

Porcine Stress Syndrome

and Its Effects on Maternal, Feedlot
and Carcass Quantitative
and Qualitative Traits



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Porcine stress syndrome (PSS) is an inherited neuromuscular disorder in pigs (6). The PSS condition is controlled by a defective gene which results in three possible genotypes (normal, carrier and positive). The PSS condition was first described by Topel et al. (38), who noted physically stressed, susceptible pigs would collapse in a shock-like state and die (Figure 1). Much attention has been given to how the PSS gene affects the 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 of terminal sires with one or two copies of the PSS gene by market hog producers, because of its perceived advantage in producing lean, heavy-muscled hogs. A large proportion of the homozygous recessive (nn) PSS-positive animals, and heterozygous (Nn) animals carrying one copy of the PSS gene, produce carcasses with inferior muscle quality (10, 32). Debates continue in the popular press concerning the use of the PSS gene, particularly in terminal sire lines.

Molecular biology advancements have resulted in the development of a simple and relatively inexpensive procedure to determine the PSS genotype of animals, with accuracies approaching 100 percent (13). 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.

Triggering Mechanisms and Symptoms

Many physical stressors 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 (29). It has also been noted that volatile anesthetics such as halothane can bring about the onset of PSS (7, 41). Because PSS can

be triggered by halothane, the gene responsible for the syndrome is often referred to as the “halothane gene.” (Figure 2) 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, collapse, muscle rigidity and eventual death (8). Once PSS is triggered, pigs exhibit



Figure 1. Rapid death of PSS positive pigs can occur when these animals are stressed.



Figure 2. The anesthetic halothane can trigger the onset of PSS symptoms.

symptoms quickly. One of only a few of the known remedies for PSS is dantrolene sodium administered intravenously (16, 22, 23, 27). Dantrolene sodium is a muscle relaxant which affects muscle cells (22), but has no effect on cardiac or smooth muscle cells (16). Symptoms of PSS are quickly alleviated after prompt administration of dantrolene sodium (16).

Mode of Inheritance

It is clear that there is a single gene responsible for the PSS condition and that it is inherited in recessive manner. Additionally, the effects of the PSS-nn genotype are not exactly the same in every pig (incomplete penetrance), as first proposed by Christian (6) and later confirmed in several other studies (23, 25, 26, 30, 34). Molecular studies have also confirmed this mode of inheritance (13).

There is speculation that a single mutation occurring in a single animal was the progenitor of the PSS condition (13, 35). O'Brien (35) suggests that this mutation occurred in Germany in the early 20th century, as 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 Belgium 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 (7). 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 (18, 20, 40). This linkage group consists of several genetic marker loci for the HAL gene that were



Figure 3. PSS nn characteristically have "basketball" shaped hams. Frequently, the seams separating muscle groups can be noted, as seen on the hams of the Pietrain pigs in this photo.

| Breed | Frequency |
|-----------------|-----------|
| Berkshire | 0.14 |
| Chester White | 0.00 |
| Duroc | 0.05 |
| Hampshire | 0.08 |
| Landrace | 0.07 |
| Poland China | 0.43 |
| Spotted | 0.09 |
| Yorkshire | 0.07 |
| Overall Average | 0.07 |

used to predict PSS susceptibility, prior to the development of the DNA test. Assignment of HAL and its linkage group was made to chromosome 6 in the pig (11, 13, 28).

The mutation responsible for PSS is also known as HAL-1843, after the discovery of the defect's location within the genome of the pig (13). Genotypic designation for stress-resistant, stress-carrier and stress-positive are NN, Nn and nn, respectively. 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, (35).

Frequency and Gene Frequency

The frequency of the nn genotype varies according to breed and country of origin. In the purebred progeny test conducted for the National Barrow Show™ from 1991 to 1993, Goodwin (14) found an overall gene frequency of 0.07 across eight pure breeds. Estimates within breeds ranged from 0.00 for the Chester White breed to 0.43 for the Poland China breed (Table 1) (14). O'Brien (35) published PSS gene frequencies by breed, and by country of origin within breed. The Pietrain breed had the highest PSS gene frequency (0.50) while the Chester White breed had the lowest frequency (0.00) (35).

