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EFFECT OF VITAMIN E AND PORCINE STRESS SYNDROME (PSS) GENOTYPE ON ANTIOXIDANT STATUS AND CARCASS CHARACTERISTICS OF PIGS

presented by

Gerry Ann McCully

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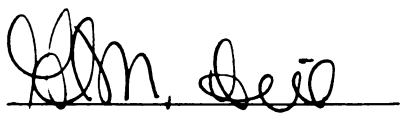

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EFFECT OF VITAMIN E AND PORCINE STRESS SYNDROME (PSS) GENOTYPE ON ANTIOXIDANT STATUS AND CARCASS CHARACTERISTICS OF PIGS

By

Gerry Ann McCully

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ABSTRACT

EFFECT OF VITAMIN E AND PORCINE STRESS SYNDROME (PSS) GENOTYPE ON ANTIOXIDANT STATUS AND CARCASS CHARACTERISTICS OF PIGS

By

Gerry Ann McCully

The effects of vitamin E (injected or fed) and porcine stress syndrome genotype on antioxidant status in young pigs and carcass characteristics in market pigs were evaluated. Vitamin E injected pigs had greater serum E than saline injected pigs for 7 d post injection. Homozygous dominant (NN) pigs had lower ($P < 0.01$) glutathione peroxidase and higher ($P < 0.05$) CuZn superoxide dismutase activities than Nn 1 d post injection. Vitamin E injection at weaning had no affect on serum E, or enzyme activities measured 28 d later. Serum ceruloplasmin activity in non-injected pigs was lower ($P < 0.05$) in nn pigs than in NN while the opposite was true in injected pigs. Carcasses of nn pigs were shorter, had less BF, larger LEA, greater kg lean, lower 45 min muscle pH, and were more pale than NN or Nn carcasses ($P < 0.05$), and nn had greater drip loss ($P < 0.05$) than NN carcasses regardless of vitamin E injection. When vitamin E was fed, muscle vitamin E was greater ($P < 0.01$). Carcasses of nn pigs were shorter, paler, had less BF, and greater drip loss ($P < 0.05$) than NN or Nn. Also, nn had lower 45 min muscle pH, larger LEA and greater kg lean ($P < 0.05$) than NN carcasses. Nn and nn pigs had altered antioxidant activity and carcass characteristics compared with NN pigs and may have different dietary requirements for nutrients associated with antioxidant capability.

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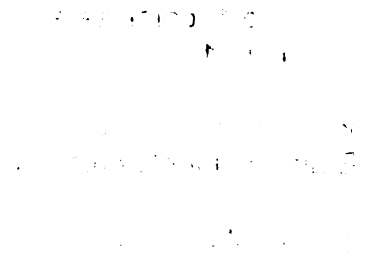


LITERATURE REVIEW

Vitamin E

Forms. The term vitamin E is used to describe all derivatives of tocopherol and tocotrienol that exhibit the biological activity of α-tocopherol. Eight naturally occurring substances have been found to have vitamin E activity: d-α-tocopherol, d-β-tocopherol, d-γ-tocopherol, d-δ-tocopherol, d-α-tocotrienol, d-β-tocotrienol, d-γ-tocotrienol and d-δ-tocotrienol. Natural forms of vitamin E are all d-stereoisomers while synthetic forms of vitamin E, typically found in commercial diets, are mixtures of stereoisomers prepared by coupling trimethlythydroquinone (TMHQ) with isophytl (Kayden and Traber, 1993).

Functions. Numerous functions have been reported for vitamin E in biological systems. It is probably best known as a non-specific anti-oxidant in lipid membranes where its activity is believed to protect membrane integrity. Vitamin E has also been reported to be involved in the synthesis of co-enzyme Q, the metabolism of vitamin C and sulfur amino acids, maintenance of structure and health of the nervous, gastro-intestinal, immune and muscular systems (Coelho, 1991). However, its role as a natural anti-oxidant is thought to be the underlying factor in most of these functions.

Vitamin E is the major lipid-soluble antioxidant in biological systems and serves as a free radical scavenger (Halliwell, 1987; Duthie et al., 1989; Dutta-Roy et al., 1994). Free radicals are molecules or molecular fragments with an unpaired electron. The presence of an unpaired electron can convey

considerable reactivity to the free radical which by hydrogen abstraction can damage a wide range of biological materials, such as DNA, nucleotide co-enzymes, proteins and lipids (Slater, 1984; DiMascio et al., 1991). Vitamin E disrupts the sequence of lipid peroxidation and prevents the formation of damaging lipid hydroperoxides.

Biological activity and biopotency. The biological activity of tocopherol has been assessed by the ability of a form of this vitamin to prevent a specific deficiency-related symptom. For example, RRR-α-tocopherol is the most effective form when activity is assessed by the rat resorption-gestation assay, but units of activity are expressed relative to that of all-rac-α-tocopheryl acetate (Chow, 2001). Therefore, the biological activity of RRR-α-tocopheryl acetate is 1.49 IU/mg compared to all-rac-tocopheryl acetate which is 1.00 IU/mg. How well a tocopherol form functions or its biopotency is related to its kinetics in a tissue. Since the activity of RRR-α-tocopherol is highest, it is believed that the phytyl tail is important for optimal biopotency. In serially sampled sows, Lauridson et al. (2002) recently reported that RRR-α-tocopherol was twice as effective in maintaining plasma α-tocopherol concentrations than synthetic all-rac-α-tocopherol.

Tissue concentrations. Tocopherols , like dietary fat, are absorbed from the small intestine in triglyceride containing chylomicrons into the lymph system to be transported to the liver. Released from the liver in lipoproteins, many tissues including the liver, muscle and adipose tissue have the ability to take-up α-tocopherol. Jensen et al. (1988) reported that skeletal muscle had higher ($P <$

0.001) vitamin E tissue concentrations after 3 wk of feeding as little as 15 mg vitamin E/kg of diet compared with pigs fed unsupplemented diets. When Cannon et al. (1996) fed diets containing 0 or 100 mg of vitamin E for 84 d, α-tocopherol concentration was increased 10-fold in the longissimus muscle. In a dietary vitamin E depletion and repletion study by Jensen et al. (1990), high levels of vitamin E (405 mg/kg diet) were fed for a 7-wk period. Following this dietary regime, a depleted vitamin E diet was fed. On d 2 of depleted diet feeding, liver vitamin E concentrations dropped by 80%. Fat and muscle vitamin E concentrations remained elevated after 7 d of feeding the depleted diet. Jensen et al. (1990) concluded that serum vitamin E and liver concentrations are short-term indicators of vitamin E status; however, adipose and skeletal muscle tissues are long-term indicators. Moreira and Mahan (2002) fed different levels of vitamin E (0, 20, 40, 60, 80, 100, 150, or 200 IU dl-α-tocopherol acetate/kg of diet) to newly weaned pigs (n = 248; age = 15 d ± 1 d; weight = 4.8 kg) for 42 days and found that serum α-tocopherol concentrations increased quadratically in the 7 d period following weaning but linearly thereafter. Serum α-tocopherol was highest for all the pigs at weaning, declining in all treatments by d 7. In a second experiment (n = 248) feeding 0, 20, 40, or 60 IU/kg diet, similar findings were reported in α-tocopherol concentrations. Additionally, tissue (liver, heart and adipose) α-tocopherol concentrations increased as dietary vitamin E increased. When 5% fat was added to the diets, liver and adipose α-tocopherol concentration increased; however, heart muscle tissue concentration was unchanged.

Effect on growth. The effect of vitamin E on growth performance was recently reported by Moreira and Mahan (2002). In two experiments (n = 496) utilizing newly weaned pigs fed various levels (up to 200 IU/kg of diet) of vitamin E (all-rac-tocopherol) they found no advantages in pigs gains, feed intake, or gain:feed for any period during the forty-two days following weaning. Chung et al. (1992) and Cannon et al. (1996) also reported that increased dietary vitamin E did not improve growth performance. Most researchers agree that supplemental vitamin E above typical NRC guidelines does not improve the overall growth rate of the pig at any stage.

Effect on meat quality. Numerous research trials have been conducted utilizing various levels of vitamin E supplementation as it pertains to meat quality, although not all agree on the effect regarding pork meat quality. Some research has shown that increased levels of dietary vitamin E (200 mg/kg) in the form of acetate improves color (Asghar et al., 1991; Monahan et al., 1992), decreases drip loss and enhances shelf life stability (Asghar et al., 1991) of pork chops. Cheah et al. (1995) reported that dietary supplementation of 500 mg vitamin E/kg of diet potentially reduced drip loss in longissimus thoracis (LT) muscle by 45% in porcine stress syndrome (PSS) carriers (Nn) and 54% in stress-free pigs (NN). They also concluded that supplementation of 1,000 mg vitamin E/kg diet for 46 d could actually prevent the formation of pale, soft, and exudative (PSE) carcasses when analyzing pre-slaughter biopsy and post-slaughter evaluation of the LT muscle. Contradicting these findings, Jensen et al. (1997) fed diets supplemented with α-tocopherol (100, 200 or 700 mg/kg feed) to pigs from

weaning to slaughter and concluded that drip loss and color stability were not affected by vitamin E treatment. Color measurement, pH, drip loss and growth were also reported by Cannon et al. (1996) to be unaltered by vitamin E supplementation in pigs fed 100 mg/kg diet for 84 d prior to slaughter compared with those fed a control diet containing no supplemental vitamin E. Corino et al. (1999) fed varying levels of vitamin E (25, 50, 100, 200 and 300 mg/kg tocopherol acetate of diet) for the last 60 d prior to slaughter and found vitamin E treatment did not alter drip loss. Past research does not fully agree on the overall effect of dietary vitamin E supplemented at varying levels and how it affects meat quality in swine.

Antioxidant System

Vitamin E is not the only antioxidant in biological systems, but it is an integral part of a network of antioxidants (Lauridsen et al., 1999a), including: ceruloplasmin (Cp; EC 1.12.3.1), catalase (CAT; EC 1.11.1.6), copper/zinc superoxide dismutase (Cu/Zn SOD; EC 1.15.1.1), and glutathione peroxidase (GPX1; EC 1.11.1.9). DiMascio et al. (1991) reported oxidative damage is controlled by a system of enzymatic and nonenzymatic antioxidants that eliminate pro-oxidants and scavenge damaging free radicals. Tocopherols are the most abundant and efficient of the antioxidants.

The protein Cp is made in the liver, binds 90 to 95% of the circulating Cu in the body, and has non-specific antioxidant activity and acts as an acute phase reactant protein. The enzyme CAT helps to rid the body of damaging peroxides. Hill et al. (1999) reported that young pigs, 3 to 14 d of age, did not have a change

in Cp activity when injected with Fe (200 mg/mL Fe) and vitamin E (300 IU) at 6 d of age, but Cp activity increased with age from 3 to 7 d of age. Milne and Matrone (1970) also reported an increase in Cp activity of the young pig through two weeks of age.

