within the muscle (Gronert, 1994; Webb et al., 1985). Increased and prolonged rise in cytoplasmic Ca^{2+} of skeletal muscle results in increases of aerobic and anaerobic metabolism and respiratory acidosis (Louis et al., 1990). Muscle cells are unable to properly regulate cytoplasmic Ca^{2+} due to improper shutting of the Ca^{2+} release channel and the inability of the Ca^{2+} pump found in the SR to replenish cytoplasmic Ca^{2+} quickly (Louis et al., 1990). Properties of the heterozygous animal (Nn) were found to be intermediate to those of normal

and PSS susceptible animals suggesting that normal and abnormal genes coding for the Ca^{2+} release channel exist in these animals (Louis et al., 1990).

The physiological mechanisms resulting in PSS have been extensively studied. It was suggested by O'Brien (1986a) that PSS in swine was initiated by a mechanism resulting in hypersensitive Ca^{2+} release by the SR that is inherited in an autosomal recessive manner. Halothane and caffeine have been shown to increase cytoplasmic Ca^{2+} (O'Brien, 1986a). Calcium release is regulated by a mechanism found in the terminal cisternae of the SR (Endo, 1977). O'Brien (1986b) suggested that PSS was not due to the reduced ability of muscle cells to sufficiently take up Ca^{2+} after the onset of PSS. Release of Ca^{2+} from the SR of muscle was shown to be stimulated in rabbits by the plant alkaloid ryanodine (Imagawa et al., 1987). Imagawa et al. (1987) demonstrated that ryanodine binds to the Ca^{2+} release channel of rabbit muscle cells, thus the channel has been termed the ryanodine receptor. The release of Ca^{2+} by the SR is controlled by micromolar Ca^{2+} or millimolar ATP and ryanodine results in the channel to open for long periods of time (Imagawa et al., 1987).

Mickelson et al. (1988) concluded that a major abnormality exists in the ryanodine receptor found in muscle cells of PSS susceptible animals. Since the ryanodine receptor regulates Ca^{2+} release from the SR, abnormalities found in the area could be responsible for differences in extracellular Ca^{2+} between PSS susceptible and non-susceptible animals (Mickelson et al., 1988). Ohta et al. (1989) found that Ca^{2+} release was induced by Ca^{2+} at much lower concentrations in PSS susceptible pigs

compared to non-susceptible pigs. Additionally, maximum rate of Ca^{2+} release was 3 times higher in PSS susceptible vs. non-susceptible animals leading to the conclusion that the cause of PSS is an enhancement of Ca^{2+}-induced Ca^{2+}-release (Ohta et al., 1989). Ohta et al. (1989) also found an increase in the release of Ca^{2+} when exposing muscle fibers from PSS susceptible and non-susceptible animals to halothane and caffeine. Muscle fibers from susceptible animals were stimulated to release Ca^{2+} by lower concentrations of halothane and caffeine when compared to non-susceptible pigs (Ohta et al., 1989). It has been reported that Ca^{2+} release from the SR of muscle fibers obtained from PSS susceptible pigs was triggered by Ca^{2+}, caffeine and ATP at one tenth the concentrations needed to cause Ca^{2+} release in control animals (O'Brien, 1990). This seems to suggest that the mechanism controlling Ca^{2+} release was hypersensitive to triggering agents.

The DNA sequence of rabbit skeletal muscle SR was deduced by Takeshima et al. (1989). It was further concluded that the Ca^{2+} release channel lies in a "foot" structure between the SR and the transverse tubules of skeletal muscle cells (Takeshima et al., 1989). The ryanodine receptor previously described (Imagawa et al., 1987) was found to be the only channel of known sequence that matched by computer survey the DNA sequence found in the rabbit muscle SR (Takeshima et al., 1989). Fill et al. (1990) proposed the actual defect responsible for PSS condition in pigs was in a low affinity Ca^{2+} binding site and suggested that a mutation in a gene coding for the Ca^{2+} release channel was likely responsible. Fill et al. (1990) also suggested that once the Ca^{2+} release channel of PSS susceptible animals is opened, it does not shut as quickly as the Ca^{2+}

release channel of normal pigs. If true, he concluded that a hypothesis of a molecular basis for the abnormal calcium release channels could be tested.

Using proteolytic digestion followed by immunoblot staining with polyclonal antibody against the rabbit Ca^{2+} release channel, Knudson et al. (1990) demonstrated that normal and PSS-susceptible pigs differed in their Ca^{2+} release channel. Trypsin, a proteolytic enzyme, was used to digest the SR of normal and PSS susceptible animals and different immunoblot patterns were observed. They concluded that the SR of PSS susceptible animals contain one or more trypsin cleavage sites not present in the SR of normal animals. Additionally, it was found that the immunoblot staining pattern of a heterozygous animal was intermediate to that of normal and PSS susceptible pigs. It was determined that halothane sensitivity is identical to the Ca^{2+} release gene with a mutation as the cause of different peptide immunoblot patterns between animals susceptible and resistant to PSS. They also suggested the mutation may have resulted in the loss or gain of a trypsin cleavage site produced by the amino acids arginine or lysine. Any mutation causing a change in the structure of the Ca^{2+} release channel could be responsible for the different immunoblot patterns, differential binding of ryanodine and the hypersensitive release of Ca^{2+} (Knudson et al., 1990). The ryanodine receptor of slow twitch muscle (cardiac and vastus intermedius skeletal muscles), as well as fast twitch muscle (normal skeletal muscle) previously described by Louis et al. (1990), show the same defect in the Ca^{2+} channel of PSS-susceptible pigs (Ervasti et al., 1991).

The previously mentioned studies of the Ca^{2+} release channel no doubt provided valuable information to Fujii and coworkers (1991) when they discovered the mutation site for the defective allele and developed a molecular test for PSS. Fujii et al. (1991) compared full length ryanodine receptor cDNAs from PSS susceptible and non-susceptible animals. Of the 18 single nucleotide polymorphisms between the two animals, one polymorphism led to an alteration in amino acid sequence. A cysteine amino acid replaced arginine at position 615 which resulted from a single base switch (cytosine (C) to thymine (T) at nucleotide 1843) of the sequence coding for the ryanodine receptor. This confirms the findings by Knudson et al. (1990) that an alteration of a tryptic cleavage site in PSS susceptible animals was responsible for PSS. Mickelson et al. (1992) supported this theory when they proposed that an 86 and a 99 kDd tryptic fragment containing the normal and mutated 615 residue could be found in the ryanodine receptor of normal and PSS-susceptible animals. Further tests were conducted on 182 pigs from six different breeds to confirm that the amino acid switch was responsible for PSS susceptibility. Genomic DNA was isolated from muscle biopsies and amplified by polymerase chain reaction (PCR). Two oligonucleotide probes were used to detect the alleles having C and T. The mutation (C to T) was found to delete a HinPI and creates a HgiAI restriction enzyme site, allowing a simple, accurate, and non-invasive molecular test for PSS (Fujii et al., 1991). They further suggested that the amino acid alteration is found on the surface of the Ca^{2+} release channel and is responsible for changes in the gating of Ca^{2+} in the SR of skeletal muscle cells. Mickelson et al. (1992) confirmed that

the domain containing this mutation is near the surface of the Ca^{2+} release channel and is important in differential regulation of Ca^{2+} release from the SR of normal and PSS-susceptible pigs.

Brenig and Brem (1992) completely sequenced subclones around the mutation at base pair 1843 previously described by Fujii et al. (1991). A 134 base pair exon that contained the mutation was found. Primers were designed to amplify the complete exon by PCR techniques. Using the Hal restriction enzyme, it was determined that the two HAL alleles from NN pigs would be cut into 50 and 84 base pair-length fragments. The DNA restriction patterns produced by nn animals would exhibit only the 134 bp fragment or remain uncut. The Nn animals would produce all three fragments (134, 84 and 50 base pairs in length), having 1 normal allele that would be cut and 1 abnormal allele that would remain uncut. A similar molecular test has been described by Otsu et al. (1992) for MH in humans.

Detection methods

Methods of detecting PSS-susceptible animals have evolved as new technology has become available. The least expensive and easiest method of detection is subjective visual appraisal. Topel et al. (1968) found stress susceptible animals to be very heavily muscled with little backfat. Christian (1974) suggested that not all heavy muscled pigs are stress susceptible but all stress susceptible animals were heavy muscled. He further suggested that pigs with tight jowls and middles, large round (basketball shaped) hams with well defined muscle separation, and of small stature are suspected to be carrying the genetic factors contributing to PSS. This method is inexpensive and easily utilized by a trained

evaluator; however, its failure to accurately classify all animals, particularly Nn animals, limits its practical use (Christian and Mabry, 1990).

Prior to 1991, the anesthetic halothane was commonly used to determine PSS susceptibility in most research settings. Christian (1974) observed typical PSS symptoms when exposing market sized (105 kg) pigs to the anesthetic halothane. He followed this study by administering 6% halothane gas for 1 minute and 2% for 2 additional minutes to weanling pigs with a face mask through a closed anesthetic machine equipped with a vaporizer. Further refinement of the procedure followed (Christian, 1974; Eikelenboom and Minkema, 1974). Briefly, 7 to 12 week old pigs are exposed to a 3 to 10% halothane-oxygen mixture for approximately 3 minutes. The pig is observed for muscle rigidity (particularly of rear legs), skin blotches, and increased respiration rates. A pig is considered PSS-resistant if relaxation and unconsciousness is observed, usually occurring within 90 seconds after the initial administration of anesthesia. Susceptible pigs usually quickly (30 to 90 seconds) show signs of PSS once the halothane gas is administered and prompt removal of it should occur to prevent death (Christian and Mabry, 1990). Halothane screening was so successful in Dutch Landrace pigs that all pigs (2944) sent to the testing station at Lochem, The Netherlands in 1975 were screened (Eikelenboom, 1979). Webb and Jordon (1979) reported similar success when screening Pietrain-Hampshire crossbred animals from a 2-generation study of divergent selection for PSS-susceptibility. Misclassifications often occur when screening sick and/or unthrifty pigs (Christian, personal communication).

Limitations to widespread halothane screening by commercial breeders include: need for trained technician, cost and bulkiness of the anesthesia machine, and inability to detect Nn animals (Christian and Mabry, 1990). Halothane screening combined with other detection methods (various erythrocyte antigen marker and CPK methods that will be discussed later) has seemed to overcome the inability to detect Nn animals (Rasmusen and Christian, 1976; Rasmusen et al., 1980).

Blood levels of creatine phosphokinase (CPK) have also been used to help ascertain a pig's susceptibility to PSS (Christian and Mabry, 1990). Christian (1974) collected blood from an ear vein of pigs at weaning and slaughter and found elevated levels of CPK in PSS-susceptible individuals. The ear vein was used as the blood collection site to avoid contamination of the sample with muscle tissue, resulting in abnormally high CPK values (Mabry et al., 1983). Mabry et al. (1983) measured 4 CPK values (Sigma CPK, Antonik CPK, log Sigma CPK, and log Antonik CPK) and compared them to halothane sensitivity (6% halothane concentration for 5 min.) to determine PSS susceptibility. PSS susceptible animals had higher ($P<.01$) Sigma (4.5 times), Antonik (10 times), log-Sigma (1.4 times), and log-Antonik (1.95 times) CPK values when compared to non-susceptible pigs. Accuracy of predicting PSS susceptibility ranged from .87 to .91 for the 4 methods of CPK measurement. Serum CPK values were higher after exposure to halothane and prior to slaughter in pigs producing pale, soft and exudative carcasses than those producing normal carcasses (Froystein et al., 1979). Difficulty in distinguishing between Nn and NN individuals is the main disadvantage which limits the use of CPK values by commercial breeders.

Several erythrocyte antigenic markers have been used to predict PSS-susceptibility. Erythrocyte antigenic marker classification can be determined by serological methods (Hojny, 1974; Hojny, 1975). The H blood group system consists of seven alleles (Hojny, 1973). Rasmusen and Christian (1976) determined the H and A-O blood type classification of 144 pigs. The following genotypes were identified in the H system: $H^{a/a}$, $H^{a/-}$, $H^{a/c}$, $H^{c/}$, and $H^{-/-}$ (Ha negative). They concluded that PSS-susceptibility could be determined and was associated most commonly with the haplotype $H^{a/a}$ (in absence of A^a or A^o and H genotypes of the H blood group system). Further work (Christian, 1976) confirmed the early results. The $H^{a/a}$ (and A^{a-},A^o negative) and $H^{-/-}$ were the H blood group haplotypes most commonly associated with halothane determined PSS susceptibility. Sigma and Antonik CPK values were also found to be nearly 4-fold higher for the $H^{a/a}$ and $H^{-/-}$ pigs than for other H blood group genotypes. The only exception to this rule was for the $H^{a/-}$ genotype which had CPK values approximately one-half as large as $H^{a/a}$ (A^a and A^o negative) or $H^{-/-}$. The intermediate CPK values found in $H^{a/-}$ may have been the result of heterozygous PSS-susceptible animals. The close association of halothane-susceptibility and CPK values with the H blood group genotype suggested that PSS-susceptibility could accurately be determined by genotyping pigs for the H blood group locus. The association between CPK values and H blood group genotype was supported by Glodek et al. (1985). Christian (1977) suggested that the H blood group was closely linked to the gene controlling PSS. Furthermore, PSS-susceptible animals and most importantly, the Nn animals might also be identified.

Scottish researcher's findings supports the conclusion that $H^{a/a}$ in absence of the A^a or A^o is associated with PSS susceptibility (Imlah and Thomson, 1979). Danish studies suggest a slightly different association between halothane responders and the H blood group genotype in Danish Landrace pigs (Jorgensen, 1979). $H^{-/-}$ commonly responded to halothane in a positive manner in U.S. studies (Rasmusen and Christian, 1976; Christian, 1976; Christian, 1977). The Danish study found that only one animal of 98 having the $H^{-/-}$ genotype was halothane sensitive (Jorgensen, 1979). This may suggest that the association between halothane susceptibility and H blood group genotype is breed dependent. Hojny et al. (1979) found that the H blood system could be utilized to predict susceptibility or resistance to PSS in populations in which all founder animals have been previously tested for PSS by halothane and blood group procedures. The H blood system has been found to be within the HAL linkage group on chromosome 6 (Archibald and Imlah, 1985).

As previously mentioned, the A-O blood system is also associated with PSS susceptibility. Rasmusen (1964) found that pigs could be classified into 3 groups (A,O, and A-O negative) based on serological tests. The A-O blood group system has been termed the S gene (Rasmusen, 1964). The S allele which allows the expression of A and O is dominant to the recessive allele s, which does not allow expression of the A or O genotypes. The S blood group genotype was often used in conjunction with the H blood group genotype to determine PSS susceptibility (Christian and Rasmusen, 1976; Christian, 1976; Christian 1977; Jorgensen, 1979; Honjy et al., 1979; Imlah and Thomson, 1979; Glodek et al., 1985). Epistasis between the S locus and

the H locus was reported by Rasmusen (1972); Hojny (1974); Hojny et al. (1979). A-O negative was most often associated with $H^{a/a}$ genotype (Rasmusen and Christian 1976; Christian, 1976; Christian, 1977). The homozygous recessive genotype (S^{ss}) at the S locus seems to prevent expression of A or O erythrocyte antigens by epistasis (Rasmusen, 1972; Hojny, 1974). Linkage of A-O (S locus) and H blood groups was indicated by recombination between the two loci (Imlah, 1980; Rasmusen 1981). Further support of the association between the S and H loci was provided by Hojny et al. (1984) and the association of these two erythrocyte antigen loci and the HAL locus was proposed by Hojny et al., 1985. Van Zeveren et al. (1985) provided further evidence when they determined that the S locus was located in the HAL linkage group to which the H blood groups locus had already been assigned.

