EFFECTS OF ORAL ADMINISTRATION OR FEEDING OF SODIUM CITRATE OR ACETATE TO PIGS ON POST-MORTEM GLYCOLYSIS, PH DECLINE, AND PORK QUALITY ATTRIBUTES

J. W. Stephens, M. E. Dikeman, J. A. Unruh, M. D. Haub[1], and M. D. Tokach

Summary

Previous studies have shown that citrate has the potential to inhibit phosphofructokinase (PFK), a key enzyme in post-mortem glycolysis. The objective of our study was to determine the effects of oral administration and feeding of citrate or acetate to pigs on post-mortem glycolysis, pH, and pork quality attributes. In Experiment 1, citrate, acetate, or water was orally administered to 30 pigs 45 min before stunning (electric plus captive bolt). In Experiment 2, citrate or acetate was fed to 30 gilts in 454 g of feed 60 min before stunning. Ante-mortem treatment had no effect ($P > 0.05$) on muscle pH or post-mortem concentrations of glycolytic metabolites: glucose-6 phosphate, fructose-6 phosphate, fructose-1,6 bisphosphate, glyceraldehyde-3 phosphate, dihydroxyacetone phosphate, or lactate. Minor, but inconsistent, differences in quality attributes were found in *longissimus* chops and inside and outside *semimembranosus* quality attributes among treatments ($P>0.05$). The reason for the lack of PFK inhibition is not known. Glycolytic-metabolite data indicate that PFK was a main regulatory enzyme in post-mortem muscle.

(Key Words: Pork, pH, Glycolysis, Citrate, Acetate.)

Introduction

Attributes that define pork quality include color, firmness, wetness, and marbling, and are of great economic importance as they affect consumer appeal, eating satisfaction, and repeat purchases. Muscle color, firmness, and wetness are dependent on the reactions of glycolysis and pH decline during the onset of rigor. Anaerobic glycolysis, which occurs in post-mortem muscle, produces lactate and hydrogen ions. These hydrogen ions accumulate in muscle and reduce pH. If this reaction occurs at an accelerated rate before adequate chilling, the combination of low pH and high temperature has the potential to denature muscle proteins, resulting in pale color, softness, and diminished water-holding capacity.

Glycolysis is regulated by the enzyme phosphofructokinase (PFK) in post-mortem muscle. This enzyme catalyzes the reaction that transfers a phosphate group from adenosine triphosphate (ATP) to fructose-6 phosphate (F6P) to form fructose-1,6 bisphosphate and adenosine diphosphate (ADP). Although ATP is a substrate for this reaction and is the source of the transferred phosphate, it also serves as an inhibitor of the enzyme. In live animals, excess ATP indicates excess energy; therefore, ATP inhibits PFK, slowing glycolysis and, ultimately, ATP production. High

[1]Department of Human Nutrition.

concentrations of ADP in the cell indicate a lack of energy, thus ADP activates PFK.

Citrate is produced in the body from Acetyl CoA and oxaloacetate as the first step of the aerobic citric-acid cycle, and has been shown to inhibit PFK in mammalian muscle. Citrate inhibits PFK by binding directly to PFK and decreasing the enzyme's affinity for its substrate, F6P, and its activator, ADP. Citrate also increases the enzyme's affinity for ATP at the substrate site and, more importantly, the inhibitory site.

In preliminary investigations in pigs, we found that oral administration of 0.75 g/kg of body weight of citrate or acetate before handling increased post-handling blood pH. Because oral administration of citrate or acetate is not feasible in an industry setting, administering the glycolytic inhibitors through feed was also studied. The objective of our study was to determine the effects of oral administration or feeding of sodium citrate or acetate to pigs on post-mortem glycolysis, pH decline, and pork quality attributes.

Procedures

Preliminary Study. Two groups of 12 finishing pigs were blocked by weight and assigned to citrate, acetate, or control treatments. The pigs were fasted for no less than 20 h. Pigs were restrained with a snout snare and given their ante-mortem treatments by placing a tube through the mouth and down the esophagus. Pigs assigned to citrate and acetate treatments were given 0.75 g/kg BW of sodium citrate or sodium acetate in a 3-to-1 solution of distilled water, followed by 60 ml of water to flush out the tube. Control pigs were given 180 ml of distilled water. Pigs were allowed to rest for 30 min, and then were moved at a trotting pace up and down the aisles in their barn (approximately 100 m).