Copper/Zinc superoxide dismutase is a copper/zinc dependent enzyme of the oxidoreductase class that catalyzes the reduction of superoxide anions to hydrogen peroxide, protecting cells against dangerous levels of superoxide. Glutathione peroxidase (GPX1) is a selenium dependent enzyme of the ligase class that catalyzes the ATP dependent-formation of glutathione from glycine and γ-glutamylcysteine. In a study by Lauridsen et al. (1999b) pigs (BW = 25 kg) were fed three levels of vitamin E: 1) basal diet (9 mg/kg vitamin E); 2) basal diet + 100 mg vitamin E; 3) basal diet + 200 mg vitamin E. They found that hepatic SOD and GPX1 activities were not affected by dietary vitamin E regime. In a second study, feeding the same diets, Lauridsen et al. (1999a) found no effect of diet on Cu/Zn SOD and GPX1 in the M. Longissimus dorsi (LD) and M. Psoas major (PM) muscles. Duthie and Arthur (1989) reported that pigs consuming a diet containing 10 IU vitamin E/kg had increased GPX1 activity in the longissimus dorsi muscle of pigs homozygous for the porcine stress syndrome (PSS) gene (nn) compared with the normal pig of similar genetics.

Malignant Hyperthermia Gene

Pigs that carry porcine stress syndrome (PSS), also known as the halothane gene, are commonly referred to as being "stress-susceptible". When exposed to relatively minor stresses, such as halothane, transportation, weaning

and co-mingling, mating and parturition, PSS homozygous recessive (nn) pigs may develop malignant hyperthermia; a rapid increase in body temperature, muscle rigidity, hyperventilation, rapid heart rate, and potentially death (Williams, 1985; Geers et al., 1992).

Relationship with vitamin E. According to research at the Rowett Institute (Duthie and Arthur, 1989), pigs that are deficient in vitamin E display similar antioxidant abnormalities to pigs with the genetic mutation known as PSS. As with vitamin E deficient pigs, PSS pigs have an increased susceptibility to free radical mediated peroxidation compared with pigs free of the stress gene (Duthie and Arthur, 1989), or "normal" (NN) pigs, and increased vitamin E supplementation may reduce the negative effects of PSS at the biochemical level.

Current HAL-1843 DNA technology (Fujii et al., 1991) makes it possible to determine the presence or absence of the PSS gene; hence, the ability to identify the heterozygote (Nn). Prior stress testing utilizing halothane was limited to only identifying the homozygous recessive (nn) individual. Therefore, past research of the heterozygote (Nn) is somewhat limited.

Genotype Characteristics

Performance. Christian and Rothchild (1981) found no differences in ADG due to presence of the PSS gene. Leach et al. (1996) studied PSS carriers (Nn) and negative (NN) pigs produced within the same litter and concluded that daily gains were similar. They also reported that Nn pigs had higher feed:gain ratio than NN pigs. Byrem et al. (1999) found improved gain in nn pigs in the finishing

stage.

Meat quality and quantity. The inheritance of PSS is thought to be through a recessive gene although not all researchers agree. Some studies indicate complete recessiveness of the PSS gene (Jensen et al., 1981; Webb, 1980) while other research has shown some dominance effects of the gene on meat quality (Sather et al., 1991b; Simpson and Webb, 1989). Cannon et al. (1995) concluded the mode of inheritance seems to be an autosomal recessive trait that demonstrates incomplete penetrance. Homozygous recessive (nn) pigs for the trait demonstrate the characteristic limb rigidity when exposed to halothane; however, the heterozygotes (Nn) do not demonstrate the limb rigidity, but produce inferior meat quality. Murray et al. (1989) reported that the heterozygote (Nn) is intermediate between both homozygotes. In terms of meat quality, Lundstrom et al. (1989) concluded that the heterozygote (Nn) is intermediate between the homozygote (nn and NN) pigs. It has also been suggested that the dominance of the PSS gene as it affects the quality of pork may rise with increased slaughter weight (Sather et al., 1991a). The interaction between the pre-slaughter handling and the genotype could be the cause of contradicting results (Oliver et al., 1993).

Carriers of PSS, both homozygous recessive (nn) and heterozygous (Nn), are leaner, heavier muscled and produce carcasses with a higher percentage of lean muscle (Oliver et al., 1993; McCully et al., 1996; Weavers et al., 1996), unfortunately their meat is more likely to be pale, soft and exudative (PSE; Casteels et al., 1995). Van Laack et al. (1993) noted that homozygous recessive

(nn) pigs produced carcasses with larger LEA and less BF, but meat quality was often sacrificed since their carcasses were more pale and exudative. On the other hand, pigs that were heterozygous (Nn) for the stress gene produced carcasses with larger LEA and a greater amount of lean product than pigs that were free of the stress gene (NN) although muscle color and drip loss did not differ (van Laack et al., 1993). Stalder et al. (1998) reported heterozygotes were intermediate for carcass traits. Leach et al. (1996) studied Nn and NN within the same litter and concluded Nn pigs had an advantage in feed efficiency and carcass yield but had a higher incidence of PSE.

According to Klont et al. (1993) PSE meat is associated with rapid decline in pH in the muscle during the first hour following slaughter. Sather et al. (1991a) reported lower pH at 45 min post mortem for nn pigs than for NN or Nn and Nn pigs were lower than NN. Greater incidence of pale color by the L* score in pigs of nn genotype was also reported by Pommier and Houde (1993) and Sather et al. (1991a) with NN loins being darker in color. Van Laack et al. (1993) stated that nn pigs (n = 4) demonstrated lower pH at 45 min post mortem and had higher L* values at 48 h than NN (n = 4) or Nn (n = 4) pigs. Klont et al. (1993), utilizing pre-slaughter anesthesia, found that differences in color and drip loss are not explained by the stress state of pigs or differences in pH and 45 min carcass temperature. Sather et al. (1991a) reported with heavier carcasses, drip loss from nn or Nn was not different. However, both differed from NN heavy carcasses.

Many factors contribute to the onset of PSE, including genetics, pre-

slaughter stress, early post-mortem pH drop, carcass temperature, chilling procedures, feed restriction during transport and additional slaughter techniques (Murray et al., 1989; Klont et al., 1993; Buckley et al., 1995). Murray et al. (1989) reported that the mixing of unfamiliar animals, physical abuse and environmental changes may increase PSE while restriction of feed during transport prior to slaughter decreases PSE. Stalder et al. (1998) wanted to determine if preslaughter handling, mixing or resting, influence the quality of muscle in pigs of differing genotypes (29 Nn; 14 NN; 9 nn). They found that 16 h of resting prior to slaughter improved the color and water holding capacity of heterozygotes (Nn). A 1995 report by the National Genetic Evaluation Board (Anonymous, 1996) suggested that 35% of carrier pigs raised under good handling and management conditions, had inferior pork quality, while only 20% of pigs free of the stress gene had inferior pork quality. Thus, the majority of PSE pork in today's marketplace is thought to be caused by the management of certain genotypes especially those carrying PSS. Because much of the reduction in pork quality is oxidative in nature, antioxidants appear to have the greatest impact in maintaining pork quality. Although many factors contribute to PSE in today's marketplace, researchers agree that PSS genetics play a major role.

Pale, soft, and exudative pork is detrimental to all aspects of the swine industry, including producers, packers and ultimately the consumer (Anonymous, 1995). Millions of dollars are estimated to be lost annually due to PSE (Kauffman and Weiler, 1992; Marbery, 1992). Lost revenue from purge and undesireable meat case display is difficult to quantify; however, the U. S. Livestock

Conservation Institute estimates the United States loses $ 32 million annually due to PSE-related losses.

It is agreed upon by past research that homozygous recessive (nn) pigs for the stress gene have increased lean, along with increased incidence of PSE; however, the overall meat quantity and quality of the heterozygote (Nn) is still in question. Also, the benefit of vitamin E on the reduction of PSE and improvement of overall meat quality in PSS pigs (nn and Nn) is not clear. Perhaps the utilization of today's molecular laboratory techniques can clarify the quantity of vitamin E needed for acceptable pork quality from the various genotypes.

INTRODUCTION

Pigs that carry an inherited genetic mutation known as porcine stress syndrome (PSS) are referred to as being stress-susceptible. Carriers of PSS, both homozygous recessive (nn) and heterozygotes (Nn), are leaner, heavier muscled and produce carcasses with a higher percent lean muscle (Oliver et al., 1993; McCully et al., 1996; Weavers et al., 1996), but their meat is more likely to be pale, soft and exudative (PSE). Leanness advantages are potentially lost due to lower yields from reduced water holding capacity in the muscle (Pommier and Houde, 1993). According to a survey of packing plants, Kauffman et al. (1992) reported 26% of pig carcasses in the United States were considered undesirable. Pale, soft and exudative pork composed up to 16% of these undesirable carcasses.

Stress-susceptible pigs have altered antioxidant activity under sustained oxidative stress from situations such as weaning, transport, mating and parturition (Duthie et al., 1989). Signs of free radical mediated damage to cell membranes, similar to normal (NN) pigs that are deficient in vitamin E, are found in stress-susceptible pigs (Duthie and Arthur, 1989). Vitamin E, the major lipid soluble antioxidant in biological systems, serves as a free radical scavenger.

Vitamin E does not function as the only antioxidant, but it is an integral part of a network of antioxidants (Lauridsen et al., 1999b), including: Cu/Zn superoxide dismutase (Cu/Zn SOD; EC 1.15.1.1), glutathione peroxidase (GPX1; EC 1.11.1.9), catalase (CAT; EC 1.11.1.6) and ceruloplasmin (Cp; EC 1.12.3.1).

Therefore, our objective in these studies was to determine the impact of vitamin E, either fed or injected, antioxidant status, growth rate, and carcass characteristics of pigs representing all stress genotypes (NN, Nn, nn) at various stages of growth.

MATERIALS AND METHODS

The All-University Committee on Animal Care and Use at Michigan State University approved (AUF number: 02/94-037-03) animal usage in all experiments.