Physiological Basis

Early research noted that PSS was associated with production of pale, soft and exudative (PSE) meat (38). It was also noted that energy stored in the muscles was rapidly depleted after slaughter in carcasses exhibiting PSE. This results in rapid pH decline in carcass muscles and the PSE condition (38).

Environmental stress results in increased muscle energy utilization and muscular contractions which lead to increased production of lactic acid, carbon dioxide and heat within the muscle (15, 43). Muscle cells in animals having the PSS gene are unable to properly regulate calcium in-flow and out-flow (23). Properties of the

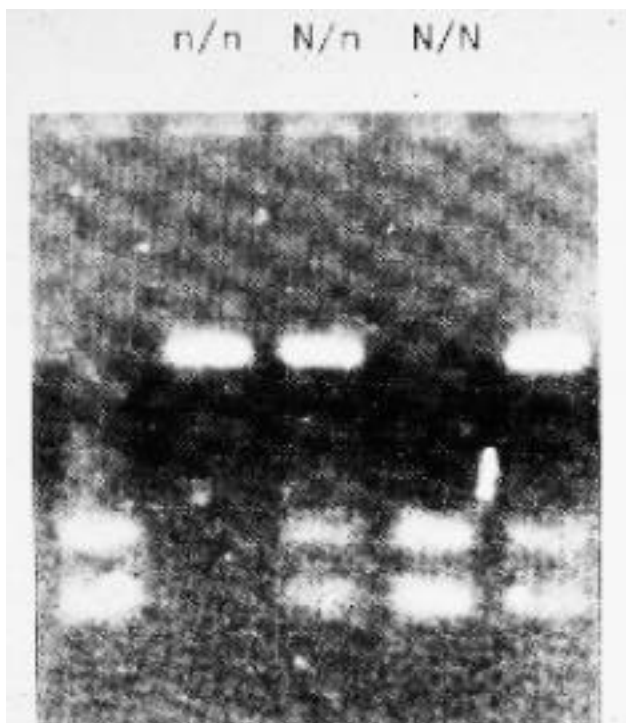


Figure 4. Characteristic banding pattern of DNA from PSS NN, Nn and nn pigs.

heterozygous animal (Nn) were found to be intermediate to those of normal and PSS susceptible animals, suggesting that both normal and abnormal gene coding for the regulation of muscle cell calcium levels exist in these animals (23).

Detection of Porcine Stress Syndrome

Methods of detecting PSS-susceptible animals have evolved as new technologies have become available. The latest tool that swine producers can utilize to determine PSS status of their breeding herd is a DNA molecular

test. Fujii et al. (13) 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 percent.

Louis et al. (24) and O'Brien and MacLennan and Phillips (27) provide a general outline of the steps used 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 or on specialized absorbent cards. Producers should contact the laboratory where they plan to submit their samples to determine the appropriate sample collection method. Use of a new needle and syringe or absorbent card for every individual is required to prevent cross contamination between pigs and inaccurate genotype determination. The sample(s) are sent to a laboratory with the ability to conduct the test and then laboratory personnel isolate DNA from the blood cells. The DNA is amplified (thousands of copies made) by polymerase chain reaction technology (PCR). The DNA is then digested with restriction enzymes. Usually, one enzyme recognizes the mutant PSS gene, while another recognizes the normal PSS gene. After digestion of the DNA occurs, the sample is placed in a gel material and exposed to an electrical current. Following this exposure, the DNA of PSS-NN, Nn and nn produce characteristic banding patterns. The distinct banding patterns allow the laboratory to determine the PSS genotype of the animal (figure 4). If procedures are followed properly and no contamination occurs at any step of the process, the accuracy of the test approaches 100 percent. The molecular test has been conducted on DNA isolated from muscle tissue, hair, adipose tissue samples and even smaller volumes (drops) of blood.