Forty-five minutes after treatment was ingested, the pigs were snared and bled via the anterior *vena cava* into a heprinized vacuum tube. Tubes were stored on ice for no more than 4 h before being analyzed for pH and lactate. Citrate and acetate treatments tended ($P = 0.18$) to increase blood pH. One h and 15 min after treatment, the pigs were bled again, and the blood parameters were similar among treatments.

Experiment 1. Two replications (one of 15 gilts and one of 5 gilts and 10 barrows) were fed a standard finishing diet containing 20 ppm ractopamine 14 d before harvest. Pigs were assigned by weight to ten groups of three. Pigs in each weight group were assigned to citrate (CIT), acetate (ACE), or control (CON) ante-mortem treatments. Before harvest, the pigs were fasted for no less than 20 h and transported to the Kansas State University Meat Laboratory. Final weights ranged from 230 to 330 lb. At 45 min before harvest, pigs were restrained with a snout snare and were given their ante-mortem treatments as described for the preliminary study. Pigs were harvested in random order within their weight group.

Experiment 2. Two replications of 15 gilts were fed a standard finishing diet containing 20 ppm ractopamine for 13 d before slaughter. Slaughter weights ranged from 235 to 305 lb. Gilts were fasted for no less than 20 h, were weighed, and were grouped by weight into 10 groups of three. Treatments (ACE, CIT, and CON) were assigned randomly to the gilts in each weight group. One h before harvest, gilts were fed 1 lb of feed containing 0.34 g / lb BW of either CIT or ACE. To counteract the salty taste of the treatments, 0.34 g of the artificial sweetener Sucram (Prince Agri. Products, Quincy, IL) was added to the feed. Control pigs received feed with Sucram, but no glycolytic inhibitor. The gilts harvested on the first replication were reluctant to eat the saltier

flavor of treated feed in the unfamiliar surroundings of the meat laboratory. Therefore, salt was gradually added to the feed for pigs in the second replication for 8 d before slaughter to familiarize them with the salty taste.

Harvest. Immediately before harvest, pigs were restrained with a snout snare and bled via the anterior *vena cava* into a heprinized vacuum tube. Tubes were stored on ice for no more than 4 h before being analyzed for pH and lactate. Bicarbonate concentrations also were calculated.

After blood samples were taken, pigs were gently moved to the abattoir, stunned by using an electric stunning wand and, in Experiment 1, stunned again by using a captive bolt stunner. Pigs then proceeded through the slaughter process according to normal procedures. After final inspection and wash, carcasses were tagged for identification, and a temperature logger was placed in the inside ham (*semimembranosus*; SM). The probe was positioned so that tip of the probe was 15 cm from the inside ham surface. The data loggers were launched to read temperature every 2.4 min for 24 h.

At 20 min post-mortem, a sample (approximately 1-inch-thick chop) was removed from the anterior portion of *longissimus* (LM) muscle (immediately posterior to the *scapula*), cut into cubes, and frozen in liquid nitrogen. This sample was used to analyze pre-rigor pH, glucose-6 phosphate (G6P), fructose-6 phosphate (F6P), fructose-1,6 bisphosphate (F16BP), combination of glyceraldehyde-3 phosphate and dihydroxyacetone phosphate (GAPDAP), and lactate.

At 24 h (Experiment 1) and 48 h (Experiment 2) post-mortem, carcasses were fabricated into loins and hams. Two chops (1 inch thick) were removed from the LM; one chop was allowed to bloom for no less than 30 min before visual evaluation. Three trained panelists evaluated each chop for color, firmness, and wetness. Color was evaluated on a six-point scale according to official color standards from the National Pork Producers Council (1 = lightest and 6 = darkest). Firmness and wetness were evaluated separately on three-point scales (1 = softest and wettest and 3 = firmest and driest). Chops were evaluated in random order within weight groups. The SM was excised from the ham, and a slice (1 inch thick) was removed. Ham slices were allowed to bloom for no less than 30 min before visual panel evaluation. Inside and outside sections of the SM were evaluated separately for color, firmness, and wetness as discussed for LM chops. After visual evaluation, L*, a*, and b* were measured on the chops and ham slices. The second loin chop was vacuum packaged until used for pH and expressible-moisture analysis. The inside and outside sections of the SM slices were separated with a knife, vacuum packaged, and held at 1°C for pH and expressible-moisture analysis.