Experiments

Experiment 1. In Exp. 1, our goal was to determine if carriers (Nn) of the PSS gene, given an exogenous antioxidant, vitamin E, demonstrate a metabolic shift in antioxidant parameters when compared to non-carriers (NN). Pigs (n = 39) were selected from the offspring of crossbred boars (Hampshire X Pietrain) and crossbred sows (Duroc X Pietrain). Genotype was established by DNA determination using the HAL 1843 method (University of Toronto, Innovations Foundation, Toronto, Ontario) at the Universal Gene Laboratories, Eldora, Iowa. Pigs (mean BW = 38.5 kg) were grouped by weight and genotype in 13 pens of three (10 pens of Nn; 3 pens of NN). They were housed in 1.5 m X 5 m pens in an open front building for 21 d prior to the initiation of the study for an adjustment period. A 16% crude protein, corn-soybean meal grower diet (Table 1) that met or exceeded National Research Council (NRC, 1988) nutrient recommendations and water were provided ad libitum.

Pigs within a pen were randomly injected i.m. (20 ga, 2.5 cm needle) in the cervical portion of the trapezius muscle, approximately 15 cm posterior to the ear with one of the following: (1) placebo (2 mL saline/22.7 kg BW); (2) 600 IU d-α-tocopherol (Vital E300; Schering-Plough Animal Health, Kenilworth, NJ)/22.7

kg BW; or (3) 1200 IU d-α -tocopherol/22.7 kg BW. Blood samples were collected, via the anterior vena cava, immediately prior to injection and at 1, 4 and 7 d post injection.

Experiment 2. To determine the effect of vitamin E and genotype on antioxidant activity and growth during and following the stress of weaning, 154 pigs (BW 7.84 kg, age 24 d) from crossbred boars (Hampshire X Pietrain) and crossbred sows (Duroc X Pietrain) and of known PSS (HAL 1843) genotype (37 NN, 80 Nn, and 37 nn) were randomly assigned by weight, litter and sex to receive either an i.m. injection of vitamin E (900 IU d-α-tocopherol) or no injection at weaning. Pigs were provided water and a complex phase I starter diet (Table 1), ad libitum, for 7 d post weaning followed by a phase II diet (Table 1) for d 8 to 28 post weaning. Both diets met or exceeded all dietary recommendations of NRC (1988). Blood was collected prior to injection and at 28 d post injection. Weights were recorded weekly.

Experiment 3. For determination of the effect of vitamin E and PSS genotype on carcass characteristics, 60 pigs (BW = 104.3 kg) of known genotype (16 NN, 32 Nn, and 16 nn) were randomly injected i.m. 24 h prior to slaughter with one of two treatments: (1) placebo, 2 mL saline/22.7 kg BW or (2) 600 IU d-α-tocopherol/22.7 kg BW. Prior to study, pigs were fed a corn-soybean meal finishing diet (Table 1) that met or exceeded NRC (1988) requirements and contained 19.3 mg α-tocopherol per kg diet by analysis. Pigs were transported for approximately 2 h to a commercial packing plant and rested for a minimum of 2 h prior to slaughter.

Experiment 4. For evaluation of dietary vitamin E and PSS genotype on carcass characteristics during the finishing phase, 56 pigs (BW = 110.7 kg) of known genotype (14 **NN**, 28 **Nn**, and 14 nn) were grouped by weight and genotype into 1.5 m X 5 m pens in an open front building. Each pen of four pigs contained 1 **NN**, 2 **Nn**, and 1 nn pig. Forty-seven days prior to slaughter, pens were allotted to one of two corn-soybean commercial diets (Table 1) meeting or exceeding NRC (1988) requirements with either: (1) 33 mg/kg vitamin E or (2) 500 mg/kg vitamin E. Growth rates were recorded weekly. Pigs were transported to a commercial packing plant as in Exp. 3.

Laboratory Assays

Experiment 1 and 2. Blood samples were collected by venipuncture in 10 mL heparinized vacutainers (Vacutainer, Becton Dickinson and Co., Frankling Lakes, NJ) and kept on ice. For serum analyses, blood was collected in vacutainers with no anticoagulant and again kept on ice. Serum was separated by centrifugation (2000 x g, 10 min, 4°C) and frozen for Cp and vitamin E analysis. The heparinized tubes were centrifuged (2000 x g, 10 min, 4°C), and the harvested red blood cells (RBC) were washed (ice-cold 0.9% saline) three times. Between washes, saline was removed by aspiration. Washed RBC were lysed in an equal volume of ice-cold deionized distilled (DD) water and frozen (-80° C) until analysis for determination of Cu/Zn SOD and CAT activities. Packed RBC's were frozen (-80°C) for later GPX1 activity measurement.

Copper/Zinc superoxide dismutase was determined by the method of Hill

et al., (1999). Units of Cu/Zn SOD activity were expressed per gram of hemolysate protein (Lowry et al., 1951). Red blood cell GPX1 activity was determined utilizing the method of Sunde and Hoekstra (1980). Red blood cell CAT activity was measured by the method by Aebi (1984), and serum Cp was determined by the method of Lehmann et al. (1974). Chemicals for antioxidant analysis were purchased from Sigma Chemical (St. Louis, MO).

Alpha-tocopherol concentration of plasma was determined by HPLC as previously described (Hill et al., 1999). Briefly, 100 μL of δ-tocopherol (25 μmoles/L) in absolute ethanol (Quantum Chemical Corporation, Tuscola, IL) was introduced into each plasma sample (100 μL) as an internal standard. Tocopherols were extracted with heptane (200 μL). Extracts were evaporated to dryness under a stream of N_2 gas, then resuspended in methanol (150 μL).

Muscle α-tocopherol determination was carried out following a procedure adapted from Taylor et al. (1976). Briefly, frozen tissue samples (0.5 g) were weighed then homogenized in 10 volumes of 10 mM EDTA that contained 100 μL of 0.054 M BHT in absolute ethanol. One mL of homogenate was then added to 2 mL of 0.057 M ascorbic acid to which 100 μL of internal standard, δ-tocopherol (25 mM/L) had previously been added. Tocopherols were extracted from the homogenates with hexane. Extracts were evaporated to dryness under a stream of N_2, then resuspended in 0.15 mL of methanol.

A 100 μL aliquot of each sample extract was injected into a high performance liquid chromatograph (Gilson HPLC) equipped with a C-18 reverse-

phase column (15 cm x 4.6 mm; 3 mm; Supelco Inc., Bellefonte, PA). The mobile phase used consisted of methanol and water (98:2, by volume) with a flow rate of 1.5 mL/min. The effluent was monitored at a UV wavelength of 292 nm (Gilson UV detector 117). Sample α-tocopherol concentration was calculated from peak area responses using a standard curve that was established from known amounts of pure α-tocopherol and δ-tocopherol. Values were corrected for losses during processing by following the recovery of the internal standards, which generally exceed 90%.

Slaughter Procedures and Carcass Characteristic Evaluation

Experiment 3 and 4. At the commercial slaughter plant, pigs were electrically stunned and exsanguinated. Carcasses were then scalded, mechanically de-haired, eviscerated, and washed. Hot carcass weights were recorded and muscle pH was measured (Solomat MPM2000, Norwalk, CT; Radiometer PHM82, Copenhagen, Denmark) on the surface of the longissimus muscle (LM) at the 10th rib, 45 min post mortem. After chilling overnight, carcasses were transported in a refrigerated truck to a commercial cutting plant.

At 24 h post mortem, loin eye area (LEA), backfat thickness (BF), carcass length and muscle pH were measured and two 10th rib chops (2.5 cm thickness) were removed for drip loss and color evaluation. Initial (24 h post mortem) chop weight and light reflectance values (L*, Minolta CR-30 Chroma Meter, Minolta Camera Corp., Osaka, Japan) were recorded. Boneless chops were stored in a –3°C cooler under florescent light. At 48, 72 and 96 h post mortem light reflectance values (L*) were again recorded. For drip loss calculation, chops

were placed on an absorbent meat-packaging pad and stored in unsealed plastic vacuum packaging bags at –3°C. At 48, 72 and 96 h post mortem chops were removed, patted dry and weighed. Percent drip loss was calculated from the weight loss of chops over the 96 h post mortem. Carcass lean (kg) was calculated (7.231 + 0.437 * adjusted warm carcass weight – 18.746 * 10^{th} rib fat depth + 3.877 * 10^{th} rib loin eye area; (NPPC, 1991)).

Statistical Analysis

Experiment 1 was analyzed as a completely randomized design with a 3 x 2 factorial arrangement of treatments and repeated measures over time. The GLM procedure of SAS (SAS Institute, Cary, NC) was used to test for treatment and genotype effects. The model included the main effects of injection and genotype and injection x genotype interaction. Mean differences between injections were ascertained by least significant difference. Effect of time was ascertained using the repeated statement.

Experiment 2 was analyzed as a completely randomized design with a 3 x 2 factorial arrangement of treatments using the GLM procedure of SAS. The model included the main effects of treatment and genotype and the interaction. Mean differences between genotypes were ascertained by least significant difference. Pen served as the experimental unit for ADG, while individual pig was the experimental unit for enzyme activity.

Experiments 3 and 4 were analyzed as completely randomized designs with a 3 x 2 factorial arrangement of treatments using the GLM procedure of SAS. In Exp. 3, pig served as the experimental unit and the model contained the

main effects of genotype and injection and the interaction. In Exp. 4, pig served as the experimental unit for carcass characteristics and pork quality. The model contained the main effects of genotype and diet and the interaction. Differences between genotypes were ascertained by least significant difference.

RESULTS AND DISCUSSION

Experiment 1. Serum vitamin E concentrations of the pigs injected with vitamin E were elevated (> 20-fold, $P < 0.001$) 24 h post injection, compared with pigs of both genotypes that were injected with saline only (Table 2). There were no differences in serum vitamin E concentration between genotypes at any sampling time. However, those injected with vitamin E had higher circulating concentrations of this antioxidant (~ two-fold). On d 7 post injection, the vitamin E concentrations of those pigs injected with vitamin E remained higher (< two-fold, $P < 0.05$) than those injected with saline which were similar to initial, pre-injection samplings. In a dietary vitamin E depletion and repletion study by Jensen et al. (1990), high levels of vitamin E (405 mg/kg diet) were fed for a 7-wk period. Following this dietary regime, a depleted vitamin E diet was fed. On d 2 of depleted diet feeding, liver vitamin E concentrations dropped by 80%. Fat and muscle vitamin E concentrations remained elevated after 7 d of feeding the depleted diet. Jensen et al. (1990) concluded that serum vitamin E and liver concentrations are short-term indicators of vitamin E status; however, adipose and skeletal muscle tissues are long-term indicators.

