Turkey Carcass Chilling and Protein Denaturation in the Development of Pale, Soft, and Exudative Meat

C. Z. Alvarado[1] and A. R. Sams*[,2]

*Texas A&M University, Department of Poultry Science, College Station, Texas 77843

ABSTRACT Pale, soft, and exudative (PSE) meat is a growing problem in the turkey industry and has been associated with processing conditions such as slow carcass chilling. The development of PSE meat is caused by protein denaturation resulting from a rapid rate of pH decline early postmortem (PM) while carcass temperatures are still elevated. This research was conducted to determine the relationship of slow chilling to protein denaturation and PSE development. A total of 48 toms were conventionally processed in 2 trials at 22.5 wk of age, and chilled at 0, 10, 20, or 30°C for either 45 or 90 min before deboning (at 60 or 105 min PM). Temperature and pH of the breast muscle was recorded at 15 min PM, at the time of deboning (60 or 90 min PM), and at 24 h PM. Color was determined at deboning and again at 24 h PM. Gel strength, cook loss, expressible moisture, total protein solubility, and bound phosphorylase quantities were determined on the fillets at 24 h PM. There was no difference in carcass temperature at 15 min PM, but by 105 min PM each temperature treatment was significantly different, with the carcasses chilled at 0 and 10°C having the lowest temperature, the 30°C-chilled birds having the highest temperature, and the 20°C-chilled carcasses being intermediate but significantly different from either extreme. The carcass temperature differences at 105 min PM indicated that the carcass experienced differing chilling rates. To varying degrees, slower rates of chilling resulted in lower pH, greater degree of lightness (L* value), greater cook loss, and reduced gel strength. However, chilling rate had no effect on total protein solubility or myofibrillar phosphorylase for any of the treatments. Chilling rate seems to contribute to PSE turkey meat characteristics but by a mechanism independent of total protein solubility or myofibrillar phosphorylase.

(*Key words*: meat color; meat functionality; muscle metabolism; pale, soft, exudative meat; water-holding capacity)

2004 Poultry Science 83:1039–1046

INTRODUCTION

With genetic selection for rapid growth and a shift from whole bird processing to further processed products, turkey processors have observed an increase in the number of meat quality problems. Specifically, processors have observed an increase in turkey products such as formed breast loaves and rolls with poor water-holding capacity, pale color, poor texture, and poor cohesiveness (Owens et al., 2000). These pale, soft, and exudative (PSE) characteristics are similar to defects observed in pork and are referred to as PSE meat (Barbut, 1996; McKee and Sams, 1998).

Products made with PSE meat are unacceptable not only to producers but also to consumers who object to the pale color and increased drip loss found in packages (Swatland, 1993; Ferket and Foegeding, 1994; Dransfield and Sosnicki, 1999). Pale, soft, and exudative meat increases the amount of purge in cook-in bags, which results in reduced cook yield, poor meat binding, and a soft product texture with poor sliceability (Owens et al., 2000). These products create economic losses for processors in reduced yield, wasted materials, extra labor required to repackage these products, and customer dissatisfaction.

The development of PSE meat is directly related to biochemical changes occurring in the muscle during rigor mortis development, specifically a rapid postmortem (PM) metabolism resulting in a rapid pH decline early PM (Penny, 1969; Pearson and Young, 1989; Sante et al., 1995; Lawrie, 1998; Dransfield and Sosnicki, 1999). Low pH combined with high carcass temperatures early PM may cause protein denaturation resulting in PSE-like characteristics (Briskey and Wismer-Pedersen, 1961; Penny, 1969).

Postmortem factors such as chilling regimens can also influence the development of PSE meat (Honikel and

Received for publication February 20, 2003.

Accepted for publication January 20, 2003.

[1]Current address: Texas Tech University, Department of Animal Science and Food Technology, Lubbock, TX 79409.

[2]To whom correspondence should be addressed: asams@poultry.tamu.edu.

Abbreviation Key: L* value = degree of lightness; PM = postmortem; PSE = pale, soft, and exudative; TBST = Tris-buffered saline with Tween.