Statistical Analysis. Blood parameters were analyzed in a randomized, complete-block design, with weight group as the block. Temperature measurements were analyzed in a randomized block design with repeated measures, and weight group as the block. Prerigor pH and glycolytic metabolites were analyzed in a split-plot design, with ante-mortem treatment as the whole plot and time post-mortem as the sub-plot. The whole plot was blocked by weight group. Instrumental color, expressible moisture, and ultimate pH were analyzed in a randomized, complete-block design, with weight group as the block. Ante-mortem treatment and time post-mortem were the fixed effects, and weight group was the random effect. Visual color, firmness, and wetness were analyzed in a randomized, complete-block design, with panelist as the block. Ante-mortem treatment was again the fixed effect; panelist was the random effect. Data

were analyzed by using PROC MIXED and means were separated by using the PDIFF test when $P<0.05$.

Results and Discussion

Blood Parameters. Mean values for blood pH, bicarbonate, and lactate concentration 45 min after oral administration (Experiment 1) and 60 min after feeding (Experiment 2) of CIT, ACE, or control are presented in Table 1. In Experiment 1, CIT or ACE ingestion did not affect blood pH level or bicarbonate concentration. Lactate concentration in pigs orally administered CIT was greater ($P<0.05$) than in controls, but ACE-treated pigs did not differ ($P>0.05$) from control or CIT-treated pigs. In Experiment 2, blood pH level and bicarbonate and lactate concentrations were not affected ($P>0.05$) by treatment with ACE or CIT. In our preliminary studies of finishing pigs, we showed an alkalizing effect on blood by both CIT and ACE. CIT is an alkaline substance that has been shown to increase blood pH, bicarbonate, and lactate in humans. In Experiment 1, CIT-treated pigs had elevated lactate concentrations, but pH was not affected by CIT or ACE in either experiment. High lactate concentrations were likely a function of the animals maintaining homeostasis and regulating blood pH.

Post-mortem Glycolysis and Pre-rigor pH. Ante-mortem treatment did not effect concentrations of glycolytic metabolites during post-mortem glycolysis (Tables 2 and 3) or pre-rigor pH ($P>0.05$; Table 4) in either Experiment 1 or 2. Time post-mortem did affect ($P<0.05$) muscle pH. In Experiment 1, pH was highest ($P<0.05$) at 20 min post-mortem. Mean values of pH were higher ($P<0.05$) at 45 min than at the remaining times post-mortem, but pH values at 3-, 6-, 12-, and 24-h post-mortem did not differ ($P>0.05$). In Experiment 2, pH decreased ($P<0.05$) with increasing post-mortem time until 12 h. This pH decline was expected and is consistent with lactate accumulation. In both experiments, temperature declined over time, and did not differ ($P > 0.05$) among treatments (data not shown).

Ingestion of CIT before exercise has been shown to increase blood pH and improve performance in human athletes, but it has also been shown to have no effect on exercise performance. Exercise physiologists theorize that athletes with more alkaline conditions in their blood during exercise have enhanced performance over athletes with neutral and acidotic blood conditions. The extreme effects of slaughter of our pigs on the body were assumed to be similar to exhaustive anaerobic exercise, but neither CIT nor ACE administration affected post-mortem muscle pH conditions in our study.

Citrate has another effect on metabolism; it is an inhibitor of PFK in muscle, thus having the potential to inhibit glycolysis and post-mortem pH decline. In rats, it has been shown that acetate ingestion increases post-mortem muscle CIT concentrations and decreases PFK activity. It is thought that ACE is converted to CIT in the body by CIT synthase, and CIT inhibits glycolysis. Nonetheless, neither CIT nor ACE altered concentrations of glycolytic metabolite, compared with CON, whether administered orally or added to feed.