The molecular test (13) has been patented by the University of Toronto. 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 (Table 2). Swine

| Table 2. Laboratories located in the U.S. that conduct the DNA test for porcine stress syndrome ¹ . | | | |
|--|--|--------------------------------|-------------------|
| Laboratory | Address | Phone Number | Contact Person |
| Marshfield Clinic | 1000 North Oak Avenue Marshfield, WI 54449 | 1-800-222-5835 715-389-3743 | Robert A. Carlson |
| PE AgGen | 1756 Picasso Avenue Davis, CA 95616-0549 | 1-800-995-2473 | Michael Miille |
| Prairie State Semen | 968 County Road 1000 N. Champaign, IL 61821 | 1-800-282-0428 | Jon Fisher |

¹ The inclusion of a laboratory in this list in no way constitutes an endorsement of the laboratory. This listing of laboratories having the ability to conduct the DNA test for PSS may not include every laboratory with this capability. Not listing laboratories with the ability to conduct the DNA test for PSS is an inadvertent error.



Figure 5. Studies have yielded different results concerning the effects of PSS on maternal performance.

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 desired. Cost of the test ranges from \$20-35 for each sample tested. Thus, breeders can accurately determine the genotype of all breeding herd animals, and manage the PSS gene frequency as they desire. Laboratories which conduct the DNA test for PSS are listed in Table 2.

Effects on Maternal Performance

Stalder (37) investigated the effects of PSS on maternal performance in a stress-susceptible line of pigs. Porcine stress syndrome Nn females farrowed more pigs at

birth than NN females ($P < 0.05$) and nn females ($P < 0.10$) (Table 3). Additionally, Nn females farrowed heavier litters ($P < 0.05$) compared to NN sows, and heavier litters than did nn ($P < 0.10$) females (Table 3).

After making parity adjustments, Nn females farrowed a larger ($P < 0.05$) number of live pigs than either NN or nn females (Table 3). Carrier females produced ($P < 0.05$) larger total birth weight of pigs born alive than did nn females and tended ($P < 0.10$) to have larger total birth weight of pigs born alive when compared to NN females. The proportion of pigs born alive that survived from birth to transfer was not different between females of differing PSS genotypes.

Normal females had more number of pigs at weaning (21 days) ($P < 0.05$) than nn females (Table 4) after adjustments were made for parity and age of the pigs at weaning. But, there was no difference between NN and Nn dams for number of pigs at 21 days. Normal females produced heavier 21-day litter weights compared with nn ($P < 0.01$) and Nn ($P < 0.05$) females. Heavier 21-day litter weight ($P < .01$) was found for Nn females when compared to 21-day litter weights from nn females (Table 4). The proportion of pigs surviving from transfer to 21 days favored ($P < 0.01$) NN sows by 13.1 percent and 9.3 percent when compared with nn and Nn sows, respectively (Table 4).

Stalder (37) also compared maternal performance of Landrace PSS-NN and -Nn females. There were no significant differences ($P > 0.05$) between NN and Nn dams for any of the traits analyzed (Table 5). Normal and

Table 3. Birth trait means and best linear unbiased estimates of mean differences (\pm SE^a) between differing porcine stress syndrome genotypes (Stalder, 1995).

| Trait | Overall Mean | Genotype Contrast | | | | | |
|---|--------------|--------------------|------------|-------------------|------------|-------|------------|
| | | NN-Nn | SE | Nn-nn | SE | NN-nn | SE |
| Number born | 10.16 | -0.94* | \pm 0.39 | 0.61 [†] | \pm 0.32 | -0.33 | \pm 0.44 |
| Birth weight, lb. | 29.94 | -2.36* | \pm 1.08 | 1.63 [†] | \pm 0.86 | -0.75 | \pm 1.15 |
| Adjusted number born alive ^b | 9.98 | -0.91* | \pm 0.39 | 0.69* | \pm 0.32 | -0.22 | \pm 0.43 |
| Born alive litter birth weight, lb. | 28.77 | -2.00 [†] | \pm 1.04 | 1.70* | \pm 0.84 | -0.31 | \pm 1.08 |
| Survival rate to transfer, % | 90.48 | -6.29 | \pm 5.30 | 2.51 | \pm 4.01 | -3.79 | \pm 5.88 |

^a Standard error of the difference between the two genotype means involved in contrast.

