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The Impact of Carcass Size, Chilling Conditions, and Electrical Stimulation on Beef Postmortem Temperature and pH Decline

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Abstract: Variability in carcass size can influence carcass chilling rates, which could result in issues associated with beef tenderness and color. Moreover, the usage of electrical stimulation can affect postmortem metabolism and meat quality. However, few studies have looked at the combined impact of chilling and electrical stimulation on temperature decline, postmortem biochemistry, and color among the current population of US beef. Beef carcasses ($N = 162$, <30 mo) were randomly selected from 2 beef processing plants. One side of each carcass was electrically stimulated, while the opposing side was not electrically stimulated. Matched sides were subjected to either conventional spray chilling (CONV) or delayed spray chilling (DELAY). Deep tissue (10.5 cm under the pelvic bone) and surface temperature (1.5 cm under the loin fat) were continuously monitored during chilling in addition to temperature and pH measurements from the *semimembranosus* (SM), *longissimus lumborum* (LL), and *psoas major* (PM) muscles at an initial time (45 to 60 min), 6 h, 12 h, and after chilling (28 to 36 h) postmortem. Further, the instrumental (L^*, a^*, and b^*) and visual color were evaluated on 14-d aged PM steaks. For data analysis, carcasses with hot carcass weights above or below the plant average were considered heavy or light, respectively. A nonlinear regression model was fitted to the continuous deep and surface temperatures, whereas other parameters were evaluated using a mixed model. Electrical stimulation improved L^* (lightness; $P < 0.05$) of PM in lightweight carcasses but not ($P > 0.05$) in heavyweight carcasses. Temperature decline was faster ($P < 0.05$) and pH decline slower ($P < 0.05$) in the SM and LL of lightweight carcasses under CONV compared to lightweight carcasses under DELAY and heavyweight carcasses under CONV and DELAY. Exponential decay models for deep and surface temperatures indicated the rate of cooling differed ($P < 0.05$) due to the combination of treatment factors. Heavyweight carcasses in DELAY had slower rates of temperature decline ($P < 0.05$). Overall, the variability in carcass size affected temperature decline and postmortem metabolism. Therefore, postmortem management practices should consider carcass weights to optimize meat quality.

Key words: beef, carcass size, chilling, electrical stimulation, nonlinear regression model, temperature decline
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Introduction

Over the last few decades, beef carcass weight has increased continuously with the range of hot carcass weight becoming wider (Lorenzen et al., 1993; Boleman et al., 1998; McKenna et al., 2002; Garcia et al., 2008; Moore et al., 2012; Boykin et al., 2017). Increased carcass weight can contribute to beef tenderness and muscle color issues, as beef packers struggle to chill beef carcasses of varying sizes more uniformly. Specifically, the large beef processing facilities have struggled to achieve acceptable deep tissue (center of the round or chuck) temperatures in heavyweight carcasses before fabrication (Maples et al., 2018). Additionally, foodservice and retail customers of beef have indicated problems

related to variation in the tenderness of top (inside) round (Guelker et al., 2013; Martinez et al., 2017) and color of the tenderloin (Seyfert et al., 2006; Mancini et al., 2018; Najar-Villarreal et al., 2021). Even though the industry has gradually moved away from very rapid chilling times over the past 10 to 15 years, with a transition from a 24 h chill to 30 to 48 h chill periods, packers are intensifying chilling conditions to more adequately chill larger beef carcasses. Quality variation in the commercial beef industry as related to carcass chilling could result in quality issues such as heat shortening, cold shortening, and heat ring.

In general, within the same commercial processing plant, carcasses are subjected to the same chilling conditions, regardless of size and weight. Keane and Allen (1998) reported that with increasing weight at slaughter, beef cattle's subcutaneous and intramuscular fat content has also increased. As fat can act as an insulating barrier (Aalhus et al., 2001) and the decreased surface area relative to the carcass mass can reduce chilling rates, significant differences in cooling rates result from chilling carcasses of varying sizes and fatness under similar conditions. Consequently, there are differences in the rate of heat dissipation and pH decline, which could explain some variability in tenderness and color of beef. In support, Lancaster et al. (2020) reported that carcass size impacted the chilling rate and pH decline of top round muscles.

Electrical stimulation is one of the postharvest management practices used to improve beef tenderness and reduce the incidence of cold shortening. Previous research has indicated that both high and low-voltage electrical stimulation, when combined with appropriate chilling practices, improve beef tenderness and muscle color (Smith et al., 2008). However, few published studies have examined the impact of chilling conditions and electrical stimulation among the current cattle population in the industry, including the heavier carcasses. Therefore, the objective of this study was to evaluate the impact of carcass size, chilling conditions, and electrical stimulation on temperature decline, pH decline, and lean color development in beef carcasses.

Material and Methods

Carcass selection and treatment application

Data were collected from 2 large-scale, commercial processing plants (Plant A and Plant B) in the United States. The last 10 to 12 carcasses in 15 separate plant lots were included in the study ($N = 162$). Each carcass's left or right side was electrically stimulated (ES), and the opposite side was not electrically stimulated (NES). Electrical stimulation parameters were unique to each packing plant. In Plant A, the carcass sides designated for stimulation received pulses of high voltage electrical stimulation applied exclusively to the middle meats of the carcass, whereas, in Plant B, carcass sides designated for stimulation received pulses of low-voltage electrical stimulation applied exclusively to the middle meats of the carcass. Following ES treatment, sides from the same carcass were matched back together, and the pair (carcass) was assigned to 1 of 2 chilling conditions: (1) conventional chilling with immediate spray chill (CONV) or (2) chilling with delayed spray chill application (DELAY). For the DELAY chilling conditions, the spray chilling was not applied to the carcasses for the first 8 h following harvest in Plant A and was not applied for the first 6 h in Plant B. For both plants, normal spray chilling consisted of cyclic intermittent (for 30 s, every 10 min) application of chilled ($4°C \pm 2$) water for the first 12 h postharvest (air velocity 2 m/s).

Carcass temperature monitoring and muscle sample collection

Once the carcasses reached the chilling coolers (30 to 45 min postmortem for Plant A, and 60 to 75 min in Plant B), deep tissue and surface temperatures were continuously monitored and recorded (every 20 s) for selected carcasses using a multiuse temperature recorder (Multitrip Green, Temprecord International Limited, Auckland, New Zealand) until chilling was completed (according to each plant's protocol). Deep tissue temperature was measured by inserting the temperature probe tip (10.5 cm) into the muscle under the pelvic bone. The surface temperature was recorded 1.5 cm beneath the fat surface at the 13th rib by inserting a temperature probe parallel with the outer surface of the carcass. Temperature recorders were removed from the carcass sides immediately before they were presented for grading at approximately 28 h postmortem for Plant A and 36 h postmortem for Plant B.

The *longissimus lumborum* (LL), *psoas major* (PM), and *semimembranosus* (SM) were used to represent muscles of different glycolytic rates (Hunt and Hedrick, 1977; Kirchofer et al., 2002). For each carcass side, using a coring tool fitted to an electric drill, 1 cm × 7.5 cm cores were removed at 4 time intervals (1 h, 6 h, 12 h, 36 h) postmortem from the LL, PM, and SM. Each muscle (LL, PM, SM) was virtually divided into 4 sections of

equal length, and the order of sampling (within carcass) was randomly assigned to the 4 time intervals. The muscle cores were immediately frozen in dry ice (carbon dioxide) to stop glycolytic processes and pH decline and were kept frozen (−80°C) until further analysis. Additional temperature measurements on the SM, PM, and LL were performed using a handheld AquaTuff 351 Wrap&Stow thermometer equipped with a Dura-Needle probe (Cooper-Atkins Corp., Middlefield, CT) inserted into the geometric center of the muscle (5 cm deep and perpendicular to the muscle surface) during the sample collection (1 h, 6 h, 12 h, 36 h).