PFK Over Time. Changes in glycolytic-metabolite concentrations over time were similar for Experiments 1 and 2. Concentrations of G6P and F6P increased ($P<0.05$) as post-mortem time increased (Tables 2 and 3); F16BP and GAPDAP decreased ($P<0.05$) with post-mortem time. Lactate is the final product of glycolysis, and it accumulated ($P<0.05$) with time. It is thought that PFK plays a major role in regulation of post-mortem glycolysis, because metabolites that serve as substrates for PFK accumulate in post-mortem muscle, and PFK products (with

the exception of lactate) decrease with time. Our glycolytic-metabolite results are in agreement with the previous research: G6P and F6P increased with post-mortem time, whereas F16BP and GAPDAP decreased. Lactate is the final product of post-mortem glycolytic reactions, and it increased with the onset of rigor in our study.

Pork Quality Traits. Mean values for visual evaluations, instrumental color, expressible moisture, and ultimate pH for the LM and inside and outside SM are presented in Tables 5 and 6. In Experiment 1, visual color scores for the LM chops from ACE-treated pigs were higher ($P<0.05$) than those from CON; scores from chops from pigs treated with CIT were intermediate. Loin chops from ACE-treated pigs also had higher ($P<0.05$) scores for wetness (drier) than those from CIT and CON carcasses. Firmness scores did not differ ($P>0.05$) among treatments. Chops from CIT-treated pigs were lighter (larger L* values; $P<0.05$) and more yellow (larger b* values, $P<0.05$) than chops from control or ACE-treated pigs were. Chops from CIT-treated pigs also had larger ($P<0.05$) a* values (more red) than chops from control pigs had. Chops from ACE-treated pigs had intermediate values. Expressible-moisture and ultimate pH values did not differ ($P>0.05$) among treatments for LM chops.

In Experiment 2, chops from CIT-fed pigs were softer and wetter ($P<0.05$) than those from CON and ACE-fed pigs (Table 6). Chops from CIT-fed pigs also had lower ($P<0.05$) ultimate pH values than those from CON and ACE-fed pigs. Nevertheless, visual color, instrumental color, and expressible-moisture data in the LM were not affected ($P>0.05$) by ante-mortem feeding of CIT or ACE.

Pork quality attributes of the inside and outside SM were unaffected ($P>0.05$) by ante-mortem oral administration or feeding of ACE or CIT. This large ham muscle was studied because of the large variation in temperature. The outer section of the SM is closer to the surface of the carcass, due to the splitting process, and chills faster than the inner portion. The inner portion, located deep within the ham, is slower to chill, and is more susceptible to protein denaturation than the outer section because of the higher temperatures. Citrate and ACE were expected to slow post-mortem glycolysis in the inside SM, allowing it to chill before the pH dropped to a protein-denaturing level.

Neither CIT nor ACE were effective at inhibiting post-mortem glycolysis or slowing pH decline and, consequently, did not positively affect pork quality traits.

The reason that CIT and ACE did not effectively inhibit pH decline and did not improve meat quality is not known. Perhaps our preliminary data were misinterpreted in that the peak of blood pH should not have been the goal. If CIT was having maximal effects on the blood, it may not have had time to cross the membrane into the muscle. Furthermore, exsanguinations may have removed most of the CIT and ACE in the blood before it had time to have an effect in muscle. Perhaps more time should have been allowed between CIT or ACE administration and stunning.

The stressful handling during bleeding and the strange surroundings associated with the pre-slaughter protocol might have had an effect on the pigs' metabolism. Moreover, pigs in Experiment 1 were stunned with a captive-bolt stunner, which induced excessive kicking during exsanguination. This extra activity may have caused the pigs to metabolize glucose stores while the blood system was still intact to remove excess lactate and to buffer pH changes. Also, the effects of slaughter on the body may be so extreme that any effect that

glycolytic inhibitors may have on a living system are overwhelmed by the severity of loss of homeostasis, loss of blood, and rigor onset.