Duthie et al. (1989) reported that carriers of the stress gene have altered antioxidant activity when compared to pigs that are free of the stress gene. In our study, pigs that carried the stress gene (Nn) had higher ($P < 0.002$) GPX1 activity 24-h after receiving the vitamin E or placebo injection than non-carriers regardless of treatment (Table 2). However, at 4 and 7 d post injection, neither

treatment nor genotype influenced the activity of this enzyme. Copper/zinc SOD activity in the red blood cell was lower ($P < 0.05$) in Nn than in NN pigs 1 d after injection with vitamin E. This change in concentration was enhanced in the 1200 IU treatment. At d 4 and 7 neither treatment nor genotype influenced Cu/Zn SOD activity. While the activity of GPX1 and Cu/Zn SOD were not affected by vitamin E injection the genotypes responded with differing activities of these antioxidant enzymes when an increase in vitamin E was administered. In a study by Lauridsen et al. (1999a) pigs (BW = 25 kg) were fed three levels of vitamin E: 1) basal diet (9 mg/kg vitamin E); 2) basal diet + 100 mg vitamin E; 3) basal diet + 200 mg vitamin E. They found that hepatic SOD and GPX1 activities were not affected by dietary vitamin E regime. In a second study, feeding the same diets, Lauridsen et al. (1999b) found no effect of diet on Cu/Zn SOD and GPX1 in the *M. Longissimus dorsi* (LD) and *M. Psoas major* (PM) muscles. Our study measured these changes in a more transient tissue (blood) and was the result of an i.m. injection as compared with a dietary intervention.

The protein Cp is made in the liver, binds 90 to 95% of the circulating Cu in the body, and has non-specific antioxidant activity. The enzyme CAT helps to rid the body of damaging peroxides (Hill et al., 1999). Ceruloplasmin and CAT activities from pigs in this study were not affected by treatment or genotype; however, Cp activity tended to increase during the 7 d post injection period while CAT activity remained unchanged (Table 2). Hill et al. (1999) reported that young pigs, 3 to 14 d of age, did not have a change in Cp activity when injected with Fe (200 mg/Ml Fe) and vitamin E (300 IU) at 6 d of age, but Cp activity increased

increased with age from 3 to 7 d of age. Similarly an increase in Cp activity was noted in this experiment using older pigs. This increase may not be an age effect, but perhaps a response to the stress of handling since Cp is also an acute phase reactant protein.

Data from Exp. 1 indicates that PSS genotype influences antioxidant activity and might affect the requirements for nutrients associated with antioxidant defense in the growing stress susceptible pig.

Experiment 2. Antioxidant enzyme activities (Table 3) 28 d post weaning were not affected by vitamin E injection. Ceruloplasmin and GPX1 activities increased during the 28 d post injection period (Table 3). This is consistent with Chung et al. (1992) who reported that varying levels of vitamin E in the diet (16, 48, 96 IU/kg) did not influence GPX1 activity or growth performance in the weanling pig. There was a treatment x genotype interaction ($P < 0.05$) for Cp activity in pigs 28 d after injection with vitamin E. Homozygous recessive pigs had higher Cp activity than homozygous dominant animals when vitamin E was not injected. The reverse was true when vitamin E was given. This may indicate that the need for added antioxidant enzyme activity protection necessary for PSS pigs is reduced by the addition of vitamin E. Catalase and Cu/Zn SOD activities remained unchanged during the experiment and were not influenced by treatment or genotype. However, nn pigs had higher initial catalase activity than NN or Nn pigs.

Homozygous recessive pigs had lower ($P < 0.05$) ADG over the 28 d when compared with Nn and NN (0.37 vs 0.41, 0.41 kg/d, respectively). Previously,

Christian and Rothchild (1981) had reported no differences in ADG, and Byrem et al. (1999) found improved gain in nn pigs. From this experiment it is apparent that injectable vitamin E (900 IU d-α-tocopherol), given only at the time of weaning, is not beneficial to the performance and antioxidant activity of the weanling pig when the activities were measured 4 wk after injection. However, increasing vitamin E may have a beneficial influence on the overall antioxidant network if measures of antioxidant status were evaluated at the time of stress (weaning).

Experiment 3. Injection of vitamin E (600 IU d-α-tocopherol/22.7 kg BW) 24 h prior to slaughter did not improve any of the measured carcass parameters in this study (Table 4). Presence of the PSS gene, however, influenced the quality and quantity of the carcass (Table 4 and 5). Homozygous recessive (nn) pigs had shorter carcasses ($P < 0.05$) with larger LEA ($P < 0.01$) and less BF ($P < 0.01$) compared with NN and Nn carcasses (Table 4). Since all carcasses were of adequate length the biological importance of this difference is probably not relevant. Huff-Lonergan et al. (2002) reported a highly significant correlation coefficient (-0.57) between LEA and BF thickness. As expected, nn pigs produced more lean pork as calculated from the National Pork Producers Council (NPPC, 1991) formula that utilizes 10th rib fat depth and LEA. Chops from homozygous recessive (nn) pigs had the highest ($P < 0.05$) L* values and lowest ($P < 0.01$) LM pH at 45 min post mortem (Table 5). A significant interaction ($P < 0.05$) between treatment and genotype was observed in pH change between 45 min post mortem and 24 h postmortem. The pH change was lowest in nn pigs

not treated with vitamin E, perhaps because the 45 min pH was already lower (5.88) in this group. If the clearance of injected vitamin E from plasma was similar to that found by Lauridsen (2002) with dietary vitamin E, perhaps this circulating antioxidant may have been important in reducing the rapid pH decline observed in nn pigs injected with vitamin E (Table 5). No pH differences were found at 24 h post mortem. Similarly, Kocwin-Podsiadla et al. (1995) found that the 30 min post mortem pH was lowest in nn pigs compared with NN and Nn, (units of pH change were similar to ours between 45 min and 24 hr post-mortem) and LM samples from nn pigs were more pale (Momcolor-D3098) compared with NN. Klont et al. (1993) found that carcasses from Nn and nn pigs had higher Hunter L* values than carcasses from NN pigs. However, they did not report a drop in pH at 45 min or 18 h post mortem. Relative to our results, the units of pH change were similar, but our initial pH at 45 min was almost one unit lower than that reported by Klont et al. (1993). In our study drip loss was higher ($P < 0.05$) in chops from nn pigs during all time periods (Table 5). Similar results were reported by Klont et al. (1993). Contrary to the results obtained when dietary vitamin E is fed to pigs (Jensen et al., 1997; Cannon et al., 1996), injection of vitamin E 24 h prior to slaughter did not increase vitamin E content of 10^{th} rib chops (Table 6). While not significant with the small sample size (n = 2 per treatment and genotype) the higher α-tocopherol concentration in the injected pigs of nn genotype may be of importance if this genotype has a higher antioxidant need as suggested by Duthie (1989). These results indicate that carrier (Nn) pigs produced carcasses with a greater amount of lean product than

NN but without sacrificing color quality and drip loss as found in nn.

Experiment 4. The vitamin E content of 10th rib chops was increased ($P <$ 0.01) when 500 mg vitamin E/kg diet was fed for 47 d (Table 6). While vitamin E is known to be rapidly cleared from the plasma to the liver, Jensen et al. (1988) reported that skeletal muscle had higher ($P < 0.001$) vitamin E tissue concentrations after 3 wk of feeding as little as 15 mg vitamin E/kg of diet compared with pigs fed unsupplemented diets. When Cannon et al. (1996) fed diets containing 0 or 100 mg of vitamin E for 84 d, α-tocopherol concentration was increased 10-fold in the longissimus muscle. Hence it is not surprising that our results reflect an increase. However, with this small sample size (n = 2 per genotype and treatment), it is not possible to discern if genotype influences tissue concentration. In our study, dietary vitamin E did not influence carcass or pork quality measurements or characteristics (Table 7 and 8). Similarly, Jensen et al. (1997) fed diets supplemented with α-tocopherol (100, 200 or 700 mg/kg feed) to pigs from weaning to slaughter and concluded that drip loss and color stability were not affected by vitamin E treatment. Color measurement, pH, drip loss and growth were also reported by Cannon et al. (1996) to be unaltered by vitamin E supplementation in pigs fed 100 mg/kg diet for 84 d prior to slaughter compared with those fed a control diet containing no supplemental vitamin E. Corino et al. (1999) fed varying levels of vitamin E (25, 50, 100, 200 and 300 mg/kg tocopherol acetate of diet) for the last 60 d prior to slaughter and found vitamin E treatment did not alter drip loss. However, not all researchers agree on the effect of vitamin E supplementation on drip loss. Cheah et al. (1995)

reported that dietary supplementation of 500 mg vitamin E/kg of diet potentially reduced drip loss in longissimus thoracis (LT) muscle by 45% in carriers (Nn) and 54% in stress-free pigs (NN). They also concluded that supplementation of 1,000 mg vitamin E/kg diet for 46 days could actually prevent PSE carcasses. Asghar et al. (1991) reported that pork chops (following three months frozen storage) from pigs supplemented with 200 mg/kg vitamin E had decreased drip loss ($P < 0.05$).

Loin pH, associated with oxidative capacity, at 45 min post mortem was highest for NN pigs ($P < 0.05$), but there were no genotype differences in LM pH at 24 h (Table 7). This is similar to the results we observed when vitamin E was injected in study 3, and typical of pigs carrying the PSE gene. Sather et al. (1991a) reported lower pH at 45 min post mortem for nn pigs than for NN or Nn and Nn pigs were lower than NN. Pommier and Houde (1993) noted that the ultimate pH of loins was significantly higher from NN pigs than from Nn pigs, but nn did not differ from the other two genotypes. Minolta light reflectance (L*) of 10^{th} rib chops at 24, 48, 72 and 96 h was the highest for nn pigs when compared with either Nn or NN genotypes (Table 7). The greater incidence of pale color by the L* score in pigs of nn genotype was also reported by Pommier and Houde (1993) and Sather et al. (1991a) with NN loins being darker in color. Van Laack et al. (1993) stated that nn pigs (n = 4) demonstrated lower pH at 45 min post mortem and had higher L* values at 48 h than NN (n = 4) or Nn (n = 4) pigs. Drip loss was higher (Table 7) in nn pigs when compared with NN. Klont et al. (1993), utilizing pre-slaughter anesthesia, found that differences in color and drip loss are

not explained by stress state of pigs or differences in pH and 45 min carcass temperature. Sather et al. (1991a) reported in heavier carcasses (85 to 105 kg), muscle drip loss from nn or Nn genotypes was not different. However, drip loss from both (nn and Nn) were greater than from NN heavy carcasses. Results of both study 3 and 4 (83 to 86 kg carcasses) support these findings.