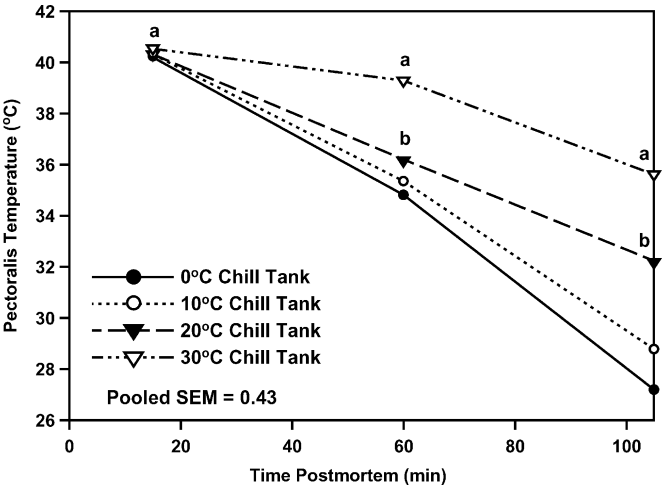

FIGURE 1. Turkey pectoralis temperature at 15, 60, and 105 min postmortem (PM) from carcasses chilled at 0, 10, 20, and 30°C. [a–c]Means within PM time with different letters are different ($P < 0.05$).

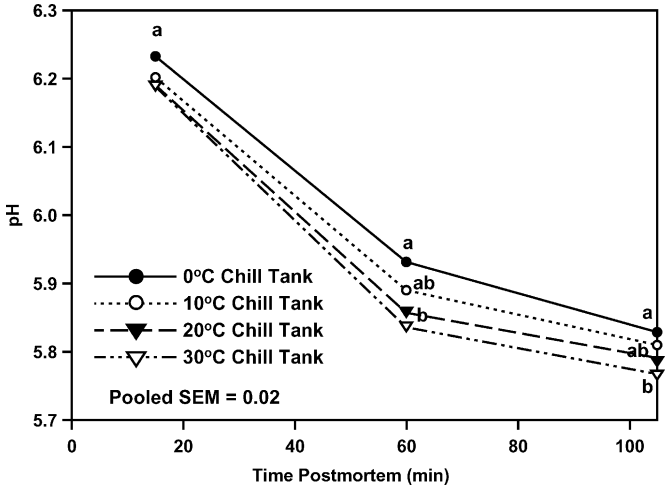

FIGURE 2. Postmortem (PM) pH decline in carcasses chilled at 0, 10, 20, and 30°C. [a,b]Means within the same PM time with different superscripts differ ($P < 0.05$).

Fischer, 1977; Offer, 1991). It is well established that PM temperature is an important factor influencing overall meat quality in pork (Lee and Choi, 1999). Studies in turkeys have indicated that PM temperatures of 30 to 37°C accelerated PM metabolism (Khan and Frey, 1971). McKee and Sams (1998) also found that holding carcasses at an elevated temperature of 40°C accelerated PM glycolysis and resulted in pale meat with increased drip loss and cook loss compared with carcasses chilled at 0°C. Alvarado and Sams (2002) found that color, water-holding capacity, texture, and product integrity are negatively affected in slow-chilled turkey carcasses. These researchers suggested that turkey carcasses should be chilled rapidly so that the pectoralis muscle temperature reaches 25°C or less by 60 min PM to prevent poor meat quality, specifically PSE-like meat characteristics.

The decreased water-holding capacity of PSE meat has been associated with denaturation of the myofibrillar proteins, whereas pale color is more often associated with denaturation of sarcoplasmic proteins (Offer, 1991). The rapid pH decline and high PM temperatures can also affect other proteins. For example, phosphorylase, an enzyme important in glycogenolysis, is present in skeletal muscle at approximately 2.5 mg of protein per g tissue (Scopes, 1970). Fischer et al. (1979) simulated PSE conditions in pork muscle and found that the solubility and activity of phosphorylase were reduced early PM at high temperatures (38 to 40°C) and low pH (< 6.0). Pietrzak et al. (1997) reported that there was a reduction in solubility of phosphorylase in PSE turkey breast muscle and that it was mainly precipitated at the Z-line and in the central portion of the A bands. Therefore, they suggested that phosphorylase was insolubilized and deposited onto myofibrils resulting in decreased myosin solubility.