Determination of pH

The pH was determined for muscle cores from every carcass at the previously described time intervals. The pH of the individual muscle cores was determined by the method described by Bendall (1973). Briefly, 1 g of frozen muscle sample (1 ± 0.1 g) was rapidly homogenized using a household blender from Oster (Boca Raton, FL) in a 10 mL solution of deionized water, sodium iodoacetate (5 mM), and potassium chloride (150 mM). Since the ultimate pH of each of the samples may not have been reached at the time of sampling and freezing, sodium iodoacetate and potassium chloride were used to prevent further pH decline during sample processing. The pH of the homogenate was measured using a calibrated tabletop pH meter equipped with an electrode (accumet model 13-620-285, Fisher Scientific, Pittsburgh, PA).

Color measurements

Objective color measurements were obtained for PM steak surface from each carcass side. Following carcass chilling and grading procedures, the short loins (NAMP 174) from paired carcass sides were collected ($N = 162$) at the time of carcass fabrication, individually vacuum packaged, boxed, and transported in a refrigerated truck (0°C to 2°C) to the Colorado State University Meat Laboratory. The short loins were stored at 2°C for 14 d postmortem aging upon arrival. Following aging, short loins were removed from their packaging, and the whole tenderloin (PM) was removed, and a single 2.5-cm steak was cut from the most posterior portion and bloomed for 30 min. Six panelists trained for color evaluation scored steaks on an 8-point color scale where 1 = light pink; 2 = pinkish red; 3 = reddish-pink; 4 = light cherry red; 5 = cherry red; 6 = dark cherry red; 7 = dark red; 8 = very dark red; and 9 = purplish red. Following panel color assessment, instrumental color measurements (Commission Internationale de l´Eclairage $L^*a^*b^*$) were taken using a portable spectrophotometer equipped with a 6-mm aperture, illuminant A, and 10° standard observer (MiniScan EZ; HunterLab, Reston, VA). The color was measured at 3 random locations on the bloomed surface of PM steaks and averaged for each steak.

Statistical analysis

Before analysis, carcasses were grouped into heavy and lightweight categories based on the average weight of the sample population in each plant. Carcasses with hot carcass weight above or below the average were considered heavyweight or lightweight, respectively. In Plant A, the average hot carcass weight was 405.45 kg, whereas in Plant B, the average hot carcass weight was 383.57 kg.

The treatment structure was a $2 \times 2 \times 2$ factorial with main effects of carcass size, chilling, electrical stimulation, and their interactions with time postmortem included in the model. Animal identity (ID) and electrical stimulation within animal ID (ID*ES) were included as random effects. Effects of carcass size (heavy vs. light), chilling conditions (CONV vs. DELAY), and electrical stimulation (ES vs. NES) on temperature and pH decline for individual muscles (PM, SM, LL), color measurements were determined using mixed models (PROC MIXED, SAS 9.4, SAS Institute Inc., Cary, NC, 2013). Residual diagnostic plots were used to confirm the model assumptions (normality and equal variance) were met. The temperature and pH data were analyzed as repeated measures with an unstructured variance-covariance matrix. The least-square means and standard errors of the means were reported for main effects and significant interactions. The PDIFF option was used to separate means with a significance level set at $\alpha = 0.05$, and Tukey adjusted pairwise comparisons were used. Trends were considered when $0.05 < P \leq 0.1$. The same analyses were performed for data obtained in both plants. Differences in the plants' logistics and chilling and electrical stimulation conditions were used as a basis for separately analyzing the data from the 2 plants.

Exponential decay model

Deep tissue and surface temperatures recorded (every 20 s) over the chilling period were downloaded from data loggers. The mixed procedure of SAS 9.4 (SAS Institute Inc., 2013) was utilized to generate least squares means for the 3-way interaction between carcass size (heavy vs. light), chilling conditions (CONV vs. DELAY), and time postmortem for deep

and surface temperatures. Fat thickness was included in the model as a covariate. Fat thickness was measured at the interface between the 12th and 13th rib, perpendicular to fat cover and at a point located three-fourths of the length of the ribeye cross section from the chine bone side (Forrest and Judge, 1994).

Exponential decay models were fitted to deep tissue, and surface temperature and temperature curves were obtained using the R package nlme. The self-starting asymptotic regression (SSasymp) function in R (Bates and Chambers, 1992) was utilized to obtain self-starting values for the nonlinear exponential decay models fitted to temperature least-square means data (deep and surface). Due to convergence issues, surface temperature data at time points at the initial time points were removed from the analysis. The models follow the equation below, and model estimations were done separately for each treatment group:

$$T = T_a + (T_0 - T_a)e^{-\lambda t}$$

where T is the temperature at time t; T_a, $(T_0 - T_a)$, and λ represent the model parameters. The parameter λ represents the rate of chilling; T_a is the asymptote and represents the value of temperature for great values of time t; and $(T_0 - T_a)$ is the difference between the initial temperature of the carcass (T_0) and the ambient temperature (T_a).

The plots of deep temperature (Figures 1 and 2) suggested a two-phase phenomenon with a rapid increase in temperature within the first hour corresponding to the first phase and a gradual decline in temperature thereafter corresponding to the second phase. Hence, a biexponential decay model was fitted to deep temperature using R package stats. The new parameterization using the biexponential model ($\lambda 1$, T_{a1} and $\lambda 2$, T_{a2}) improved model fit and predictions (Table 1): $T = T_{a1} + (T_0 - T_{a1})\ e^{-\lambda 1 t} + T_{a2} + (T_0 - T_{a2})\ e^{-\lambda 2 t}$.

The least-square means for deep tissue and surface temperature of the combination of carcass size (heavy vs. light) and chilling conditions (CONV vs. DELAY) were plotted against time and presented in Figures 1 and 2 for Plant A and Plant B, respectively. The plots show both deep tissue and surface temperature curves follow an exponential decay pattern and are evidence for the geometrical behavior of observed temperature decline in beef carcasses. In addition, a relationship between deep tissue temperature and surface temperature was developed using a spline model in R. The approximation of the observed relationship between deep and surface temperature was achieved by a b-spline model. Model parameters were set as follows: degree = 2 and number of interior knots = 4. The knots were sets using the quantiles.

Results and Discussion

Internal temperature decline in different muscles

The carcass data from Plant A and B are presented in Tables 2 and 3, respectively. Further, the internal temperature decline in the LL, PM, and SM in Plant

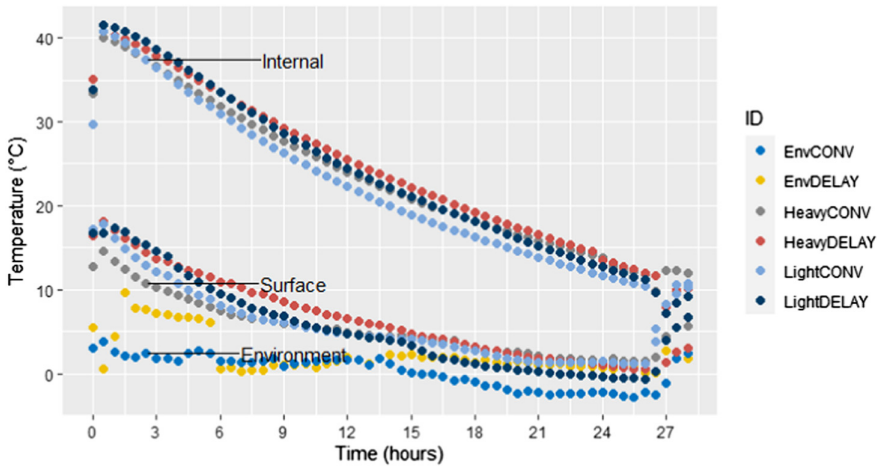

Figure 1. Deep tissue (*semimembranosus*) temperature decline of beef carcasses and environmental temperature (°C) of the coolers in Plant A. Deep temperature was measured by inserting the probe tip (10.5 cm) into the *semimembranosus* under the pelvic bone. The environmental temperature was recorded with the probe hung in the close vicinity of carcasses. EnvCONV = environmental temperature in conventional spray chilling; EnvDELAY = environmental temperature in delay-spray chilling; HeavyCONV = heavyweight conventionally spray-chilled carcass sides; HeavyDELAY = heavyweight delayed spray-chilled sides; LightCONV = lightweight conventionally chilled sides; LightDELAY = lightweight delayed spray-chilled sides.

