

Creatine monohydrate supplementation and the quality of fresh pork in normal and halothane carrier pigs¹

R. J. Maddock^{*2}, B. S. Bidner[†], S. N. Carr[†], F. K. McKeith[†], E. P. Berg[‡], and J. W. Savell^{*}

^{*}Department of Animal Sciences, Texas A & M University, College Station 77843-2471;

[†]Department of Animal Sciences, University of Illinois, Urbana 61801; and

[‡]Department of Animal Sciences, University of Missouri, Columbia 65211

ABSTRACT: The objective of this research was to examine the impact of supplementation with creatine monohydrate (CMH) on the quality of various muscles from normal and heterozygous halothane carrier pigs. Twenty-nine crossbred pigs, 16 normal (NN) and 13 halothane carrier (Nn) genotypes, were supplemented with 0 or 25 g·pig⁻¹·d⁻¹ of CMH for 5 d before slaughter. Supplemented pigs gained 2.26 kg more weight ($P < 0.05$) during 5 d of supplementation. There were trends ($P < 0.10$) toward higher objective marbling scores and lower cooking loss for supplemented pigs. The 45-min pH was 0.27 units higher ($P < 0.05$) for supplemented

pigs in the semimembranosus; CMH supplementation did not influence ($P > 0.05$) drip loss or muscle composition. Supplementation with CMH also resulted in lower L* values in two ham muscles, semitendinosus (5.15 units) ($P < 0.05$) and semimembranosus (1.95 units) ($P < 0.10$) for Nn carcasses. Genotype had significant effects on most quality indicators, with Nn carcasses producing lower-quality lean as evidenced by less desirable subjective and objective color and higher drip losses. Genotype also affected the composition of several muscles, with the NN carcasses having more fat and less moisture.

Key Words: Creatine, Meat Quality, Pigs

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Introduction

In an effort to improve the lean quality of pork, Berg et al. (1999) and O'Quinn et al. (2000) have studied the dietary supplementation of creatine monohydrate (CMH). Creatine monohydrate provides ATP for muscle contraction and metabolism. Increasing the amount of available energy for ATP production that does not involve glycolysis or the production of lactic acid may improve pork quality by slowing pH declines immediately postmortem.

Variation in the lean quality of pigs is a major concern to the pork industry. Estimates of economic losses due to poor quality reach \$100 million or more per year (Cannon et al., 1995). The presence of pale, soft, and exudative (PSE) lean is the major contributor to the economic loss associated with poor quality pork carcasses. The PSE condition can result from several fac-

tors that modulate metabolism, including genetic susceptibility, preslaughter stress, and other factors (Sutton et al., 1997). Muscle metabolism around the time of slaughter has a major effect on pork quality (Klont and Lambooy, 1995). An important genetic factor that alters muscle metabolism is the presence of the recessive halothane gene. Interventions that alter muscle metabolism have been investigated as methods to improve pork quality. The objective of this research was to examine the impact of supplementation with CMH on the meat quality of various muscles from normal and heterozygous halothane carrier pigs and to determine whether a supplementation × genetic type interaction occurred.

Materials and Methods

Animals and Experimental Design

The protocol for this study was approved by the University of Illinois Laboratory Animal Care Advisory Committee. For each of two replications, 16 Duroc-cross pigs (approximately 110 kg) were placed in individual pens and allowed to acclimate for 48 h before the start of creatine supplementation. Eight pigs per replication were recessive halothane gene carriers (Nn genotype) and eight were homozygous dominant (NN genotype)

¹The authors thank Lori Engel for assistance with data collection.

²Correspondence and current address: Dept. of Anim. and Range Sci., South Dakota State Univ., Box 2170, Brookings 57007 (phone: 605-688-5439; fax: 605-688-6170; E-mail: robert_maddock@sdstate.edu).

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as determined by DNA analysis (Fujii et al., 1991). An equal number of gilts and barrows were used in this study. Animals were distributed by weight across treatment to reduce initial weight variation. Upon entering the individual feeding pens, pigs were restricted to 2.75 kg of a corn-based diet per day. The ration was divided into two equal parts and fed at consistent times in the morning and afternoon. Twenty-five grams of CMH (Wilke International, Lenexa, KS) was divided into two equal portions (for morning and afternoon feedings) and supplemented to eight pigs per replication stratified equally across genotype. This provided 25 g·pig⁻¹·d⁻¹ of CMH to the treated pigs. Creatine was fed by top dressing at the time of feeding. Refused feed was removed prior to the morning feeding and weighed. Following 5 d of supplementation, all pigs were transported to the University of Illinois abattoir and conventionally slaughtered.

Abattoir Measurements

Temperature at 45 min was measured with a handheld digital thermometer (Koch Suppliers, Kansas City, MO) placed approximately into the center of the semimembranosus. The pH at 45 min was measured with a calibrated, portable pH meter (pH-Star, SFK Technology, Denmark) inserted directly into the semimembranosus and longissimus thoracis at approximately the 10th rib.