^b Adjusted using combined breed factors according to Brubaker et al. (1994).

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

[†] Indicates differences are approaching significance ($P < 0.10$).

Table 4. Means and best linear unbiased estimates of mean differences (\pm SEa) for 21- and 42-day reproductive traits between sows of different porcine stress syndrome genotypes (Stalder, 1995).

| Trait | Overall Mean | Genotype Contrast | | | | | |
|---|--------------|-------------------|------------|--------|------------|---------|------------|
| | | NN-Nn | SE | Nn-nn | SE | NN-nn | SE |
| Adjusted number at 21 days | 10.41 | 0.28 | \pm 0.21 | 0.21 | \pm 0.17 | 0.49* | \pm 0.24 |
| Adjusted 21-day litter weight, lb. ^b | 111.66 | 6.31* | \pm 3.22 | 7.36** | \pm 2.62 | 13.67** | \pm 3.66 |
| Survival rate to 21 days, % | 88.06 | 9.33* | \pm 3.71 | 3.75 | \pm 0.15 | 13.07** | \pm 4.42 |
| Number at 42 days | 7.35 | 0.24 | \pm 0.23 | 0.33 | \pm 0.20 | 0.57* | \pm 0.28 |
| Adjusted 42-day litter weight, lb. | 179.48 | 6.75 | \pm 6.72 | 11.38* | \pm 5.49 | 18.12* | \pm 7.78 |
| Survival rate to 42 days, % | 82.39 | 3.21 | \pm 2.72 | 4.48* | \pm 2.22 | 7.69* | \pm 3.11 |

^aStandard error the difference between the two genotype means involved in contrast.

^bAdjusted using combined breed factors according to Brubaker et al. (1994).

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

**Indicates highly significant difference between genotypes ($P < 0.01$).

Nn females had nearly identical adjusted number of pigs-born-alive records. Adjusted number of pigs alive at 21 days was very similar for NN and Nn sows. Though not significantly different, NN sows produced litters that averaged .99 lb heavier at 21 days than those of Nn sows. The average percent of pigs surviving from birth to 21 days was nearly identical for NN and Nn dams. Similarly, farrowing interval was only .44 days different (not significant) for NN and Nn dams.

Previous work strongly discourages the use of nn females in most commercial breeding herds. The NN and Nn females clearly have a reproductive performance advantage compared to nn sows. Additionally, when the poor maternal performance of nn females is combined with increased gilt development costs (associated with high death loss of nn animals), their use in commercial swine breeding programs is not recommended. It is less clear whether Nn females should be retained for breeding purposes. 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. The economic advantage is required to recoup potential economic losses from increased death loss and inferior muscle quality incurred when Nn females are mated to Nn or nn terminal sires.

Table 5. Reproductive trait means and best linear unbiased estimates of mean differences (\pm SEa) between porcine stress syndrome (PSS) normal (NN) and carrier (Nn) dams (Stalder, 1995).

| Trait | Overall Mean | Genotype Contrast | |
|---|--------------|-------------------|----------|
| | | NN-Nn | \pm SE |
| Adjusted number born alive ^b | 10.82 | -0.003 | 0.14 |
| Adjusted number at 21 days | 12.17 | -0.03 | 0.05 |
| Adjusted 21-day litter weight, lb. ^b | 151.48 | 0.99 | 1.06 |
| Survival to 21 days, % | 95.48 | 0.06 | 0.44 |
| Farrowing interval, days | 172.0 | 0.45 | 1.80 |

^aStandard error of the difference between the genotype means.