Numerous isozymes markers have also been used in an attempt to determine PSS genotypes of pigs. The phosphohexose isomerase (PHI) locus has 2 co-dominant alleles, PHI^A and PHI^B, and encodes the glucosephosphate isomerase enzyme (Christian and Mabry, 1990). Close linkage between the PHI and H blood group loci was determined by Andresen (1970a). Rasmusen et al. (1980) reported very close linkage association between the PHI, H and HAL loci. A pig's genotype for the PHI locus is determined by electrophoresis of plasma proteins (Gahne and Juneja, 1985). The PHI^B allele is most often associated with PSS-susceptible animals, as suggested by Jorgensen (1981) and supported by Imlah (1984) and Grashorn and Muller (1985). Vogeli (1989) formally determined the location of PHI within the HAL linkage group.

The isozyme 6-phosphogluconate dehydrogenase (6-PGD) has also been found to be associated with halothane sensitivity (Rasmusen et al., 1980). Saison (1968) discovered the existence of two co-dominant variants (PGD^A and PGD^B) by electrophoretically separating plasma proteins. Andresen (1970b) determined that the H and the 6-PGD loci were linked in the pig. It appears that the PGD^B allele is most often associated with PSS susceptibility (Vogeli et al., 1985). The association of the PGD marker and HAL is not as strong as some of the other markers used as indicated by the highest recombination rates among HAL and 6-PGD when compared to other loci (Gahne and Juneja, 1985). Rasmusen (1981) suggested that 6-PGD is the furthest removed marker from the HAL locus. This was confirmed by Vogeli (1989) by further refining the order of the markers within the HAL linkage group. An additional variant, C, was found in wild pig populations of East Asia, in addition to the A and B alleles (Kurosawa and Tanka, 1992).

Juneja et al. (1983) assigned the postalbumin 2 (PO-2) locus to the HAL linkage group. This locus encodes the PO-2 protein found in the serum of pigs and other species (Juneja et al., 1983). Electrophoresis of pig serum revealed two autosomal co-dominant PO-2 alleles (F and S). Furthermore, it was noted that PSS susceptibility was most often associated with the S PO-2 allele. The PO-2 locus has been identified as belonging to HAL linkage group (Cepica, et al., 1986). Rasmusen (1983) has summarized the application of isozymes in swine breeding programs. Vogeli (1989) place the PO-2 locus between the H and 6-PGD loci in the HAL linkage group.

Advances in electrophoretic methodology allow for the simultaneous PHI, 6-PGD and PO-2 haplotyping (Gahne and Juneja, 1985). Limitation to

widespread use of any of the marker loci in determining PSS susceptibility include: need for haplotyping foundation animals and current breeding herd to ensure linkage between marker loci and the HAL locus (Honjy et al., 1979), inaccurate classification due to crossing over, locating qualified labs to perform the test, and costs of haplotyping.

The latest tool that swine breeders can utilize to determine PSS status of their breeding herd is a DNA molecular test. As previously mentioned, Fujii et al. (1991) developed a quick, simple, and accurate molecular test for PSS that can distinguish between all three PSS genotypes (NN, Nn, and nn) with an accuracy approaching 100%. The exact location of the mutation responsible for PSS was determined as previously described (Fujii et al., 1991). Louis et al. (1992) and O'Brien (1992) provide a general outline of the steps utilized in determining PSS genotype by molecular methods. The procedure involves the breeder collecting a blood sample in a sterile-heparinized test tube with a needle and syringe. Use of a new needle and syringe for every individual is recommended to prevent cross contamination between pigs and inaccurate genotype determination. The sample(s) is sent to a laboratory with the ability to conduct the test. Laboratory personnel isolate DNA from nucleated leukocytes. The DNA is amplified (thousands of copies made) by PCR technology. The DNA is then digested with restriction enzymes. Usually one enzyme recognizes the mutant allele while another recognizes the normal allele. After digestion of the DNA occurs, the samples are electrophoretically separated on agarose or polyacrylamide gels containing ethidium bromide stain. After separation is complete, the stained bands of each genotype form characteristic

patterns and differentiation of the three genotypes can be made. If procedures are followed properly and no contamination occurs at any step of the process, the accuracy of the test approaches 100%. The molecular test has been conducted for DNA isolated from muscle tissue, hair, and adipose tissue samples (Christian, personal communication).

The molecular test developed by Fujii and co-workers (1991) has been patented by the University of Toronto and licensing for commercial labs and breeders is available through Innovations Foundation, Toronto, Ontario Canada (Howard Bartlett, personal communication). Several commercial laboratories are conducting the DNA test for PSS. Swine breeders can send their samples to any of the available commercial labs and PSS genotype can be determined on as many individuals within their herd as they desire. Cost of the test ranges from 20-35 dollars for each sample tested. Breeders can accurately determine the genotype of all breeding herd animals and manage the PSS gene frequency as they desire.

Effects on maternal performance

Studies that examine the effects of PSS on the reproductive performance of females are not as plentiful as those concerning its effects on other characteristics. Additionally, the majority of research studies completed have looked at the performance of PSS-susceptible (determined by halothane) and non-susceptible females, due to the difficulty in distinguishing between Nn and NN animals. Mabry (1977) examined maternal performance between halothane susceptible and non-susceptible females from a stress-susceptible composite line for two farrowing seasons. Similar performance was found for number of pigs farrowed, number of pigs raised to

21 d, 21-d litter weight, number of pigs alive at 56 d, 56-d litter weight, and survival rate. Trends though not significant, suggested, some superiority in maternal ability for the PSS non-susceptible females for number of pigs at 21 and 56 d, and litter weight at 21 and 56 d. Webb and Jordon (1978) utilized females from a Pietrain-Hampshire synthetic line. No significant differences were observed between halothane positive (HP) and halothane negative (HN) females for any of the maternal traits measured. Trends indicated that HN females produced 0.4 more pigs at birth and raised 0.6 more pigs to weaning (56 d). Birth weight was 0.89 kg heavier at birth and 12.6 kg heavier at weaning for HN females when compared to HP females.

Schneider et al. (1980) measured maternal performance of 1056 Swedish Landrace litters farrowed by females with known PSS genotypes. No differences were observed between NN and Nn females for number born alive, birth weight of the litter or % mortality at weaning (28 d). Nn females were superior ($P<0.05$) to NN or nn females for number of pigs raised to weaning. However, NN females produced heavier ($P<0.05$) litters at weaning than did Nn or nn females. Females classified as nn were generally inferior to NN and Nn females.

Willeke et al. (1984) utilized 731 litter records from 206 German Landrace females to examine the effects of PSS genotype on litter size at birth and weaning (28 d), and mortality from birth to weaning. No differences, for any of the traits, was observed between NN, Nn or nn females. However, there was a tendency for NN females to be superior to Nn

and nn females for all traits. Additionally, Nn females were intermediate to NN and Nn females for all traits studied.

Lampo et al. (1985) collected maternal performance data on 1413 Belgian Landrace litters from 494 females farrowed on 7 commercial farms. No significant differences between HP and HN females were observed for age at first farrowing, number born alive (regardless of parity), number at weaning (28 to 42 d depending on herd), or farrowing interval. On average, there was a trend for HN sows to farrow more live pigs, particularly in second parity and beyond. Litter size at weaning and survival rate followed similar trends, favoring HN females.

A Hampshire-Pietrain synthetic line was used by Carden et al. (1985) to examine differences in maternal performance due to PSS classification. Divergent lines were formed by selecting for and against halothane sensitivity and litter data were recorded. During years 3-5 of the experiment, stress resistant (SR) and stress susceptible (SS) females were mated to SR males providing two distinct data sets. Stress resistant female litter size was 1.16 pigs larger ($P<0.05$) at birth and 1.76 pigs larger ($P<0.01$) at weaning (approximately 50 d) compared to SS farrowed litters. When data from the 2 data sets were combined, piglets farrowed by SS or SR females did not differ in their average weight at birth or weaning but tended to favor the SR females. Stress resistant females had 18% less ($P<0.001$) piglet mortality from birth to weaning compared to SS females.

Simpson et al. (1986) collected maternal performance data on 300 British Landrace females involved in a halothane sensitivity selection experiment. HP positive females had 0.10, 0.23, and 0.28 smaller litter

sizes at birth, 21, and 42 d, respectively; however, these differences were not significant. HN females had heavier birth (1.19 kg) ($P<0.01$), 21-d (3.22 kg) ($P<0.01$), and 42-d (6.7 kg) ($P<0.05$) litter weights compared to HP females. Conception rates were not different between HP and HN females.

A comparison of maternal performance was made between NN and Nn Swedish Yorkshire females (Nystrom and Andersson, 1993). No number born or number born alive differences were observed between the two genotypes. However, both tended to favor the carrier animal (.88 pig born and .08 pigs born alive). NN females had .80 fewer ($P<0.05$) stillborn pigs than did Nn females. Results favored NN females by 0.85, 0.89, and 0.99 for number of live pigs at 21, 42 and 63 d, respectively, compared to Nn females, but the differences were not significant. Average weight of fully developed pigs born and pigs born alive did not differ between NN and Nn females. Average differences of litter weights at 21 and 63 d were 0.26 and 0.64 kg heavier ($P<0.05$) for NN compared to Nn females.

Previous work strongly discourages the use of a HP female in most commercial breeding herds. It is less clear whether Nn females should be retained for breeding purposes. The Nn animals would fall under the HN category along with NN animals which clearly have a reproductive performance advantage compared to nn or HP animals. Studies comparing NN and Nn females for maternal performance are few and involve small numbers (Nystrom and Andersson, 1993), or were conducted when methods of distinguishing between the NN and Nn genotypes were less precise (Schnieder et al., 1980; Willeke et al., 1984).

Effects on quantitative performance and carcass traits

Christian (1974) reported that HP pigs grew faster and had similar feed gain ratios (F/G) compared to HN pigs. When these pigs were slaughtered, HP animals had on average .29 cm^2 larger ($P<0.05$) loin muscle area (LMA) than did HN animals. HP were not different from HN pigs but tended to have shorter carcasses and less backfat (BF) than did HN pigs.

Webb and Jordan (1979) found no differences between HP and HN pigs for F/G, average daily gain (ADG), BF, LMA, dressing percentage (DP). Carcass length favored ($P<0.05$) HN pigs by 11.5 cm.

Eikelenboom et al. (1980) investigated PSS genotypic differences in Dutch Landrace market hogs. No differences were observed between NN, Nn and nn pigs for ADG or F/G. Animals with nn genotype were found to have better DP, BF, ham%, and loin% than did either NN or Nn animals, although not always significantly better. Generally, the carcass traits from Nn pigs were intermediate to those of NN or nn pigs.

Christian and Rothschild (1981) found similar ADG, FG, and feed consumption among pigs of all three PSS genotypes. Carcasses from Nn and nn carcasses had less BF at the tenth rib (BF10), larger LMA, thus a higher percent of carcass lean (differences were not always significant). Dressing percent was similar for all three genotypes. Webb et al. (1982) summarized numerous studies which evaluated various traits. Generally, HP pigs grew slower and ate less feed, but had better F/G. Halothane positive pigs had a more favorable average percent carcass lean, percent ham, and LMA compared to HN pigs. The HN pigs averaged less BF and more carcass

length compared to HP pigs. Similar results were reported for British Landrace by Simpson and Webb (1989).

Differences between HP and HN British Landrace and Pietrain-Hampshire market hogs were evaluated (Webb and Simpson, 1986). Superior ADG, F/G BF, LMA, DP, percent lean was found for HP pigs. However, HN pigs had higher daily food consumption, carcass length, and calculated economic return than did HP animals.

Jensen and Barton-Gade (1985) evaluated growth and carcass traits in Danish Landrace pigs of all PSS genotypes. Normal pigs had higher ADG ($P<0.05$) than Nn or nn pigs. Similar F/G were found among pigs of all PSS genotypes. Shorter carcass length ($P<0.05$) was found in nn pigs compared to NN or Nn pigs. A linear effect was observed ($P<0.05$) with nn pigs producing carcasses with superior, Nn intermediate and nn the poorest percent loin, percent ham, BF, and LMA.

The same linear effect was observed for carcass length and loin depth (LD) by Jones et al. (1988). Additionally, the Nn pigs were not intermediate to nn and NN pigs as previously described for other traits. Similar LMA and midback BF was observed for nn and Nn pigs, while NN and Nn had similar BF measured at point of maximum muscle depth. Aalhus et al. (1991) found a significant ($P<0.05$) linear effect for age at slaughter in 805 Lacombe pigs. The NN pigs were found to be superior, Nn intermediate and nn the poorest for age at marketing. A linear effect ($P<0.05$) was also observed for carcass weight. Normal animals produced the smallest carcasses while Nn produced intermediate weight and nn animals produced the largest carcasses. The nn pigs were superior to Nn and NN pigs for

relative lean growth and BF. The relative BF and lean percent superiority of nn pigs decreased with increasing live weight. Pommier et al. (1992) utilized NN and Nn pigs to study PSS genotype effects on carcass and performance traits in crossbred pigs. No difference was observed between the NN and Nn animals for d to slaughter, ADG, carcass length, percent loin, percent ham, BF, and LD regardless of sex (only barrows and gilts were included in the study). Dressing percentage slightly favored ($P<0.04$) the Nn individuals. One hundred thirty six pigs representing seven different breed groups were utilized to examine performance and carcass trait differences between NN and nn pigs (Zhang et al., 1991). Carrier animals grew faster ($P<0.05$) from 24.5 kg to market weight. Similar DP, percent carcass lean, LMA, BF, and percent ham were found between the two genotypes.

Goodwin et al. (1994) estimated differences between 1298 NN and 181 Nn PSS genotypes for numerous performance and carcass traits. The NN animals had superior ($P<0.05$) ADG when compared to Nn animals. Carrier animals had a leaner ($P<0.05$) mean BF10, larger ($P<0.05$) LMA, and higher ($P<0.05$) DP than did NN animals. The carcass length of NN and Nn animals did not differ.

Effects on qualitative carcass traits

Kauffman et al. (1992) suggested that muscle color, firmness/wetness, and marbling are important fresh pork quality parameters which influence consumer acceptability. Fresh pork should be reddish pink and uniform in color. Soft, flaccid and exudative pork has poorer water-holding capacity and will shrink as much as 7% during handling and processing, making it

undesirable for packers and consumers. Slight marbling or intramuscular fat is desirable for a juicy, tasteful cooked product (Kauffman et al., 1992). Kauffman et al. (1992) estimated the incidence of poor quality pork in the U.S. to be 26%. Pale, soft and exudative (PSE) and dark, firm and dry (DFD) pork products are of lower value to the packing industry because of a loss of water holding ability and their use in less expensive, further processed products rather than being sold as higher priced, fresh products. This cost is estimated to be 30 million dollars annually (USDA - FSIS reports, 1994).

PSE pork is primarily due to rapid pH decline occurring after stunning (Hofmann, 1988) caused by genetic and environmental factors. Eikelenboom (1985) described many ways to alleviate some of the environmental factors contributing to poor pork quality.

PSS is one of the genetic factors that can cause the development of PSE pork. Rapid pH decline during the first 45 minutes post mortem resulting from excessive metabolic anaerobic glycolysis and buildup of lactic acid are the major factors causing PSE and DFD pork (Cassens et al., 1975; Mitchel and Heffron, 1982; and Bendall and Swatland, 1988).