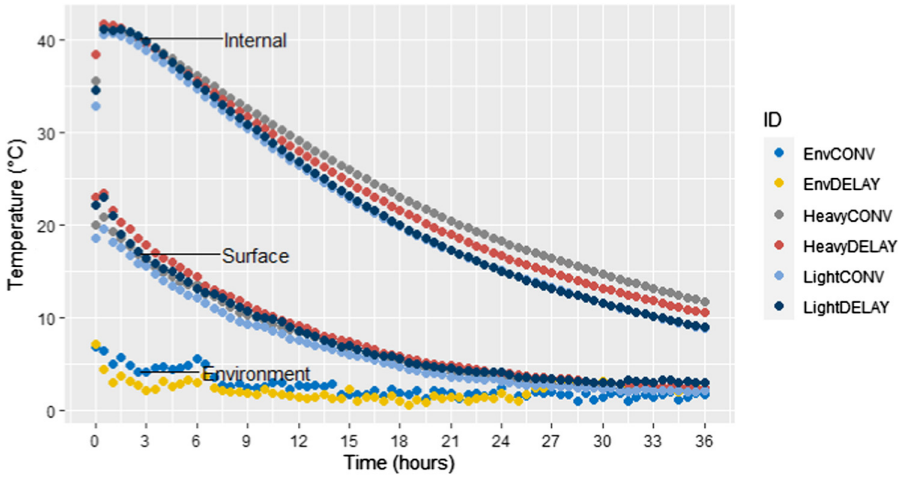

Figure 2. Deep tissue (*semimembranosus*) temperature decline of beef carcasses and environmental temperature (°C) of the coolers in Plant B. Deep temperature was measured by inserting the probe tip (10.5 cm) into the *semimembranosus* under the pelvic bone. The environmental temperature was recorded with the probe hung in the close vicinity of carcasses. EnvCONV = environmental temperature in conventional spray chilling; EnvDELAY = environmental temperature in delay-spray chilling; HeavyCONV = heavyweight conventionally spray-chilled carcass sides; HeavyDELAY = heavyweight delayed spray-chilled sides; LightCONV = lightweight conventionally chilled sides; LightDELAY = lightweight delayed spray-chilled sides.

Table 1. Parameter estimates of the exponential (expo) and biexponential (biexpo) cooling model fitted to deep[1] (semimembranosus) and surface[1] temperature of beef carcasses in Plant A and B

Location	Treatment Groups	*n*	R^2 Expo	R^2 Biexp	[3]T_0 Est Exp[4]	T_0 Observed	T_0 Est Biexp[5]	λ Exp	λ1 Biexp	λ2 Biexp
Plant A										
Deep	[2]Light CONV	57	0.97	0.99	40.1	34.38	34.31	0.035	1.78	0.055
	Heavy CONV	57	0.98	0.99	40.15	33.38	35.39	0.032	3.21	0.045
	Heavy DELAY	57	0.99	0.99	40.97	35.45	35.73	0.015	1.48	0.048
	Light DELAY	57	0.98	0.99	41.92	33.81	34.44	0.022	4.93	0.058
Surface	Light CONV	57	0.95		18.03	17.23		0.146		
	Heavy CONV	57	0.97		13.87	12.74		0.104		
	Heavy DELAY	57	0.99		17.66	16.37		0.061		
	Light DELAY	57	0.98		18.8	16.75		0.094		
Plant B										
Deep	[2]Light CONV	73	0.98	0.99	42.14	32.9	29.74	0.031	5.33	0.055
	Heavy CONV	73	0.99	0.99	42.54	35.59	33.39	0.024	3.89	0.047
	Heavy DELAY	73	0.99	0.99	43.41	38.41	35.6	0.032	1.46	0.048
	Light DELAY	73	0.98	0.99	43.21	34.6	34.09	0.033	1.98	0.053
Surface	Light CONV	73	0.99		19.64	18.51		0.089		
	Heavy CONV	73	0.99		20.72	19.92		0.082		
	Heavy DELAY	73	0.99		23.21	22.94		0.089		
	Light DELAY	73	0.99		22.26	22.19		0.097		

[1]Deep temperature was measured by inserting the probe tip (10.5 cm) into the *semimembranosus* under the pelvic bone. The surface temperature was recorded 1.5 cm beneath the fat surface at the 13th rib.

[2]CONV = conventionally chilled sides; DELAY = delay spray–chilled sides.

[3]λ = constant rate of cooling (°C·h^{-1}); R^2 = pseudo R-square; T_0 = initial temperature of the body.

[4]Est expo = estimates of the exponential model.

[5]Est biexpo = estimates of the biexponential model.

SEM = standard error of the mean.

A and Plant B are shown in Table 4. There was a carcass size × time postmortem interaction ($P < 0.0001$) for temperature decline in the LL, PM, and SM in Plant A and LL and PM in Plant B. However, electrical stimulation did not influence ($P > 0.05$) the temperature of the carcasses. In Plant A, the initial internal

Table 2. Carcass data (mean ± standard deviation) from Plant A

	Heavyweight				Lightweight			
	DELAY[1]		CONV[1]		DELAY[1]		CONV[1]	
	NES[2]	ES[2]	NES[2]	ES[2]	NES[2]	ES[2]	NES[2]	ES[2]
n	20	20	22	22	20	20	20	20
HCW (kg)	447.46 ± 29.41	447.46 ± 29.41	445.2 ± 19	445.2 ± 19	362.49 ± 31.24	362 ± 31.24	360.01 ± 36.69	360.01 ± 36.69
REA	16.08 ± 2.33	16 ± 2.02	16.64 ± 1.52	16.53 ± 1.46	13.98 ± 1.97	14.4 ± 1.92	14.95 ± 1.9	14.67 ± 1.93
FT (mm)	18.38 ± 8.37	18.5 ± 8.74	14.9 ± 5.54	15.88 ± 6.43	13.46 ± 8.6	14.1 ± 9.73	11.02 ± 4.29	11.28 ± 4.67
PYG	3.81 ± 082	3.82 ± 0.86	3.47 ± 0.55	3.56 ± 0.63	3.33 ± 0.85	3.39 ± 0.96	3.09 ± 0.42	3.11 ± 046
adjPYG	3.86 ± 0.84	3.9 ± 0.84	3.6 ± 0.53	3.63 ± 0.69	3.34 ± 0.95	3.41 ± 1.05	3.13 ± 0.48	3.13 ± 0.53
Marbling	407.39 ± 73.37	400 ± 75.59	426.3 ± 84.74	432.1 ± 88.41	429.5 ± 83.76	429 ± 79.03	385 ± 55.39	398.5 ± 60.55
SMat	69.57 ± 51.39	75.2 ± 47.08	53.33 ± 29.44	49.58 ± 29.26	49 ± 35.08	66.5 ± 63.02	76 ± 88.46	71.5 ± 90.51
LMat	107.39 ± 126.68	106 ± 122.1	82.5 ± 104.3	77.08 ± 93.78	43 ± 41.18	45 ± 43.59	26 ± 24.58	30 ± 28.84
OMat	84.13 ± 64.11	86.7 ± 59.57	69.17 ± 59.14	64.17 ± 58.55	48.25 ± 31.84	63 ± 60.18	53 ± 51.67	50 ± 50.86
%KPH	3.74 ± 0.47	3.91 ± 0.39	4.19 ± 1.06	3.85 ± 0.52	3.55 ± 0.92	3.58 ± 0.89	3.7 ± 0.77	3.58 ± 0.73
YG	3.66 ± 1.61	3.72 ± 1.49	3.21 ± 0.88	3.27 ± 0.95	3.1 ± 1.38	3.05 ± 1.48	2.56 ± 0.84	2.65 ± 0.85

[1]CONV = conventional spray-chilling; DELAY = delayed spray-chilling.