In conclusion, neither ante-mortem oral administration of citrate or acetate at 45 min before harvest nor feeding them 60 min before harvest were effective at inhibiting post-mortem glycolysis, altering pre-rigor pH, or improving pork quality. More research should be conducted to determine if earlier administration would improve their effects. Phosphofructokinase was the rate-limiting step in post-mortem glycolysis in pork, but the length of time that the rate-limiting effects last is questionable when pigs are fed before slaughter. Glycolytic inhibitors may have potential to affect post-mortem metabolism in pork, but they must reach the enzyme in time to be effective, and the inhibition must be accomplished early post-mortem.

Table 1. Mean Values For Blood pH and Bicarbonate and Lactate Concentrations of Pigs 45 min after Oral Administration (Experiment 1) and 60 min after Feeding (Experiment 2) of Sodium Citrate, Sodium Acetate, or Control Diets

	Acetate	Citrate	Control	S.E. [a]
Experiment 1				
pH	7.48	7.47	7.48	0.01
Bicarbonate (mmol/L)	37.36	37.08	37.31	0.65
Lactate (mmol/L)	2.51[bc]	3.36[b]	1.51[c]	0.45
Experiment 2				
pH	7.48	7.46	7.46	0.02
Bicarbonate (mmol/L)	34.84	33.97	34.00	1.09
Lactate (mmol/L)	2.54	2.99	3.96	0.49

[a]Largest standard errors for ante-mortem treatments.
[bc]Means lacking a common superscript letter differ ($P<0.05$).

Table 2. Mean Values and Standard Errors of Glycolytic Metabolite Concentrations from the *Longissimus*, Removed at 45 min and 3 and 12 h Post-mortem, from Pigs Given Sodium Citrate, Sodium Acetate, or Water as Control 45 min Ante-mortem (Experiment 1)

Metabolite	Treatment	45 min	3 h	12 h	Mean	S.E.[d]
Glucose-6 Phosphate (μmole/g)						
	Acetate	4.88	6.72	8.16	6.59	0.42
	Citrate	4.84	7.80	8.15	6.93	0.42
	Control	4.48	6.92	7.43	6.27	0.42
	Mean	4.73[c]	7.15[b]	7.91[a]		
	S.E.[e]	0.29	0.29	0.29		
Fructose-6 Phosphate (μmole/g)						
	Acetate	0.73	1.03	0.89	0.89	0.05
	Citrate	0.71	0.89	0.92	0.84	0.05
	Control	0.73	0.89	0.86	0.83	0.05
	Mean	0.73[b]	0.94[a]	0.89[a]		
	S.E.[e]	0.04	0.04	0.04		
Fructose-1,6 Bisphosphate (nmole/g)						
	Acetate	90.8	23.9	14.4	43.0	5.6
	Citrate	86.7	22.1	18.8	42.5	5.7
	Control	85.0	6.7	15.9	35.9	5.7
	Mean	87.5[a]	17.6[b]	16.4[b]		
	S.E.[e]	5.3	5.2	5.2		
Glyceraldehyde-3 Phosphate and Dihydroxyacetone Phosphate (nmole/g)						
	Acetate	38.6	11.1	15.9	21.9	3.4
	Citrate	46.3	11.0	18.8	25.4	3.4
	Control	33.6	6.7	8.9	16.4	3.4
	Mean	39.5[a]	9.6[b]	14.5[b]		
	S.E.[e]	3.0	3.0	3.0		
Lactate (μmole/g)						
	Acetate	9.28	11.37	11.85	10.83	0.34
	Citrate	9.26	11.58	11.44	10.76	0.34
	Control	9.33	12.75	11.45	11.18	0.34
	Mean	9.29[b]	11.90[a]	11.58[a]		
	S.E.[e]	0.34	0.34	0.34		

[abc]Means for post-mortem time means of metabolites lacking common superscript letters differ ($P<0.05$).
[d]Standard error of the ante-mortem treatment main-effect means.
[e]Standard error of the post-mortem time main-effect means.