Carcass Quality Attributes and Fabrication

Following 24 h of chill at 4°C in a cooler with high air movement velocity, the right side of the carcass was ribbed between the 10th and 11th rib and subjective quality scores were assigned to the longissimus thoracis using National Pork Producers Council (NPPC, 2000) standards by a trained individual. Color and firmness were evaluated using a 6-point scale (1 = pale pinkish gray to white, or very soft and very watery and 6 = dark purplish red, or very firm and dry), and marbling was evaluated using the NPPC scale with assigned number corresponding to the estimated percentage of intramuscular fat content (NPPC, 2000). Carcass fat depth was measured at the first and last rib, and fat thickness was measured adjacent to the 10th rib. Loin eye area and carcass length also were measured. The right side was then separated into individual muscles: longissimus lumborum et thoracis (**LT**), semimembranosus (**SM**), biceps femoris (**BF**), semitendinosus (**ST**), quadriceps group (**QD**), triceps brachii (**TB**), and serratus ventralis (**SV**).

Muscle Quality Attributes

At 24 h, muscle pH was obtained as described for 45-min measurements. Objective color was measured using a portable Minolta Chroma Meter CR 310 (Minolta, Tokyo, Japan) with a D-65 light source and a zero angle reflection to obtain L* measurements. Mus-

cle groups were cut across the muscle fibers in consistent locations for each muscle, and two to five measurements that would adequately cover the majority of the lean surface were taken at random points on the split surface of the lean.

Drip loss was measured according to McCaw et al. (1997). Briefly, a slice of each muscle approximately 1.3 cm thick was excised, weighed, and suspended by hooks in plastic bags for 24 h at 4°C. The sample was reweighed and the drip loss was expressed as the percentage of weight lost.

Muscle samples were obtained at approximately 24 h postmortem, ground within 48 h, and frozen for subsequent proximate analysis determination of the water and lipid content of each muscle using AOAC (1990) methods. A single rib chop was saved from the 10th rib location, aged for 7 d, frozen for 14 d, thawed overnight at 4°C, and used for cooking loss and tenderness determination using Warner-Bratzler shear force (AMSA, 1995). Briefly, chops were weighed raw, placed on a Farberware Open-Hearth grill (Kidde, Bronx, NY), and cooked to an internal temperature of 71°C; chops were turned once. Cooked weights were recorded and cooking loss was determined as a percentage of raw weight. Chops were allowed to cool to room temperature and six cores were removed parallel to the muscle fibers; each core was sheared once with a Warner-Bratzler shear machine.

Statistical Analysis

Data were analyzed as a 2 × 2 factorial with creatine supplementation and genotype and their interaction as the main effects utilizing the general linear models (GLM) procedure of SAS (SAS Inst., Cary, NC). Means were separated using the PDIF option of the GLM procedure. To ensure all pigs had ingested adequate amounts of CMH, pigs that did not consume at least 2.25 kg of feed per day were not included in any analysis.

Due to differences in weight between NN and Nn genotype, slaughter weight was analyzed as a covariate using ANCOVA. Harvest weight was found to be a significant covariate for most carcass traits, but not other quality traits. Therefore, weight was included as a covariate in the analysis of harvest and carcass traits, unless otherwise mentioned in the text. However, a harvest weight × genotype interaction was not found to be significant.

Sex class and replication were analyzed and found to not be significant for any analyzed trait. These variables were subsequently removed from all models of analysis.

Results and Discussion

Live Animal Traits

Live animal traits are summarized in Table 1. Due to limited numbers of available Nn animals and a desire

Table 1. Least squares means (\pm SE) for weights, feed intake, creatine intake, and weight change of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^{ab}

Trait	Genotype		P	Treatment		P
	NN (n = 16)	Nn (n = 13)		C (n = 16)	S (n = 13)	
Start weight, kg	114.7 \pm 2.0 ^c	104.2 \pm 2.2 ^d	0.0017	110.5 \pm 2.0	108.3 \pm 2.2	0.47
Feed intake/d, kg	2.6 \pm 0.04	2.6 \pm 0.04	0.74	2.6 \pm 0.04	2.6 \pm 0.04	0.35
Creatine intake/d, g	12.0 \pm 0.15	12.3 \pm 0.17	0.31	0 ^c	24.3 \pm 0.17 ^d	0.0001
Harvest weight, kg	118.9 \pm 1.7 ^c	109.6 \pm 1.9 ^d	0.011	114.9 \pm 1.7	114.9 \pm 1.9	0.63
Weight increase, kg	5.4 \pm 0.7	5.6 \pm 0.8	0.83	4.4 \pm 0.7 ^c	6.6 \pm 0.8 ^d	0.047
Hot carcass weight, kg	89.7 \pm 1.4 ^c	81.7 \pm 1.6 ^d	0.0007	85.6 \pm 1.4	85.8 \pm 1.6	0.98

^aThere were no significant genotype \times treatment interactions.

^bDiets were supplemented with 25 g/d of creatine monohydrate.

^{c,d}Least squares means for the same main effect (genotype or treatment) without a common superscript differ ($P < 0.05$).

to have approximately equal weights between control and supplemented pigs, it was not possible to exactly match weights of NN and Nn genotypes. As a result, NN pigs were heavier than Nn pigs at the start of the trial and at harvest and had higher hot carcass weights.