[2]ES = stimulated; NES = nonstimulated.

%KPH = percentage kidney, pelvic, heart fat; adjPYG = adjusted preliminary yield grade; FT = fat thickness; HCW = hot carcass weight; LMat = lean maturity; OMat = overall maturity; PYG = preliminary yield grade; REA = ribeye area; SD = standard deviation; SMat = skeletal maturity; YG = yield grade.

Table 3. Carcass data (mean ± standard deviation) from Plant B

	Heavyweight				Lightweight			
	DELAY[1]		CONV[1]		DELAY[1]		CONV[1]	
	NES[2]	ES[2]	NES[2]	ES[2]	NES[2]	ES[2]	NES[2]	ES[2]
n	25	25	20	20	16	16	19	19
HCW (kg)	418.07 ± 27.15	418.07 ± 27.15	420.55 ± 25.59	420.55 ± 25.59	357.15 ± 23.32	357.15 ± 23.32	353.8 ± 24.24	353.8 ± 24.24
REA	15.29 ± 1.55	15.12 ± 1.14	14.28 ± 0.79	14.49 ± 1.1	13.54 ± 1.31	13.68 ± 1.5	13.53 ± 0.93	13.44 ± 1.29
FT (mm)	18.38 ± 3.52	14.94 ± 3.72	17.54 ± 4.05	16.67 ± 5.99	14.34 ± 4.69	13.56 ± 4.73	13.42 ± 4.19	13.7 ± 4.38
PYG	3.75 ± 0.35	3.46 ± 0.37	3.73 ± 0.4	3.64 ± 0.59	3.4 ± 0.46	3.33 ± 0.47	3.32 ± 0.42	3.35 ± 0.43
adjPYG	3.67 ± 0.38	3.45 ± 0.4	3.86 ± 0.53	3.73 ± 0.56	3.44 ± 0.46	3.42 ± 0.42	3.36 ± 0.55	3.43 ± 0.48
Marbling	462.5 ± 70.29	475.6 ± 77.28	492.6 ± 98.99	505.3 ± 95.76	484.23 ± 92.31	475.4 ± 81.1	484.21 ± 85.78	483.16 ± 89.2
SMat	41.25 ± 37.57	43.75 ± 40.48	50.53 ± 43.01	54.21 ± 50.7	33.85 ± 25.93	34.23 ± 25.8	24.21 ± 18.95	26.32 ± 22.41
LMat	46.25 ± 27.29	45 ± 28.05	52.63 ± 30.7	50.53 ± 31.18	43.85 ± 20.41	45 ± 17.72	42.11 ± 17.51	41.05 ± 15.6
OMat	43.75 ± 31.49	44.38 ± 32.09	51.58 ± 34.24	52.37 ± 36.64	38.85 ± 21.04	40 ± 21.07	*32.63 ± 12.18*	33.68 ± 16.32
%KPH	3.09 ± 1.76	3.45 ± 1.44	4.18 ± 1.54	3.92 ± 1.53	3.68 ± 1.65	3.96 ± 1.65	3.74 ± 1.16	3.92 ± 1.52
YG	3.66 ± 1.24	2.76 ± 1	3.98 ± 0.76	3.77 ± 0.71	3.22 ± 0.84	3.16 ± 0.86	3.2 ± 0.69	3.29 ± 0.7

[1]CONV = conventional spray-chilling; DELAY = delayed spray-chilling.

[2]ES = stimulated; NES = nonstimulated.

%KPH = percentage kidney, pelvic, heart fat; adjPYG = adjusted preliminary yield grade; FT = fat thickness; HCW = hot carcass weight; LMat = lean maturity; OMat = overall maturity; PYG = preliminary yield grade; REA = ribeye area; SD = standard deviation; SMat = skeletal maturity; YG = yield grade.

temperature (1 h postmortem) was lower ($P < 0.05$) in heavy than lightweight carcasses across all muscles. This temperature difference is unexpected and contrary to the results of Agbeniga and Webb (2018). The difference in temperature between light and heavyweight muscle temperatures in this plant may be specific to the plant. In the PM, this difference is likely due to the application of a hot water carcass wash intervention (83°C to 93°C) and the removal of kidney fat, which allowed for hot water to directly contact the PM in Plant A (Dickson and Acuff, 2017). For instance, in Plant B, hot water was applied but much earlier in the process (relatively 30 to 35 min before reaching the hotboxes), which may have allowed the extra heat to dissipate before temperature data collection. In Plant A, temperature data collection occurred sooner after hot water spray (10 to 15 min) than in Plant B. Also, the specific electrical stimulation settings may have contributed to the temperature difference between light and heavyweight carcasses. In addition, in Dickson and

Table 4. Effect of beef carcass size on temperature (°C) decline in the longissimus lumborum (LL), psoas major (PM), and semimembranosus (SM) in Plant A and B

Muscle	Weight Class	1 h	6 h	12 h	36 h	*n*	SEM	*P* Value
Plant A								
LL	Heavy	36.16^{ax}	21.14^{bx}	12.19^{cx}	1.86^{dx}	42	0.51	<0.0001
	Light	39.56^{ay}	23.42^{by}	13.22^{cx}	2.93^{dx}	40	0.51	
PM	Heavy	32.70^{ax}	17.26^{bx}	9.23^{cx}	0.72^{dx}	42	0.49	<0.0001
	Light	37.73^{ay}	18.59^{bx}	9.35^{cx}	0.71^{dx}	40	0.49	
SM	Heavy	37.03^{ax}	24.01^{bx}	15.21^{cy}	3.48^{dy}	42	0.42	<0.0001
	Light	39.60^{ay}	22.85^{bx}	12.60^{cx}	2.04^{dx}	40	0.42	
Plant B								
LL	Heavy	39.66^{ax}	24.36^{by}	13.21^{cy}	4.22^{dx}	45	0.39	<0.0001
	Light	39.11^{ax}	22.93^{bx}	11.26^{cx}	3.71^{dx}	35	0.39	
PM	Heavy	38.64^{ax}	25.54^{by}	16.16^{cy}	4.92^{dy}	45	0.42	<0.0001
	Light	38.88^{ax}	23.30^{bx}	13.30^{cx}	3.87^{dx}	35	0.42	
SM		39.42^{a}	29.61^{b}	18.09^{c}	5.67^{d}	80	0.49	0.013

$^{a–d}$Least squares means within a row for the same muscle with different letters are different ($P < 0.05$).

^{x,y}Least squares means within a column for the same muscle with different letters are different ($P < 0.05$).

SEM = standard error of the mean.

Acuff (2017) reported that the muscle and surface temperatures of the lightweight carcasses were increased by the hot water wash. Barkate et al. (1993) reported that spraying beef carcasses with 95°C hot water for 20 s increased the carcass surface temperature. Since lighter carcasses offer more surface area for hot water per kilogram than the heavier counterparts, they are more likely to have a higher temperature than lighter carcasses. However, there was no difference ($P > 0.05$) in initial temperature between heavy and lightweight carcasses in Plant B.

Overall, the lightweight carcasses had a faster ($P < 0.0001$) temperature decline than heavyweight carcasses (Table 4) as a result of a lesser mass. The temperature of the LL of lightweight carcasses dropped—on average—2°C faster than that of heavyweight counterparts between 0 h and 12 h. The rate of temperature decline in the LL was not different ($P = 0.72$) following 12 h of chilling. Similar results were observed in the PM. In Plant A, the SM had a more rapid temperature decline ($P < 0.0001$) in lightweight carcasses than heavyweight carcasses, even though they started with higher ($P < 0.01$) temperatures. This was consistent with a previously published study that reported leaner (lighter) carcasses chill more rapidly than fatter (heavier) carcasses (Hippe et al., 1991). In agreement, Lochner et al. (1980) also reported differences in chilling rates between larger and fatter carcasses compared to lighter (leaner) carcasses. In the present study, only time postmortem significantly influenced ($P = 0.013$) temperature decline in the SM in Plant B. For all the muscles, the rate of internal temperature decline was greatest within the first 6 h postmortem for both plants. On average, there was a 15°C drop in temperature within the first 6 h compared to 9°C decreases between 6 and 12 h and between 12 and 36 h for all muscles.