Eikelenboom and Minkema (1974) reported lower ($P<.001$) pH and higher ($P<.05$) temperature values 45 minutes post mortem in carcasses from HP pigs compared to HN pigs. Reflectance values were higher ($P<01$) indicating a higher degree of paleness in HP pigs compared to control animals (Christian, 1976). Subjective color scores (1-3) (NPPC, 1988) were lower ($P<0.01$) in carcasses produced by HP animals compared to those produced by HN pigs (Christian, 1976). Higher 45 minute pH values, better water

holding capacity, and superior objective color scores were observed in a HP line of pigs when compared to controls (Froystein et al., 1979). Eikelenboom et al. (1980) found lowest ($P<0.05$) subjectively appraised muscle scores in nn carcasses, while Nn carcasses were intermediate and NN carcasses were superior ($P<0.05$). Webb et al. (1982) summarized several studies and concluded HP pigs produce 46% more PSE and 15% poorer colored carcasses than HN pigs. Additionally, pH values averaged .31 lower for HP compared to HN carcasses across the studies summarized. Jensen and Barton-Gade (1985) reported two, six, and 54% PSE incidence of visually appraised carcasses from NN, Nn and nn pigs (1-4 scale). A significant ($P<0.05$) linear effect was noted for 45 minute carcass pH with Nn carcasses being lower than NN but higher than nn. Significantly lower 45 minute pH and higher temperatures were found in HP compared to HN carcasses (Eikelenboom and Costa, 1988). The same study found better ($P<0.01$) subjective color scores (1-4 scale), higher ($P<0.001$) reflectance values and similar fat percentage within the loin muscle of HP carcasses when compared to those from HN pigs. Murray et al. (1989) accessed pork quality of carcasses produced by pigs of all PSS genotypes. The carcasses produced by NN were superior, Nn intermediate and nn were inferior ($P<0.05$) for 45 minute pH, percent drip loss, and intramuscular fat content. Simpson and Webb (1989) found lower ($P<0.01$) visual muscle scores and higher electronic color scores (EEL reflectance) in HP compared to HN carcasses. Lundstrom et al. (1989) reported Nn carcasses to be intermediate, NN superior and nn poorest for percent drip loss ($P<0.001$) and EEL reflectance ($P<0.001$). Nn pigs produced carcasses with superior ($P<0.05$) subjective color (1-5 scale),

firmness (1-5 scale), and marbling (1-5 scale) than did nn pigs (Zhang et al., 1991). Oliver et al. (1993) found that HP animals produced carcasses with poorer fiber optic probe color and higher 45 minute pH when compared to HN animals. Christian et al. (1993) used subjective and objective measures of muscle quality to evaluate PSS genotype difference. Significant ($P<0.01$) linear effects, always favoring NN animals with Nn animals being intermediate, were observed for visual color, marbling and firmness, Minolta reflectance, Hunter color, 24 hour drip loss and various pH values.

One of the more comprehensive evaluations of pork quality occurring in the U.S. was conducted by the National Pork Producers Council. The National Genetic Evaluation is the most extensive U.S. carcass quality evaluation and was conducted by the National Pork Producers Council in cooperation with Iowa State University, University of Minnesota, Purdue University, Western Illinois University, George H. Hormel Foods, Inc. Rochelle Foods Inc., Carroll's Food Inc. and the Minnesota and Iowa Pork Producers Associations. This project involved over 3200 market hogs. Complete feedlot performance, carcass composition and quality, and sensory evaluations were recorded on each animal. Normal animals were found to have superior ($P<0.05$) Minolta scores (an objective method to measure color), and subjective color, marbling and firmness scores (1-5 scale) when compared to Nn animals. Additionally, NN animals produced carcasses with higher ($P<0.05$) lipid content of the loin muscle and better ($P<0.05$) Instron values, a measure of tenderness. No differences between NN and Nn animals were found for ADG, d to 250 lbs., soundness, and BF10.

Differences ($P<0.05$) favoring the NN animals were observed for carcass length, last rib backfat, visual color, marbling and firmness, loin lipid content, drip loss, Minolta and Hunter color and Instron tenderness. Differences ($P<0.05$) favoring Nn animals were observed for lean gain on test and LMA (Goodwin, personal communication).

Research clearly indicates that nn (HP) pigs produce carcasses having poorer muscle quality than do NN or Nn pigs. More recent research even suggests that Nn animals also produce carcasses with less desirable quality measures (Christian et al., 1993; Goodwin, 1994; Murray et al., 1988) than do NN pigs. Production of Nn pigs in a quality conscious marketplace would have to be questioned.

Ways to objectively measure pork quality have been evaluated (Chizzolini et al., 1993a) and their application to on-line packing plant systems examined (Chizzolini et al., 1993b). If meat packers discover a way to objectively evaluate pork quality at line speed at the packing plant, muscle quality is likely to become a larger issue. Market hog producers are likely to see discounts placed on carcasses having inferior muscle quality by packing companies.

Genetic Aspects of Sow Productivity

Economically important aspects of sow productivity include: number of piglets born alive (NBA), litter birth weight, number of pigs alive at 21 d (NO21), adjusted 21-d litter weight, and litters per sow per year (MacKay, 1992).

These traits have tremendous impact on the profitability of commercial and seedstock swine producers (Mabry et al., 1990) and should determine breeding herd longevity (MacKay, 1972). These traits have been found to be heavily influenced by environmental factors and to a much lesser extent by genetics as indicated by the low magnitude of reproductive heritabilities.

Heritability estimates from various studies have been summarized by Lamberson (1990). Heritability estimates range from -.06 to .76 and the mean of all studies was .10 for total number born (NB) and ranged from -.07 to .66 for NBA with all studies with a mean of .07. Mean heritability was estimated to be .05 with a range of -.08 to .97 for survival from birth to weaning. Number weaned (NOWN) heritability estimates ranged from -.01 to 1.02 and had a mean value of .06. Litter birth weight estimates of heritability averaged .29 with a range from .00 to .54. Heritability estimates for 21-d litter weight averaged .15 with a range from .07 of .38. Lamberson (1990) also summarized estimates of genetic correlations between numerous traits. Positive genetic correlations ranging from .63 to .88 were noted among NBA and number weaned, litter birth weight, and litter 21-d weight. Southwood and Kennedy (1990) estimated heritabilities for NB, NBA and NOWN of .13, .13, and .10, respectively, in Yorkshire females and .13, .09, and .07, respectively, in Landrace females. See et al. (1993) estimated the heritability of NBA to be approximately .13 for the Spotted, Hampshire, and Landrace breeds.

Maternal effects can be described as genetic aspects of a mother which influence the phenotypic values of her offspring (Falconer, 1989).

Willham (1972) suggested that there are maternal influences affecting weaning weight and litter size in pigs. Robison (1972) suggested that maternal effects exist in many production and reproduction traits in swine. Negative environmental correlations exist between the litter size of dam and daughter (Revelle and Robinson, 1973). Maternal effects might partially explain lower reproductive trait heritabilities in swine and make selection less effective for those traits (Revelle and Robinson, 1973). They also suggested that gilts from large litters would be unable to express their full genetic superiority due to the stress and competition prior to weaning. If the effect of being reared in a large litter is removed by the transfer of pigs shortly after birth, in a manner in which all litter sizes are standardized for each female, the post-natal maternal effect would be equal for all litters (Revelle and Robinson, 1973). Bereskin et al. (1974) reported maternal effects influenced total litter weight at birth, 21, and 56 d of age, but found no maternal effects influencing litter size and total litter weight at birth, 21, and 56 d of age. Southwood and Kennedy (1990) found a negative genetic covariance between direct additive and maternal genetic effects. Maternal effects can bias estimates of genetic and environmental parameters and influence selection response for litter size in swine (Roehe and Kennedy, 1993).

Inbreeding reduces phenotypic values of traits related to fitness such as reproductive traits (Falconer, 1989). Bereskin et al. (1968) reported that litter inbreeding did not affect NBA but depressed litter birth weight ($P<0.05$). Dam inbreeding had a negative effect ($P<0.05$ or less) on NB, NBA, and litter birth weight. Traits at weaning showed

inbreeding of litter and dam had a negative impact on NO21 and 21-d litter weight ($P<0.01$). Survival of piglets was substantially lowered ($P<0.05$) as inbreeding of the litter increased. However, survival rates were not affected by inbreeding of the dam (Bereskin et al., 1973). Schneider et al. (1982) reported a reduction of 0.016 for NBA and 0.033 for NO21 for each 1% litter inbreeding. Furthermore, litter weights at birth and 21-d could be expected to be reduced by 0.05 kg and 0.33 kg, respectively, for each 1% inbreeding. Johnson (1990) suggested that .10 inbreeding in the dam would reduce NBA by .63 and NO21 by .43 pigs. A reduction of 1.12 kg for birth and 4.33 kg for 21-d litter weights could be expected of dams having an inbreeding coefficient of 0.10 (Johnson, 1990). A 0.10 inbreeding coefficient of the litter reduced litter size at birth and 21 d by 0.20 and 2.78 pigs, respectively, compared to litters with no inbreeding. Birth and 21-d litter weights were reduced by .64 kg and 3.35, respectively, with a 0.10 inbreeding coefficient (Johnson, 1990).

Crossbreeding is the mating of animals from two or more breeds or lines. The benefits of crossbreeding include heterosis or hybrid vigor and complementary matings (MacKay, 1992). Crossbreeding has also been shown to influence reproductive traits. O'Ferrall et al. (1968) reported that NBA was similar for inbred and crossbred litters. The advantage of the crossbred litter was evident for litter size at 21 and 56 d of age (.8 and 1.0 more pigs, respectively). No differences were reported between crossbred and inbred litters for birth weight although crossbred litters tended to be heavier. Litter weight at 21 and 56 d of age was 6.0 kg and 23.8 kg higher ($P<0.05$), respectively, for crossbred litters compared to

inbred litters. Young et al. (1976) reported significant heterosis effects for litter size and weight at 42 d.

Johnson et al. (1978) reported that conception and ovulation rates were similar among purebred and crossbred gilts. However, crossbred gilts had .71 more embryos than did purebred gilts when slaughtered 30 d post-breeding. Crossbred females farrowed .93 and weaned 1.24 more pigs per litter and had nearly 20% heavier litters at weaning than did purebred dams. Johnson (1980) reported a .46, .64 and .58 litter size advantage P($P<0.05$) at birth, 21 and 42 d, respectively, for crossbred dams compared to purebred dams. Similar birth, 21-d, and 42-d litter weight trends were observed between purebred and crossbred dams but were only significant at 42-d.

Sow Productivity Environmental Adjustment Factors

Environmental effects are those non-genetic factors affecting phenotypic performance of an animal. Environmental effects can mask true genetic ability and pose a problem in measuring true genetic effects or breeding value of an animal. Accounting for all environmental factors affecting the phenotypic record of an animal is not possible. There are, however, many environmental factors that can be accounted for and their effects reduced through the use of adjustment factors. The purpose of adjustment factors is to negate environmental influences on the phenotypic record of an animal. Adjusted phenotypic records are suited to more accurate determination of genetic effects and the evaluation of each animal's genetic capability for selection purposes (Kemp and Rothschild,

1988). Sow productivity traits are typically adjusted for parity of female, number of pigs allowed to nurse and age of litter when 21-d weight is taken (NSIF, 1987).

Bereskin et al. (1973) found that location, year-season, location by year-season, and birth weight of pig, each accounted for 5% or more of survival rate variation. Significant breed and breed cross differences were observed for number of pigs and litter weight at 21 and 42 d by Young et al. (1976). Significant first and second parity differences were observed for NBA, NO21, and litter weight at birth and 21 d (Schneider et al., 1982). Similar results were found by Fahmy et al. (1971) and Holtman et al. (1975). Swiger and Irvin (1977) observed NB, NBA, NO21, and 21-d litter weight differences between first and second parity Duroc females. Twenty-one d litter weight adjustments for age at which 21-d litter weight was measured and number allowed to nurse were also recommended (Swiger and Irvin, 1977.)

Adjustment factors for 21-d litter weight were developed for purebred and crossbred dams (Wilson and Johnson, 1980). Season, parity, and number of pigs allowed to nurse each accounted for a significant ($P<0.01$) amount of variation. Linear regression coefficients for number allowed to nurse and 21-d litter weight were similar in Duroc and Hampshire females and lower for Yorkshire females, suggesting that the adjustments for number after transfer may be breed dependent for 21-d litter weight.

Yen et al. (1987) found breed, parity, and year-season effects on various litter traits in Yorkshire, Duroc, Chester White, Hampshire, Spotted and Landrace females. Number born alive was found to be superior

for parities 3 through 6 when compared to parities 1, 2, and 7 and higher. Number at 21 d and 21-d litter weight was found to be superior for parities 2 through 6 when compared to parities 1 and 7. It was further observed that increased 21-d litter weights were produced by increasing the number of pigs allowed to nurse up through 13 pigs per litter. Similar environmental effects were noted for sow productivity index.

Wood et al. (1990) developed a regression equation to standardize litter weaning weights to 21 d. It was suggested that, in order to fairly evaluate the productivity of each female NSIF, additive parity and number after transfer adjustments be made after first adjusting for the age of the litter when weighed.

Irvin et al. (1991), using similar methodology, developed 21-d litter weight adjustment factors for age at which the weight was taken. Different multiplicative adjustment factors were found among Landrace, Duroc, Duroc x Landrace, and three or four breed crossbred dams. Irvin et al. (1991) suggested that breed-specific adjustments would more accurately select which females should remain in the breeding herd based on sow productivity. Alternatively, to avoid producer confusion, Irvin et al. (1991) also suggested that multiplicative adjustments could be developed across breeds so that only one set of adjustments is needed.

Landrace and Yorkshire breed specific adjustment factors and combined adjustment factors were developed by Brubaker et al. (1994). Weaning age multiplicative adjustment factors developed by Brubaker et al. (1994) differed little from current NSIF recommendations, but tended to give litters younger than 21 d a smaller adjustment than those older than 21 d

compared to NSIF adjustments. Landrace, Yorkshire, and combined NBA parity adjustments differ ($P<0.05$) from current NSIF parity adjustment factors. Parities three through five were considered the mature base parity which differed from Irvin et al. (1991) who suggested that parities three through six were mature equivalent. Parity adjustment factors currently recommended by NSIF (1987) and those proposed by Brubaker et al. (1994) were similar for 21-d litter weight. Adjustments for NOWN are not currently available from NSIF. Brubaker et al. (1994) suggested that adjustment of NOWN for parity and number of pigs allowed to nurse is warranted. They also concluded that separate adjustment factors for this trait are needed for Landrace and Yorkshire dams. Many of the adjustment factors suggested by Brubaker et al. (1994) are being implemented in the latest revision of NSIF guidelines.

This review suggests that adequate adjustment factors exist for numerous environmental items that impact sow productivity. Accuracy of selection can be enhanced by the appropriate adjustment of phenotypic records for environmental influences.

Mixed Model Methodology

Henderson (1963) proposed the use of linear mixed models to predict an animal's genetic worth. The general form of the mixed model is:

$$y = Xb + Zu + e$$

where: y = vector of phenotypic performance values for a given trait of an animal,

X = incidence matrix relating fixed effects to the phenotypic performance values, y,

b = vector of unknown fixed effects,

Z = incidence matrix relating random effects to the phenotypic values, y,

u = unknown vector of random effects,

e = vector of random residual effects,

where, typically the variance of u is any general covariance matrix and the variance of e is $I\sigma^2_e$.

Estimates of fixed effects are best linear unbiased estimators (BLUE) of some estimable function of the fixed effects. Estimates of the random factors are best linear unbiased predictors (BLUP) of some estimable function of the random effects (Henderson, 1973). Mixed model evaluation is considered to have an advantage over selection index (Hazel, 1943) because it is unbiased. Selection index required that fixed effects be known without error resulting in best linear predictions (BLP) of random effects. Mixed model methodology does not have this requirement resulting in advancement to BLUP (Henderson, 1973). Henderson (1974) pointed out the following desirable properties of mixed models:

1. evaluations are unbiased (the expectation of the predictor and true value are the same);
2. evaluations minimize the variance of prediction errors;
3. method is easy to learn;
4. it is easy to modify when conditions change;
5. its properties are clearly defined;

6. it takes advantage of modern statistical and computing techniques developed for linear models;
7. variance of prediction error is obtained;
8. it can eliminate bias due to selection and culling and provides a mechanism for checking for the existence of bias (pp. 963-964).

Including the inverse of the relationship matrix (A^{-1}) (where diagonal elements are inbreeding coefficients and off-diagonal elements are Wright's (1922) relationship coefficients between animals) in mixed model procedures was described by Henderson (1976). He concluded that the inclusion of A^{-1} reduces prediction error variance and thus improves accuracy of genetic evaluations. Henderson (1985) concluded that fixed effect estimates are BLUE and random effects are BLUP even when selection has occurred in the population being evaluated if the following conditions are met:

1. random variables have a multivariate random distribution,
2. proportionality of variances and covariances are known,
3. selection decisions were based on linear function of phenotypic records, y,
4. the data used when making selection decisions are used in the mixed model equations (p. 445).