Because of the weight difference, a significant difference in weights is noted for all weight measures. Other research has not shown weight disadvantages associated with the halothane genotype; therefore, this weight difference is likely due to preslaughter CMH treatment allocation. However, because there have been reports of a genotype \times carcass weight interaction (Sather et al., 1991) affecting quality, slaughter weight, when proven to be significant, was included as a covariate in the analysis of carcass traits. Slaughter weight was used as a covariate for first and last rib fat thickness, 10th rib fat depth, carcass length, and objective quality traits. Slaughter weight was not a significant covariate for any other traits examined.

Intake data showed that there was no difference in feed or creatine intake across genotype or treatment. This is consistent with Leach et al. (1996), who did not find a difference in feed intake between genotypes. The CMH used for supplementation was tasteless and odorless and likely did not affect the palatability of feed. In addition, feed level was restricted to 2.75 kg/d (approximately 85% normal intake) to ensure complete use of all of the ration and complete ingestion of all supplemented CMH.

Of most interest concerning live traits was the significant increase in weight displayed by CMH-treated pigs vs controls. Supplemented pigs gained 34% more weight in the 5-d supplementation period than controls. This is consistent with data from human creatine trials, which have shown an increase of up to 1 kg in a 6-d supplementation period (Balsom et al., 1993). The authors attributed the increase in body weight to water retention in muscle tissue and not to protein accretion but did not examine why this might occur. Proximate analysis results from this trial did not show a significant increase in moisture content due to CMH treatment, so body weight increase was likely not due to water retention by muscle. Berg et al. (1999) reported

that pigs supplemented with CMH had a lowered muscle protein:moisture ratio and hypothesized that this was due to myofibrillar hydration. Muscle protein content was not measured in this study, so this same conclusion cannot be drawn or refuted. O'Quinn et al. (2000) found numeric increases in ADG and gain:feed (G/F) during a 10-d CMH supplementation. Another possibility explaining the weight increase is actual muscle protein accretion. Ingwall et al. (1974) reported that muscle cells supplemented with CMH in vitro increased accretion of myosin heavy chain. In addition, Kreider et al. (1998) observed higher fat/bone free mass in humans consuming a CMH supplement for 28 d.

Carcass Traits

Table 2 shows carcass traits. Least squares means were adjusted for these traits using harvest weight as a covariate. Dressing percentage and liver weights were not significantly affected by genotype or CMH treatment. This is in contrast with other research that has found halothane carrier and reactor pigs to have higher dressing percentages and a greater proportion of muscle yield (Sather et al., 1991; Leach et al., 1996). The lack of difference in dressing percentage may be a result of the variation in weights, or the small sample size. No fat measures were affected by CMH treatment or genotype. Due to the short feeding period, no CMH treatment differences were expected. When carcass weight was included as a covariate, genotype did not significantly affect fat measurements; other studies have not found differences in the fat thickness or depth of carrier or reactor pigs (Pommier et al., 1992; Leach et al., 1996). Carcasses from NN pigs were significantly longer than those from Nn pigs, likely related to the higher harvest weights of NN pigs. Loin eye areas were not affected by CMH supplementation or genotype. If, as previously hypothesized by the authors, muscle protein accretion occurred, it was not detectable as an increase in loin muscle size. There were several carcass composition differences between genotypes. Other reports have investigated the effects of genotype on composition more thoroughly than this experiment (Sather et al., 1991; Leach et al., 1996).

Table 2. Least squares means (\pm SE) for harvest and carcass traits of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^{a,b,c}

Trait	Genotype		<i>P</i>	Treatment		<i>P</i>
	NN (n = 16)	Nn (n = 13)		C (n = 16)	S (n = 16)	
Dressing percentage	75.0 \pm 0.3	74.4 \pm 0.4	0.25	74.4 \pm 0.3	75.0 \pm 0.4	0.31
Liver weight, g	1,688 \pm 66	1,661 \pm 71	0.78	1,618 \pm 64	1,730 \pm 74	0.26
First rib fat, mm	36.0 \pm 1.5	37.3 \pm 1.7	0.60	38.3 \pm 1.4	35.0 \pm 1.6	0.13
Tenth rib fat, mm	22.0 \pm 1.6	21.0 \pm 1.8	0.68	22.1 \pm 1.5	21.0 \pm 1.65	0.62
Last rib fat, mm	20.5 \pm 0.9	20.7 \pm 1.0	0.92	20.1 \pm 0.9	21.1 \pm 1.0	0.50
Loin eye area, cm ²	41.5 \pm 1.1	39.0 \pm 1.2	0.14	40.6 \pm 1.1	39.8 \pm 1.2	0.59
Carcass length, cm	82.4 \pm 0.4 ^x	80.0 \pm 0.5 ^y	0.001	80.9 \pm 0.4	81.6 \pm 0.5	0.38
Color ^d	2.6 \pm 0.1 ^x	2.3 \pm 0.1 ^y	0.050	2.4 \pm 0.1	2.5 \pm 0.1	0.35
Firmness ^d	2.5 \pm 0.1	2.3 \pm 0.1	0.21	2.3 \pm 0.1	2.5 \pm 0.1	0.12
Marbling ^e	2.2 \pm 0.2 ^x	1.5 \pm 0.20 ^y	0.029	1.7 \pm 0.2	2.1 \pm 0.2	0.08
Cooking loss, %	25.2 \pm 0.9	25.6 \pm 1.0	0.78	26.6 \pm 0.9	24.1 \pm 1.0	0.095
Warne-Bratzler shear force, kg	3.7 \pm 0.1	4.1 \pm 0.2	0.054	4.0 \pm 0.1	3.8 \pm 0.2	0.35

^aFor carcass traits, harvest weight was utilized as a covariate when it was significant ($P < 0.05$).