Because slow chilling is a factor that can contribute to the PSE condition, developing a time and temperature range for chilling turkey carcasses may decrease problems associated with water-holding capacity and pale meat. Therefore, the objectives of this study were to evaluate the relationships between muscle chilling rates, myosin denaturation, and the development of PSE-like meat characteristics. In the present study, chiller water temperatures of 0, 10, 20, and 30°C were selected to simulate possible carcass temperatures observed in the industry. Water-holding capacity, color, gel strength, total protein solubility, and phosphorylase quantities were evaluated to determine the time and temperature relationship between muscle chilling rate and protein denaturation.

MATERIALS AND METHODS

At 22.5 wk of age, 48 Nicholas male turkeys weighing between 25 and 27 kg were processed in each of 2 trials. Feed was withdrawn from the pens at 12 h before processing, but the birds had access to water until transportation to the slaughter facility (2 h before processing). In each trial, the birds were hung on a shackle line and exsanguinated by a unilateral neck cut severing the right carotid artery and jugular vein. Preslaughter stunning was not used in this experiment because it is not required for poultry by US law (Goodwin et al., 1960; Bilgili, 1992; USDA, 1997), and it interferes with rigor mortis development (Murphy et al., 1988; Papinaho and Fletcher, 1995; Poole and Fletcher, 1998). After a 3-min bleeding period, the turkeys were scalded at 61°C for 45 s, defeathered in a rotary drum picker[2] for 25 s, and manually eviscerated. Following evisceration, the carcasses were chilled in 0, 10, 20, or 30°C agitated immersion water tanks for 45 or 90 min before deboning. The left turkey breast fillet was deboned after 45 min of chilling (60 min PM), leaving the skin and underlying fascia on the remaining fillet undisturbed at the keel bone. The right fillet was har-

[2]Model SP3055, Brower Corp., Haughton, IA.

vested after 90 min of chilling (105 min PM). These times were selected to mimic typical deboning times in commercial plants visited by the authors. Muscle characteristics in this early PM time period are important as the meat is frequently used directly in product formulation.

Temperature and pH of the turkey breast muscle were recorded at 15 min PM (following picking), at deboning, and at 24 h PM using a portable pH meter[3] and a piercing probe.[4] At the time of deboning (60 or 105 min PM) and at 24 h PM, degree of lightness (L* value)[5] was measured on the medial surface of each fillet. The middle portion of the fillet was used for determination of probe penetration gel strength and weight loss during cooking (Alvarado and Sams, 2002) at the time of deboning and at 24 h PM. A separate sample from the middle portion of the fillet was frozen in liquid nitrogen for determination of protein solubility and bound phosphorylase by electrophoresis and densitometry.

Myofibril Extraction

Myofibrils were extracted using modified procedures of Warner et al. (1997) and Bee et al. (1999). Briefly, 3 g of turkey breast muscle was homogenized (on ice) 1:7 (wt/vol) with rigor buffer (10 m*M* immidazole, 75 m*M* KCl, 2 m*M* ethylene glycol tetra acetic acid, 2 m*M* $MgCl_2$, and 2 m*M* NaN_3, pH 7.2) using a Polytron Tissuemizer[6] for 15 s on and 15 s off for 2 cycles. The homogenate was centrifuged for 10 min at 1,500 × *g* at 4°C. The supernatant was discarded and the pellet was resuspended in another 7 vol of rigor buffer and centrifuged again for 10 min at 1,500 × *g* at 4°C. The supernatant was discarded, the pellet was resuspended 1:1.5 (wt/vol) in extraction buffer (0.1 *M* $Na_4P_2O_7$, 5 m*M* ethylene glycol tetra acetic acid, 5 m*M* 1,4-Dithio-DL-threitol, and 5 μg/mL leupeptin), and incubated on ice for 1 h. Following incubation, the samples were centrifuged at 40,000 × *g* for 30 min at 4°C. The myofibrillar supernatant fraction was removed for SDS-PAGE analysis. The pellet was resuspended 1:3 (wt/vol) in extraction buffer. Protein solubility of the myofibrillar fraction and the pellet were determined using the Bradford protein analysis (Bradford, 1976).