Temperature decline is related to postmortem meat quality development. The rate and extent of heat dissipation, especially in the early postmortem period, has been studied and its influence on postmortem metabolism has been demonstrated (Ferguson et al., 2001; Bruce, 2004). Although a two-way interaction ($P < 0.0001$) between chilling conditions and time postmortem was detected in the LL in Plant A, the rate of temperature decline was similar ($P > 0.05$) between the 2 chilling groups (CONV vs. DELAY). In the CONV group, the temperature of the LL dropped from 37.7°C to a final 2.85°C, whereas it declined from 38.35°C to 2.93°C in the DELAY group. These results agreed with previous reports where time postmortem had more influence on temperature decline during chilling than the chilling conditions used (Hippe et al., 1991; Strydom and Buys, 1995). On the other hand, others have reported that varying spray-chilling durations (4, 8, or 12) increased the rate of chilling of *longissimus dorsi* (LD) and *longissimus thoracis* (Jones and Robertson, 1988; Prado and de Felício, 2010).

In general, electrical stimulation minimally influenced temperature decline in all 3 muscles. There was no impact of electrical stimulation ($P > 0.05$) on temperature decline in the LL (Plant B, $P = 0.77$), PM (Plant A, $P = 0.19$; Plant B, $P = 0.71$), and SM

(Plant A, $P = 0.17$; Plant B, $P = 0.53$) except for the LL in Plant A ($P = 0.019$) where high voltage was applied. The stimulated sides had a lower mean temperature ($P = 0.019$) than nonstimulated sides (data not presented). In support, Mombeni et al. (2013) reported lower temperatures in the LD of stimulated sides compared to nonstimulated counterparts. It is likely that high voltage electrical stimulation induced early postmortem depletion of metabolic energy (Chrystall and Devine, 1978), as illustrated by the rapid decline in pH. The early depletion of the energy may explain the lower temperature in ES carcasses since the heat generated by metabolism could no longer influence the temperature (de Meis, 2001).

The current study highlighted significant differences in the rate of internal temperature decline between heavy and lightweight carcasses. More specifically, the muscles of the round (represented by SM) of heavyweight carcasses showed the slowest chilling rates compared to the middle meats represented by LL and PM. In agreement, Agbeniga and Webb (2018) also reported slower temperature decline and faster pH decline in heavier beef carcasses compared to lighter ones. The differences in the rate of chilling (and potentially pH decline) in heavyweight carcasses' SM may be due to its location in the thickest and heaviest section of the carcass in addition to its bulk density and fat cover. It has been previously suggested that increased mass and/or a combination of fat cover and marbling acting as an insulator may contribute to differences in the rate of chilling and temperature decline in heavier carcasses (Aalhus et al., 2001; Savell et al., 2005; Juárez et al., 2016). However, marbling was not a significant factor in this study ($P > 0.05$). Hence, it was not included in the final model. Also, there were no significant Pearson correlations between marbling and the temperature of the LD in Plant A (0.058, P value = 0.49) and Plant B (0.01, P value = 0.9).

pH decline

Before electrical stimulation and chilling, samples were collected within 15 to 20 min postmortem from LL of 31 and 36 carcass sides in Plant A and Plant B, respectively, for initial pH measurement. The mean initial pH in Plant A was 6.38 (ranged from 6.12 to 6.71), whereas, in Plant B, the mean initial pH was 6.58 with a range of 6.15 to 7.02. These pH ranges captured animal to animal variability and were similar to values reported in the literature (Bendall, 1978; Prado and de Felício, 2010; Mombeni et al., 2013).

In Plant A, a four-way interaction (carcass size × chilling conditions × electrical stimulation × time postmortem, $P = 0.021$) for pH was detected in LL (Table 5). A four-way interaction could be challenging to interpret because of the difficulty in distinguishing the factors that had the most significant influence on pH decline. In general, pH fall was 0.1 unit faster in stimulated sides than in nonstimulated over the first 12 h regardless of the chilling conditions and carcass size. The initial decrease in pH at 6 h averaged 0.4 to 0.5, which is similar to the pH decline ranges reported by Chrystall and Devine (1978) for stimulated *sternomandibularis*. This range of pH decline was observed in both stimulated and nonstimulated sides, suggesting that electrical stimulation may not be solely responsible for the initial drop in pH observed in Plant A. Previous research suggested that high temperatures early during postmortem could have a confounding effect with electrical stimulation in decreasing the pH (Chrystall and Hagyard, 1976; Ferguson et al., 2001). As shown in Table 4, heavyweight carcasses had slower chilling rates, which is consistent with a faster pH decline, and similar results were previously reported (Rybarczyk et al., 2015; Agbeniga and Webb, 2018).

Typically, electrical stimulation is used to offset the toughening effect of overly aggressive chilling conditions on beef tenderness (cold shortening). In this study, high voltage ES (Plant A) had an interactive effect ($P < 0.05$) on pH decline in the LL, but low-voltage ES (Plant B) did not (Table 5). Previous research has demonstrated that electrical stimulation influenced the rate and extent of pH decline in different livestock species (Chrystall and Hagyard, 1976; Chrystall and Devine, 1978; Chrystall et al., 1980; Chrystall and Devine, 1985; Eikelenboom et al., 1985). Although electrical stimulation had a significant effect ($P < 0.05$) on pH decline in the LL in Plant A, there was no evidence that it accelerated pH decline in the PM and SM ($P > 0.05$). One of the reasons for this difference could be that most of the currently used electrical systems are designed to exert a minor effect, if any, on the muscles of the round (Roeber et al., 2000). In Plant B, an interaction ($P < 0.05$) between carcass size and time postmortem was observed for LL ($P = 0.038$) and PM ($P = 0.0015$; Table 5). For SM, carcass weight seems to have a tendency ($P = 0.05$) to influence pH decline.

In general, heavyweight carcasses showed a more rapid pH decline within 12 h of exsanguination than lightweight carcasses. Initial pH was lower ($P < 0.05$) in heavyweight carcasses across all muscles. This is

Table 5. Effect of carcass size, chilling conditions, and electrical stimulation on pH decline in longissimus lumborum (LL), psoas major (PM), and semimembranosus (SM) in Plant A and B