^bThere were no significant genotype \times treatment interactions.

^cDiets were supplemented with 25 g/d of creatine monohydrate.

^d1 = pale, pinkish gray or very soft and very watery and 6 = dark purplish red or very firm and dry.

^eMarbling is an estimation of the percentage of intramuscular fat.

^{x,y}Least squares means for the same main effect (genotype or treatment) without a common superscript differ ($P < 0.05$).

Supplementation with CMH did not affect the levels of most subjective quality assessments. Treatment with CMH did not affect color or firmness scores, although marbling score did tend to be higher in CMH-treated pigs. It is not likely that 5 d of CMH supplementation can increase marbling amount. This is also shown in the composition data from this study, which show no difference in fat content of the LT, and therefore an explanation for the increased marbling score is unclear. Possible reasons for the increased marbling may include darker lean in the longissimus leading to increased visual acuity of intramuscular fat; however, this is not shown by L* values collected in this study.

Shear force and cooking loss data showed either a trend toward genotype, treatment, or a treatment \times genotype interaction. A trend toward a genotype \times CMH treatment effect was suggested because the untreated Nn genotype loin chops had the highest shear force value (4.40 kg), which tended ($P = 0.095$) to be higher than that of CMH-treated Nn loin chops (3.82 kg). This is a fairly large (0.57 kg) improvement in shear force of Nn chops; however, the data were not consistent enough for a conclusion to be drawn. Factors related to tenderness in pork loin including muscle composition, pH, and color were not found to be different in this study. This result requires an explanation beyond these factors.

Cooking loss of loin chops showed a trend ($P = 0.095$) toward being affected by CMH treatment; treated loin chops had a lower cooking loss than controls. The primary factors that affect cooking loss are pH and composition of the chop. There were no differences found in these factors in this experiment.

Genotype was significant for color and marbling, but not firmness. As previously shown (Leach et al., 1996;

Sutton et al., 1997; Baas and Mabry, 1999), subjective quality measures were significantly affected by genotype, with NN genotypes having a higher color and marbling score. Warner-Bratzler shear force of the LT tended to be higher for Nn genotype (4.11 kg) than for NN (3.67 kg), which disagrees with findings of Leach et al. (1996) and De Smet et al. (1996), who did not show a genotype effect on shear force. However, Sather et al. (1991) reported a significant difference between Nn and NN pigs.

Cooking loss was not affected by genotype. The trend toward similar cooking losses has been reported before (De Smet et al., 1996). Leach et al. (1996) suggested that moisture (purge) lost before cooking was higher in carrier pigs, resulting in less water that could be lost during cooking.

pH and Temperature Decline

The pH and temperature measurements are recorded in Table 3. There were no differences in internal ham temperature (measured in the semimembranosus) at 45 min or 24 h across genotype or treatment. There would be no apparent reason why CMH would increase body temperature; this also agrees with Prevost et al. (1997), who found no difference in the body temperature of humans taking creatine supplement, and Klont and Lambooy (1995), who were unable to detect a difference in temperature due to genotype. For measurements taken in the SM, 45-min pH was not affected by genotype but was by treatment: carcasses from CMH-treated pigs had a 45-min pH that was 0.27 units higher. The CMH treatment effect may be explained as a persistence of postmortem aerobic glycolysis. Greenhaff (1996) reported that humans supplemented

Table 3. Least squares means (\pm SE) of temperature and pH declines of the semimembranosus (SM) and longissimus thoracis (LT) of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^{a,b}

Trait	Genotype		P	Treatment		P
	NN (n = 15)	Nn (n = 13)		C (n = 16)	S (n = 12)	
45-min temp SM, °C	41.1 \pm 0.15	41.1 \pm 0.16	0.85	41.6 \pm 0.15	41.7 \pm 0.16	0.89
45-min pH SM	6.1 \pm 0.1	6.0 \pm 0.09	0.19	5.9 \pm 0.08 ^c	6.2 \pm 0.09 ^d	0.039
45-min pH LT	6.1 \pm 0.08 ^c	5.7 \pm 0.08 ^d	0.0006	5.9 \pm 0.08	5.9 \pm 0.09	0.42
24-h temp SM, °C	21.3 \pm 1.2	22.5 \pm 1.3	0.75	21.2 \pm 1.2	19.6 \pm 1.5	0.68
24-h pH SM	5.6 \pm 0.04	5.5 \pm 0.05	0.11	5.5 \pm 0.04	5.6 \pm 0.05	0.11
24-h pH LT	5.5 \pm 0.04	5.5 \pm 0.04	0.31	5.5 \pm 0.03	5.5 \pm 0.04	0.18

^aThere were no significant genotype \times treatment interactions.

^bDiets were supplemented with 25 g/d of creatine monohydrate.