The development of full animal models (a type of mixed model) to conduct genetic evaluations was a major advancement in animal breeding technology. Improvements in computer technology have allowed implementation of the full animal model to conduct across-herd genetic

evaluation on large field data sets (Wiggans et al., 1989). The animal model has several advantages compared to other genetic evaluation methods. Animal models incorporate A^{-1} and when it is multiplied by additive genetic variance (σ^2_a), the model describes the variance - covariance structure among additive breeding values of all animals evaluated (Kennedy et al., 1988). The inclusion of A^{-1} accounts for changes in means and variances brought about by selection and linkage disequilibrium (Kennedy et al., 1988). Wiggans et al. (1988) described the advantages of the animal model compared to the modified contemporary group evaluation previously used by the USDA when conducting national dairy genetic evaluations:

1. merit of mates is considered,
2. all animals are simultaneously evaluated,
3. relationship matrix is more complete by the inclusion of dams,
4. more precise definition of fixed effects (p. 62).

Additionally, breeding values obtained under an animal model are not influenced by selection or inbreeding (Kennedy et al., 1988). Effects of inbreeding depression can be accommodated in an animal model by including inbreeding coefficients as a covariate.

Animal models have been used for conducting genetic evaluations of various livestock species. Swine testing and genetic evaluation systems (STAGES) was developed by Purdue University and is currently used by some purebred swine organizations to conduct genetic evaluations (Lofgren et al., 1994). Another system, purebred across-herd genetic evaluation (PAGE 1), developed by the University of Georgia is currently used by purebred swine organizations (Mabry and Middleton, 1994). The University of Georgia

also conducts genetic evaluations for many of the purebred beef cattle organizations. The animal model has also been applied to multibreed data (Arnold et al., 1992).

Restricted maximum likelihood (REML) procedures are considered current state-of-the-art methodology to estimate variance and covariance components of unbalanced animal breeding data (Boldman et al., 1993). This method will also yield least squares estimates of the fixed effects included in the model used for analysis (Boldman et al., 1993). Improvements in computer technology and the development of a derivative-free (DF) REML algorithm (Graser et al., 1987) has resulted in more widespread use of REML procedures. Meyer (1989) used a DFREML algorithm to simultaneously estimate variance components and environmental effects. Mistal (1990) and Boldman and Van Vleck (1991) reported improved computer efficiency when a sparse-matrix solver is used with DFREML procedures to estimate variance components. Boldman et al. (1993) developed a manual outlining the use of the DFREML program to estimate fixed affects, random effects, and variance components in either a single or a multiple trait setting. The DFREML programs (Boldman et al., 1993) were utilized to estimate the fixed effect of PSS genotype on various reproductive traits described later in this dissertation.

EFFECT OF PORCINE STRESS SYNDROME GENOTYPE ON THE MATERNAL PERFORMANCE OF A COMPOSITE LINE OF STRESS-SUSCEPTIBLE SWINE.

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Abstract

A porcine stress syndrome (PSS) susceptible herd was established at Iowa State University in 1975. Females used to start the herd were primarily of Yorkshire descent. The herd currently exists as a composite line of Yorkshire, Hampshire, Duroc, Spotted, Landrace, and Pietrain pure breeds. The goal of this herd has been to produce all possible PSS genotypes within each litter. No selection emphasis has been placed on reproductive performance or carcass traits. The PSS genotype classification procedures have evolved over time with the development of new technology. Visual appraisal, halothane sensitivity, post-stress blood creatine phosphokinase levels, erythrocyte antigen and serum protein haplotyping, and most recently DNA typing have been the methods utilized to determine the PSS genotype of pigs in this herd. Three generation pedigrees were recorded for each female for which records were analyzed in this study. Reproductive data was collected on 61 PSS normal (NN), 140 PSS carrier (Nn), and 132 PSS positive (nn) females farrowing 145, 358, and 362 litters, respectively. Data were adjusted for parity, number after transfer, and age of litters not weighed exactly at 21 d using the combined adjustments from Brubaker et al. (1994) and analyzed using multiple trait derivative-free restricted maximum likelihood (MTDFREML) and best linear

unbiased prediction or estimation (BLUP or BLUE) procedures fitting the sows' PSS genotype as a fixed effect in the model. Carrier females farrowed significantly more pigs per litter ($P<0.05$) than either NN or nn females. Normal females had more pigs at 21 d compared to nn females. Litters from NN females were heavier ($P<0.05$) at 21 d than litters from Nn or nn females. Additionally, Nn females had heavier litters at 21 d when compared to nn females. Normal females raised a higher proportion of pigs from transfer to 21 d than either Nn or nn females. Normal females had more pigs, heavier litters, and raised a higher proportion of their pigs to 42 d than did nn females ($P<0.05$). Carrier females had heavier litter weights ($P<0.05$) and a higher proportion of pigs raised to 42 d ($P<0.05$) when compared to nn females. The results of this study indicate that the detrimental effects of the PSS gene should preclude its use in commercial-production-maternal lines.

Introduction

Porcine stress syndrome (PSS), an inherited neuromuscular disorder, was first described in the U.S. by Topel et al. (1968). He noted PSS susceptible pigs, when stressed, would collapse in a shock-like state and die. Knudson et al. (1990) demonstrated that PSS susceptible and non-susceptible animals differed in their calcium (Ca^{2+}) release channel of muscle cells. Fill et al. (1990) suggested that the Ca^{2+} release channel fails to close properly once it is opened, leading to the commonly observed symptoms of PSS susceptible animals.

Since the discovery of its monogenic, autosomal recessive inheritance (Christian, 1972) and its location on chromosome six (Davies et al., 1988), much attention has been given to the effects of PSS genotype on muscle quality and performance traits of market hogs, while the effect of PSS genotype on maternal performance has been largely ignored. Frequency of the PSS gene varies in different breeds of swine found throughout the world. Goodwin (1994) reported a range of PSS gene frequencies from 0.00 to 0.42 among eight pure breeds of U.S. swine. Kallweit (1985) summarized various worldwide estimates of PSS gene frequency and reported some lines of Landrace and Pietrain to have PSS gene frequencies approaching 1.00.

Maternal performance traits can greatly influence the profitability of commercial and seedstock swine producers (Mabry et al., 1990). The most economically important maternal traits in swine are generally considered to be: number of pigs born alive, litter birth weight, number of pigs weaned, 21-d litter weight, and litters per sow per year (MacKay, 1993).

Few investigations have examined the effect of the PSS gene on maternal performance. The majority of the maternal trait investigations have focused on differences between stress susceptible (nn) and stress resistant (NN or Nn) females because of the difficulty distinguishing between normal (NN) and carrier (Nn) animals. Those studies having reproductive performance results for NN, Nn, and nn females are few and involve small numbers (Nystrom and Anderson, 1993) or were conducted when less precise methods were available to distinguish between NN and nn females (Schneider et al., 1980; Willeke et al., 1984). Molecular biology advancements have resulted in the development of a simple and cost

effective procedure to determine the PSS genotype of animals with an accuracy approaching 100% (Fujii et al., 1991). Swine producers can submit blood samples from individual pigs to a licensed laboratory using the procedure and have the PSS genotype determined quickly and accurately.

Individual swine producers should use all information available to them to make an informative decision concerning the appropriate use of the PSS gene in their breeding programs. The information required to make this decision should include the effect of PSS genotype on the following traits: reproduction, feedlot performance, carcass cutability, and meat quality.

The objectives of this study were to determine the maternal performance differences at birth, 21 d and 42 d between the PSS normal, carrier, and positive females.

Materials and Methods

Animals

A stress susceptible herd was established in 1975 at the Iowa State University Bilsland Memorial Swine Breeding Farm located near Madrid, IA. Females used to start this herd were primarily of Yorkshire descent. In the early years, females were mated to Yorkshire and Hampshire males thought to be carrying 1 or more copies of the PSS positive allele, and in later years sires of various other breeds have been mated to females in the PSS susceptible herd. The sow herd has remained closed except for the occasional introduction of females suspected to be carrying one or more copies of the PSS positive allele from other projects at the Bilsland Farm. Semen from PSS carrier (Nn) and positive (nn) males from outside the herd

has been utilized in recent years to reduce inbreeding and to introduce industry representative genetics into the population. The selection goal of this herd was to produce the most possible PSS genotypes within the litter with each mating. No selection emphasis has been placed on reproductive, carcass, or performance traits. The herd exists today as a composite line of Yorkshire, Hampshire, Duroc, Spotted, Landrace, and Pietrain pure breeds.

Data description

Reproductive data from the PSS susceptible herd has been collected since the first sow farrowed in the spring of 1975. Farrowing occurs in the spring and fall of each year to avoid the extreme summer heat detrimental to nn females. Reproductive traits recorded include number born (NB), number born alive (NBA), birth weight (BW), birth weight of pigs born alive (BABW), number after transfer (NAT), number at 21 d (NO21), 21-d litter weight (LWT21), number at 42 d (NO42),and 42-d litter weight (LWT42). Additionally, the proportion of pigs surviving from birth to transfer (SURVT), transfer to 21 d (SURV21), and transfer to 42 d (SURV42) were calculated from the data. Complete three generation pedigrees were recorded for each female. Number of dams, records, records per dam, and the number of service sires used during the existence of the PSS susceptible herd are listed in Table 1 by genotype.

The PSS genotype of animals has been determined by a variety of methods during the twenty year existence of the PSS susceptible herd. Visual appraisal and the anesthetic halothane (Christian, 1974) were the detection methods of choice in the early years. These procedures along

Table 1. Distribution of dams, records, average number of records per dam, and service sires by porcine stress syndrome (PSS) genotype.

	Genotype			
	NN	Nn	nn	total
dams	61	140	132	333
records	145	358	362	865
records per dam	2.38	2.56	2.74	2.60
service sires	16	37	76	129

with elevated blood creatine phosphokinase (CPK) (Mabry et al., 1983) were used in the intermediate years. Halothane screening results coupled with linkage disequilibrium at the H and S erythrocyte antigen loci (Rasmusen and Christian 1976; Honjy, 1974), phosphohexose isomerase (PHI) loci, 6-phosphogluconate dehydrogenase (6-PGD) isoenzyme loci, and at the post albumin-2 (PO-2) serum protein loci (Archibald and Imlah, 1985) were used until 1991. All of these markers have been placed within the same linkage group on chromosome 6 of the pig (Vogeli, 1989). It was difficult to distinguish between NN and Nn animals with many of the detection methods if used alone.

These methods when used in combination were reasonably accurate, but the occasional crossover of marker loci would lead to inaccurate determination of the animal's PSS genotype. Furthermore, the need for trained personnel and costly equipment prevented widespread use of these detection methods. The DNA molecular test (Fujii et al., 1991) detects the actual mutation responsible for PSS with an accuracy approaching 100% and is the current method of choice when determining PSS genotype of an animal. The molecular test results are periodically verified with the response to halothane. A frequency of 0.61 was calculated for the n PSS allele among females retained for breeding purposes in the PSS susceptible herd since its beginning.

Statistical analysis

Adjustments for parity, NAT, and age at weighing (Brubaker et al., 1994) were applied to NBA, LWT21, and NO21 prior to the analysis of these traits. All 42 d litter weights not taken at exactly 42 d were adjusted to

a constant 42 d age using the average daily gain of the litter from birth to d weighed. All reproductive data were analyzed using the full animal model described as :

$$y = Xb + Zu + e$$

where y = vector of phenotypic performance values for animal,

X = incidence matrix relating fixed effects to the phenotypic performance values, y,

b = unknown vector of fixed effects,

Z = incidence matrix relating random effects to the phenotypic performance values, y,

u = unknown vector of random animal effects and,

e = vector of random residual effects,

where the variance u is $A\sigma^2_a$ (A is the relationship matrix) and the variance of e is $I\sigma^2_e$.

The model was incorporated using multiple-trait DFREML procedures (MTDFREML) (Boldman and Van Vleck, 1993). The MTDFREML programs will also yield estimates of the fixed effects included in the model. The relationship matrix included in the animal model accounts for the known variance-covariance associations that exist among additive breeding values of all animals evaluated. The inclusion of the relationship matrix allows for more precise estimation of fixed effects included in the model (Wiggans et al., 1989). A total of 630 animals, 333 dams with records and 297 base animals, were included in the relationship matrix used in these analyses.

Fixed effects included in all models include contemporary group (year-season of farrowing), type of mating (artificial insemination - natural service), and PSS genotype of the female with record. The number of contemporary groups included in the analysis were 38. Additionally, the fixed effect for parity of the dam was included in the models for NB, BW, BABW, SURVT, SURV21, NO42, LWT42, and SURV42 because no prior parity adjustment was available. Estimates of fixed effects are empirically best linear unbiased estimators (BLUE) of some estimable function of the fixed effects (Henderson, 1973).

Covariates for inbreeding of female (animal), inbreeding of litter, and inbreeding of service sire were included in the model for all traits. Number after transfer was used as a covariate in the models for SURV21, NO21, LWT42, and SURV42.

The random effects of animal, sire, dam, service sire, repeated record and maternal effects were included in the models for all traits. The solutions for the random effects are empirically best linear unbiased predictors (BLUP) of some estimable function of the random effects (Henderson, 1973).

All traits were analyzed singly and evaluated to the recommended convergence (1.E-9) for the variance of the simplex function (Boldman et al., 1993). All models were "cold restarted" with preceding variance and covariance values. Convergence to a global maximum was determined when two successive cold restarts yielded the same variances, covariances, and -2 log likelihood of the simplex function (Boldman et al., 1993). The globally maximized values were used to obtain solutions for the mixed model

equations (solutions for all covariates fixed and random effects are obtained). Planned contrasts between all PSS genotypes were made in the final MTDFREML run. If the difference between genotypes was larger than two times the standard error of the two means involved in the contrast, they were considered different ($P<0.05$) as suggested by Boldman et al. (1993).

Results and Discussion

Porcine stress syndrome carrier females farrowed 0.94 ($P<0.05$) more pigs at birth compared to NN females (Table 2). Additionally, Nn females farrowed 1.07 kg and 0.74 kg heavier litters ($P<0.05$) than did NN or nn females, respectively. After making parity adjustments, Nn females had a larger number of pigs born alive, than did either NN or nn females, 0.91 and 0.69, respectively. No differences in the litter birth weight of pigs born alive were observed between females of any PSS genotype. Carrier females, however, tended to have larger litter weights of pigs born alive than did either NN or nn females. Proportion of pigs born alive surviving from birth to transfer was not different between females of differing PSS genotypes.

Mabry (1977), Lampo et al. (1985), and Simpson et al. (1986) all report no NBA differences between halothane positive (HP) (nn genotype) and halothane negative (HN) (NN or Nn genotype) females. Webb and Jordan (1978) and Carden et al. (1985) reported that HN females farrowed 1.6 ($P<0.01$) and 1.2 ($P<0.05$) more pigs, respectively, when compared to HP females.

Table 2. Birth trait means and estimated mean differences (± SE[1]) between differing porcine stress syndrome genotypes.

Trait	Overall Mean	Genotype Contrast					
		NN-Nn	SE	Nn-nn	SE	NN-nn	SE
Number born	10.16	-0.94*	±0.39	0.61	±0.32	-0.33	±0.44
Birth weight, kg	13.58	-1.07*	±0.49	0.74*	±0.39	-0.34	±0.52
Adjusted number born alive[2]	9.98	-0.91*	±0.39	0.69*	±0.32	-0.22	±0.43
Born alive litter birth weight, kg	13.05	-0.91	±0.47	0.77	±0.47	-0.14	±0.49
Survival rate to transfer, %	90.48	-6.29	±5.30	2.51	±4.01	-3.79	±5.88

[1] Standard error of the difference between the two genotype means contrasted.

[2] According to Brubaker et al. (1994).

* Indicates significant difference between genotypes ($P < 0.05$).