				1 h	6 h	12 h	36 h	*n*	SEM	*P* Value
Plant A										
	Weight Class	**Chilling Conditions**	**Electrical Stimulation**							
LL	Heavy	CONV[1]	ES[2]	6.11^{aw}	5.66^{bxy}	5.57^{cwx}	5.45^{cw}	22	0.041	0.021
			NES[2]	6.20^{awx}	5.59^{bwx}	5.56^{bwx}	5.45^{cw}	22	0.041	
		DELAY[1]	ES	6.17^{awx}	5.55^{bw}	5.50^{bw}	5.45^{cw}	20	0.041	
			NES	6.24^{ax}	5.61^{bwx}	5.53^{cw}	5.43^{cw}	20	0.041	
	Light	CONV	ES	6.23^{ax}	5.67^{bxyz}	5.63^{bxy}	5.48^{cw}	20	0.041	
			NES	6.16^{awx}	5.75^{bz}	5.67^{by}	5.48^{cw}	20	0.041	
		DELAY	ES	6.17^{awx}	5.75^{bz}	5.68^{by}	5.45^{cw}	20	0.041	
			NES	6.23^{ax}	5.73^{byz}	5.55^{cwx}	5.49^{cw}	20	0.041	
PM	Heavy	CONV		5.73^{aw}	5.68^{abwx}	5.68^{abw}	5.66^{bw}	22	0.036	0.005
		DELAY		5.8^{aw}	5.60^{bw}	5.61^{bw}	5.60^{bw}	20	0.036	
	Light	CONV		5.76^{aw}	5.64^{bw}	5.65^{bw}	5.64^{bw}	20	0.036	
		DELAY		5.71^{aw}	5.66^{abw}	5.64^{abx}	5.61^{bw}	20	0.036	
SM		CONV		6.32^{aw}	5.94^{bw}	5.73^{cw}	5.49^{dw}	42	0.033	0.002
		DELAY		6.28^{aw}	5.75^{bx}	5.60^{vx}	5.47^{dw}	40	0.033	
Plant B										
LL	Heavy			6.16^{aw}	5.54^{bw}	5.48^{cw}	5.49^{bcw}	45	0.032	0.038
	Light			6.27^{ax}	5.61^{bx}	5.5^{cw}	5.51^{cw}	35	0.032	
PM	Heavy			5.69^{aw}	5.69^{aw}	5.62^{bw}	5.63^{bw}	45	0.037	0.002
	Light			5.81^{bw}	5.67^{aw}	5.64^{aw}	5.62^{aw}	35	0.037	
SM	Heavy			6.34^{aw}	5.86^{bw}	5.66^{cw}	5.54^{dw}	80	0.035	0.05
	Light			6.42^{ax}	6.02^{bx}	5.76^{cx}	5.56^{dw}	80	0.035	

[1]CONV = conventional spray chilling, DELAY = delay spray chilling.

[2]ES = electrically stimulated, NES = nonstimulated.

[a–d]Least squares means within the same row with different letters are different ($P < 0.05$).

[w–z]Least squares means within a column with different letters for the same muscle are different ($P < 0.05$).

SEM = standard error of the mean.

consistent with temperature decline, highlighting the close relationship between temperature decline and pH decline, and agrees with previous research (Ferguson et al., 2001; Bruce, 2004). A plot of pH versus temperature (figure not included) showed the LL of lightweight carcasses in Plant A could be at risk of heat shortening. This finding was unique to Plant A, in which the muscle temperatures of the lightweight carcasses were significantly elevated over that of the heavier carcasses, presumably as a result of the hot water carcass wash. The risk of heat shortening may be lower in heavyweight carcasses even with a slower rate of temperature decline since the temperature at 1 h was on average 3°C lower than in lightweight carcasses (Table 4).

Electrical stimulation did not influence ($P > 0.05$) the rate of pH decline in the PM and SM in both plants. The pH of the PM in both plants fell below 6 within an hour postmortem and to its lowest values within 6 h postmortem. Given that PM has been classified as red oxidative muscle, such a significant decline in pH early postmortem was unexpected. Postmortem pH decline has been shown to occur faster in glycolytic muscles than in oxidative muscles (Lebret and Guillard, 2005; Listrat et al., 2016). However, Listrat et al. (2016) also suggested that the relationship is not systemic because they observed much lower pH values at 45 min in pork PM compared to LD. Similarly, Lyon et al. (1983) reported the pH of the PM at 1 h postmortem was much lower than that of *triceps brachii,* an intermediary muscle that has both glycolytic and oxidative metabolism. Lower levels of pH within an hour of exsanguination in the PM were also reported by McCollum and Henrickson (1977). A comparative study of postmortem muscle glycolysis between the PM and LD of Korean native cattle concluded that

glycolysis proceeded at a higher velocity in PM than in the LD (Kim et al., 2000). The extent of pH decline in PM has been known to be lesser than that of glycolytic muscles because levels of glycogen in muscles play an important role in determining the rate and extent of glycolysis. For example, the ultimate pH of the PM reported in the present study was 5.61, while those of the LL and SM were 5.45 and 5.47, respectively.

The muscle fiber composition and metabolic characteristics of the PM, its location in the carcass, and its inherent buffering capacity may be contributing to its response to electrical stimulation and postmortem pH fall. In addition, the PM is usually insulated by the kidney and pelvic fat, and, in the case of Plant A in this study, in which the kidney fat was removed, the PM was directly exposed to the hot water wash. Thus, its location might be likely contributing to a microclimate that could promote a more rapid postmortem metabolism. Between the initial temperature measurement and 6 h postmortem, the rate of temperature decline was 1°C and 2°C slower in the PM than the LL for heavy and lightweight carcasses, respectively.

The electrical stimulation did not influence pH decline ($P > 0.05$) in SM in Plant A or B. As indicated earlier, the electrical systems used in this study were designed to deliver electrical inputs to the muscles of the middle cut, and hence, it was not expected to have a significant effect on the postmortem metabolism of SM (Roeber et al., 2000). In Plant A, the pH of SM declined faster ($P = 0.01$) in DELAY carcasses than in CONV carcasses within the first 6 h postmortem. This was not expected since no temperature differences were observed between the DELAY and CONV treatment groups. However, the extent of pH decline in the SM of both groups was similar over the chilling period (Table 5).

Objective color of PM

Electrical stimulation had a significant impact ($P < 0.05$) on PM color panel scores in both plants (Table 6). Specifically, the ES sides had greater ($P < 0.05$) PM color scores (redness) than nonstimulated sides in Plant A. Overall, 82% of the stimulated sides were ranked light cherry red or cherry red, while only 66% of nonstimulated sides were ranked as such in Plant A (data not presented). There were twice as many nonstimulated sides (34%) classified as dark red or very dark red compared to stimulated sides (17%). In Plant B, 62% of stimulated (and 55% of nonstimulated) sides were classified as bright cherry red or cherry red. There were fewer (30% vs. 41%) stimulated sides ranked as dark or very dark red. There was also an interaction between carcass size and electrical stimulation ($P = 0.0036$) for color scores in Plant B. While electrical stimulation increased color scores in heavyweight carcasses, it had no impact on the color scores for lightweight carcasses. However, the impact of electrical stimulation on the color score in plant B is relatively inconsequential since the scores of both heavy and lightweight groups fall in the bright cherry to a cherry red category, which consumers find acceptable. Regardless of the plant, carcass size and chilling conditions did not influence ($P = 0.11$) panel color scores of the PM (Table 6).

Table 6. Effect of carcass size, chilling conditions, and electrical stimulation on subjective color scores[1] of *psoas major* in Plant A and B

Treatment/ Weight Class		Color Scores	*n*	SEM	*P* Value
Plant A					
ES[2]		5.34[y]	41	0.15	0.002
NES		4.78[x]	41	0.15	
CONV[3]		5.11[x]	42	0.27	0.76
DELAY		5.02[x]	40	0.27	
Heavy		5.02[x]	42	0.27	0.76
Light		5.10[x]	40	0.27	
Plant B					
ES	**Heavy**	5.25[x]	20	0.15	0.0036
	Light	4.92[y]	19	0.15	
NES	**Heavy**	5.08[x]	25	0.19	0.30
	Light	4.98[x]	16	0.19	

[1]Scores were on an 8-point scale with 1 being light pink and 8 being very dark red.

[2]ES = electrically stimulated, NES = nonstimulated.

[3]CONV = conventional spray chilling, DELAY = delay spray chilling.

[a–d]Least squares means within the same row with different letters are different ($P < 0.05$).

[x,y]Least squares means within a column with different letters are different ($P < 0.05$).

SEM = standard error of the mean.