^{c,d}Least squares means for the same main effect (genotype or treatment) without a common superscript differ ($P < 0.05$).

with 20 g of CMH for 5 to 6 d increased muscular creatine phosphate concentrations by 25%. Higher levels of creatine phosphate provide greater substrate for rephosphorylation of ADP to ATP and may result in a delay in metabolic conversion of glucose to lactic acid in postmortem muscle. In addition, Prevost et al. (1997) reported that intramuscular loading of creatine phosphate may serve as a lactic acid buffer and improve exercise recovery time from short-duration, maximum-intensity activity. Other studies in humans have shown a higher muscle pH following intense exercise in humans supplemented with creatine (Williams and Branch, 1998). The difference in CMH treatment effects between the LT and SM may be explained by the muscle fiber types of each muscle. The semimembranosus tends to have high proportion of fast glycolytic and glycolytic fibers (α -white or Type II), whereas the LT tends to have a greater amount of oxidative fibers (β -red and α -red) (Solomon et al., 1994; Aalhus et al., 1997). Creatine also has been shown to provide a greater amount of energy to type II fibers, and the availability of phosphocreatine has been suggested as a limiting factor for maintaining muscle force during high-intensity exercise (Balsom et al., 1994). Klont et al. (1993) also showed that phosphocreatine was more rapidly expended in NN and nn genotypes, revealing the importance of creatine to postmortem muscle metabolism and pH decline. Creatine may exert a stronger influence on pH in muscles with a greater amount of type II fibers. The 45-min pH of the SM was not affected by genotype, which is different from findings of several other studies (Klont and Lambooy 1995; De Smet et al., 1996).

For the LT, CMH treatment did not affect 45-min pH across genotype. Genotype was very important, with NN carcasses having a pH of 6.14 and Nn 5.67, which agrees with others, including Klont and Lambooy (1995) and De Smet et al. (1996).

At 24 h, the pH in the SM was similar for CMH-treated (5.57) and control (5.47) carcasses. In addition, at 24 h, NN genotype carcasses also were similar in pH (5.57) to

Nn carcasses (5.47). The 24-h pH of both muscles (LT and SM) was not affected by genotype or treatment. Higher ultimate pH for NN than for Nn genotypes is widely seen (Sather et al., 1991; Leach et al., 1996; Stadler et al., 1998). Conversely, others have reported no difference in 24-h or ultimate pH between NN and Nn genotypes (Pommier et al., 1992; Klont et al., 1993).

Drip Loss

Table 4 reports the drip loss data. Supplementation with CMH had no effect on the level of drip loss of any of the muscles evaluated in this study, including the SM, despite the significant influence CMH treatment had on 45-min pH. All of the drip losses were lower for the CMH-treated carcasses; however, no levels of significance were reached.

There was a strong trend toward higher drip loss from Nn carcasses in the LT, and significantly more drip loss from Nn carcasses in the BF, but the SM, ST, and TB did not show any difference. Most studies have only reviewed the LT and SM when evaluating genotype effects on most quality traits, including drip loss. These studies tend to find decreased drip loss in NN genotypes compared to Nn genotypes (Cisneros et al., 1996; DeSmet et al., 1996). The LT from Nn genotypes in this study had a lower 45-min pH, suggesting there was more protein denaturation occurring in muscle from Nn carcasses. In addition, the water-holding capacity of muscles can be affected by ultimate pH due to approaching the isoelectric point. It should be noted that genotype was not significant for 45-min pH in the SM, and genotype did not affect drip loss of the SM.

L*-Values

All ham and loin muscles had a genotype or a genotype \times treatment interaction. Neither genotype nor treatment affected objective color evaluation of muscles of the shoulder.

Table 4. Least squares means (\pm SE) of drip loss (%) of the longissimus thoracis (LT), semimembranosus (SM), biceps femoris (BF), semitendinosus (ST), and triceps brachii (TB) of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^{a,b}

Muscle	Genotype		P	Treatment		P
	NN (n = 16)	Nn (n = 13)		C (n = 16)	S (n = 13)	
LT	5.8 \pm 0.5	7.2 \pm 0.5	0.054	6.8 \pm 0.5	6.3 \pm 0.5	0.47
SM	6.0 \pm 0.4	5.9 \pm 0.5	0.65	5.9 \pm 0.4	5.6 \pm 0.45	0.56
BF	6.4 \pm 0.4 ^a	8.3 \pm 0.5 ^b	0.0079	7.5 \pm 0.4	7.1 \pm 0.5	0.61
ST	6.3 \pm 7.4	7.4 \pm 0.9	0.33	7.6 \pm 0.8	6.1 \pm 0.9	0.21
TB	2.2 \pm 0.2	2.5 \pm 0.3	0.41	2.4 \pm 0.2	2.4 \pm 0.3	0.98

^aThere were no significant genotype \times treatment interactions.

^bDiets were supplemented with 25 g/d of creatine monohydrate.

^{c,d}Least squares means for the same main effect (genotype or treatment) without a common superscript differ ($P < 0.05$).