Schneider et al. (1980) and Willeke et al. (1984) reported that NN, Nn and nn females farrowed similar numbers of live pigs, while Nystrom and Andersson (1993) reported that NN and Nn females had similar litter sizes at birth. These studies differ from those of the present one in which Nn females farrowed higher NBA compared to NN and nn females.

Schneider et al. (1980) reported heavier ($P<0.05$) litter birth weights for NN and Nn dams compared to nn dams. Similar results were reported by Simpson et al. (1986) who found HN females to produce 1.19 kg heavier ($P<0.01$) litter birth weights compared to HP females. Nystrom and Andersson (1993) found similar litter birth weights between NN and Nn dams. Webb and Jordon (1978) and Carden et al. (1985) found similar litter weights at birth when comparing HN and HP sows which is in agreement with the present findings.

Normal females had more ($P<0.05$) adjusted NO21 compared to nn females. There was no difference between NN and Nn dams. Normal females produced 6.20 kg and 2.86 kg heavier ($P<0.05$) LWT21 compared to nn and Nn females, respectively. A 3.34 kg advantage ($P<0.05$) for LWT21 was found for Nn females compared to nn females. The proportion of pigs surviving from transfer to 21 d favored ($P<0.05$) NN sows by 13.1% and 9.3% when compared to nn and Nn sows, respectively (Table 3).

The results of the present study agree closely with the previous findings. Mabry (1977) found no NO21 or LWT21 differences between HP and HN dams. Schneider et al. (1980) reported Nn females had more pigs per litter at 28 d ($P<0.05$) than did NN or nn females, but NN females had heavier ($P<0.05$) litters than did either NN or nn females. Additionally,

Table 3. 21-day trait means and estimated mean differences (± SE[1]) between differing porcine stress syndrome genotypes.

Trait	Overall Mean	Genotype Contrast NN-Nn	SE	Nn-nn	SE	NN-nn	SE
Adjusted number at 21 d[2]	10.41	0.28	±0.21	0.21	±0.17	0.49*	±0.24
Adjusted 21-d litter weight, kg[2]	50.65	2.86*	±1.46	3.34*	±1.19	6.20*	±1.66
Survival rate to 21 d, %	88.06	9.33*	±3.71	3.75	±3.15	13.07*	±4.42

[1] Standard error the difference between the two genotype means contrasted.

[2] According to Brubaker et al. (1994).

* Indicates significant difference between genotypes ($P<0.05$).

Schneider et al. (1980) found that NN and Nn dams had lower ($P<0.05$) mortality from birth to 28 d than did nn females. Willeke et al. (1984) found no significant differences in the number of pigs per litter or mortality rates at 28 d between NN, Nn, or nn sows. But, NN dams tended to have more pigs and lower mortality percentage than did Nn or nn dams. Nn females were intermediate for both traits. Simpson et al. (1986) found similar NO21 between HP and HN females, but found that HN sows had heavier ($P<0.01$) LWT21 compared to HP females. Nystrom and Andersson (1993) reported no NO21 differences between NN and Nn females, but NN dams had heavier ($P<0.05$) average pig weight at 21 d than did Nn dams.

Normal females had 0.57 more ($P<0.05$) pigs at 42 d compared to nn females. No significant differences were found between NN and Nn or Nn and nn females for NO42. Normal and Nn females produced 8.22 kg and 5.16 kg, respectively, heavier ($P<0.05$) LWT42 when compared to nn females. No significant LWT42 differences were found between Nn and NN females, but LWT-42 tended to favor NN females. A 7.69% and 4.48% SURV42 advantage was found for NN and Nn females, respectively, when compared to nn females. Normal and Nn females had similar survival rates to 42 d (Table 4).

The 42 d findings of the present study generally agree with previous investigations of Webb and Jordon (1978), who observed no differences in the number of pigs per litter or litter weight at 50 d between HN and HP females. Similar results were reported by Carden et al. (1985) and Lampo et al. (1985). Simpson et al. (1986) reported similar average NO42 between HN and HP sows, but HN dams had superior ($P<0.05$) LWT42 compared to

Table 4. 42-day trait means and estimated mean differences (± SE[1]) between differing porcine stress syndrome genotypes.

Trait	Overall Mean	Genotype Contrast NN-Nn	SE	Nn-nn	SE	NN-nn	SE
Number at 42 d	7.35	0.24	±0.23	0.33	±0.20	0.57*	±0.28
Adjusted 42 d litter weight, kg[2]	81.41	3.06	±3.05	5.16*	±2.49	8.22*	±3.53
Survival rate to 42 d, %	82.39	3.21	±2.72	4.48*	±2.22	7.69*	±3.11

[1] Standard error of the difference between the two genotype means contrasted.

[2] Prior adjustment for age at which 42-d weight was taken.

* Indicates significant difference between genotypes ($P < 0.05$).

HP dams. Nystrom and Andersson (1993) found NN and Nn sows to have similar NO42 and LWT42.

The decision most commercial producers will make is whether to retain Nn females for breeding purposes or to cull them. Research suggests that nn animals would suffer a 5% to 17% higher post-weaning death loss when compared to Nn or NN animals, which would likely increase gilt development costs of nn females substantially (Webb et al., 1982). The inability of nn animals to handle more stressful conditions and their poor maternal performance seems to preclude their use in most breeding programs. It appears that Nn females have as good, and in some cases better, maternal performance than do NN females, particularly for some traits measured at birth (Table 2). But, LWT21 of NN females excelled that of both the Nn and nn females. This suggests that the PSS gene impairs the milk producing ability of both Nn and nn females. The NN females had a superior ability to raise a higher proportion of the pigs to 21 d and 42 d than did Nn females; however, the difference was only significant at 21 d. Beyond 21 d, the litter survival rates may be a function of the individual pig within the litter rather than the dam's mothering ability.

If Nn females are retained in the breeding herd, the resulting offspring are expected to average 50% NN and 50% Nn if mated to NN males, 50% Nn and 25% nn if mated to nn males, and 50% Nn 50% nn if mated to nn males. The previously mentioned increase in post-weaning death loss of nn offspring would likely increase production costs greatly. It has been reported that nearly all nn and 30-50% of Nn offspring will produce carcasses with inferior muscle quality (Christian et al., 1993). The ever

increasing quality conscious consumer would likely discriminate against this type of meat quality. If pork consumption is to increase over time, producers and meat processors will be much more concerned with the muscle quality of pork, and thus, it would be undesirable to have the PSS gene in a commercial producer's herd.

Implications

If Nn females are to be retained in the breeding herd, there would have to be a substantial advantage in maternal performance when compared to NN females. Economic losses would likely result from increased death loss and poor meat quality of offspring produced, particularly when Nn females are mated to Nn or nn males. The results of this research combined with the previous work in the areas of pork quality would indicate that the negative effects of the PSS gene would preclude its use in commercial swine breeding programs. The advent of the molecular test for PSS now allows the commercial producer to determine which breeding females have the PSS gene and eliminate them.

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Appendix

Table 5. Distribution of sow records within each year-season of farrowing by porcine stress syndrome (PSS) genotype.

Year-Season[1]	Genotype NN	Nn	nn	total	Year-Season	Genotype NN	Nn	nn	total
1975-S	4	8	7	19	1984-F	0	4	3	7
1975-F	7	5	10	22	1985-S	0	2	8	10
1976-S	6	8	11	25	1985-F	3	8	9	20
1976-F	11	8	11	30	1986-S	0	7	11	18
1977-S	15	33	22	70	1986-F	1	5	11	17
1977-F	13	18	19	50	1987-S	1	8	8	17
1978-S	12	23	14	49	1987-F	1	6	8	15
1978-F	8	18	13	39	1988-S	2	15	9	26
1979-S	8	17	13	38	1988-F	4	20	4	28
1979-F	1	8	15	24	1989-S	3	21	10	34
1980-S	5	3	13	21	1989-F	6	17	9	32
1980-F	4	3	14	21	1990-S	2	10	10	22
1981-S	0	1	12	13	1990-F	3	9	11	23
1981-F	5	0	8	13	1991-S	2	8	11	21
1982-S	3	2	4	9	1991-F	1	13	10	24
1982-F	3	1	5	9	1992-S	2	10	9	21
1983-S	1	1	2	4	1992-F	3	13	8	24
1983-F	1	2	6	9	1993-S	3	11	7	21
1984-S	0	6	4	10	1993-F	1	6	3	10
					TOTAL	145	358	362	865

[1] S = spring and F = fall farrowing seasons.

Table 6. Distribution of records by parity.

Parity	Number of Records
1	300
2	216
3	162
4	104
5	50
6	24
7	9

Table 7. Inbreeding coefficients of sows, litters farrowed, and service sires by porcine stress syndrome genotype.

Mean inbreeding coefficient of:	Genotype			
	NN	Nn	nn	total
Sows	0.043	0.037	0.078	0.055
Litters farrowed	0.071	0.072	0.090	0.079
Service sires	0.065	0.035	0.065	0.056

$$\text{Adjusted 21-d litter weight} = \text{Weight when weighed} * \left[2.662665 - \left(.11436 * \text{Age at weighing}\right) + \left[.001757 * \left(\text{Age at weighing}\right)^2\right]\right]$$

Equation 1. Formula for adjusting 21-d litter weight for those litters not weighed at exactly 21-d from Brubaker et al. (1994).

$$\text{Adjusted 42-d litter weight} = \left[\left[\left(\frac{\text{Weight when weighed}}{\text{number of piglets weaned}}\right) - \left(\frac{\text{born alive birth weight}}{\text{number born alive}}\right)\right] * \left(\frac{42}{\text{Age when weighed}}\right)\right] + \left(\frac{\text{born alive litter birth weight}}{\text{number born alive}}\right) * \text{number when weighed}$$

Equation 2. Formula for adjusting 42-d litter weight for those litters not weighed at exactly 42-d.

Table 8. Parity adjustment for number of pigs born alive, number of pigs alive at 21 days, and 21-day litter weight (kg) from Brubaker et al. (1994).

Parity	Number born alive adjustment, pigs	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1	0.67	-0.01	2.82
2	0.27	0.00	0.00
3	0.00	0.13	0.49
4	0.00	0.27	1.72
5	0.00	0.40	2.80
6	0.47	0.53	4.31
7	0.74	0.62	5.27
8	1.00	0.74	6.89
≥9	1.84	0.95	9.76

Table 9. Number after transfer adjustment for number of pigs alive at 21 days and 21-day litter weight (kg) from Brubaker et al. (1994).

Number after transfer	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1-2	9.43	47.3
3	7.96	34.5
4	6.95	27.8
5	6.07	23.1
6	5.18	18.6
7	4.25	13.6
8	3.38	9.5
9	2.49	7.7
10	1.68	0.0
11	0.91	0.0
12	0.84	0.0
≥13	0.00	0.0

MATERNAL PERFORMANCE DIFFERENCES BETWEEN PORCINE STRESS SYNDROME NORMAL AND CARRIER LANDRACE FEMALES

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Abstract

Differences between porcine stress syndrome (PSS) normal (NN) and carrier (Nn) Landrace dams were determined for adjusted number of pigs born alive, adjusted number of pigs at 21 d, adjusted 21 d litter weight, proportion of pigs surviving to 21 d, and farrowing interval. Registration number and PSS genotype were obtained from nine purebred Landrace herds and the maternal performance data and pedigree information were obtained from the American Landrace Association. Porcine stress syndrome genotypes were determined by the molecular test described by Fujii et al. (1991). A total of 841 females, 623 NN and 218 Nn having 2231 and 869 records, respectively, were analyzed. Three nn females, two from herd 8 and one from herd 3, were dropped from the analysis because of their low contribution to the total number of records. No other herds reported retaining nn females. Frequency of the n allele ranged from 0.07 to 0.28 in the nine herds involved in this study. When determined by Chi-square analysis, PSS genotypic frequencies within each herd did not differ from expected values based on gene frequency within the same herd. Data were adjusted using the Landrace breed-specific adjustments from Brubaker et al. (1994), and analyzed using derivative-free restricted maximum likelihood procedures fitting the dams' PSS genotype as a fixed effect in the model.

Farrowing interval was calculated as the number of d between successive farrowings. Only females having two or more successive parities were used in this analysis, resulting in a reduction of total records analyzed to 2201 (1564 NN and 637 Nn records) from 632 females (445 NN and 187 Nn females). The proportion of pigs surviving to 21 d was calculated by dividing the number of pigs alive at 21 d by the number of pigs after transfer for each litter farrowed. No differences between NN and Nn dams were observed for adjusted number of pigs born alive, adjusted number of pigs at 21 d, adjusted 21 d litter weight, proportion of pigs surviving to 21 d and farrowing interval. The results of this investigation indicate no significant maternal performance differences between PSS NN or Nn dams. Factors other than maternal performance should determine whether PSS Nn females are retained for breeding purposes on U.S. swine farms.

Introduction

The maternal performance of breeding herd females greatly influences the profitability of commercial and seedstock swine producers (Mabry et al., 1990). The traits generally considered to be the most economically important include: number of pigs born alive, litter birth weight, number of pigs weaned, 21 d litter weight, and litters per sow per year (MacKay, 1993). Increased consumer demand for healthful food products has led meat processors to reward commercial swine producers for lean, heavy-muscled market hogs. Some commercial swine producers have used porcine stress syndrome (PSS) carrier (Nn) or positive (nn) sires to produce leaner market hogs. Reports suggest that nn pigs produce carcasses with superior percent

loin, percent ham, backfat, and loin muscle area when compared to carcasses from either Nn or NN pigs, while Nn animals produced carcasses superior to NN animals (Jensen and Barton-Gade, 1985). Many swine producers still raise their own replacement females. The potential exists for an increase of the PSS positive allele (n) in the breeding herd female population if producers use Nn and nn terminal sires and raise their own breeding herd replacement females.

Porcine stress syndrome was first described in the U. S. by Topel et al. (1968). He noted that some pigs, when stressed, would suddenly collapse and then die within a few minutes. Pigs prone to the condition were then termed PSS susceptible. The physiological basis for PSS was described by Knudson et al. (1990). He demonstrated the existence of differences in the calcium (Ca^{2+}) release channel of muscle cells. Fill et al. (1990) suggested that the Ca^{2+} release channel, once opened, fails to close properly resulting in the symptoms commonly associated with PSS.

Since the discovery of its monogenic autosomal recessive inheritance (Christian, 1972) and the location on chromosome six (Davies et al., 1988), much attention has been given to the effects of PSS genotype on muscle quality and performance traits of market hogs. The effects of PSS genotype on maternal traits has largely been ignored. Frequency of the PSS positive allele varies in different breeds of swine throughout the world. Goodwin (1994) reported a range of PSS gene frequencies from 0.00 to 0.42 among eight pure breeds of swine. He reported a 0.06 PSS frequency in the Landrace breed. Southwood et al. (1988) reported a 0.33 PSS gene frequency in Canadian Landrace pigs while Houde et al. (1993) reported a PSS gene

frequency of 0.15 in British Landrace pigs. O'Brien (1995) reported PSS gene frequencies of 0.18, 0.21, and 0.198 in Canadian, U.S., and English Landrace pigs, respectively.

Few investigations have examined the effects of the PSS gene on maternal performance. The majority of the investigations have focused on differences between stress susceptible (nn) and stress negative (NN and Nn) dams because of the difficulty distinguishing between normal (NN) and carrier (Nn) animals. Those studies having maternal performance results for NN, Nn, and nn sows are few and involve small numbers (Nystrom and Andersson, 1993), or were conducted when less precise methods were available to distinguish between NN and Nn animals (Schneider et al., 1980; Willeke et al., 1984).

Technological advancements in molecular biology led to a molecular DNA test (Fujii et al., 1991) to determine the PSS genotype of individual pigs. This simple and cost effective procedure has the ability to classify individual pigs into one of the three PSS genotypes with an accuracy approaching 100%. Swine producers can submit blood samples from individual pigs to a licensed laboratory and have the PSS genotype of that animal determined by this method.