^bLandrace breed-specific adjustments (Brubaker et al., 1994).

| Table 6. Feed, growth, carcass composition and qualitative carcass trait performance means (\pm SE) of porcine stress syndrome normal, carrier and positive market hogs (Christian and Rothschild, 1981). | | | |
|---|-----------------|-----------------|-----------------|
| Trait | Stress Genotype | | |
| | NN | Nn | nn |
| Feed consumption, lb./day | 6.01 \pm 0.15 | 5.72 \pm 0.13 | 5.72 \pm 0.15 |
| Feed efficiency, F/G | 3.60 \pm 0.18 | 3.50 \pm 0.08 | 3.45 \pm 0.09 |
| Average daily gain, lb./day | 1.69 \pm 0.03 | 1.69 \pm 0.02 | 1.72 \pm 0.03 |
| Average back fat, in. | 1.57 \pm 0.03 | 1.54 \pm 0.03 | 1.62 \pm 0.04 |
| 10th rib fat, in. | 1.54 \pm 0.04 | 1.45 \pm 0.04 | 1.45 \pm 0.05 |
| Loin muscle area, sq. in. | 5.53 \pm 0.15 | 5.70 \pm 0.12 | 6.20 \pm 0.18 |
| Dressing percentage | 73.6 \pm 0.30 | 74.1 \pm 0.20 | 74.7 \pm 0.30 |
| Percentage lean | 42.0 \pm 0.90 | 43.1 \pm 0.09 | 44.7 \pm 1.00 |
| Percentage fat | 40.7 \pm 1.10 | 39.5 \pm 1.10 | 39.4 \pm 1.20 |
| pH (45 min.) | 6.42 \pm 0.06 | 6.15 \pm 0.05 | 5.73 \pm 0.06 |
| Color reflectance | 22.5 \pm 0.80 | 24.6 \pm 0.70 | 29.0 \pm 0.90 |

Effects on Quantitative Performance and Carcass Traits

Christian and Rothschild (9) found similar average daily gain (ADG), feed efficiency (F/G) and feed consumption among pigs of all three PSS genotypes (Table 6). Carcasses from Nn and nn animals had less BF at the tenth rib (BF10) and larger loin muscle area (LMA). Thus, NN and nn carcasses had a higher percent of carcass lean. Dressing percent was similar for all three genotypes. Though the values reported in Table 6 for a given trait are not representative of the performance of today's market hogs, the relative performance differences between the three PSS genotypes is likely similar.

Jensen and Barton-Gade (19) 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



Figure 6. Porcine stress syndrome can have positive and negative effects, depending on the feedlot and carcass trait of interest.