Table 3. Mean Values and Standard Errors of Glycolytic Metabolite Concentrations from the *Longissimus*, Removed at 45 min and 3 and 12 h Post-mortem, from Pigs Fed Sodium Citrate, Sodium Acetate, or a Control Diet at 60 min Ante-mortem (Experiment 2)

Metabolite	Treatment	45 min	3 h	12 h	Mean	S.E.[d]
Glucose-6 Phosphate (µmole/g)						
	Acetate	3.92	6.37	8.68	6.32	0.32
	Citrate	4.18	7.79	9.22	7.06	0.33
	Control	4.02	7.29	8.28	6.53	0.31
	Mean	4.04[c]	7.15[b]	8.73[a]		
	S.E.[e]	0.32	0.33	0.32		
Fructose-6 Phosphate (µmole/g)						
	Acetate	0.70	1.14	1.53	1.12	0.07
	Citrate	0.83	1.49	1.52	1.28	0.07
	Control	0.76	1.35	1.40	1.17	0.07
	Mean	0.76[b]	1.32[a]	1.48[a]		
	S.E.[e]	0.07	0.07	0.07		
Fructose-1,6 Bisphosphate (nmole/g)						
	Acetate	89.4	67.8	19.5	58.9	6.3
	Citrate	75.1	58.7	19.6	51.1	6.4
	Control	78.9	68.8	11.1	53.0	6.1
	Mean	81.1[a]	65.1[b]	16.7[c]		
	S.E.[e]	5.6	5.8	5.6		
Glyceraldehyde-3 Phosphate and Dihydroxyacetone Phosphate (nmole/g)						
	Acetate	66.6	32.7	22.7	40.7	5.9
	Citrate	43.3	31.9	25.6	33.6	5.9
	Control	44.9	36.3	19.9	33.7	5.7
	Mean	51.6[a]	33.6[b]	22.7[b]		
	S.E.[e]	5.2	5.5	5.4		
Lactate (µmole/g)						
	Acetate	6.94	10.67	12.83	10.15	0.33
	Citrate	7.41	10.59	12.03	10.01	0.34
	Control	6.92	10.66	11.96	9.85	0.32
	Mean	7.09[c]	10.64[b]	12.27[a]		
	S.E.[e]	0.32	0.33	0.32		

[abc]Means for post-mortem times of metabolites with different superscript letters differ ($P<0.05$).
[d]Standard errors for the ante-mortem treatment main-effect means.
[e]Standard errors for time main-effect means.

Table 4. Mean Values and Standard Errors of Pre-rigor pH from *Longissimus* Muscle, Removed at 45 min and 3 and 12 h Post-mortem, from Pigs 45 min after Oral Administration (Experiment 1) and 60 min after Feeding (Experiment 2) of Sodium Citrate, Sodium Acetate, or Controls

Treatment	20 min	45 min	3 h	6 h	12 h	24 h	Mean	S.E.[f]
Experiment 1								
Acetate	6.17	5.92	5.53	5.50	5.50	5.46	5.68	0.04
Citrate	6.21	5.94	5.47	5.44	5.45	5.39	5.65	0.04
Control	6.20	5.95	5.39	5.41	5.44	5.44	5.64	0.04
Mean	6.19[a]	5.94[b]	5.46[c]	5.45[c]	5.46[c]	5.43[c]		
S.E. [g]	0.03	0.03	0.03	0.03	0.03	0.03		
Experiment 2								
Acetate	6.39	6.32	5.84	5.55	5.53	5.43	5.84	0.03
Citrate	6.40	6.19	5.71	5.52	5.46	5.41	5.78	0.03
Control	6.36	6.27	5.76	5.54	5.41	5.47	5.80	0.02
Mean	6.38[a]	6.26[b]	5.77[c]	5.54[d]	5.47[e]	5.43[e]		
S.E. [g]	0.02	0.02	0.02	0.02	0.02	0.02		

[abcde]Means for post-mortem time means lacking a common superscript letter differ ($P<0.05$).
[f]Standard error of the ante-mortem treatment main-effect means.
[g]Standard error of the post-mortem time main-effect means.