In Plant A, an interaction ($P = 0.04$) between carcass size and electrical stimulation was observed ($P < 0.05$) for PM lightness (L^*; Table 7). Electrical stimulation increased ($P < 0.05$) L^* values in the PM from lightweight carcasses (54.66 for stimulated sides vs. 49.94 for nonstimulated sides) but not in heavyweight carcasses. The PM redness (a^*) was not affected ($P > 0.05$) by treatment factors, unlike the panel scores. Previously, Ripoll et al (2012) reported significant correlations between panel color scores and instrumental chroma. However, these authors did not find any relationship between panelists' scores and instrumental

Table 7. Effect of carcass size (weight class), chilling conditions, and electrical stimulation on *L** (lightness) *a** (redness), and *b** (yellowness) of *psoas major* in Plant A and Plant B

	*L**		*a**		*b**	
Plant A						
Weight Class	**Heavy**	**Light**			**Heavy**	**Light**
ES	53.52^{ax} ($n = 18$)	54.66^{ay} ($n = 20$)	23.14^{x} ($n = 38$)			
NES	52.18^{ax} ($n = 24$)	49.92^{ax} ($n = 20$)	23.19^{x} ($n = 44$)			
CONV					17.55^{ax} ($n = 22$)	18.41^{ay} ($n = 20$)
DELAY					17.6^{ax} ($n = 20$)	16.41^{ax} ($n = 20$)
SEM		1.1	0.61			0.58
***P*value**		0.04	0.14			0.04
Plant B						
Weight Class	**Heavy**	**Light**	**Heavy**	**Light**	**Heavy**	**Light**
	40.1^{a} ($n = 45$)	40.43^{a} ($n = 35$)	22.37^{b} ($n = 45$)	21.09^{a} ($n = 35$)	16.32^{b} ($n = 45$)	15.23^{a} ($n = 35$)
SEM		0.65		0.49		0.34
***P*value**		0.62		0.011		0.002

$^{a–d}$Least squares means within a trait with different letters are different ($P < 0.05$).

^{x,y}Least squares means within a column with different letters are different ($P < 0.05$).

CONV = conventional spray chilling, DELAY = delay spray chilling; ES = stimulated; NES = nonstimulated; SEM = standard error of the mean.

redness, lightness, or hue values. Similar conflicting reports on correlations between panelist appraisals and instrumental coordinates have been reported by others as well (Chan et al., 1996; Hulsegge et al., 2001). There was an interaction ($P = 0.04$) between carcass size and chilling conditions for PM yellowness (*b**). Delay chilling improved PM *b** values ($P = 0.04$) from lightweight carcasses compared to heavyweight carcasses. In Plant B, heavyweight carcasses had redder PM (higher *a**; $P = 0.011$) than lightweight carcasses (Table 7). Also, carcass size influenced PM *b** values, with heavyweight carcasses having more yellow-colored PM ($P = 0.0021$) than lightweight carcasses. The interaction of chilling conditions and electrical stimulation for lightness (*L**) in Plant B approached significance ($P = 0.0732$). ES and delayed chilled carcass sides tended to be lighter ($L^* = 53.30$) than their nonstimulated and conventionally chilled counterparts ($L^* = 49.40$). The color differences between the 2 plants are likely a result of cattle and plant-specific differences.

These results are in agreement with previous research on the impact of electrical stimulation on meat color. Electrical stimulation generally results in a brighter lean color of the *longissimus* muscle (LL) as evaluated at the 12th /13th rib interface at grading (Savell et al., 1978a; Savell et al., 1978b; Roeber et al., 2000). Moreover, improvements in objective color parameters (*L**, *a**, and *b**) have been reported with electrical stimulation (Roeber et al., 2000). These authors reported that stimulated sides had a *longissimus* muscle eye that was brighter, redder, and more yellow than nonstimulated sides.

Modeling of temperature decline

The purpose of modeling temperature decline for livestock species is to have unbiased, reliable estimates and predictions of carcass chilling with high confidence for an individual or group of carcasses. A good model should be versatile enough to accommodate different sets of data and allow extrapolations beyond the data. The model developed in this study is a simple and appropriate fit for temperature decline in heavy and lightweight carcasses under 2 different cooling regimes observed in Plant A and Plant B.

Figures 1 and 2 indicate that the temperature decline in deep tissue (SM) and at the surface of carcasses was curvilinear. The R squares of the models and predicted versus observed temperatures (Table 1) suggested that the model explained most of the variability in the data and was a good fit. An asymptotic exponential decay model was used to fit the temperature decline data because it captures the Newtonian behavior of carcass chilling. The self-starting Nls asymptotic regression has an initial attribute that allowed an evaluation of initial estimates of the parameters T_a, T_0, and λ for each temperature data set (Table 1).

In both plants, during the first hour, deep tissue temperature rose to a certain level, where it plateaued and decreased thereafter for all treatment groups

(Figure 1 and 2). However, the temperature rise was more pronounced in Plant A than in Plant B. An initial increase in temperature has been previously reported and explained by an increase in temperature generated by postmortem glycolysis (Mallikarjunan and Mittal, 1994). According to de Meis (2001), beef carcass temperature could rise by 2.1°C to 2.7°C above body temperature during the first hours following death. The increase in temperature results from postmortem hydrolysis of ATP, which is responsible for an enthalpy release of approximately $\Delta H_{ATP} = 134$ kJ/mol Pi (de Meis, 2001). While the rise in temperature observed in the present study could be due to the metabolic activity and electrical stimulation following death, the difference observed between the 2 plants is more likely the result of the application of hot water carcass wash in Plant A. The increase in temperature suggested a two-stage (biexponential) model. By fitting a biexponential model, initial temperatures (T_0) for deep tissue estimated by the model were closer to observed values (Table 1). Similarly, pseudo-R squares were improved, and fitted values matched more closely observed values. The surface temperature did not show an increase in temperature. Therefore, the surface temperature was not fitted to a biexponential model.

Comparisons between parameter estimates were conducted only on the exponential decay model because it matches Newton's law of cooling and is closer to previous models developed to assess temperature decline in biological systems (Overholt et al., 2019). Table 1 shows parameter estimates for the fitted model for Plant A and Plant B. The rate of temperature decline in each group as represented by λ was used to compare the different groups to lightweight carcass sides conventionally chilled (Light CONV; considered as a reference group) for both deep and surface temperatures (Table 8). A greater cooling constant (λ) for a treatment group means that the temperature decline in that group was more rapid than that of the reference group.

Parameter estimates for deep tissue and surface temperature in Plant A (Table 8) indicated that the heavy delay-chilled group (DELAY) had the slowest ($P = 0.007$) rate of chilling in deep tissue among all

Table 8. Test comparing the rate of decline (λ) for deep[1] (semimembranosus) and surface[1] temperature of treatment groups against the reference group (Light CONV) in Plant A and B

Location	Parameter Estimates[2]	Treatment Groups	Estimates	Standard Error	*t* Value	*P* Value	Cooler Temperature	Humidity[6]	Observed $^5T_{28}$	Predicted T_{28}
Plant A										
Deep	λ	Intercept[3] (Light CONV)	0.035	0.005			0.59	90.47	14.4761	14.65
		Heavy CONV[4]	−0.003	0.008	−0.43	0.669			13.81	13.82
		Heavy DELAY	−0.02	0.007	−2.72	0.007			17.92	18.22
		Light DELAY	−0.013	0.007	−1.85	0.066			14.45	14.58
Surface	λ	Intercept (Light CONV)	0.15	0.01	16.25				2.46	2.52
		Heavy CONV	−0.04	0.01	−3.09	0.002			3.18	3.36
		Heavy DELAY	−0.08	0.01	−7.84	<0.0001			3.31	3.23
		Light DELAY	−0.05	0.01	−4.84	<0.0001			2.68	2.77
Plant B										
Deep	λ	Intercept (Light CONV)	0.03	0.003			2.3	90.05	12.63	12.71
		Heavy CONV[4]	−0.01	0.004	−1.87	0.06			15.77	15.91
		Heavy DELAY	0.0003	0.004	0.09	0.93			14.22	14.37
		Light DELAY	0.001	0.004	0.34	0.73			12.59	12.73
Surface	λ	Intercept (Light CONV)	0.09	0.001	55.56				1.70	1.81
		Heavy CONV	−0.007	0.002	−3.15	0.002			4.20	4.64
		Heavy DELAY	0.0001	0.002	0.08	0.93			2.67	2.99
		Light DELAY	0.01	0.002	4.07	<0.0001			1.2	1.53

[1]Deep temperature was measured by inserting the probe tip (10.5 cm) into the *semimembranosus* under the pelvic bone. Surface temperature was recorded 1.5 cm beneath the fat surface at the 13th rib.