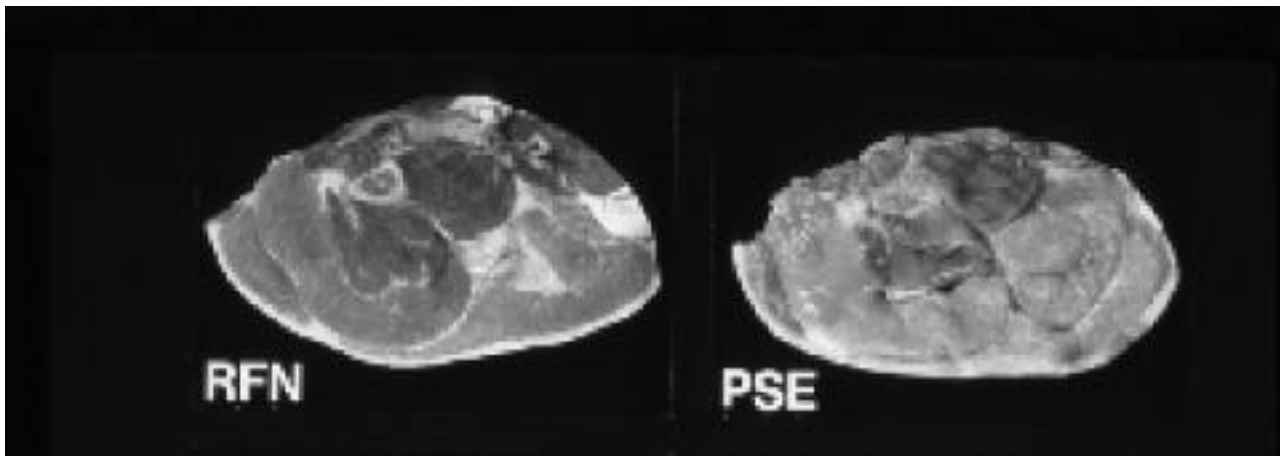


Figure 7. Desirable pork quality is shown in the ham on the left. It is reddish pink, the muscle is firm and holds its shape and has a normal amount of exudate (i.e. RFN). The ham on the right is pale and is exudative (has poor water-holding capacity). The muscles are soft and tend to droop (i.e., PSE).

Table 7.
Feed, growth, carcass quantitative, carcass qualitative trait difference between porcine stress syndrome normal (NN) and carrier (Nn) market hogs from the National Genetic Evaluation (National Pork Producers Council, 1995).

| Trait | Normal Minus Carrier Differences |
|-----------------------------|----------------------------------|
| Average daily gain, lb./day | -0.01 NS ^a |
| Days to 250 lb, days | 0.13* |
| Lean gain on test, lb./day | -0.017* |
| Tenth rib backfat, in. | -0.001NS |
| Loin muscle area, sq. in. | -0.29* |
| Carcass length, in. | 0.18* |
| Carcass yield, % | -0.376* |
| Loin color score, 1-5 | |
| Loin marbling score, 1-5 | 0.21* |
| Loin firmness score, 1-5 | 0.24 |
| Minolta reflectance, % | -1.707* |
| Ultimate pH | 0.018* |
| Loin drip loss, % | -0.48* |
| Loin intramuscular fat,% | 0.33* |

^a NS Indicates that porcine stress syndrome normal (NN) minus carrier (Nn) differences were non-significant.
^b * Indicates that porcine stress syndrome normal (NN) minus carrier (Nu) differences were significant ($P < 0.05$).

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, backfat (BF) and LMA.

Aalhus et al. (1) found a significant ($P < 0.05$) linear PSS gene 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 animals 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.

Goodwin et al. (14) estimated differences between 1298 NN and 181 Nn PSS genotypes for numerous performance and carcass traits (Table 7). 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. (21) stated that muscle color, firmness/wetness and marbling are important fresh-pork quality parameters which influence consumer acceptability. Fresh pork should be uniformly reddish pink. Soft and exudative

pork has poorer water-holding capacity and will shrink as much as 7 percent during handling and processing, making it undesirable for packers and consumers. Slight marbling or intramuscular fat is desirable for a juicy, tasteful cooked product (21). Kauffman et al. (21) estimated the incidence of poor-quality pork in the U.S. to be 26 percent. 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 (Figure 7). They are used in less-expensive, further-processed products rather than being sold as higher-priced, fresh products. These costs associated with discounted pork are estimated to be \$30 million annually (39).

PSE pork is primarily due to rapid pH decline in the muscles of pigs following stunning (17), which is caused by genetic and environmental factors. Eikelenboom (12) 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 PSE pork. Rapid pH decline during the first 45 minutes post-mortem resulting from excessive muscle energy depletion and buildup of lactic acid in the muscle are the major factors causing PSE and DFD pork (2, 3, 31).

Christian et al. (10) used subjective and objective measures of muscle quality to evaluate PSS genotype differences. Significant ($P < 0.01$) linear effects, always favoring NN animals with Nn animals being intermediate,

were observed for visual color, marbling and firmness, instrumental color values, 24 hour drip loss and pH values.