Objective color is used as an indicator of consumer acceptability of fresh meats and as a means of quality classification of fresh pork. Significant interactions were found for L*-values for the ST and QD, with a trend found in the SM (Table 5). For the ST and SM, it appears that CMH supplementation improved the L*-values of Nn pigs. This result fits with the higher 45-min pH of the SM for supplemented pigs because objective color is closely related to the pH and drip loss of fresh pork (Warriss and Brown, 1987). Because pH levels were not measured in the ST, it is difficult to determine whether CMH treatment affected the pH decline. It is unlikely that CMH supplementation affected color beyond the influence of pH; this is also evidenced by the lack of treatment effect on color of the LT, which was not affected by CMH supplementation. On the other hand, the QD had a higher L*-value for NN genotypes (unchanged for Nn), with a significant interaction. This increase in

L* is difficult to explain. However, the value is well below the range of PSE, and in fact the untreated QD could actually be considered dark, firm, and dry.

In this study, the loin muscle was consistently pale with mean L*-values greater than 52. There was a nearly significant ($P = 0.055$) genotype effect in the LT with NN being darker than Nn, but the NN was still in a range considered to be pale. Several other studies have found genotype to be an important influence in color development of the longissimus, with Nn genotypes having higher L*-values (Sather et al., 1991; Pommier et al., 1992; Leach et al., 1996). Some other studies have shown no genotype influence on color (De Smet et al., 1996; Sutton et al., 1997).

Muscle Moisture and Fat Content

Moisture and fat content of muscles is shown in Table 6. There were no CMH treatment effects on composition

Table 5. Least squares means (\pm SE) of objective color (L*) of the longissimus thoracis (LT), semimembranosus (SM), biceps femoris (BF), semitendinosus (ST), quadriceps group (QD), triceps brachii (TB), and serratus ventralis (SV) of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^a

Muscle	Genotype				Type ^{b,c}	Treat ^{b,c}	Type \times treat ^{b,c}
	NN		Nn				
	C (n = 8)	S (n = 7)	C (n = 8)	S (n = 6)			
LT	52.8 \pm 0.9 ^x	53.7 \pm 0.9 ^x	55.5 \pm 0.9 ^x	55.2 \pm 1.2 ^x	0.055	0.77	0.55
SM	46.3 \pm 0.9 ^x	47.9 \pm 0.9 ^{xy}	50.5 \pm 0.9 ^y	48.5 \pm 1.2 ^{xy}	0.022	0.86	0.083
BF	45.6 \pm 1.1 ^x	46.9 \pm 1.2 ^{xy}	50.4 \pm 1.1 ^y	49.5 \pm 1.4 ^z	0.004	0.87	0.38
ST	51.2 \pm 1.4 ^x	53.0 \pm 1.4 ^x	57.3 \pm 1.4 ^y	52.1 \pm 1.8 ^z	0.095	0.28	0.032
QD	43.0 \pm 0.9 ^x	47.6 \pm 0.9 ^y	45.7 \pm 0.9 ^x	45.1 \pm 1.2 ^x	0.89	0.061	0.018
TB	42.7 \pm 0.6 ^x	42.3 \pm 0.6 ^x	43.2 \pm 0.6 ^x	43.1 \pm 0.9 ^x	0.36	0.81	0.88
SV	47.6 \pm 1.0 ^x	47.8 \pm 1.0 ^x	49.4 \pm 1.0 ^x	48.6 \pm 1.3 ^x	0.25	0.82	0.68

^aDiets were supplemented with 25 g/d of creatine monohydrate.

^bP-value of differences between main effects or interaction.

^cType refers to genotype and Treat refers to dietary treatment.

^{x,y,z}Within a row, least squares means without a common superscript letter differ ($P < 0.05$).

Table 6. Least squares means (\pm SE) of moisture and fat content of the longissimus thoracis (LT), semimembranosus (SM), biceps femoris (BF), semitendinosus (ST), quadriceps group (QD), triceps brachii (TB), and serratus ventralis (SV) of normal (NN) and halothane carrier (Nn) pigs treated with a control (C) or creatine monohydrate-supplemented (S) diet^a

