Porcine Stress Syndrome
Michigan State University
Cooperative Extension Service

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January 2000
4 pages

Extension Bulletin E-1127 • Revised January 2000

pork industry handbook

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Although pigs naturally respond to the conditions around them with varying degrees of stress, researchers in the late 1960s described a syndrome in pigs that exhibit an abnormal amount of stress under some conditions. The porcine stress syndrome (PSS) became a major concern to the pork industry in the 1960s and the 1970s, and today continues to cause significant economic loss.

When PSS is present, it is typically associated with lean, heavily muscled animals and results in sudden, unexplained death losses. Usually, the postmortem muscles of PSS pigs are pale, soft, and exudative (PSE). Animals with PSS often show signs of nervousness and may have muscle tremors indicated by a rapid quivering of the tail and back and leg muscles.

When exposed to stressful situations such as a change in surroundings, comingling with new hogs, a sudden change in weather, vaccination, castration, estrus, or mating, the pigs often respond by becoming overly excited and developing reddish blotches on their skin. They begin to experience muscle rigidity followed by rapid, labored breathing. Their body temperature also begins to rise and they show signs of heat stress even in cold weather. At this point, many producers have attempted to save them by spraying with water, but the condition progresses so rapidly that it is virtually impossible to cool the pigs fast enough. In the final stages of the syndrome, pigs experience total collapse and extreme muscle rigidity, usually followed by death. In humans, a similar condition is called malignant hyperthermia.

Death losses from PSS usually occur during the process of sorting and delivering animals for slaughter. In addition, death losses are greater in the summer months when temperatures are higher and pigs are unable to rid themselves of body heat. Research has revealed many characteristics of PSS animals and the genetic basis for the disorder. Some of these findings are summarized in this fact sheet.

The Underlying Causes of PSS

The Biology of PSS

PSS pigs cannot cope well with stressful situations. When exposed to stress, they undergo several metabolic and biological reactions, including a very rapid depletion of their muscle energy stores. As their muscle energy stores are being depleted, there is also a corresponding increase in lactic acid in both muscles and blood. Normal pigs can remove the lactic acid from muscles and blood fast enough to prevent excessive build-up, but PSS pigs have such great quantities of lactic acid produced that they cannot remove it from the muscle fast enough. Therefore, following a stressful situation, the levels of lactic acid increase in PSS pigs which increases blood acidity creating a condition known as metabolic acidosis. Accompanying the acidosis is a build-up of heat due to a wasteful process of using the muscle glycogen for energy.

The primary defect of PSS pigs is likely to be in the muscle structure itself. Certain muscle organelles lack the ability to bind calcium. Higher levels of unbound calcium trigger muscle contraction and the breakdown of energy-rich phosphates. This initiates the series of reactions outlined above that produces excessive amounts of lactic acid.

The Genetics of PSS

No breed is entirely free of the PSS problem and, likewise, no breed can be termed categorically stress-susceptible. In some European breeds, the incidence is extremely high and in others extremely low. The trait is inherited in a simple recessive manner meaning that both the sire and dam must be at least carriers of the gene mutation to produce stress-susceptible offspring. On the average, when carrier parents are mated (Nn x Nn), one of four offspring will be PSS (nn), two of four will be

carriers (Nn), and one of four will be free of the defective gene (NN). Therefore, if there is a problem in the herd, the quickest and most economical step is to replace the sire with one that is totally free of the defective gene.

Although the PSS condition is sometimes found in animals with superior muscling and leanness, these traits reflect the joint effects of many genes; consequently, it is not necessary to sacrifice desirable carcass composition for freedom from the PSS problem. Instead, one should incorporate meat-type animals into the breeding herd that have tested free of the defective gene. PSS pigs normally have a superior composition, but they have several disadvantages including smaller frame size, lower feed intake, lower daily gain, smaller litters born and reared, and, as is discussed below, inferior meat quality.

Tests for PSS

Until recently, the most accurate test for evaluating PSS status required that pigs be treated with the anesthetic gas, halothane. PSS animals respond to halothane anesthesia by showing signs of extreme muscle rigidity, usually within 3 minutes from the start of the treatment. This test provides immediate results, but the equipment involved is expensive and the operator requires training. Also, the halothane test only detects stress-susceptible pigs and cannot distinguish between normal carriers (Nn) and non-carriers (NN). Therefore, the test is an expensive and limited selection tool that cannot be used to completely eradicate the defective gene from a population.

Rapid advancement in genetic technology, along with growing knowledge of the underlying causes of PSS, has led researchers to the identification of the gene responsible for the condition. Initially, blood typing was used to predict an animal's genotype for PSS since several genes that determine blood type are on the same chromosome and are closely linked to the gene causing PSS. However, the distance on the chromosome between these genes left room for errors when using blood type to predict PSS genotype.

In 1991, the specific mutation that causes PSS was located and identified. The DNA mutation is a single-base pair change of cytosine to thymine that creates an amino acid change of arginine to cysteine. This mutation is at nucleotide 1843 in a gene that codes for a skeletal muscle receptor that is known to bind a drug called ryanodine. The receptor, called the sarcoplasmic reticulum calcium channel protein, controls movement of calcium from the sarcoplasmic reticulum into the muscle cytoplasm. The defect in the PSS pig appears to be a hypersensitive gating of this channel resulting in it being more easily opened than normal and preventing or making difficult its closure. The result is muscle contraction, hypermetabolism, and a hyperthermia characteristic of this syndrome.