Table 5. Visual Evaluations, Instrumental Color, Expressible Moisture, and Ultimate pH Measurements of *Longissimus,* Inside and Outside *Semimembranosus* Muscles from Pigs Treated 45 min Ante-mortem with Sodium Acetate, Sodium Citrate, or a Water Control (Experiment 1)

Muscle		Ante-mortem Treatment			
		Acetate	Citrate	Control	S.E.[c]
Longissimus					
	Color[a]	2.91[y]	2.73[yz]	2.55[z]	0.08
	Firmness[b]	2.28	2.13	2.15	0.11
	Wetness[b]	2.38[y]	2.08[z]	2.11[z]	0.11
	L*	56.41[z]	60.34[y]	57.68[z]	0.84
	a*	3.36[yz]	4.11[y]	2.57[z]	0.43
	b*	12.36[z]	13.53[y]	12.18[z]	0.44
	Exp. moisture	21.34	23.75	23.34	1.03
	Ultimate pH	5.53	5.38	5.53	0.05
Inside *Semimembranosus*					
	Color	2.59	2.53	2.73	0.15
	Firmness	1.98	2.00	2.02	0.09
	Wetness	2.03	2.05	2.08	0.13
	L*	57.14	56.82	58.24	1.09
	a*	7.05	7.36	6.96	0.61
	b*	15.48	15.70	15.50	0.50
	Exp. moisture	22.48	20.13	19.81	0.89
	Ultimate pH	5.59	5.58	5.60	0.04
Outside *Semimembranosus*					
	Color	2.98	2.91	3.08	0.38
	Firmness	2.26	2.22	2.32	0.11
	Wetness	2.30	2.17	2.27	0.15
	L*	55.75	55.97	56.07	0.93
	a*	5.20	5.17	4.73	0.53
	b*	14.32	14.64	14.21	0.51
	Exp. moisture	20.98	21.66	20.48	1.18
	Ultimate pH	5.64	5.60	5.62	0.03

[a]Color was evaluated on a six-point scale according to official color standards from the National Pork Producers Council (1 = lightest and 6 = darkest).
[b]Firmness and wetness were evaluated separately on three-point scales (1 = softest and wettest and 3 = firmest and driest).
[c]Standard error of the means.
[yz]Means, within a row, lacking common superscript letters differ ($P<0.05$).

Table 6. Visual Evaluations, Instrumental Color, Expressible Moisture, and Ultimate pH Values of *Longissimus,* Inside and Outside *Semimembranosus* Muscles from Pigs Fed Feed Containing Sodium Acetate, Sodium Citrate, or a Control Diet at 60 min Ante-mortem (Experiment 2)

		Ante-mortem Treatment			
Muscle		Acetate	Citrate	Control	S.E.[c]
Longissimus					
	Color[a]	3.73	3.56	3.77	0.17
	Firmness[b]	2.53[y]	2.20[z]	2.45[y]	0.13
	Wetness[b]	2.34[y]	1.97[z]	2.38[y]	0.18
	L*	52.32	53.48	54.11	0.87
	a*	3.32	3.43	3.07	0.38
	b*	11.58	12.10	11.94	0.44
	Exp. moisture	23.43	25.12	23.84	1.39
	Ultimate pH	5.56[y]	5.43[z]	5.54[y]	0.05
Inside *Semimembranosus*					
	Color	3.58	3.26	3.57	0.30
	Firmness	2.01	1.91	2.10	0.10
	Wetness	2.15	1.98	2.10	0.14
	L*	54.97	56.44	55.77	1.40
	a*	7.51	7.93	7.57	0.46
	b*	15.73	16.15	15.85	0.54
	Exp. moisture	18.86	20.13	20.74	1.36
	Ultimate pH	5.50	5.46	5.53	0.04
Outside *Semimembranosus*					
	Color	3.20	3.00	2.98	0.35
	Firmness	2.50	2.52	2.55	0.11
	Wetness	2.39	2.20	2.37	0.19
	L*	54.05	55.15	54.74	0.58
	a*	4.41	5.45	4.68	0.30
	b*	13.78	14.50	13.96	0.33
	Exp. moisture	16.17	19.13	19.49	1.09
	Ultimate pH	5.54	5.46	5.51	0.04

[a]Color was evaluated on a six-point scale according to official color standards from the National Pork Producers Council (1999, 1 = lightest and 6 = darkest).
[b]Firmness and wetness were evaluated separately on three-point scales (1 = softest and wettest and 3 = firmest and driest).
[c]Largest standard error for the means.
[yz]Means, within a row, lacking common superscript letters differ ($P<0.05$).