[2]Parameter estimates: λ = the constant rate of cooling (°C·h^{-1}).

[3]Intercept = the intercept represents Light CONV used as the reference group; the model was significant ($P < 0.0001$).

[4]CONV = conventionally chilled sides; DELAY = delay spray–chilled sides; heavy = heavyweight; light = lightweight.

[5]T^{28} is the temperature at time $t = 28$ h, observed or predicted by the model parameters.

[6]Humidity = represents average humidity measured in the coolers.

groups when compared to the reference. Lightweight carcasses with delayed spray chilling tended ($P = 0.066$) to have the second slowest rate of temperature decline. Heavy and lightweight carcasses chilled under conventional chilling were the most similar ($P = 0.127$ and $P = 0.116$, respectively) in their rate of temperature decline compared to the reference group. Surface temperature declined slowly ($P < 0.0001$) in heavyweight groups, regardless of chilling conditions in comparison to the reference group. Heavyweight carcasses that were conventionally chilled also had a slower rate ($P = 0.002$) than the reference group.

The rate of deep tissue temperature decline in Plant B (Table 8) was not different ($P > 0.05$) between the treatment groups. However, the heavyweight, conventionally chilled carcasses (Heavy CONV) had a tendency ($P = 0.06$) to fall more rapidly than the reference group (Light CONV). Due to the logistics in Plant B, it was not possible to keep the 2 chilling groups (conventional vs. delay spray) in 2 separate chillers. Hence, the 2 chilling groups were separated onto different rails in the same coolers, with the spray chill held off for the rail holding the DELAY group. This may not have been sufficient to significantly alter the rate of deep tissue temperature decline for the chilling groups and could have led to a lack of difference in the chilling rate. Nonetheless, differences ($P < 0.05$) in surface chilling rate were observed between the lightweight delay-spray carcasses and the reference group as well as between heavyweight conventionally chilled carcasses and the reference group ($P = 0.02$). The heavyweight, delay spray–chilled carcasses had a similar rate of temperature decline ($P > 0.05$) to that of the reference group.

The relationship between deep tissue and surface temperature was also examined in the current study. Deep tissue temperature linearly regressed with the surface temperature and strong positive relations (Plant A: R-squared = 0.79, $P < 0.0001$; Plant B: R-squared = 0.90, $P < 0.0001$) were found over time between deep tissue and surface temperature. However, a closer look into the relationship showed a curvilinear trend. Therefore, a spline model was fitted to improve the goodness of fit (Figures 3 and 4; Plant A: R-squared = 0.81, $P < 0.0001$; Plant B: R-squared = 0.97, $P < 0.0001$). Moreover, Akaike information criterion (AICc) indicated that the spline model was a better fit than the linear model (Plant A: AICc = 1,297 vs. 1,314; Plant B: 1,142 vs. 1,487). Early in the chilling process, the spline curve (Figures 3 and 4) showed a plateau, which corresponds to a weak relationship between deep tissue and surface temperatures. When the surface

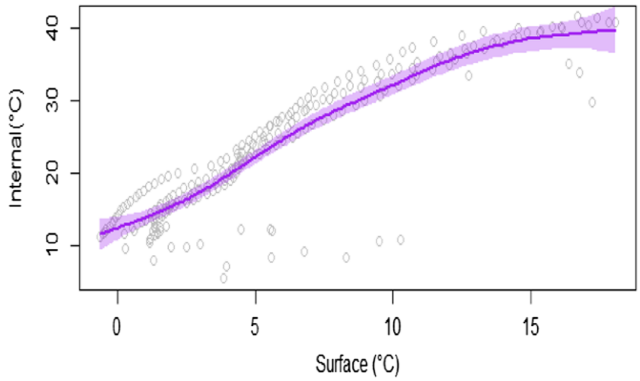

Figure 3. The spline model demonstrating the relationship between deep tissue (*semimembranosus*) and surface temperature (°C) of beef carcasses in Plant A.

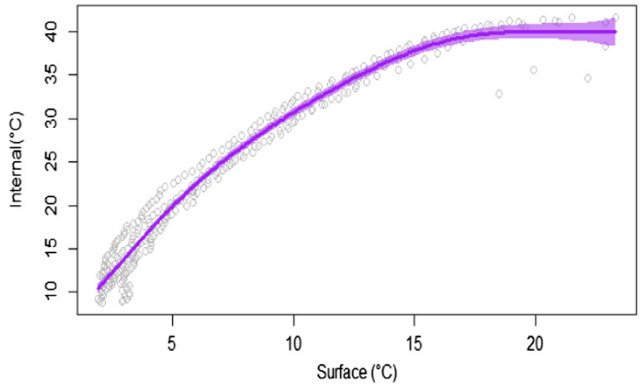

Figure 4. The spline model demonstrating the relationship between deep tissue (*semimembranosus*) and surface temperature (°C) of beef carcasses in Plant B.

temperature dropped below 17°C, about 5 h into the chilling process, the curve declined more abruptly, and deep tissue temperature decline was linearly and strongly related to surface temperature decline.

One of the primary objectives of the beef carcass chilling process is to reduce carcass temperature, within a reasonable time postmortem, to a level where bacterial growth is slowed. In the US, many beef processors target a carcass surface temperature of 4°C within 24 h. This temperature has been used in most plants as a critical control point for the control of pathogens in plant HACCP plans (Savell et al., 2005). Most carcasses in this study, whether they were lightweight or heavyweight, reached 4°C within 24 h postmortem. However, deep tissue temperatures did not reach similar temperatures. There were significant differences in deep tissue temperature between the 2 weight groups by 24 h postmortem. Deep tissue temperature of most lightweight carcasses dropped below 10°C by 24 h while that of heavyweight counterparts remained above 13°C in Plant A and Plant B. This is in agreement with

Klauer (2019), who found that deep tissue temperature in heavyweight carcasses did not fall below 7°C even after 28 h of chilling. Although it is assumed that surface temperature could suffice as an indicator for food safety, the present study, in corroboration with Klauer (2019), suggests that heavyweight carcasses may take longer to achieve adequate internal temperatures necessary to ensure food safety. The additional time carcasses are allowed to chill in holding coolers following the grading procedures may limit the risks of having hot carcasses on the fabrication floor. Nevertheless, should chilling periods be reduced, appropriate attention should be paid to heavier carcasses to avoid muscles of the round being chilled inadequately. The addition of hot/warm carcasses in coolers during early postmortem periods contributes to an increase in the cooler temperature, as evidenced by the environmental temperature data (Figures 1 and 2). Such environmental temperature fluctuations occur regularly in coolers where carcass turnover is high and can influence carcass temperature decline during chilling.

Chilling carcasses of varying sizes and masses under the same chilling conditions could exacerbate issues related to postmortem metabolism and subsequent beef quality. As postmortem metabolism influences the tenderness development and the tenderization process, the variation in carcass sizes may influence the postmortem eating quality. It could be argued that segregating carcasses by size or mass and carcass fatness in chilling coolers could help mitigate differences in the rate of cooling and postmortem glycolysis and subsequently ensure uniformity in beef quality.

Conclusion

The results of the current study indicated that there are differences in temperature and pH decline among the current cohort of beef cattle processed in the US based on their weight. Moreover, postmortem quality management practices such as electrical stimulation and chilling conditions influenced the rate of temperature and pH decline, but the extent of this varied between plants depending on their management. Such variations can contribute to the beef quality and tenderness inconsistencies, which could be economically significant for the beef industry. However, the current industry practices treat all carcasses the same regardless of the carcass weight and size. As such, increased chilling time to allow for heavier carcasses to reach temperature could be beneficial for beef quality. Further research is necessary to examine whether quality inconsistencies could be mitigated through carcasses sorting by weight/size before chilling.

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