Knowledge of the DNA mutation responsible for PSS has resulted in the development of a relatively simple DNA test to determine the exact PSS genotype of a pig. This non-invasive test only requires a small amount of blood or tissue (for example, a blood sample, some hair follicles or an ear notch) for extraction of DNA. The affected part of the ryanodine receptor gene is amplified from the DNA sample using the polymerase chain reaction (PCR), and the product is digested with a restriction enzyme that makes a specific cut at the point of the mutation. The digested DNA is then observed using electrophoresis to determine if the animal providing the sample has genotype nn, Nn or NN.

This diagnostic test for PSS has been patented (trademark: HAL-1843) and is available to all producers through licensed laboratories. Laboratories typically require a 5 ml blood sample or a blood spot on a special blood collection card from each pig to be tested. The cost of the test is approximately $30 per sample. The ability to determine the exact PSS genotype of a pig provides breeders with the opportunity to eradicate the defective gene from a population of pigs or regulate its use in a breeding program.

PSS and Meat Quality

Because of changes in marketing systems in the US, and developing international markets, meat quality has become an important factor in the selection of breeding stock. It is true that most PSS animals will yield PSE muscles. However, not all pigs that produce normal quality carcasses are free of PSS, and not all PSE pork originates from stress-susceptible pigs. The quality of fresh pork is the result of the genetic make-up of the animal and the conditions under which the animal is handled during the slaughter process.

It has been demonstrated that much of PSE pork is the end result of PSS, but research shows the genetic and environmental influences to be closely related. Those animals that are stress-susceptible may die enroute to market or, if they survive until slaughter, produce a high incidence of PSE muscle. In populations with a low incidence of PSS, preslaughter and slaughter conditions are relatively more influential in producing PSE pork.

High-quality, uncured pork is reddish pink in color, firm in texture, relatively free of surface juices, and (for some consumers) contains modest amounts of marbling. These characteristics result in a juicy, tender, flavorful, nutritious product when properly cooked. In addition, high-quality pork will retain most of its juices during cutting, packaging, freezing, and cooking and also during curing, smoking, and emulsifying in the making of manufactured products.

On the other hand, PSE pork is low in quality for the following reasons:

- It is soft, mushy, loose-textured, floppy, pale, and unappealing.
- The muscles become acidic, especially during early stages after slaughter, and consequently the proteins lose their ability to retain juices.
- The condition appears more frequently in the loin and outer ham muscles, giving a two-toned appearance in many pork cuts.
- Affected muscles appear to have little or no marbling.
- In the unprocessed, fresh condition, it releases juices during cutting and handling (shrinkage is sometimes greater than 7%) as well as possessing excess purge in the retail package, quickly turns gray in color and is unattractive to consumers, and thus has a shorter shelf-life than normal pork.
- When used for processing (smoked cuts, sausage products), it shrinks excessively (3% to 10% above normal for fully cooked hams), lacks uniform cured color, shows separation of individual muscles, and may be difficult to slice.
- Frozen cuts lose excessive amounts of juice upon thawing.

In some instances PSS pigs do not produce PSE muscle. Several factors may interfere with the usually close relationship. For example, the particular stage of stress response developed by the pig at the moment of slaughter will dictate the conditions

within the muscles. If an animal is stress-susceptible but survives a stress that occurs well in advance of slaughter, the muscles may be depleted of their energy reserves. In this instance, the meat may appear dark, firm, and dry (DFD) because there is very little energy supply to produce acid after death. The DFD condition is undesirable in appearance and is more subject to spoilage due to higher ultimate pH (less acidity), but it does not have the other disadvantages of PSE muscle. If preslaughter conditions are right, PSS pigs can yield normal appearing muscles. These complicating factors suggest that it is more reliable to base animal selection on direct measurements on the animals in question rather than on meat quality characteristics of their littermates' carcasses.

Because it recently has become possible to accurately distinguish non-carrier (NN) from normal carrier (Nn) pigs, new information regarding the carcass characteristics and muscle quality associated with these genotypes is available. In some genetic backgrounds, Nn pigs exhibit an advantage over NN pigs for dressing percentage and acceptable composition. This result has led some breeders to use a system in which carrier (Nn) market hogs are produced. However, some studies also have indicated a slightly greater incidence of PSE and thus a slightly poorer meat quality in carrier (Nn) than in non-carrier (NN) pigs.

Handling of Pigs to Prevent PSE Pork

Some environmental conditions may be comfortable to a normal, stress-resistant animal but stressful to the pig with PSS. Consequently, it may be impossible to handle pigs under practical conditions without imposing some stress.

Some of the undesirable meat characteristics can be minimized by observing simple management practices at marketing time. The following are suggestions for reducing losses associated with handling market hogs:

- Avoid crowding in holding pens and trucks. Make sure loading and unloading facilities are well designed to minimize excitement. Personnel that handle pigs should be trained in animal behavior.
- Eliminate the opportunity for fighting. Do not co-mingle pigs that have not been reared together. When handling pigs, treat them quietly at all times, refrain from use of an electric prod.
- Avoid extremes in temperature and other environmental conditions. Do not move pigs during the hottest part of the day.
- Use general precautions in all phases of the marketing process. Do not require pigs to walk long distances; avoid driving pigs over slippery surfaces; do not feed pigs 12 hours before marketing; and allow longer periods of adjustment to changes in conditions.
- Avoid slaughter immediately after arrival at the plant. Include a 2- to 4-hour resting period in preslaughter handling. Use sprinklers for cooling if temperatures are high. Move pigs from holding pens to the stunning location as carefully as possible to minimize crowding and exciting the pigs.

Related Publications

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Related Fact Sheets

PIH-97, Swine Genetic Abnormalities
PIH-127, Pork Quality

Acknowledgement

The authors of the previous version of this factsheet are acknowledged for much of the information included in the present revision. They include Drs. Max Judge, Gijs Eikelenboom, and Dennis Marple. The late Dr. Lauren Christian contributed heavily to this revision.

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