Muscle trait	Genotype				Type ^{b,c}	Treat ^{b,c}	Type \times treat ^{b,c}
	NN		Nn				
	C (n = 8)	S (n = 7)	C (n = 8)	S (n = 6)			
LT/moisture	70.9 \pm 0.8	71.6 \pm 0.8	71.8 \pm 0.8	72.7 \pm 1.0	0.27	0.37	0.90
LT/fat	6.1 \pm 0.9	5.8 \pm 0.9	5.4 \pm 0.9	4.2 \pm 1.2	0.27	0.44	0.62
SM/moisture	74.2 \pm 0.4	74.0 \pm 0.4	73.7 \pm 0.4	74.7 \pm 0.5	0.83	0.30	0.10
SM/fat	3.2 \pm 0.3	3.5 \pm 0.3	3.0 \pm 0.3	2.7 \pm 0.4	0.17	0.98	0.42
BF/moisture	72.3 \pm 0.6	72.4 \pm 0.6	73.4 \pm 0.6	73.2 \pm 0.8	0.19	0.93	0.82
BF/fat	6.1 \pm 0.8	6.0 \pm 0.8	4.5 \pm 0.8	4.5 \pm 1.0	0.097	0.99	0.97
ST/moisture	71.7 \pm 0.4 ^x	72.3 \pm 0.4 ^y	73.4 \pm 0.4 ^y	73.5 \pm 0.5 ^y	0.003	0.41	0.65
ST/fat	8.0 \pm 0.5 ^x	7.8 \pm 0.5 ^x	5.8 \pm 0.5 ^y	5.4 \pm 0.6 ^y	0.001	0.58	0.81
QD/moisture	74.8 \pm 0.2	74.7 \pm 0.2	75.3 \pm 0.2	75.3 \pm 0.3	0.039	0.84	0.86
QD/fat	3.0 \pm 0.2 ^x	2.8 \pm 0.2 ^{x,y}	2.3 \pm 0.2 ^{y,z}	2.1 \pm 0.3 ^z	0.002	0.31	0.87
TB/moisture	73.2 \pm 0.7	72.4 \pm 0.7	73.9 \pm 0.7	74.5 \pm 0.9	0.062	0.93	0.34
TB/fat	5.3 \pm 0.8	6.4 \pm 0.8	4.6 \pm 0.8	3.9 \pm 1.0	0.088	0.82	0.32
SV/moisture	71.8 \pm 0.4 ^x	71.7 \pm 0.4 ^x	73.3 \pm 0.4 ^y	72.9 \pm 0.6 ^{xy}	0.008	0.62	0.70
SV/fat	7.5 \pm 0.6 ^{xy}	7.9 \pm 0.6 ^x	5.9 \pm 0.6 ^z	5.8 \pm 0.7 ^{yz}	0.005	0.82	0.78

^aDiets were supplemented with 25 g/d of creatine monohydrate.

^bP-value of differences between main effects or interaction.

^cType refers to genotype and Treat refers to dietary treatment.

^{x,y,z}Within a row, least squares means without a common superscript letter differ ($P < 0.05$).

and only a trend ($P = 0.10$) toward a greater moisture content of the supplemented Nn semimembranosus. Genotype did have an effect on many composition traits. Generally, NN muscle had a lower moisture content and a higher fat content. The LT and the SM were the only muscles not affected by genotype for moisture or fat. In general, a 1 to 3% increase in moisture content was observed in Nn muscles, whereas NN muscles had a 2 to 4% higher fat content.

For the ST, QD, TB, and SV, Nn genotypes had higher moisture and lower fat contents. Other studies, focusing on the longissimus, reported similar results with lower fat and higher moisture amounts (Sather et al., 1991). On the other hand, Leach et al. (1996) and De Smet et al. (1996) did not find differences in fat content due to genotype. Warner et al. (1993) compared the relationship of pork muscles within the same carcass to the longissimus. They found that when the longissimus was classified as PSE, the muscles of the ham, with the exception of the rectus femoris, were PSE a large majority of the time, whereas the muscles of the shoulder were not affected. In addition, Nold et al. (1999) found that the muscles of the shoulder tended to have a greater amount of fat and lower drip losses. Because this study did not find differences in the LT, but rather in other muscles not usually examined, it is hard to determine whether genotype had an influence on the composition of the muscles of the shoulder and the quadriceps.

Implications

Methods to improve the lean quality of pork need to be easy, effective, and financially feasible for widespread

use. These data do not provide convincing evidence for the widespread use of creatine monohydrate supplementation to increase the quality of fresh pork of normal and halothane carrier genotypes. There were enough positive data to indicate a need for further study to determine whether a different level or timing of supplementation may be feasible.

Literature Cited

- Aalhus, J. L., D. R. Best, F. Costello, and A. L. Schaefer. 1997. The effect of porcine somatotropin on muscle fiber morphology and meat quality of pigs of known stress susceptibility. *Meat Sci.* 45:283–295.
- AMSA. 1995. Research guidelines for cookery, sensory evaluation, and instrumental tenderness measurements of fresh meat. Natl. Live Stock and Meat Board, Chicago, IL.
- AOAC. 1990. Official Methods of Analysis. 15th ed. Association of Official Analytical Chemists, Arlington, VA.
- Baas, T. J., and J. W. Mabry. 1999. The impact of genetics on pork quality. In: *Pork Facts*. Natl. Pork Producers Council, Des Moines, IA.
- Balsom, P. D., B. Ekblom, and K. Soderlund. 1993. Creatine supplementation and dynamic high-intensity intermittent exercise. *Scand. J. Med. Sci. Sports* 3:143–149.
- Balsom, P. D., K. Soderlund, and B. Ekblom. 1994. Creatine in humans with special reference to creatine supplementation. *Sports Med.* 18(4):268–280.
- Berg, E. P., J. D. Spencer, and G. L. Allee. 1999. Dietary supplementation of creatine monohydrate in swine finishing diets improves fresh pork quality. *J. Anim. Sci.* 77(1):46 (Abstr.).
- Cannon, J. E., J. B. Morgan, J. Heavner, F. K. McKeith, G. C. Smith, and D. L. Meeker. 1995. Pork quality audit: A review of the factors influencing pork quality. *J. Muscle Foods* 6:369–402.
- Cisneros, F., M. Ellis, F. K. McKeith, J. McCaw, and R. L. Fernando. 1996. Influence of slaughter weight on growth and carcass char-