SDS-PAGE

Samples were subjected to SDS-PAGE for quantitation of phosphorylase in the supernatant and pellet fractions. Samples were diluted to a final concentration of 1 μg/μL. Samples were then further diluted (1:1) with SDS buffer (62.5 m*M* Tris HCl, 2% SDS, 2% glycerol, and 0.05% bromophenol blue), heated for 3 min at 100°C, cooled to room temperature, and loaded on the gel at 10 μL (5 μg of protein) per lane. A high molecular weight standard mixture[7] was applied to each gel as well as 2 concentrations of phosphorylase (0.05 and 0.1 μg/μL). The reservoir buffer used during electrophoresis was 1× Tris/glycine/SDS buffer.[8] The Linear Gradient gels[9] (15-well, 15 μL/well, 4%–15% T Tris-HCl) were run for 1.5 h using a 107 mA current at room temperature. Gels were stained in Gelcode Blue Stain[10] for 1 h and destained overnight with deionized, distilled H_2O.

Densitometry

Gels were placed between 2 cellulose sheets and dried with Gel-Dry Drying Solution[11] for 20 min. Gels were allowed to dry overnight before scanning with an imaging densitometer.[12] The program Molecular Analyst[13] was used to analyze the image and determine concentrations of phosphorylase. Results were expressed in nanograms per five micrograms of total protein applied to the gel in each lane.

Western Blot Analysis

Proteins were transferred from an unstained gel to a piece of 0.2 μm polyvinylidene fluoride[14] using a modified procedure described by Harlow and Lane (1998). The transfer buffer contained 25 m*M* Tris, 192 m*M* glycine, 20% methanol, and 0.05% SDS. The current was 30 mA for 12 h, 60 mA for 2 h, and 90 mA for 30 min, at 4°C. Following transfer, the membranes were blocked in Tris buffered saline with Tween (TBST) containing 5% bovine serum albumen (BSA) overnight. Following incubation, the membrane was washed 3 times with TBST and incubated for 3 h with the primary antibody (polyclonal anti-phosphorylase[15]) diluted 1:1,000 with TBST. The membrane was then washed 3 times with TBST and once with 1× alkaline phosphatase buffer. Following washing, the membrane was incubated for 1 h with alkaline phosphatase-labeled secondary antibody[16] diluted 1:30,000 with TBST. The membrane was then transferred to a solution containing 5-bromo-4-chloro-3-indolyl phosphate/nitroblue tetrazolium for color development. The reaction was stopped with 20% EDTA and the membrane rinsed in deionized, distilled H_2O.

Statistics

The data were subjected to ANOVA using the GLM procedure of SAS (1990). Means were separated using Duncan's multiple range test with a significance level of $P < 0.05$ (SAS Institute, 1990). Because there were no trial by treatment and replication by treatment interactions, the data from the trials were pooled by treatment.

[3]IQ 240 pH meter, IQ Scientific Instruments, Inc., San Diego.
[4]pH 26-SS, IQ Scientific Instruments, Inc., San Diego.
[5]Minolta Chroma Meter, Model CR-200, Minolta Corp., NJ.
[6]Polytron Tissuemizer, Model ST1018, Tekmar Co., Cincinnati, OH.
[7]Precision Protein Standards, 161-0372, Bio-Rad, Hercules, CA.
[8]Tris/glycine/SDS Buffer, 161-0732, Bio-Rad.
[9]Linear Gradient gels, 161-1122, Bio-Rad.
[10]Gelcode Blue Stain, 24590, Pierce, Rockford, IL.
[11]Gel-Dry Drying Solution LC4025, Invitrogen, Carlsbad, CA.
[12]Model G6-690, Bio-Rad.
[13]Molecular Analyst program, Bio-Rad, Hercules, CA.
[14]Trans-Blot, 162-0180, Bio-Rad.
[15]Polyclonal Antiphosphorylase, Biogenesis, Kingston, NH.
[16]Phosphatase Labeled Secondary Antibody, A5187, Sigma, St. Louis, MO.

TABLE 1. Means of meat quality parameters at deboning or 24 h postmortem (PM) for turkey pectoralis fillets deboned at 60 min PM

Parameter	Chiller temperature (°C)				Pooled SEM
	0	10	20	30	
pH (at 24 h PM)	5.85[a]	5.85[a]	5.83[a]	5.74[b]	0.02
L* value[1] (at 24 h PM)	53.12	53.63	53.38	53.52	0.24
Cook loss (%) (at deboning)	16.87[b]	18.52[ab]	17.82[ab]	21.40[a]	0.72
Cook loss (%) (at 24 h PM)	18.9	19.25	18.68	20.51	0.44
Gel strength (kN) (at deboning)	21.45[a]	18.38[ab]	19.3[a]	13.24[b]	1.12
Gel strength (kN) (at 24 h PM)	19.94[a]	17.46[ab]	12.64[b]	16.98[ab]	0.85

[a,b]Means with no common superscript within each row differ ($P < 0.05$).