In the present study, genotype influenced both carcass measurements and quality. Homozygous recessive pigs had shorter length ($P < 0.05$) and less BF ($P < 0.01$) than NN pigs. Both Nn and nn pigs produced carcasses with larger ($P < 0.05$) LEA and a greater amount ($P < 0.05$) of lean pork when compared to NN genotypes (Table 8). Similar results were reported by van Laack et al. (1993). These researchers noted that homozygous recessive (nn) pigs produced carcasses with larger LEA and less BF, but meat quality was often sacrificed since their carcasses were more pale and exudative. On the other hand, pigs that were heterozygous (Nn) for the stress gene produced carcasses with larger LEA and a greater amount of lean product than pigs that were free of the stress gene (NN) although muscle color and drip loss did not differ (van Laack et al., 1993). These findings are similar to ours in both Exp. 3 and 4.

While initial bleeding vitamin E concentrations in serum (Table 9) appeared to vary, there were no genotype differences. Serum vitamin E increased (approximately 1.5-fold) in the three genotypes fed the 33 mg/kg vitamin E diets as pigs approached market weight. In humans and rodents, the strong relationship between serum tocopherol and serum lipid has been recognized for many years (Horwitt et al., 1972). While it is reasonable to

assume that pigs would be similar to humans and rodents in this regard, we are not aware of any data in the literature that directly support this conclusion. Pigs fed the diet containing 500 mg/kg vitamin E had 3- to 5-fold greater ($P < 0.01$) serum vitamin E concentrations than those fed the 33 mg/kg vitamin E diet. In addition, as they approached market weight, pigs fed the 500 mg/kg vitamin E diet had increased (5- to 6-fold) serum vitamin E concentrations with no differences between the genotypes. Duthie and Arthur (1989) previously reported that when 10 or 235 IU vitamin E/kg were fed, plasma vitamin E concentrations did not differ for stress-susceptible vs stress-resistant pigs. However, Moreira and Mahan (2002) reported a linear increase in serum vitamin E when nursery pigs were fed 0 to 200 IU vitamin E /kg of diet. Lauridsen et al. (2002) recently reported in sows, plasma vitamin E, when supplied as deuterated all–rac-α-tocopheryl acetate, reached maximum concentrations 12 h post-prandially and returned to pre-ingestion concentrations by 36 h. Our data does not indicate that dietary vitamin E is managed differently due to differing stress genotype in market weight pigs.

IMPLICATIONS

These results indicate that feeding 500 mg/kg of vitamin E or injecting 600 IU/22.7 kg BW does not consistently influence carcass characteristics. Further research is needed to determine if higher amounts of (e.g. 1,000 mg/kg) vitamin E could be fed to prevent PSE. Form and duration of feeding must be evaluated with these genotypes that appear to have altered antioxidant capability. Hence not only vitamin E needs should be evaluated, but also other nutrients involved in antioxidant responses such as Se, Cu, and Zn.

Table 1. Experimental diets[a] (as-fed basis)

Ingredient, % of diet	Exp. 1, 3 and 4[a,b]	Exp. 2 (Phase I)[c]	Exp. 2 (Phase II)[d]
Dent yellow corn	68.88	36.40	37.96
Dried whey	-	20.00	22.50
Dehulled soybean meal, 44%	22.87	14.46	32.54
Dried skim milk	-	10.00	-
Spray-dried animal plasma[e]	-	7.50	-
Oat groats	-	5.00	-
Tallow	4.03	-	-
Soybean oil	-	3.00	3.00
Monocalcium phosphate, 21% P	1.71	1.04	1.48
Limestone	1.06	0.90	0.87
Vitamin and trace mineral premix[f]	0.50	0.50	0.50
Selenium and vitamin E premix[g]	0.50	0.50	0.50
NaCl	0.30	0.30	0.30
Antibacterial premix[h]	-	0.25	0.25
L-Lysine HCL	0.15	-	-
Copper sulfate	-	0.10	0.10
DL-methionine	-	0.05	-

[a]Formulated to contain 16 % CP, 0.95 Lysine, 0.8 % Ca and 0.7 % total P.

[b]Corn was removed for additional vitamin E (500 mg/kg) diet. By analysis, diet contained 568 mg α-tocopherol per kg of diet.

[c]Pelleted diet formulated to contain 23 % CP, 1.5 % Lysine, 0.89 % Ca, 0.79 % total P and 0.56 % available P.

[d]Formulated to contain 22 % CP, 1.3 % Lysine, 0.89 % Ca, 0.79 % total P and 0.50 % available P.

[e]AP-920, American Protein Corporation, Ames, IA.

[f]Provided per kg of complete diet: 3300 IU vitamin A, 660 IU vitamin D, 2.2 mg menadione, 3.3 mg riboflavin, 17.6 mg niacin, 13.2 mg pantothenic acid, 111.7 mg choline, 19.8 mg vitamin B_{12}, 37.4 mg Mn, 59.4 mg Fe, 9.9 mg Cu, 0.5 mg I, and 74.8 mg Zn.

[g]Provided per kg of complete diet: 16.5 IU vitamin E and 0.3 mg Se. By analysis, the diet contained 19.3 mg α-tocopherol per kg diet.

[h]Mecadox-10, 2.2 % carbadox antibacterial agent, Pfizer Animal Health Company, Exton, PA.

Table 2. Effect of treatment[a], genotype[b] and time post injection on serum vitamin E and ceruloplasmin and red cell glutathione peroxidase 1, catalase and Cu/Zn superoxide dismutase – experiment 1

Bleeding	Saline		600 IU vitamin E		1200 IU vitamin E					
	NN (n = 3)	Nn (n = 10)	NN (n = 3)	Nn (n = 10)	NN (n = 3)	Nn (n = 10)	SEM	*P*-value Trt[c]	*P*-value Geno[d]	*P*-value Trt x geno[c,d]
					Serum vitamin E, μL^{-1}					
Initial	2.79	2.24	2.90	2.39	2.35	2.16	0.14	0.600	0.199	0.884
1 d	3.22	2.68	35.45	60.18	113.75	101.80	3.00	0.001	0.563	0.105
4 d	3.75	3.10	5.91	5.57	6.37	6.22	0.20	0.001	0.413	0.906
7 d	2.51	1.78	2.90	2.94	3.72	3.22	0.15	0.012	0.250	0.646
					RBC glutathione peroxidase 1, U/g hemoglobin					
Initial	7.65	7.93	7.77	8.05	7.79	7.95	0.06	0.738	0.078	0.908
1 d	6.78	7.26	6.87	7.09	6.76	7.25	0.05	0.968	0.002	0.569
4 d	7.29	7.12	6.86	7.10	6.90	7.42	0.07	0.484	0.229	0.224
7 d	5.81	5.88	6.23	6.03	6.00	6.13	0.09	0.508	0.998	0.784
					RBC Cu/Zn superoxide dismutase, U/mg protein					
Initial	89.74	116.59	115.41	106.97	118.63	105.14	1.83	0.197	0.704	0.001
1 d	105.43	100.06	106.27	96.46	113.77	88.70	2.68	0.977	0.040	0.414
4 d	95.89	107.40	96.34	89.09	105.31	87.80	2.86	0.558	0.515	0.216
7 d	130.15	143.19	133.49	139.27	150.67	138.14	2.76	0.523	0.748	0.265
					RBC catalase, k					
Initial	0.02	0.03	0.03	0.03	0.03	0.03	> 0.001	0.646	0.510	0.673
1 d	0.03	0.03	0.03	0.03	0.03	0.03	> 0.001	0.541	0.172	0.140
4 d	0.03	0.03	0.03	0.04	0.03	0.03	> 0.001	0.471	0.743	0.839
7 d	0.04	0.03	0.03	0.03	0.03	0.03	> 0.001	0.631	0.563	0.807

Table 2 (cont'd)

					Serum ceruloplasmin, U/mL					
Initial	0.23	0.22	0.20	0.19	0.22	0.19	0.01	0.400	0.364	0.894
1 d	0.22	0.22	0.21	0.22	0.25	0.21	0.01	0.798	0.676	0.587
4 d	0.27	0.23	0.26	0.24	0.27	0.25	0.01	0.873	0.120	0.948
7 d	0.24	0.28	0.23	0.21	0.24	0.22	0.01	0.234	0.963	0.364

[a]Pigs (BW = 38.5 kg) received either 1) a placebo injection (2 mL saline/22.7 kg BW); 2) 600 IU d-α-tocopherol/22.7 kg BW (Vital E® 300; Schering-Plough Animal Health, Kenilworth, NJ); or 3) 1200 IU d-α-tocopherol/22.7 kg BW.

[b]Homozygous dominant (NN) non-carriers or heterozygous carriers (Nn) of the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Trt = treatment.

[d]Geno = genotype.

Table 3. Effect of treatment[a], genotype[b] and time post injection on serum vitamin E and ceruloplasmin and red cell glutathione peroxidase 1, catalase and Cu/Zn superoxide dismutase - experiment 2

Bleeding	No Injection			Injection			SEM	*P*-value Trt[c]	*P*-value Geno[d]	*P*-value Trt x Geno[c,d]
	NN (n = 13)	Nn (n = 47)	nn (n = 16)	NN (n = 23)	Nn (n = 33)	nn (n = 21)				
				RBC glutathione peroxidase 1, U/g hemoglobin						
Initial	7.48	7.84	7.75	7.16	7.74	7.75	0.18	0.721	0.559	0.952
28 d	9.89	11.16	10.36	10.89	10.86	10.32	0.25	0.681	0.443	0.586
				RBC Cu/Zn superoxide dismutase, U/mg protein						
Initial	173.50	183.18	184.88	172.31	189.28	187.15	2.99	0.720	0.197	0.886
28 d	173.53	172.37	170.56	173.75	168.36	173.21	1.68	0.919	0.740	0.713
				Serum ceruloplasmin, U/mL						
Initial	0.13	0.13	0.14	0.14	0.12	0.13	0.003	0.697	0.813	0.430
28 d	0.19	0.21	0.21	0.22	0.20	0.19	0.003	0.539	0.484	0.013
				RBC catalase, k						
Initial	0.03	0.03	0.05	0.03	0.03	0.03	0.002	0.159	0.032	0.071
28 d	0.03	0.03	0.03	0.03	0.03	0.04	0.001	0.557	0.159	0.506

[a]Pigs (BW = 7.8 kg, age = 24 d) received no injection -No E or 900 IU d-α-tocopherol at weaning - E (Vital E® 300; Schering-Plough Animal Health, Kenilworth, NJ).

[b]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Trt = treatment.

[d]Geno = genotype.