Individual swine producers should use all of the information available to them to make an informative decision concerning the proper use of the PSS gene in their breeding programs. The decision most producers will need to make concerns the use of Nn females for breeding purposes. The inability of nn females to handle stressors associated with parturition

and the increased post weaning death loss of nn females (Webb et al., 1982), would preclude their use in most breeding programs.

The objective of this study was to determine maternal trait performance differences at birth and 21 d between PSS normal and carrier females.

Materials and Methods

Animals

Nine purebred Landrace herds from across the U.S. provided registration numbers and all known PSS genotypes of sows within their herd. These herds were selected because the PSS genotype of all breeding females were known. Porcine stress syndrome genotypes were determined by commercial laboratories using the DNA molecular procedure described by Fujii et al. (1991). This test detects the actual mutation responsible for PSS and can determine PSS genotypes of individual animals with an accuracy approaching 100%.

Records from a total of 844 sows (623 NN, 218 Nn, and 3 nn females) with known PSS genotypes were provided by these nine herds. The smallest herd provided records for 36 different sows while the largest herd provided records for 235 different sows. The distribution of females by herd and genotype are in Table 1. Frequency of the PSS positive allele (n) ranged from 0.07 to 0.28 in the herds providing information for this study (Table 2). A Chi-square analysis was performed to evaluate the distribution of dams within each PSS genotype. The observed PSS genotype distribution of

Table 1. Distribution of sows by herd and porcine stress syndrome genotype.

Herd	Genotype			Total
	NN	Nn	nn	
1	28	8	0	36
2	28	14	0	42
3	135	57	1	193
4	173	62	0	235
5	60	12	0	72
6	52	9	0	61
7	28	10	0	38
8	22	25	2	49
9	97	21	0	118
Total	623	218	3	844

Table 2. Porcine stress syndrome gene frequency by herd.

	Gene Frequency	
Herd	N	n
1	0.89	0.11
2	0.83	0.17
3	0.85	0.15
4	0.87	0.13
5	0.92	0.08
6	0.93	0.07
7	0.87	0.13
8	0.72	0.28
9	0.91	0.09
Total	0.87	0.13

dams within each herd did not differ from the expected distribution given the PSS gene frequency within each herd.

Data description

Sow productivity records and registration numbers were obtained from the American Landrace Association (ALA) and were merged with the PSS genotypes provided by each Landrace breeder. A total of 3100 records (2231 NN and 869 Nn) were obtained. The three nn dams were dropped from the analysis because of the insufficient number of records to adequately evaluate dams possessing this PSS genotype. The distribution of records by herd and genotype is found in Table 3. The maternal litter information provided by the ALA included: number of pigs born alive (NBA), number of pigs after transfer (NAT), number of pigs at 21 d (NO21), and litter weight taken at approximately 21 d (LWT21). The proportion of pigs surviving from transfer to 21 d (SURV21) was calculated from the data provided by the ALA. Additionally, farrowing interval between from two consecutive parities was calculated. A total of 2201 records from 632 sows (Table 4) were used in the analysis of farrowing interval.

Statistical analysis

Landrace breed-specific adjustments for parity, NAT, and age at weighing (Brubaker et al., 1993) were applied to NBA, NO21, and LWT21 prior to the analysis of these traits. All reproductive data were analyzed using the full animal model described as:

$$y = Xb + Zu + e$$

where y = vector of phenotypic performance values for an animal,

Table 3. Distribution of sow records by herd and porcine stress syndrome genotype.

Herd	Genotype		Total
	NN	Nn	
1	131	49	180
2	157	90	247
3	472	257	729
4	848	277	1125
5	95	22	117
6	172	29	201
7	52	25	77
8	74	70	144
9	230	50	280
Total	2231	869	3100

Table 4. Distribution of dams (and the number of records) having two or more consecutive records by herd and porcine stress syndrome genotype.

Herd	Genotype NN		Nn		Total	
1	24	(103)	8	(41)	32	(144)
2	27	(128)	14	(76)	41	(204)
3	85	(329)	53	(192)	138	(521)
4	163	(656)	60	(210)	223	(866)
5	27	(31)	8	(9)	35	(40)
6	42	(116)	8	(19)	50	(135)
7	15	(24)	7	(15)	22	(39)
8	18	(49)	20	(47)	38	(96)
9	44	(128)	9	(28)	53	(156)
Total	445	(1564)	187	(367)	632	(2201)

X = incidence matrix relating fixed effects to the phenotypic performance values, y,

b = unknown vector of fixed effects,

Z = incidence matrix relating random effects to the phenotypic performance values, y,

u = unknown vector of random effects, and

e = vector of random residual effects,

where the variance u is $A\sigma^2_a$ (A is the relationship matrix) and the variance of e is $I\sigma^2_e$.

The model was incorporated using multiple-trait DFREML procedures (MTDFREML) (Boldman and Van Vleck, 1993). The MTDFREML algorithms provide estimates of fixed effects included in the model. The relationship matrix was included in the animal model to account for the known variance-covariance structure that exists among additive breeding values of the animals evaluated. The inclusion of the relationship matrix allows for more precise definitions of fixed effects included in the model (Wiggans et al., 1989). A total of 1702 animals, 641 with records and 1061 base animals, were included in the relationship matrix. Each herd was genetically tied, through related individuals, to at least one other herd such that all herds were connected. Contemporary groups (CG) within each herd were tied by one or more females. More precise estimates of fixed effects and predictions of random effects are obtained when herds and contemporary groups are adequately connected.

Contemporary group was defined as herd-year-season of farrowing. A total of 132 contemporary groups were formed and groups were included as a fixed effect in all models. In order to minimize the number of contemporary groups having fewer than five records, season was defined as four consecutive months of time. Other fixed effects included in all models included the type of litter allowed to nurse (purebred or crossbred) and PSS genotype of the sow producing the record. Additionally, parity was included in the models for SURV21 and farrowing interval because no published adjustments for these traits were available. Estimates of fixed effects are empirically best linear unbiased estimators (BLUE) of some estimable function of the fixed effects (Henderson, 1973).

Inbreeding of the female (animal) was used as a covariate in the model for all traits. Additionally, NAT was used as a covariate in the SURV21 model and weight of the previous litter adjusted only for age at weighing was used as a covariate for farrowing interval.

Random effects included in all models were animal, sire, dam, service sire, repeated record, and maternal effects. The solutions for the random effects are empirically best linear unbiased predictors (BLUP) of some estimable function of the random effects (Henderson, 1973).

All traits were analyzed singly and evaluated to the recommended convergence (1.E-9) for the variance of the simplex function (Boldman et al., 1993). All models were "cold restarted" with previous variance - covariance values. Convergence to a global maximum was determined when two successive cold restarts yielded the same variances, covariances, and -2 log likelihood of the simplex function (Boldman et al., 1993). The

globally maximized values were used to obtain solutions for the mixed model equations (solutions for all covariates, fixed, and random effects). The contrast between NN and Nn PSS genotype for all traits was made in the final MTDFREML run. If the difference between the two genotypes for a particular trait was larger than two times the standard error of the two means involved in the contrast, they were considered different ($P<0.05$) as suggested by Boldman et al. (1993).

Results and Discussion

There were no significant differences between NN and Nn dams for any of the traits analyzed (Table 5). Normal and Nn females averaged nearly identical adjusted NBA. Adjusted number at 21 d was very similar for NN and Nn sows. Though not significantly, NN sows produced litters that averaged 0.45 kg heavier at 21 d than those of Nn sows. The average SURV21 was nearly identical when the averages produced by NN and Nn dams were compared. Similarly, farrowing interval was only 0.44 d different (not significant) for NN and Nn dams.

Nystrom and Andersson (1993) reported similar NBA and NO21 results in a study involving NN and Nn Yorkshire sows. But, they found NN dams to produce litters with higher average pig weight at 21 d ($P<0.05$) compared to average pig weight in litters from Nn dams.

Willeke et al. (1984) reported that NN, Nn, and nn German Landrace sows produced litters with similar NBA, number at 28 d, and mortality (survival) rate. Normal dams tended to produce more NBA, number of pigs at

Table 5. Reproductive trait means and estimated mean differences (± SE[1]) between porcine stress syndrome normal and carrier dams.

Trait	Overall Mean	Genotype Contrast NN-Nn	±S.E.	
Adjusted number born alive[2]	10.82	-0.003	0.14	N.S.[3]
Adjusted number at 21 d[2]	12.17	-0.03	0.05	N.S.
Adjusted 21-d litter weight, kg[2]	68.71	0.45	0.48	N.S.
Survival to 21 d, %	95.48	0.0006	0.0004	N.S.
Farrowing interval, d	172.00	0.45	1.80	N.S.

[1] Standard error of the difference between the genotype means.

[2] Landrace breed-specific adjustments (Brubaker et al., 1994).

[3] N.S. - all differences were non-significant.

28 d, and lower mortality rates compared to Nn or nn females. Carrier sows were intermediate for these traits. Schneider et al. (1980) reported identical NBA for NN and Nn PSS genotypes in a study involving Swiss Landrace sows. They also reported similar birth weights and mortality rates between NN and Nn dams. They reported that Nn sows had 0.11 more pigs ($P<0.05$) at 28 d than did NN sows. Normal dams, however, produced litters 1.50 kg heavier ($P<0.05$) at 28 d than Nn dams.

Other studies involving British and Belgian Landrace dams (Simpson et al., 1986); Lampo et al., 1985) have focused on comparisons between halothane positive (HP) (PSS genotype nn) and halothane negative (HN) (PSS genotypes NN or Nn) females. These studies generally found HP sows to produce poorer litter weights at birth and 21 d, while having litters with similar NBA and NO21 as HN females.

Studies involving a synthetic Hampshire - Pietrain line also focused on differences between HP and HN females (Carden et al., 1985; Webb and Jordon, 1978). Both studies reported HN sows to produce significantly ($P<0.05$ or higher) higher NBA than HP females. Neither study found significant LWT21 differences between HP and HN dams.

Mabry (1977) found HN and HP sows to have similar NBA, NO21, LWT21, number of pigs per litter at 56 d, and survival rates to 56 d in a Yorkshire-based PSS susceptible herd. There was some tendency for HP dams to produce poorer LWT21 compared to HN females.

The results of this investigation suggest there to be no difference in the maternal performance of NN or Nn Landrace females. The previously mentioned research regarding the maternal performance of nn females and the

higher post weaning death loss associated with nn animals (Webb et al., 1982) would seem to preclude the use of nn females in the breeding herd by commercial swine producers. The decision, however, most commercial producers must make is whether to retain Nn females in their breeding herds. If retained, the resulting offspring would on average be 50% Nn if mated to NN males, 50% Nn and 25% nn if mated to Nn males, and 50% Nn and 50% nn if mated to nn males. Previous research indicates that nearly all nn and 30-50% of the Nn animals will produce carcasses with inferior muscle quality (Goodwin, 1994). Research also suggests that Nn and nn pigs have similar average daily gain and feed efficiency when compared to NN animals (Christian and Rothschild, 1981). Similar performance results were reported between PSS genotypes by Simpson and Webb (1989) in a study of British Landrace market hogs. Jensen and Barton-Gade (1985) evaluated growth and carcass traits in Danish Landrace pigs of all possible PSS genotypes. They reported similar feed efficiency between NN, Nn, and nn animals. A linear effect was observed with nn pigs having carcasses with superior ($P<0.05$) percent loin, percent ham, backfat, and loin muscle area. The NN animals produced carcasses that were the poorest, while Nn animals produced intermediate carcasses for these traits.

Implications

Swine producers should use all available information when determining the appropriate use of the PSS gene in their herd. This research indicates no maternal performance difference between PSS NN and Nn sows. Hence, a swine producer's decision regarding the use of the PSS gene should be based

on factors other than maternal performance. Economic losses would likely result from the increased death loss and poor meat quality of offspring produced when Nn females are mated to Nn or nn males. If Nn females are retained in the breeding herd, a substantial advantage in maternal performance would be required to make up for the economic losses associated with the PSS gene when compared to NN females. The results of this research combined with the previous work in the areas of pork quality would indicate that the negative effects of the PSS gene would prevent its use in commercial breeding programs. The advent of the molecular test for PSS now allows the commercial producer to identify those females with the mutated PSS allele and eliminate them from the breeding herd.

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Appendix

Table 6. Observed and (expected) number of dams of each porcine stress syndrome genotype by herd.

Herd	Genotypes						Total	χ^2
	NN	χ^2	Nn	χ^2	nn	χ^2		
1	28 (29)	0.034	8 (7)	0.143	0 (0)	0.000	36	0.177
2	28 (29)	0.034	14 (12)	0.333	0 (1)	1.000	42	1.367
3	135 (140)	0.179	57 (49)	1.306	1 (4)	2.250	193	3.725
4	173 (178)	0.140	62 (53)	1.528	0 (4)	4.000	235	5.668
5	60 (61)	0.016	12 (11)	0.091	0 (0)	0.000	72	0.017
6	52 (53)	0.019	9 (8)	0.125	0 (0)	0.000	61	0.144
7	28 (29)	0.034	10 (9)	0.111	0 (1)	1.000	38	1.145
8	22 (25)	0.360	25 (20)	1.250	2 (4)	1.000	49	2.610
9	97 (98)	0.010	21 (19)	0.211	0 (1)	1.000	118	1.221
Total	623 (630)	0.826	218 (191)	5.098	3(14)	10.250	844	16.174

Table 7. Records per sow by porcine stress syndrome genotype and herd.

Herd	Genotype		Total
	NN	Nn	
1	4.68	6.13	5.00
2	5.61	6.43	5.88
3	3.50	4.51	3.80
4	2.73	4.47	4.79
5	1.58	1.83	1.63
6	3.31	3.22	3.30
7	1.86	2.50	2.03
8	3.36	2.80	3.06
9	2.37	2.38	2.37
Total	3.58	3.99	3.69

Table 8. Distribution of sow records by parity and herd.

Parity	1	2	3	4	5	6	7	8	9	Total
1	36	41	192	224	31	58	38	38	109	767
2	32	40	138	220	58	50	22	31	56	647
3	31	37	121	183	28	38	11	26	40	515
4	27	32	96	140	0	22	5	20	30	372
5	24	28	71	108	0	12	1	14	19	277
6	15	23	46	80	0	10	0	10	15	199
7	8	19	27	60	0	5	0	2	6	127
8	4	10	19	40	0	3	0	1	3	80
9	2	8	13	25	0	2	0	1	2	53
10	1	6	2	21	0	1	0	1	0	32
11	0	1	4	9	0	0	0	0	0	14
12	0	1	0	6	0	0	0	0	0	7
13	0	1	0	6	0	0	0	0	0	7
14	0	0	0	1	0	0	0	0	0	1
15	0	0	0	1	0	0	0	0	0	1
16	0	0	0	1	0	0	0	0	0	1
Total	180	247	729	1125	117	201	77	144	280	3100

Table 9. Distribution of records from dams having two or more consecutive parities.

Parity	1	2	3	4	5	6	7	8	9	Total
1	32	39	138	208	21	46	22	28	44	578
2	31	37	120	176	19	35	11	22	38	489
3	27	31	92	137	0	22	5	19	29	362
4	24	28	69	106	0	12	1	14	19	273
5	15	23	43	77	0	10	0	8	15	191
6	8	19	26	56	0	4	0	2	6	121
7	4	12	17	39	0	3	0	1	3	79
8	2	6	12	25	0	2	0	1	2	50
9	1	6	2	20	0	1	0	1	0	31
10	0	1	2	9	0	0	0	0	0	12
11	0	1	0	4	0	0	0	0	0	5
12	0	1	0	6	0	0	0	0	0	7
13	0	0	0	1	0	0	0	0	0	1
14	0	0	0	1	0	0	0	0	0	1
15	0	0	0	1	0	0	0	0	0	1
Total	144	204	521	866	40	135	39	96	156	2201

Table 10. Average sow inbreeding coefficient by porcine stress syndrome genotype and herd.