One of the more comprehensive evaluations of pork quality was conducted by the National Pork Producers Council (33). This project involved more than 3200 market hogs. Complete feedlot performance, carcass composition and quality and sensory evaluations were recorded for each animal. Normal animals were found to have superior ($P < 0.05$) instrumentally measured color scores, visual color, marbling and firmness scores when compared to Nn animals (Table 7). Additionally, NN animals produced carcasses with higher ($P < 0.05$) lipid content of the loin muscle and better ($P < 0.05$) mechanically measured tenderness (Figure 8). No differences between NN and Nn animals were found for ADG, days to 250 lbs., soundness and BF10. Differences ($P < 0.05$) favoring the NN animals were observed for carcass length and last rib backfat. Differences ($P < 0.05$) favoring Nn animals were observed for lean gain on test and LMA (Goodwin, personal communication). Previously mentioned research clearly indicates that the production of Nn pigs in a quality conscious marketplace should be questioned.

Methods to objectively measure pork quality have been evaluated (4) and their application to on-line packing plant systems examined (5). 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 may see packing companies place discounts on carcasses boasting inferior muscle quality.

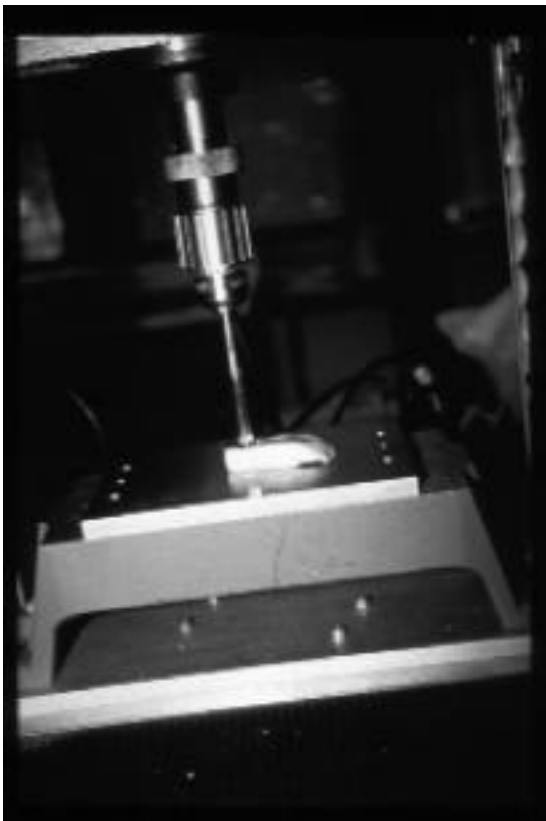


Figure 8. Tenderness of meat samples can be mechanically measured using a Universal™ testing machine.

Summary

Swine producers should use all available information when determining the appropriate use of the PSS gene in their herd. The results of research cited in this brochure indicate that the negative effects of the PSS gene on reproductive and carcass quality traits would preclude its use in commercial swine breeding programs. The advent of the molecular test for PSS allows the commercial producer to determine which breeding females have the PSS gene. Females with the PSS *n* allele can be eliminated from the breeding herd. Additionally, commercial producers should purchase or produce females free of the PSS gene and gradually replace older sows. By doing so, a producer can use PSS Nn or nn terminal sires if he/she thinks they substantially improve carcass cutability of the offspring. However, the National Pork Producers Council adopted a resolution in 1997 to rid the U.S. pork population of the PSS gene. If commercial producers choose to use PSS Nn or nn terminal sires, they should retain only PSS-NN females for breeding. As the production of high quality pork becomes increasingly important, producers can replace their PSS-Nn or nn terminal sires with terminal sires free of the PSS gene. This breeding system ensures that all offspring are free of the stress gene by simply replacing boars with the PSS gene with those who do not. Additionally, this mating system becomes more valuable when packers offer incentives or discounts based on the quality of pork produced from individual carcasses.

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