Table 4. Effect of injected vitamin E and genotype on carcass characteristics – experiment 3[a,b]

Parameter	No E			Injected E			SEM	*P*-value Trt[c]	*P*-value Geno[d]	*P*-value Trt x geno[c,d]
	NN (n = 7)	Nn (n = 16)	nn (n = 7)	NN (n = 7)	Nn (n = 16)	nn (n = 7)				
Hot carcass wt, kg	88.1	84.7	79.8	82.1	88.7	84.9	1.0	0.632	0.235	0.092
Carcass length, cm	80.6	78.6	77.3	78.4	78.6	76.4	0.3	0.153	0.024	0.395
10th rib loin eye area, cm^2	38.4	40.5	46.1	40.1	43.3	49.4	0.7	0.105	0.001	0.924
10th rib backfat, cm	2.5	1.8	1.4	1.9	2.0	1.2	0.1	0.401	0.010	0.211
Dressing %	81.3	82.0	81.0	80.1	82.1	83.4	0.3	0.444	0.082	0.074
Lean pork[e], kg	44.0	45.4	46.2	43.9	47.1	49.8	0.5	0.083	0.015	0.408

[a]Pigs (BW = 104.3 kg) received a sterile saline injection (2 mL/22.7 kg BW) - No E; or 600 IU d-α-tocopherol/22.7 kg BW 24 h prior to slaughter - E (Vital E® 300; Schering-Plough Animal Health, Kenilworth, NJ).

[b]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Trt = treatment.

[d]Geno = genotype.

[e]As calculated by the following formula: (7.231 + 0.437 * adjusted warm carcass wt – 18.746 * 10th rib fat depth + 3.877 * 10th rib loin eye area) converted to kg of lean pork, National Pork Producers Council (1991).

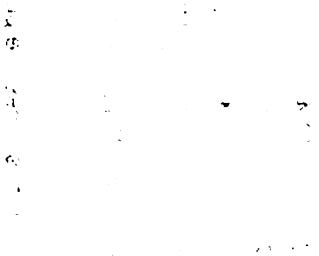


Table 5. Effect of injected vitamin E and genotype on pork quality – experiment 3[a,b]

Parameter[c]	No E			Injected E			SEM	*P*-value Trt[d]	*P*-value Geno[e]	*P*-value Trt x geno[d,e]
	NN (n = 7)	Nn (n = 16)	nn (n = 7)	NN (n = 7)	Nn (n = 16)	nn (n = 7)				
pH										
45 min post-mortem	6.48	6.62	5.88	6.57	6.34	6.09	0.04	0.914	0.001	0.016
24 h post-mortem	5.61	5.59	5.61	5.61	5.59	5.59	0.01	0.690	0.478	0.843
Change between 45 min and 24 h postmortem	-0.87	-1.03	-0.26	-0.96	-0.76	-0.50	0.04	0.840	0.001	0.016
Minolta L* value										
24 h post-mortem	58.4	57.0	59.9	56.8	59.0	61.3	0.40	0.465	0.022	0.181
48 h post-mortem	58.2	57.6	61.4	57.2	58.9	61.4	0.36	0.872	0.001	0.406
72 h post-mortem	58.2	57.4	61.0	57.3	58.7	60.7	1.40	0.978	0.006	0.397
96 h post-mortem	58.2	57.6	60.3	57.4	59.1	61.2	0.34	0.482	0.008	0.367
Drip loss, %										
24 – 48 h	1.25	1.73	2.81	1.24	2.34	2.28	0.16	0.948	0.033	0.371
48 – 72 h	2.01	2.27	3.81	1.61	2.90	3.17	0.17	0.718	0.008	0.253
72 – 96 h	2.26	2.80	3.94	2.12	3.33	3.93	0.19	0.758	0.011	0.727

[a]Pigs (BW = 104.3 kg) received a sterilized saline injection (2 mL/22.7 kg BW) - No E; or 600 IU d-α-tocopherol/22.7 kg BW 24 h prior to slaughter - E (Vital E® 300; Schering-Plough Animal Health, Kenilworth, NJ).

[b]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Measurements made on 10th rib chops (2.5 cm thickness).

[d]Trt = treatment.

[e]Geno = genotype.

Table 6. Effect of vitamin E (injected[a] or fed[b]) and genotype[c] on 10th rib chop[d] vitamin E concentration (μmol/g tissue) in experiments 3 and 4

Experiment	No E			Injected E			SEM	*P*-value Trt[e]	*P*-value Geno[f]	*P*-value Trt x geno[e,f]
	NN (n = 2)	Nn (n = 2)	nn (n = 2)	NN (n = 2)	Nn (n = 2)	nn (n = 2)				
Experiment 3	2.45	2.80	2.45	2.84	3.38	4.03	0.31	0.204	0.714	0.696
	33 mg/kg vitamin E diet			500 mg/kg vitamin E diet			SEM	*P*-value Trt[e]	*P*-value Geno[f]	*P*-value Trt x geno[e,f]
	NN (n = 2)	Nn (n = 2)	nn (n = 2)	NN (n = 2)	Nn (n = 2)	nn (n = 2)				
Experiment 4	3.25	2.49	4.46	10.80	10.27	9.95	0.40	0.001	0.675	0.463

[a]Pigs (BW = 104.3 kg) received a sterilized saline injection (2 mL/22.7 kg BW) - No E; or 600 IU d-α-tocopherol/22.7 kg BW 24 h prior to slaughter - E (Vital E® 300; Schering-Plough Animal Health, Kenilworth, NJ).

[b]Pigs (initial BW = 69.0 kg, final BW = 110.7) were fed either 1) 33 mg/kg vitamin E ; or 2) 500 mg/kg vitamin E in the diet.

[c]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[d]Randomized sampling of 10th rib chops representing all treatments and genotypes (n = 2 per each treatment x genotype combination).

[e]Trt = treatment.

[f]Geno = genotype.

Table 7. Effect of feeding vitamin E and genotype on pork quality – experiment 4[a,b]

Parameter[c]	33 mg/kg vitamin E			500 mg/kg vitamin E			SEM	*P*-value Trt[d]	*P*-value Geno[e]	*P*-value Trt x geno[d,e]
	NN (n = 7)	Nn (n = 14)	nn (n = 7)	NN (n = 7)	Nn (n = 14)	nn (n = 7)				
pH										
45 min post-mortem	6.37	6.16	6.19	6.43	6.17	6.17	0.04	0.834	0.026	0.919
24 h post-mortem	5.53	5.53	5.55	5.56	5.52	5.52	0.01	0.939	0.705	0.405
Change between 45 min and 24 h postmortem	-0.84	-0.62	-0.64	-0.87	-0.65	-0.67	0.04	0.745	0.056	1.000
Minolta L* value										
24 h post-mortem	56.2	56.6	61.3	56.6	57.6	60.0	0.36	0.933	0.001	0.457
48 h post-mortem	57.1	57.2	62.1	57.5	58.0	61.8	0.39	0.764	0.001	0.850
72 h post-mortem	57.7	57.5	62.7	57.9	58.5	61.7	0.37	0.954	0.001	0.552
96 h post-mortem	57.8	57.4	62.3	57.8	58.5	61.4	0.38	0.874	0.001	0.563
Drip loss, %										
24 – 48 h	2.32	3.21	5.34	3.10	3.91	5.77	0.27	0.281	0.002	0.973
48 - 72 h	3.03	4.46	6.44	4.00	4.74	6.87	0.28	0.353	0.001	0.879
72 – 96 h	3.62	5.33	7.29	4.67	5.35	7.51	0.29	0.489	0.001	0.762

[a]Pigs were fed (from 69.0 to 110.7 kg BW) either 1) 33 mg/kg vitamin E; or 2) 500 mg/kg vitamin E in the diet.

[b]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Measurements made on 10th rib chops (2.5 cm thickness).

[d]Trt = treatment.

[e]Geno = genotype.

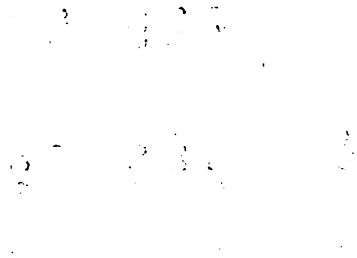


Table 8. Effect of feeding vitamin E and genotype on carcass characteristics – experiment 4[a,b]

Parameter	33 mg/kg vitamin E			500 mg/kg vitamin E			SEM	*P*-value Trt[c]	*P*-value Geno[d]	*P*-value Trt x geno[c,d]
	NN (n = 7)	Nn (n = 14)	nn (n = 7)	NN (n = 7)	Nn (n = 14)	nn (n = 7)				
Hot carcass wt, kg	84.4	85.7	84.6	83.3	85.4	83.1	0.7	0.500	0.443	0.926
Carcass length, cm	75.3	77.1	73.8	77.6	75.6	74.8	0.3	0.346	0.020	0.033
10th rib loin eye area, cm^2	42.4	45.8	48.8	43.7	47.7	48.0	0.7	0.571	0.020	0.713
10th rib backfat, cm	2.1	1.7	1.3	1.8	1.8	1.3	0.1	0.547	0.003	0.519
Dressing %	79.6	80.7	80.7	80.6	79.4	79.4	0.4	0.535	0.998	0.469
Lean Pork[e], kg	44.6	47.5	49.2	45.6	47.8	48.4	0.4	0.877	0.015	0.756

[a]Pigs were fed (from 69.0 to 110.7 kg BW) either 1) 33 mg/kg vitamin E; or 2) 500 mg/kg vitamin E in the diet.

[b]Homozygous dominant (NN) non-carriers, heterozygous carriers (Nn), or homozygous recessive positive (nn) for the stress gene as determined by the HAL 1843 method (Universal Gene Laboratories, Eldora, IA).

[c]Trt = treatment.

[d]Geno = genotype.

[e]As calculated by the following formula: (7.231 + 0.437 * adjusted warm carcass wt – 18.746 * 10th rib fat depth + 3.877 * 10th rib loin eye area) converted to kg of lean pork, National Pork Producers Council (1991).

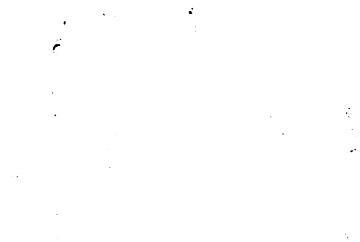


Table 9. Effect of feeding vitamin E on serum vitamin E concentration (μmol L^{-1}) – experiment 4[a]

	33 mg/kg vitamin E			500 mg/kg vitamin E						
Time	NN (n = 6)	Nn (n = 16)	Nn (n = 9)	NN (n = 8)	Nn (n = 11)	Nn (n = 6)	SEM	*P*-value Trt[b]	*P*-value Geno[c]	*P*-value Trt x geno[b,c]
Initial bleeding[d]	0.91	1.03	0.72	1.37	1.10	1.04	0.06	0.031	0.297	0.421
Final bleeding[e]	1.35	1.86	1.01	6.84	6.74	6.47	0.13	0.001	0.202	0.521

[a]Pigs were fed (from 69.0 to 110.7 kg BW) either 1) 33 mg/kg vitamin E ; or 2) 500 mg/kg vitamin E in the diet.