	Genotype		
Herd	NN	Nn	Total
1	0.007	0.004	0.006
2	0.005	0.001	0.004
3	0.018	0.009	0.015
4	0.012	0.015	0.012
5	0.005	0.000	0.004
6	0.032	0.028	0.031
7	0.001	0.000	0.001
8	0.016	0.056	0.037
9	0.002	0.004	0.002
Total	0.012	0.015	0.012

$$\text{Adjusted 21-d litter weight} = \text{Weight when weighed} * \left[2.9619 - \left(0.14367 * \text{Age at weighing}\right) + \left[0.00239 * \left(\text{Age at weighing}\right)^2\right]\right]$$

Equation 1. Landrace breed-specific age adjustment formula for those litters not weighed at 21 days of age from Brubaker et al. (1994).

Table 11. Landrace breed-specific parity adjustment for number of pigs born alive, number of pigs alive at 21 days, and 21-day litter weight (kg) from Brubaker et al. (1994).

Parity	Number born alive adjustment, pigs	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1	0.57	0.10	4.57
2	0.29	0.00	0.00
3	0.00	0.08	0.08
4	0.00	0.20	0.81
5	0.00	0.27	1.51
6	0.32	0.40	3.19
7	0.64	0.45	3.91
8	0.84	0.58	5.83
≥9	2.09	0.78	8.74

Table 12. Landrace breed-specific number after transfer adjustment for number of pigs alive at 21 days and 21-day litter weight (kg) from Brubaker et al. (1994).

Number after transfer	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1-2	10.06	51.65
3	8.65	40.10
4	7.61	32.67
5	6.91	29.18
6	5.94	24.54
7	5.02	18.57
8	4.11	14.12
9	3.19	9.36
10	2.38	0.00
11	1.66	0.00
12	0.98	0.00
≥13	0.00	0.00

RESTRICTED MAXIMUM LIKELIHOOD GENETIC PARAMETER ESTIMATES FOR MATERNAL SWINE TRAITS FROM LANDRACE FEMALES WITH KNOWN PORCINE STRESS SYNDROME GENOTYPES

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Abstract

Heritabilities, additive and maternal genetic variances, and environmental variances were estimated from purebred Landrace field data. Porcine stress syndrome (PSS) genotype of all dams with records was known. Maternal performance traits were evaluated using a full animal model. A total of 3100 records from 841 sows were analyzed. Traits evaluated included number born alive (NBA), number at 21 d (NO21), 21-d litter weight (LWT21), survival rate to 21 d (SURV21), and farrowing interval. Each trait was analyzed individually using MTDFREML procedures. Fixed effects included in the model for each trait were contemporary group, type of litter (purebred or crossbred), and PSS genotype of dam (animal with record). Random effects included in the model were animal, sire, dam, service sire, repeated records, and maternal influence. Number at 21 d was preadjusted for parity and number after transfer (NAT) prior to evaluation according to Brubaker et al. (1994). Number born alive and LWT21 were adjusted for parity, age at weighing, and number after transfer according to Brubaker et al. (1994) and current National Swine Improvement Federation (NSIF) recommendations. Additionally, covariates for parity, age at weighing, and number after transfer were added to the models for number

born alive, number at 21 d, and 21-d litter weight to develop data set specific adjustments for these environmental effects. The model for SURV21 included the additional fixed effect for parity and a covariate for NAT. Farrowing interval was calculated as the number of d between consecutive farrowings. A total of 632 dams with 2201 records were evaluated in the farrowing interval analysis. An additional fixed effect for parity and a covariate for 21 d litter weight adjusted for age were included in the model for the analysis of farrowing interval. Heritabilities for NBA were 0.05, 0.06, and 0.04 when adjusted according to Brubaker et al. (1994), NSIF (1987), or when data specific adjustments were used, respectively. The heritability estimate for number at 21 d was 0.32 when adjusted according to Brubaker et al. (1994), and 0.21 when the records were adjusted by including the appropriate environmental effects in the model for analysis. Heritabilities for LWT21 were 0.21, 0.19, and 0.14 when adjusted according to Brubaker et al. (1993), NSIF (1987), or when specific adjustments were used, respectively. The heritability estimate for SURV21 was 0.21 and the heritability for farrowing interval was estimated as 0.02.

Introduction

Genetic improvement for economically important production traits relies on accurate estimates of heritability. Maternal performance traits are generally lowly heritable (Lamberson, 1990). Genetic improvement through selection, however, is still possible because of the large standard deviation found for most reproductive traits. Maternal performance traits greatly influence profitability of commercial swine production (Mabry et

al., 1990). The traits having the greatest impact on profitability include number of pigs born alive, litter birth weight, number of pigs weaned, 21 d litter weight, and litters per sow per year (MacKay, 1993). These maternal traits are considerably more economically important when compared to growth and carcass traits (See, 1994). Genetic improvement of these traits can help seedstock and commercial swine producers become more efficient and profitable. Knowledge of the genetic and environmental variances and heritabilities can assist producers in deciding which traits should receive selection emphasis. This knowledge may also allow producers to estimate the amount of time it will take to improve a given trait.

The majority of previous investigations have used traditional analysis of variance procedures to estimate the heritability of various reproductive traits and are summarized by Lamberson (1990). Advancements in computer and algorithm technology allow more efficient estimation of heritability from large data sets.

Restricted maximum likelihood methods (REML) (Patterson and Thompson, 1971) have been used to estimate components of variance and heritability. Estimates of variance components are influenced by the choice of data, method of analysis, and model utilized (Mistal, 1990). This method has considerable computational requirements and is difficult to program (Mistal, 1995). A derivative-free (DF) REML procedure was developed by Graser et al. (1987) to estimate variance components which reduces the computational needs and is less difficult to program when compared to prior REML procedures. Meyer (1989) outlined the use of DFREML procedures to estimate variance components using a model with more than one random effect

in the model. The development of DFREML procedures allowed the use of more complex models to estimate genetic parameters from animal breeding data. Computational needs are further reduced when a direct sparse matrix solver is utilized with DFREML procedures to obtain the log-likelihood function. The use of the direct sparse matrix solver along with the DFREML procedures still empirically yield best linear unbiased estimators (BLUE) and best linear unbiased predictors (BLUP) of fixed and random effects included in the model for analysis (Boldman and Van Vleck, 1991). This method allows the use of the full animal model which incorporates the relationship matrix of all animals when estimating variance components. The use of the full animal model and its corresponding relationship matrix accounts for the known variance-covariance structure which exists among relative and allows for more accurate parameter estimation (Wiggans et al., 1989).

The purpose of adjustment factors is to negate environmental influences on the phenotypic record of an animal (Kemp and Rothschild, 1988). Adjusted phenotypic records are suited to more accurate determination of genetic effects and the evaluation of each animal's genetic capability for selection purposes (Kemp and Rothschild, 1988). The adjustment factors are applied to the records of various traits prior to conducting the analysis of the data. This reduces the number of equations to be solved in mixed model evaluation by reducing the number of covariates and fixed effects in the model. The National Swine Improvement Federation has published general adjustment factors that can be applied to maternal data collected from any breed. Brubaker et al. (1994) published Landrace and Yorkshire breed-specific adjustment factors for several maternal traits. It is generally

accepted that adjustment factors may differ between breeds and that breed-specific adjustments can more adequately account for environmental effects when compared to general adjustments.

The objectives of this study were: 1) to estimate maternal trait genetic parameters from Landrace females with known porcine stress syndrome (PSS) genotypes; 2) to numerically compare genetic parameter estimates when data are adjusted using current NSIF (1987) recommendations or Landrace breed-specific adjustments, or when the data is adjusted by fitting various environmental effects into the model for maternal traits.

Materials and Methods

Animals

Nine purebred Landrace breeders from across the U.S. provided litter performance records, registration numbers and PSS genotype of sows from within their herds. These herds were chosen because of a knowledge of the PSS genotype for all of their breeding animals. Porcine stress syndrome genotype of animals within these herds had been determined by commercial laboratories using the DNA molecular procedure described by Fujii et al. (1991). This test detects the mutation responsible for PSS and determines the PSS genotype of individual animals with an accuracy approaching 100% (Fujii et al., 1991).

Records from total of 844 dams (623 NN, 218 Nn, and 3 nn females) with known PSS genotype were provided by these nine herds. The smallest herd provided records of 36 sows while the largest herd provided records of 235 sows. The distribution of females by herd and genotype is found in

Table 1. Frequency of the PSS positive allele (n) ranged from 0.07 to 0.28 in the various herds providing information for this study (Table 2). A Chi-square analysis was performed to evaluate the distribution of dams within each PSS genotype. The observed PSS genotype distribution of dams within each herd did not differ from the expected distribution given the PSS gene frequency within each herd.

Data description

The actual sow productivity records were obtained from the American Landrace Association (ALA) and were merged with the respective sow's PSS genotype provided by each Landrace breeder. A total of 3100 (2231 NN and 869 Nn) records were obtained. The three nn dams were dropped from the analysis because of the insufficient number of records available to adequately evaluate dams having this PSS genotype. The distribution of records by herd and genotype can be found in Table 3. Litter information provided by the ALA included: number of pigs born alive (NBA), number of pigs after transfer (NAT), number of pigs at 21 d (NO21), and litter weight taken at approximately 21 d (LWT21). The proportion of pigs surviving from transfer to 21 d (SURV21) was calculated from the data provided by the ALA. Additionally, farrowing interval between two consecutive parities was calculated. A total of 2201 records from 632 females (Table 4) was used in the analysis of farrowing interval.

Statistical analysis

The adjustment factors recommended by NSIF (1987) and Landrace breed-specific adjustments (Brubaker et al., 1993) for parity, NAT, and age at weighing were applied to NBA, NO21, and LWT21 prior to the analysis of

Table 1. Distribution of dams by herd and porcine stress syndrome genotype.

Herd	Genotype			Total
	NN	Nn	nn	
1	28	8	0	36
2	28	14	0	42
3	135	57	1	193
4	173	62	0	235
5	60	12	0	72
6	52	9	0	61
7	28	10	0	38
8	22	25	2	49
9	97	21	0	118
Total	623	218	3	844

Table 2. Porcine stress syndrome gene frequency by herd.

Herd	Gene Frequency	
	N	n
1	0.89	0.11
2	0.83	0.17
3	0.85	0.15
4	0.87	0.13
5	0.92	0.08
6	0.93	0.07
7	0.87	0.13
8	0.72	0.28
9	0.91	0.09
Total	0.87	0.13

Table 3. Distribution of sow records by herd and porcine stress syndrome genotype.

Herd	Genotype		Total
	NN	Nn	
1	131	49	180
2	157	90	247
3	472	257	729
4	848	277	1125
5	95	22	117
6	172	29	201
7	52	25	77
8	74	70	144
9	230	50	280
Total	2231	869	3100

Table 4. Distribution of dams (and the number of records) having two or more consecutive records by herd and porcine stress syndrome genotype.

Herd	Genotype				Total	
	NN		Nn			
1	24	(103)	8	(41)	32	(144)
2	27	(128)	14	(76)	41	(204)
3	85	(329)	53	(192)	138	(521)
4	163	(656)	60	(210)	223	(866)
5	27	(31)	8	(9)	35	(40)
6	42	(116)	8	(19)	50	(135)
7	15	(24)	7	(15)	22	(39)
8	18	(49)	20	(47)	38	(96)
9	44	(128)	9	(28)	53	(156)
Total	445	(1564)	187	(367)	632	(2201)

these traits. Additionally, data set-specific adjustments were determined by fitting these effects into the models for the various traits. A fixed effect for parity was included in the models used for the analysis of NBA, NO21, and LWT21. A covariate for NAT was included in the models for NO21 and LWT21. Age at weighing was also added as a fixed effect in the model for LWT21. All reproductive data were analyzed using the full animal model described as:

$$y = Xb + Zu + e$$

where y = vector of phenotypic performance values for an animal,

X = incidence matrix relating fixed effects to the phenotypic performance values, y,

b = unknown vector of fixed effects,

Z = incidence matrix relating random effects to the phenotypic performance values, y,

u = unknown vector of random effects, and

e = vector of random residual effects,

where the variance u is $A\sigma^2_a$ (A is the relationship matrix) and the variance of e is $I\sigma^2_e$.

The model was incorporated using multiple-trait DFREML procedures (MTDFREML) (Boldman and Van Vleck, 1993). Restricted maximum likelihood procedures (DFREML) are considered state-of-the-art methodology to estimate variance and covariance components of unbalanced animal breeding data (Boldman et al., 1993). The DFREML procedures have the ability to

implement more complex models and are more computationally efficient than methods previously utilized to estimate variance components.

The MTDFREML algorithms also yield estimates of fixed effects included in the model. The relationship matrix was included in the animal model to account for the known variance-covariance structure that exists among additive breeding values of the animals evaluated. The inclusion of the relationship matrix allows for more precise definitions of the effects included in the model and more precise estimates of genetic parameters (Wiggans et al., 1989). Genetic variance is generally biased downward when relationships are ignored in the genetic analysis (Dong et al., 1988).

A total of 1702 animals, 641 sows with records and 1061 base animals, were included in the relationship matrix. Each herd was genetically tied, through related individuals, to at least one other herd such that all herds were connected. Contemporary groups (CG) within each herd were tied by one or more females. More precise estimates of fixed effects, predictions of random effects, and genetic parameter estimates can be made when herds and contemporary groups are adequately connected.

Contemporary groups were defined as herd-year-season of farrowing. A total of 132 contemporary groups were included as fixed effects in all models. In order to minimize the number of contemporary groups having less than five records, a season was defined as four consecutive months. Other fixed effects included in all models included the type of litter allowed to nurse (purebred or crossbred) and the PSS genotype of the female with the record. Additionally, parity was included in the models for SURV21 and farrowing interval because no previous parity adjustments have been

published for these traits. Estimates of fixed effects are empirically best linear unbiased estimators (BLUE) of some estimable function of the fixed effects (Henderson, 1973).

A covariate for NAT was used for SURV21 and farrowing interval while a covariate for weight of the previous litter, adjusted only for age, and was used for the analysis of farrowing interval.

Random effects included in all models were animal, sire, dam, service sire, repeated record, and maternal effects. The solutions for the random effects are empirically best linear unbiased predictors (BLUP) of some estimable function of the random effects (Henderson, 1973).

All traits were analyzed individually and evaluated to the recommended convergence (1.E-9) for the variance of the simplex function (Boldman et al., 1993). All models were "cold restarted" with previous variance - covariance values. Convergence to a global maximum was determined when two successive cold restarts yielded the same variances, covariances, and -2 log likelihood of the simplex function (Boldman et al., 1993). The globally maximized values were used to obtain solutions for the mixed model equations (solutions for all covariates, fixed, and random effects).

Results and Discussion

Overall means and standard deviations for the maternal traits included in this study are listed in Table 5. Means are numerically different for NBA, NO21, and LWT21 are dependent on the adjustment factors applied. The base for parity and for number after transfer from which adjustments are

Table 5. Reproductive trait means and standard deviations.

Trait	Overall Mean	SD[1]
Adjusted number born alive (Brubaker et al., 1994)	10.82	2.76
Adjusted number born alive (NSIF, 1987)	11.12	2.75
Adjusted number born alive (adjusted with own data)	10.47	2.79
Adjusted number at 21 d (Brubaker et al., 1994)	12.17	0.86
Adjusted number at 21 d (adjusted with own data)	9.51	1.78
Adjusted 21-d litter weight (Brubaker et al., 1994), kg	68.71	9.02
Adjusted 21-d litter weight (NSIF, 1987), kg	64.31	9.35
Adjusted 21-d litter weight (adjusted with own data), kg	61.65	13.77
Survival rate to 21 d, %	95.48	8.16
Farrowing interval, d	172.00	36.17

[1] SD - Standard deviation.

not required differ between the adjustment methods. These factors contribute to the differences in the overall means for NBA, NO21, and LWT21. Heritabilities for NBA, NO21, and LWT21 were 0.05, 0.32, and 0.21, respectively, when Landrace breed-specific adjustments were utilized (Brubaker et al. 1994). When the data were adjusted using NSIF (1987) recommendations, the heritability estimates were 0.06 for NBA and 0.19 for LWT21. The heritability estimates for NBA, NO21, and LWT21 were 0.04, 0.21, and 0.14, respectively. Additionally, the heritability estimate for SURV21 was 0.21 and the heritability of farrowing interval was 0.02. Heritability and other parameter estimates are summarized in Table 6. The heritability estimates for NBA were similar regardless of the method used to adjust the data for environmental influences. The LWT21 heritability estimates were similar when the data were adjusted using Landrace breed-specific or NSIF adjustment recommendations and both were numerically higher than the estimate for LWT21 when parity, age at weighing, and NAT were included into the analysis model.