- acteristics, commercial cutting and curing yields, and meat quality of barrows and gilts from two genotypes. *J. Anim. Sci.* 74:925–933.
- DeSmet, S. M., H. Pauwels, S. De Bie, D. I. Demeyer, J. Callewier, and E. Eeckhout. 1996. Effect of halothane genotype, breed, feed withdrawal, and lairage on pork quality of Belgian slaughter pigs. *J. Anim. Sci.* 74:1854–1863.
- Fujii, J., K. Otsu, F. Zorzato, S. De Leon, V. K. Khanna, J. E. Weiler, P. J. O'Brien, and D. H. MacLennan. 1991. Identification of a mutation in porcine ryanodine receptor associated with malignant hypothermia. *Science (Wash DC)* 253:448–451.
- Greenhaff, P. L. 1996. Creatine supplementation: Recent developments. *Br. J. Sports Med.* 30:281–282.
- Ingwall, J. S., C. D. Weiner, M. F. Morales, E. Davis, and F. E. Stockdale. 1974. Specificity of creatine in the control of muscle protein synthesis. *J. Cell Biol.* 63:145–151.
- Klont, R. E., and E. Lambooy. 1995. Effects of preslaughter muscle exercise on muscle metabolism and meat quality studied in anesthetized pigs of different halothane genotype. *J. Anim. Sci.* 73:108–117.
- Klont, R. E., E. Lambooy, and J. G. van Logtestijn. 1993. Effects of preslaughter anesthesia on muscle metabolism and meat quality of pigs of different halothane genotypes. *J. Anim. Sci.* 71:1477–1485.
- Kreider, R. B., M. Ferreira, M. Wilson, P. Grindstaff, S. Plisk, J. Reinardy, E. Cantler, and A. L. Almada. 1998. Effects of creatine supplementation on body composition, strength, and sprint performance. *Med. Sci. Sports Exerc.* 30:73–98.
- Leach, L. M., M. Ellis, D. S. Sutton, F. K. McKeith, and E. R. Wilson. 1996. The growth performance, carcass characteristics, and meat quality of halothane carrier and negative pigs. *J. Anim. Sci.* 74:934–943.
- McCaw, J., M. Ellis, M. S. Brewer, and F. K. McKeith. 1997. Incubation temperature effects on physical characteristics of normal, dark, firm, and dry, and halothane-carrier pork longissimus. *J. Anim. Sci.* 75:1547–1552.
- Nold, R. A., J. R. Romans, W. J. Costello, and G. W. Libal. 1999. Characterization of muscles from boars, barrows, and gilts slaughtered at 100 or 110 kilograms: Differences in fat, moisture, color, water-holding capacity, and collagen. *J. Anim. Sci.* 77:1746–1754.
- NPPC. 2000. Pork Composition and Quality Assessment Procedures. National Pork Producers Council, Des Moines IA.
- O'Quinn, P. R., B. S. Andrews, R. D. Goodband, J. A. Unruh, J. L. Nelssen, J. C. Woodworth, M. D. Tokach, and K. Q. Owen. 2000. Effects of modified tall oil and creatine monohydrate on growth performance, carcass characteristics, and meat quality of growing-finishing pigs. *J. Anim. Sci.* 78:2376–2382.
- Pommier, S. A., A. Houde, F. Rousseau, and Y. Savoie. 1992. The effect of the malignant hypothermia genotype as determined by a restriction endonuclease assay on carcass characteristics of commercial crossbred pigs. *Can. J. Anim. Sci.* 72:973–976.
- Prevost, M. C., A. G. Nelson, and G. S. Morris. 1997. Creatine supplementation enhances intermittent work performance. *Res. Q. Exerc. Sport.* 68:233–240.
- Sather, A. P., S. D. M. Jones, A. K. W. Tong, and A. C. Murray. 1991. Halothane genotype by weight interactions on pig meat quality. *Can. J. Anim. Sci.* 71:645–658.
- Solomon, M. B., T. J. Caperna, R. J. Mroz, and N. C. Steele. 1994. Influence of dietary protein and recombinant porcine somatotropin administration in young pigs: III. Muscle fiber morphology and shear force. *J. Anim. Sci.* 72:615–621.
- Stadler, K. J., J. Maya, L. L. Christian, S. J. Moeller, and K. J. Prusa. 1998. Effects of preslaughter management on the quality of carcass from porcine stress syndrome heterozygous market hogs. *J. Anim. Sci.* 76:2435–2443.
- Sutton, D. S., M. Ellis, Y. Lan, F. K. McKeith, and E. R. Wilson. 1997. Influence of slaughter weight and stress gene genotype on the water holding capacity and protein gel characteristics of three porcine muscles. *Meat Sci.* 46:173–180.
- Warner, R. D., R. G. Kauffman, and R. L. Russell. 1993. Quality attributes of major porcine muscles: A comparison with the longissimus lumborum. *Meat Sci.* 33:359–372.
- Warriss, P. D., and S. N. Brown. 1987. The relationship between initial pH, reflectance and exudation in pig muscle. *Meat Sci.* 20:65–74.
- Williams, M. H., and J. D. Branch. 1998. Creatine supplementation and exercise performance: an update. *J. Am. Coll. Nutr.* 17:216–234.