[b]Trt = treatment.

[c]Geno = genotype.

[d]Sample collected prior to vitamin E dietary treatments.

[e]Sample collected 38 or 45 d after initial vitamin E dietary treatment.

APPENDIX

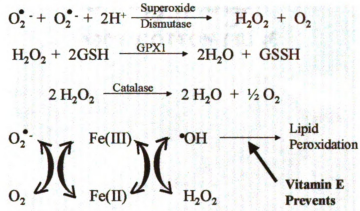
Effect of Vitamin E and Porcine Stress Syndrome (PSS) on Antioxidant Status and Carcass Characteristics of Pigs

Presented by:
Gerry Ann McCully

Vitamin E

- Vitamin E is the major lipid-soluble antioxidant in biological systems. It is an integral part of a network of antioxidants, including Cu/Zn SOD, GPX1, CAT and Cp (Lauridsen et al., 1999).

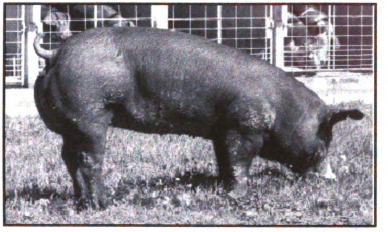
Antioxidant Reactions



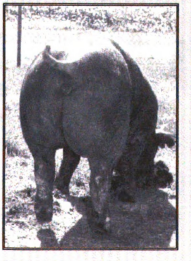
Porcine Stress Syndrome (PSS)

- Halothane or Stress gene
- DNA testing (Fuji et al., 1991)
  - Ability to identify heterozygote (Nn)
  - Limited research on heterozygote (Nn)
- Increased muscle and lean
- Increased incidence PSE
- Increased mortality when exposed to relatively minor stresses

Stress Positive (nn)



Stress Positive (nn)



Introduction

- PSS shows certain etiologic similarities to inadequacies in cellular antioxidant defense mechanisms, such as may be incurred in vitamin E deficiency (Duthie et al., 1987)

Objective

- Four studies were conducted to determine the effects of Vitamin E and the halothane gene on antioxidant status and carcass characteristics

Objective – *Study 1*

- To determine if carriers (Nn) of the halothane gene given an exogenous antioxidant (d-α-tocopherol) demonstrate a metabolic shift in antioxidant parameters

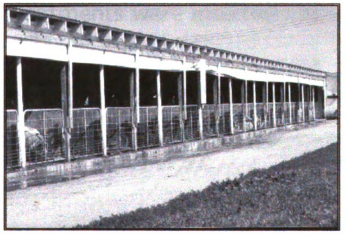
Methods – *Study 1*

- Crossbred pigs (n = 39)
  - Pietrain x Duroc Sows and Pietrain x Hampshire Boars
  - Average weight = 38.5 kg
- Genotype established by DNA determination (HAL 1843*)
  - 30 "Carriers" (Nn)
  - 9 "Normal" (NN)

Methods *cont.* – *Study 1*

- Housed in an open front building
- 13 pens (1.5 m x 5 m)
  - 10 pens "Carriers" (Nn)
  - 3 pens "Normal" (NN)

Housing Facility – *Study 1*



Methods *cont.* – *Study 1*

- Corn-soybean diet (16% crude protein)
- Vitamin E (α-tocopherol)
  - Formulated: 16.5 IU per kg diet
  - Actual: 19.3 IU per kg diet

Methods *cont.* – *Study 1*

- Treatments were injected i.m.
  - Control = 2 ml sterilized saline/22.7 kg of BW
  - 1X Vit E = 600 IU d-α-tocopherol/22.7 kg of BW
  - 2X Vit E = 1200 IU d-α-tocopherol/22.7 kg of BW

Methods *cont.* – *Study 1*

- Blood samples were collected prior to injection and 1, 4 and 7 day:
  - RBC Superoxide Dismutase (SOD)
  - RBC Glutathione Peroxidase (GPX1)
  - RBC Catalase (CAT)
  - Serum Ceruloplasmin (CP)
  - Serum Vitamin E (Vit E)

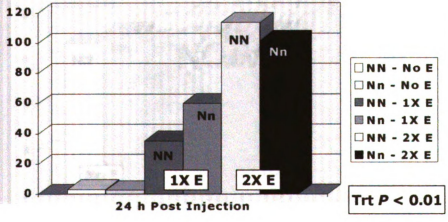
Statistical Analysis – *Study 1*

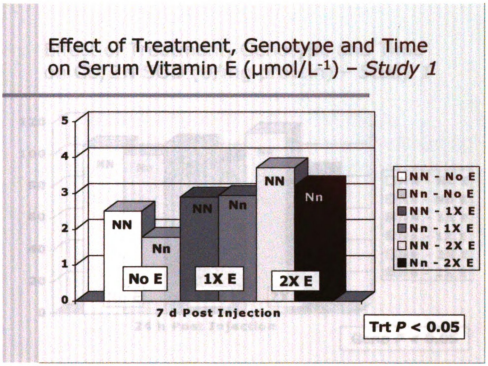
- Completely randomized design with a 3 x 2 factorial arrangement of treatment and repeated measures over time
- GLM procedure of SAS was used to test for treatment and genotype effects

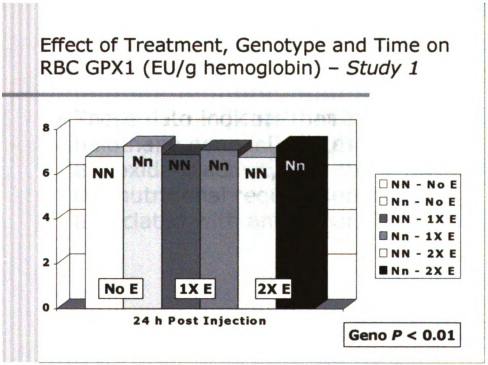
Statistical Analysis – *Study 1*

- Model included main effects of injection and genotype and injection x genotype interaction

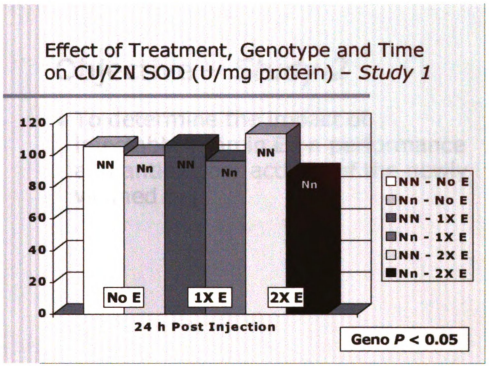
Effect of Treatment, Genotype and Time on Serum Vitamin E (μmol/L^{-1}) – *Study 1*




Effect of Treatment, Genotype and Time on Serum Vitamin E (μmol/L-1) – Study 1
5
4
3
2
1
0
NN
Nn
NN
Nn
NN
Nn
No E
1X E
2X E
7 d Post Injection
NN - No E
Nn - No E
NN - 1X E
Nn - 1X E
NN - 2X E
Nn - 2X E
Trt P < 0.05


Effect of Treatment, Genotype and Time on RBC GPX1 (EU/g hemoglobin) – Study 1
8
6
4
2
0
NN
Nn
NN
Nn
NN
Nn
No E
1X E
2X E
24 h Post Injection
NN - No E
Nn - No E
NN - 1X E
Nn - 1X E
NN - 2X E
Nn - 2X E
Geno P < 0.01


IXE
SXE



Implications – *Study 1*

- These data indicate that the halothane gene influences antioxidant activity and might affect the nutritional requirements associated with antioxidant defense

Objective – *Study 2*

- To determine the impact of injectable vitamin E on performance and antioxidant activity of the newly weaned pig

Methods *cont.* – *Study 2*

- Crossbred pigs (n = 154)
  - Pietrain x Duroc sows and Pietrain x Hampshire boars
  - Average weight = 7.84 kg
  - Average age = 24 days

Methods *cont.* – *Study 2*

- Genotype established by DNA determination (HAL 1843*)
  - 37 "Positive" (nn)
  - 80 "Carriers" (Nn)
  - 37 "Normal" (NN)

Methods *cont.* – *Study 2*

- At the time of weaning
  - Control = no injection
  - Vit E = i.m. injection of 900 IU d-α-tocopherol
- Randomly allotted to treatment by weight, litter and sex
- All genotypes and treatments were represented within each pen

Methods *cont.* – *Study 2*

- Weights were recorded weekly
- Blood samples collected at weaning and 28 day post-weaning:
  - RBC Superoxide Dismutase (SOD)
  - RBC Glutathione Peroxidase (GPX1)
  - RBC Catalase (CAT)
  - Serum Ceruloplasmin (CP)

Methods *cont.* – *Study 2*

- Diets (met or exceeded NRC)
  - 0 – 7 d: complex pelleted starter diet that contained spray-dried plasma and dried skim milk
  - 8 – 28 d: phase II diet containing primarily corn/soybean

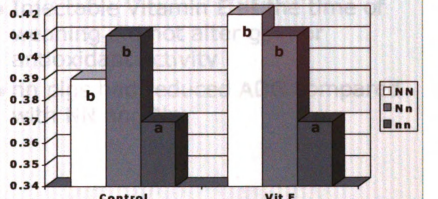
Statistical Analysis – *Study 2*

- Completely randomized design with a 3 x 2 factorial arrangement of treatments using the GLM procedure of SAS
- Model included the main effects of treatment and genotype and treatment x genotype interaction

Statistical Analysis – *Study 2*

- Mean differences between genotypes ascertained by least significant difference
- Pen served as the experimental unit for growth data, while individual pig was the experimental unit for enzyme activity

ADG – *Study 2*



Results – *Study 2*

- Vitamin E injection at weaning did not alter antioxidant activity
- CP activity increased with time
- GPX1 increased with time
- CAT and Cu/Zn SOD remained unchanged

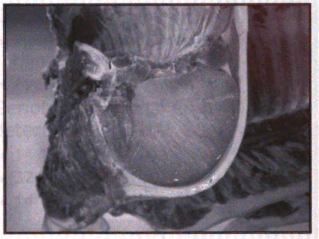
Summary – *Study 2*

- Injectable Vitamin E at the time of weaning did not alter gain or antioxidant activity
- nn pigs had reduced ADG compared with NN and Nn

Objective – *Study 3 and 4*

- To determine the impact of genotype and vitamin E on carcass characteristics of pigs (NN, Nn, nn)

PSE Carcass



Objective – *Study 3*

- To determine the impact of the halothane gene and injectable vitamin E 24 hours prior to slaughter on carcass characteristics of pigs of known genotype (nn, Nn, NN)