The additive variance estimate for NBA was 0.34 when Landrace breed-specific adjustments were applied to the data. When the current NSIF (1987) adjustments were applied to the data, the additive variance for NBA was 0.42 and the estimate was 0.27 when parity was included into the model. The additive variance for NO21 was 0.33 when the data were adjusted prior to analysis using the Landrace breed-specific adjustments and 0.18 when parity and NAT were included into the model. The estimates of LWT21 additive variance were 16.07 and 15.10, respectively, when Landrace breed-specific and NSIF adjustments were applied to the data. When parity and

Table 6. Landrace reproductive genetic parameter estimates.

Trait	Parameter Estimates[1]					
	V_A	V_M	V_E	V_T	h^2	h^2_M
Adjusted number born alive (Brubaker et al., 1994)	0.34	0.04	5.92	7.08	0.05	0.01
Adjusted number born alive (NSIF, 1987)	0.42	0.00	5.87	6.92	0.06	0.00
Adjusted number born alive (adjusted with own data)	0.27	0.05	5.87	6.91	0.04	0.01
Adjusted number at 21 d (Brubaker et al., 1994)	0.33	0.04	0.54	1.02	0.32	0.04
Adjusted number at 21 d (adjusted with own data)	0.18	0.07	0.51	0.88	0.21	0.08
Adjusted 21-d litter weight (Brubaker et al., 1994)	16.07	2.88	59.73	75.25	0.21	0.04
Adjusted 21-d litter weight (NSIF, 1987)	15.10	4.17	63.09	78.21	0.19	0.05
Adjusted 21-d litter weight (adjusted with own data)	13.69	3.19	82.61	97.25	0.14	0.03
Survival rate to 21 d	0.13	0.26	0.45	0.69	0.21	0.39
Farrowing interval	23.67	0.08	1032.83	1055.25	0.02	0.00

[1] V_A - additive genetic variance.

V_M - maternal genetic variance.

V_E - environmental variance.

V_T - total phenotypic variance.

h^2 - heritability.

h^2_M - maternal heritability.

NAT were included in the model for LWT21, an additive variance of 13.69 was obtained. Additive variances were higher when the data were adjusted using the NSIF or Brubaker et al. (1994) recommendations compared to those obtained when the various environmental effects were included in the models. Additive variance estimates of 0.13 and 23.67 were obtained for SURV21 and farrowing interval, respectively. Variance estimates are summarized in Table 6. The small amount of additive variance for SURV21 indicates that improvement through selection would be difficult even though the heritability is 0.21. Similarly, the results indicate that farrowing interval is largely influenced by environmental factors (as indicated by the large environmental variance) and improvement of this trait by selection would likely be slow. The remaining traits appear to have sufficient heritabilities and additive variance to be improved by selection.

Maternal genetic variance accounted for less than 1% of the total phenotypic variance for NBA regardless of the method used to adjust the data for parity (Table 6). The maternal variance for NO21 accounted for 3.9% of the total phenotypic variance when Landrace breed-specific adjustments were applied prior to conducting the analysis, and accounted for 7.9% when parity and NAT were included into the model for the analysis (Table 6). The maternal variance for LWT21 ranged from 3.3% to 5.3% when expressed as a proportion of total phenotypic variance depending on the adjustment method applied to the data (Table 6). Maternal variance accounted for 38% of the total phenotypic variance for SURV21 (Table 6).

Young et al. (1978) reported heritability estimates of 0.66 for NBA, 0.29 for NO21, and 0.38 for weaning weight in a synthetic population. Their reported heritability estimates for NBA and LWT21 were considerably higher than those estimates found in this study (Table 6). Strang and Smith (1979) reported heritability estimates for numerous maternal traits in British Landrace females. They found heritability estimates of 0.07, -0.02, 0.01, and 0.07 for NBA, NO21, SURV21, and LWT21, respectively. The most striking difference between these estimates and those found in this investigation were for NO21 and LWT21, the current estimates being larger.

Irgang and Robison (1982) reported heritability estimates of 0.27 for farrowing interval when estimated by paternal half sib analysis and 0.24 when estimated by maternal half sib analysis. These estimates are higher than those found in the present study. Vangen (1986) reported farrowing interval heritability estimates for first to second, second to third, third to fourth, and fourth to fifth parities. All of the estimates he reported were higher than those of this investigation. The current data were obtained from purebred seedstock herds. Purebred seedstock breeders may tolerate longer farrowing intervals, particularly with females producing outstanding maternal records. Commercial producers may be more likely to cull females having rebreeding problems.

Irvin and Swiger (1984) reported heritabilities of 0.20 for NBA, 0.30 for NO21, and 0.15 for LWT21 from data that included records from purebred Yorkshire, Hampshire, and Duroc females. MacCarter et al. (1987) estimated maternal trait heritabilities from field data obtained from the American Yorkshire Club and found the heritabilities to be 0.13 for NBA and 0.15 for

LWT21. Gu et al. (1989) estimated heritabilities from two Cotswold synthetic lines, one of Landrace origin and one of Large White origin. Heritability estimates for NBA were 0.067 for the line originating from Landrace and 0.118 for the line having a Large White origin. Kaplon et al. (1991) estimated maternal trait heritabilities from data obtained from Polish Large White nucleus herds and found heritabilities of 0.07 for NBA, 0.06 for NO21, and 0.06 for LWT21. See et al. (1993) reported NBA heritability estimates of 0.13, 0.13, and 0.12 from Hampshire, Spotted, and Landrace field data, respectively.

Lamberson (1990) summarized heritability estimates for various maternal performance traits. These studies were conducted on several breeds or lines found throughout the world. Some of the previously cited investigations were included in his summary. He reported a mean heritability of 0.07 for NBA, a value very similar to the estimate found in this study. He also reported a mean heritability of 0.06 for number weaned which is considerably lower than the estimates from this investigation. The LWT21 estimates found in this study were reasonably close to the summarized average reported by Lamberson (1990). He also reported an average heritability of 0.05 for survival rate to weaning which is lower than the 0.21 estimate found in this study.

In general the results from the present study fall within the range of heritabilities summarized by Lamberson (1990). The results of previous work and those found in the present study indicate that heritabilities are likely to differ depending on breed and the estimation method used to obtain the estimates. The advancements made in computer hardware and

software technologies give researchers the capability of more accurately estimating heritability for a given trait by application of more sophisticated methods of analysis. The incorporation of the relationship matrix into the full animal model also provides more accurate estimates of heritability by taking into account factors such as merit of mates and prior selection.

Implications

In this sample of the U.S. Landrace population heritabilities of NBA and farrowing interval were found to be low and those for NO21, LWT21 and SURV21 in the moderate range. Maternal genetic effects generally accounted for only a small amount of the total phenotypic variance. The exception to this generalization was for SURV21 where maternal genetic effects accounted for 38% of the total phenotypic variance. These traits appear to have sufficient heritability and additive genetic variance to be improved by selection. The method used to adjust the data for known environmental effects does not appear to drastically change the heritability estimates of the traits. But, estimates were generally higher when Landrace breed-specific adjustments were applied. Heritability estimates may need to be breed or population specific when conducting genetic evaluations. More research needs to be conducted to compare general and breed-specific adjustment factors for other pure breeds of swine.

Improvement through selection of the most economically important traits including NBA, NO21, and LWT21 would seem possible based on the results of this study. Improvement in SURV21 may be difficult due to the

small amount of additive variation available. Farrowing interval appears to be substantially influenced by environmental effects and improvement in this trait via selection is likely to be difficult.

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Appendix

Table 7. Reproductive trait means and estimated mean differences (± SE[1]) between porcine stress syndrome normal and carrier dams.

Trait	Overall Mean	Genotype Contrast NN-Nn	±S.E.
Adjusted number born alive (Brubaker et al., 1994)	10.82	0.02	0.14
Adjusted number born alive (NSIF, 1987)	11.12	0.04	0.14
Adjusted number born alive (adjusted with own data)	10.47	0.04	0.14
Adjusted number at 21 d (Brubaker et al., 1994)	12.17	-0.03	0.05
Adjusted number at 21 d (adjusted with own data)	9.51	-0.10*	0.05
Adjusted 21-d litter weight (Brubaker et al., 1994)	68.71	0.96	1.06
Adjusted 21-d litter weight (NSIF, 1987)	64.31	1.67	1.06
Adjusted 21-d litter weight (adjusted with own data)	61.65	1.17	1.13
Survival rate to 21 d	95.48	-0.0004	0.004
Farrowing interval	172.00	0.65	1.67

[1] Standard error of the difference between the two genotype means.

Table 8. Observed and (expected) number of dams of each porcine stress syndrome genotype by herd.

Herd	Genotypes						Total	χ^2
	NN	χ^2	Nn	χ^2	nn	χ^2		
1	28 (29)	0.034	8 (7)	0.143	0 (0)	0.000	36	0.177
2	28 (29)	0.034	14 (12)	0.333	0 (1)	1.000	42	1.367
3	135 (140)	0.179	57 (49)	1.306	1 (4)	2.250	193	3.725
4	173 (178)	0.140	62 (53)	1.528	0 (4)	4.000	235	5.668
5	60 (61)	0.016	12 (11)	0.091	0 (0)	0.000	72	0.017
6	52 (53)	0.019	9 (8)	0.125	0 (0)	0.000	61	0.144
7	28 (29)	0.034	10 (9)	0.111	0 (1)	1.000	38	1.145
8	22 (25)	0.360	25 (20)	1.250	2 (4)	1.000	49	2.610
9	97 (98)	0.010	21 (19)	0.211	0 (1)	1.000	118	1.221
Total	623 (630)	0.826	218 (191)	5.098	3(14)	10.250	844	16.174

Table 9. Distribution of sow records by parity and herd.

Parity	1	2	3	4	5	6	7	8	9	Total
1	36	41	192	224	31	58	38	38	109	767
2	32	40	138	220	58	50	22	31	56	647
3	31	37	121	183	28	38	11	26	40	515
4	27	32	96	140	0	22	5	20	30	372
5	24	28	71	108	0	12	1	14	19	277
6	15	23	46	80	0	10	0	10	15	199
7	8	19	27	60	0	5	0	2	6	127
8	4	10	19	40	0	3	0	1	3	80
9	2	8	13	25	0	2	0	1	2	53
10	1	6	2	21	0	1	0	1	0	32
11	0	1	4	9	0	0	0	0	0	14
12	0	1	0	6	0	0	0	0	0	7
13	0	1	0	6	0	0	0	0	0	7
14	0	0	0	1	0	0	0	0	0	1
15	0	0	0	1	0	0	0	0	0	1
16	0	0	0	1	0	0	0	0	0	1
Total	180	247	729	1125	117	201	77	144	280	3100

Table 10. Distribution of records from dams having two or more consecutive parities.

Parity	1	2	3	4	5	6	7	8	9	Total
1	32	39	138	208	21	46	22	28	44	578
2	31	37	120	176	19	35	11	22	38	489
3	27	31	92	137	0	22	5	19	29	362
4	24	28	69	106	0	12	1	14	19	273
5	15	23	43	77	0	10	0	8	15	191
6	8	19	26	56	0	4	0	2	6	121
7	4	12	17	39	0	3	0	1	3	79
8	2	6	12	25	0	2	0	1	2	50
9	1	6	2	20	0	1	0	1	0	31
10	0	1	2	9	0	0	0	0	0	12
11	0	1	0	4	0	0	0	0	0	5
12	0	1	0	6	0	0	0	0	0	7
13	0	0	0	1	0	0	0	0	0	1
14	0	0	0	1	0	0	0	0	0	1
15	0	0	0	1	0	0	0	0	0	1
Total	144	204	521	866	40	135	39	96	156	2201

Table 11. Landrace breed-specific parity adjustment for number of pigs born alive, number of pigs alive at 21 days, and 21-day litter weight (kg) from Brubaker et al. (1994).

Parity	Number born alive adjustment, pigs	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1	0.57	0.10	4.57
2	0.29	0.00	0.00
3	0.00	0.08	0.08
4	0.00	0.20	0.81
5	0.00	0.27	1.51
6	0.32	0.40	3.19
7	0.64	0.45	3.91
8	0.84	0.58	5.83
≥9	2.09	0.78	8.74

Table 12. Landrace breed-specific number after transfer adjustment for number of pigs alive at 21 days and 21-day litter weight (kg) from Brubaker et al. (1994).

Number after transfer	Number at 21 days adjustment, pigs	21-day litter weight adjustment, kg
1-2	10.06	51.65
3	8.65	40.10
4	7.61	32.67
5	6.91	29.18
6	5.94	24.54
7	5.02	18.57
8	4.11	14.12
9	3.19	9.36
10	2.38	0.00
11	1.66	0.00
12	0.98	0.00
≥13	0.00	0.00

$$\text{Adjusted 21-d litter weight} = \text{Weight when weighed} * \left[2.9619 - \left(0.14367 * \text{Age at weighing}\right) + \left[0.00239 * \left(\text{Age at weighing}\right)^2\right]\right]$$

Equation 1. Landrace breed-specific age adjustment formula for those litters not weighed at 21 days of age from Brubaker et al. (1994).

$$\text{Adjusted 21-d litter weight} = \text{Weight when weighed} * \left[2.218 - \left(0.0811 * \text{Age at weighing}\right) + \left(0.0011 * \left(\text{Age at weighing}\right)^2\right)\right]$$

Equation 2. Age adjustment formula for those litters not weighed at 21 days of age (NSIF, 1987).

Table 13. Parity adjustment for number born alive and 21-day litter weight (kg) (NSIF, 1987).

Parity	Number born alive adjustment, pigs	21-day litter weight adjustment, kg
1	1.5	2.95
2	0.9	0.00
3	0.3	0.00
4	0.0	0.68
5	0.0	2.04
6	0.0	2.04
7	0.0	2.04
8-10	0.4	3.86
>10	1.6	5.44

Table 14. Number of pigs after transfer adjustment for 21-day litter weight (NSIF, 1987).

Number after Transfer	Adjustment, kg
3	29.48
4	22.68
5	16.78
6	11.79
7	7.71
8	4.54
9	1.81
10	0.00

GENERAL SUMMARY

One of two investigations revealed maternal performance differences to exist between females with different PSS genotypes. The first study involved a composite line of NN, Nn, and nn dams. It was discovered that nn sows have poorer maternal productivity compared to NN and Nn sows. The previously reported increased development costs of nn females and their poor maternal performance prevent their use in commercial swine breeding programs.

The more difficult question facing many commercial producers is whether to keep Nn females in the breeding herd. In the study of the composite line, Nn dams farrowed more pigs per litter and litters were heavier at birth when compared to those from NN dams. Litter size at 21 d (NO21) of NN dams was similar to that of Nn dams; however, NN dams had heavier ($P<0.05$) litters at 21 d than Nn dams.

The second study involved only NN and Nn Landrace dams. No significant differences between NN and Nn dams were observed for any of the maternal traits measured.

To economically justify the retention of Nn females in the breeding herd, a substantial advantage in maternal performance above that of NN females would need to exist. This is because losses are likely to result from a higher death rate and from poor meat quality of offspring produced, when these females are mated to Nn or nn males. This research combined with results of previous work on pork quality indicate that the negative effects of the PSS gene would prevent its use in commercial swine breeding programs.

Genetic parameters were estimated from Landrace females of known PSS genotype. Heritabilities for NBA, NO21, LWT21, and SURV21 were similar to those previously reported. Genetic variation appears to be of sufficient magnitude and heritability large enough for selection response to improve the economically important maternal traits of in NBA, NO21, and LWT21.

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