Methods – *Study 3*

- Crossbred pigs (n = 60)
  - Pietrain x Duroc Sows and Pietrain x Hampshire Boars
  - Average weight = 69.25 kg
- Genotype established by DNA determination (HAL 1843*)
  - 14 "Positive" (nn)
  - 32 "Carriers" (Nn)
  - 14 "Normal" (NN)

Methods *cont.* – *Study 3*

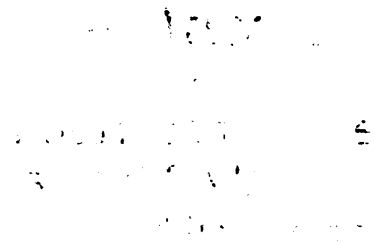
- 24 hours prior to slaughter pigs were injected i.m.
  - Control = 2 ml sterilized saline/22.7 kg of BW
  - Vit E = 600 IU d-α-tocopherol/22.7 kg of BW

Methods *cont.* – *Study 3*

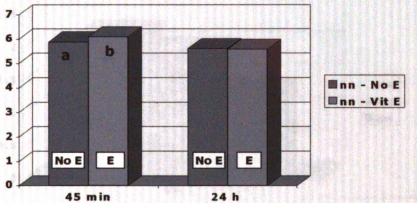
- Pigs were transported for approximately 2 h to a commercial harvesting facility
- Pigs rested for a minimum of 2 h
- Carcasses were chilled overnight and transported to a commercial cutting plant

Methods *cont.* – *Study 3*

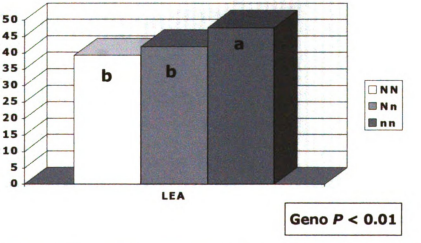
- pH at 45 min and 24 h on the surface of the LM at the 10^{th} rib
- At 24 h, two boneless chops (2.5 cm)
  - Minolta light reflectance (L*)
  - Drip loss (DL)
- 10^{th} rib loin eye area (cm^2)
- 10^{th} backfat (BF)
- Lean kg of pork produced



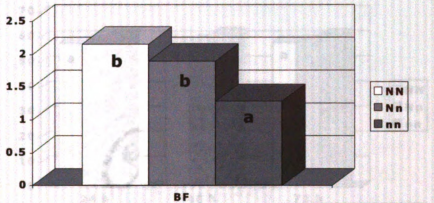
Effect of Vitamin E on Loin pH in nn pigs– *Study 3*



Effect of Genotype on 10th Rib Loin Eye Area, cm² – *Study 3*

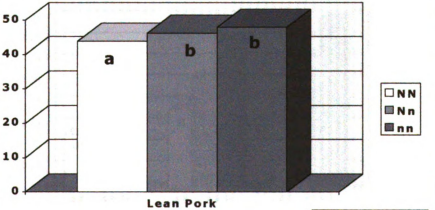


Effect of Genotype on 10th Rib Backfat, cm – *Study 3*



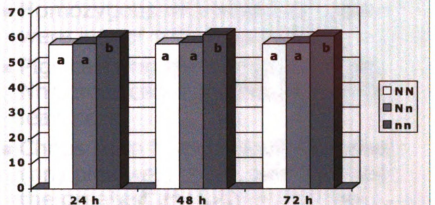
Geno $P < 0.01$

Effect of Genotype on Lean Pork (kg) – *Study 3*

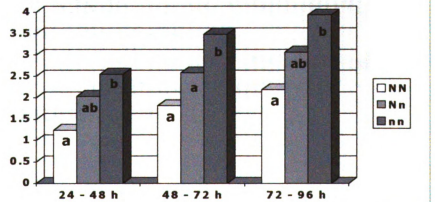


Geno $P < 0.05$

Effect of Genotype of Minolta L* Values – *Study 3*



Effect of Genotype on Drip Loss (%) – *Study 3*



Summary – *Study 3*

- Homozygous recessive (nn) pigs were leaner with larger LEA
- Pigs carrying the stress gene (nn, Nn) produced a greater amount of lean pork
- Chops from homozygous recessive (nn) pigs were more pale and had the greatest amount of drip loss

Summary *cont.* – *Study 3*

- Injectable vitamin E did not affect drip loss or color
- Injectable vitamin E did improve pH at 45 min in the homozygous recessive (nn) pig

Objective – *Study 4*

- To determine the impact of halothane genotype and dietary vitamin E on carcass characteristics of pigs of know genotype (nn, Nn, NN)

Methods – *Study 4*

- Crossbred pigs (n = 56)
  - Pietrain x Duroc Sows and Pietrain x Hampshire Boars
- Genotype established by DNA determination (HAL 1843*)
  - 14 "Positive" (nn)
  - 28 "Carriers" (Nn)
  - 14 "Normal" (NN)

Methods *cont.* – *Study 4*

- Two dietary treatments fed 42 days prior to slaughter
  - Control = finisher diet containing 33 IU Vitamin E/kg diet
  - High E = Formulated: control diet plus 500 IU Vitamin E/kg diet
- Blood samples were collected 72 h prior to slaughter for serum Vitamin E analysis

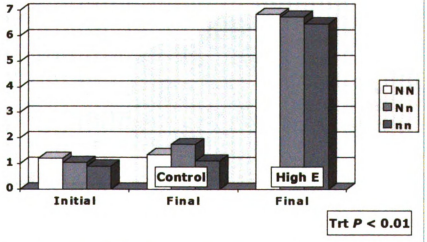
Methods *cont.* – *Study 4*

- Pigs were transported for approximately 2 h to a commercial harvesting facility
- Pigs rested for a minimum of 2 h
- Carcasses were chilled overnight and transported to a commercial cutting plant

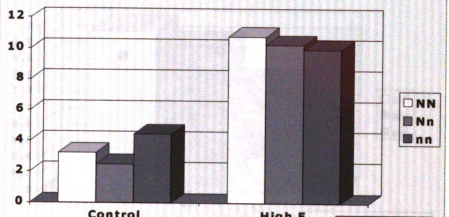
Methods *cont.* – *Study 4*

- pH at 45 min and 24 h on the surface of the LM at the 10th rib
- At 24 h, two boneless chops (2.5 cm)
  - Minolta light reflectance (L*)
  - Drip loss (DL)
- 10th rib loin eye area (cm^2)
- 10th backfat (BF)
- Lean kg of pork produced

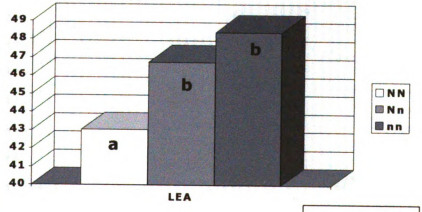
Effect of Genotype, Treatment and Time on Serum Vitamin E – *Study 4*



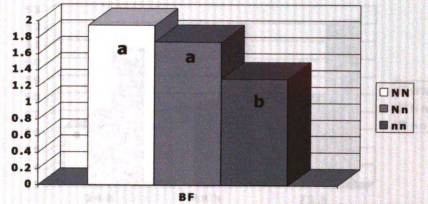
Effect of Dietary Vitamin E on Vitamin E Content (μmol/g tissue) in Chops – *Study 4*



Effect of Genotype on 10th Rib Loin Eye Area, cm² – *Study 4*

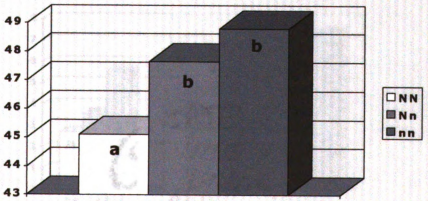


Effect of Genotype on 10th Rib Backfat, cm – *Study 4*



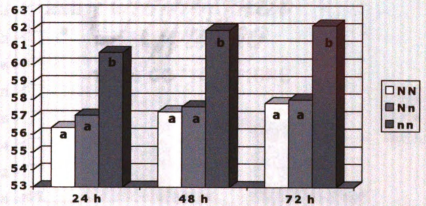
Geno $P < 0.01$

Effect of Genotype on Lean Pork (kg) – *Study 4*



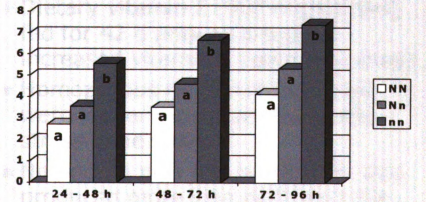
Geno $P < 0.05$

Effect of Genotype of Minolta L* Values – *Study 4*



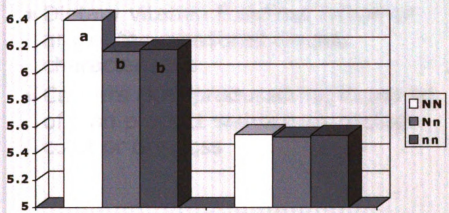
Geno $P < 0.01$

Effect of Genotype on Drip Loss (%) – *Study 4*



Geno $P < 0.05$

Effect of Genotype on pH in chops – *Study 4*



Summary – Study 4

- Dietary vitamin E (500 mg/kg diet) fed for 42 d prior to slaughter increased vitamin E content in chops
- Homozygous recessive (nn) pigs were leaner than pigs free of the stress gene (NN)
- Nn and nn pigs had larger LEA and produced more lean pork than NN

Summary *cont.* – *Study 4*

- Dietary vitamin E did not influence any of the measured carcass characteristics
- Carriers (Nn) produced increased kg of lean product without sacrificing color or drip loss

Implications

- 500 mg/kg vitamin E or injecting 600 IU/22.7 kg BW does not consistently influence carcass characteristics
- Further research is needed to determine if higher amounts of vitamin E could prevent PSE

Implications *cont.*

- Stress positive (nn) and carriers (Nn) appear to have altered antioxidant capability and may have differing dietary requirement than pigs that are free of the stress gene (NN)

Implications *cont.*

- Not only should vitamin E be evaluated, but also other nutrients involved in antioxidant responses such as Se, Cu, and Zn

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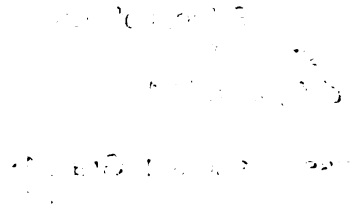

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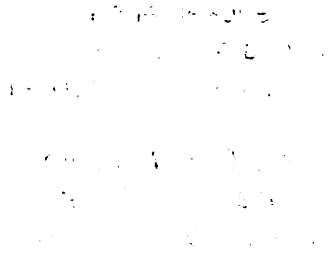

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