[1]L* = degree of lightness (color).

RESULTS AND DISCUSSION

The effect of chilling rates on temperature decline of the pectoralis muscle in this study is shown in Figure 1. At 15 min PM, there were no temperature differences among any of the treatment groups. At 60 min PM, the temperature of the carcasses in the 30°C chill tank was significantly higher (39°C) than in the remaining treatments. By 105 min PM, the temperature of the carcasses chilled at 20 and 30°C was significantly higher than the carcasses chilled at 0 and 10°C, which were not significantly different from each other. A similar pattern of carcass temperature decline was noted by Alvarado and Sams (2002). However, the overall carcass temperatures were greater in this study compared with Alvarado and Sams (2002) due to the increased size and age of the birds in this study. With the increase in BW and therefore, breast size, there is a decrease in chilling rate which can influence pH decline, water-holding capacity, and gel strength.

A decline in pH is related to the accumulation of lactic acid within the muscle due to PM glycolysis (Lawrie, 1991). There were no differences in pH between temperature treatments at 15 min PM (Figure 2). However, by 60 min PM, the carcasses chilled at 20 and 30°C had a significantly lower pH compared with those chilled at 0°C. At 105 min PM, the carcasses chilled at 30°C had significantly lower pH than those chilled at 0°C. These results indicated that carcasses held at 30°C during chilling had a more rapid pH decline than those held at 0°C. Warris and Brown (1987) reported that the rate of pH decline within 1 h PM was the most important factor in PSE meat, with a faster decline at higher temperatures resulting in PSE pork. On the other hand, Fernandez et al. (1994) reported that ultimate pH had a significant effect on the development of PSE meat. In the present study, the carcasses with the lowest ultimate pH at 24 h PM were those chilled at 30°C for either 60 or 105 min (Tables 1 and 2). However, both carcass temperature and PM pH decline are important in determining the extent of protein denaturation. Breast fillets with an internal temperature of approximately 36°C or higher reached an ultimate pH earlier PM, suggesting a faster rate of rigor development compared with more rapidly chilled carcasses (Figure 3). In the carcasses chilled for 90 min, there was a more rapid decline in pH until muscle temperature reached approximately 28°C (Figure 4).

Color is an important attribute for customer satisfaction. The L* value of the meat depends upon the amount of light scattered. Swatland (1993) reported that an increased scattering of light due to denaturation of the sarcoplasmic proteins and an increase in the amount of extracellular water was responsible for pale meat. The effects of chilling at 0, 10, 20, and 30°C on turkey breast meat color are shown in Tables 1 and 2. In fillets deboned at 60 min PM, the carcasses chilled at 30°C had significantly higher L* values, indicating lighter fillets than the carcasses chilled at 0°C (Figure 5). At 105 min PM, carcasses chilled at 10, 20, and 30°C had significantly higher L* values than those chilled at 0°C (Figure 6). However, by 24 h PM, there were no differences among any of the treatment groups chilled for either 45 or 90 min (Tables

TABLE 2. Means of meat quality parameters at deboning or 24 h postmortem (PM) for turkey pectoralis fillets deboned at 105 min PM

Parameter	Chiller temperature (°C)				Pooled SEM
	0	10	20	30	
pH (at 24 h PM)	5.85[a]	5.81[ab]	5.78[b]	5.73[c]	0.01
L* value[1] (at 24 h PM)	53.22	53.06	53.47	54.2	0.28
Cook loss (%) (at deboning)	20.21[b]	23.75[a]	20.82[b]	22.10[ab]	0.48
Cook loss (%) (at 24 h PM)	18.15[b]	21.23[a]	21.58[a]	21.67[a]	0.39
Gel strength (kN) (at deboning)	20.78[a]	19.44[ab]	15.96[b]	16.67[ab]	1.03
Gel strength (kN) (at 24 h PM)	17.23	15.93	16.68	15.99	1.15

[a–c]Means with no common superscript within each row differ ($P < 0.05$).

[1]L* = degree of lightness (color).

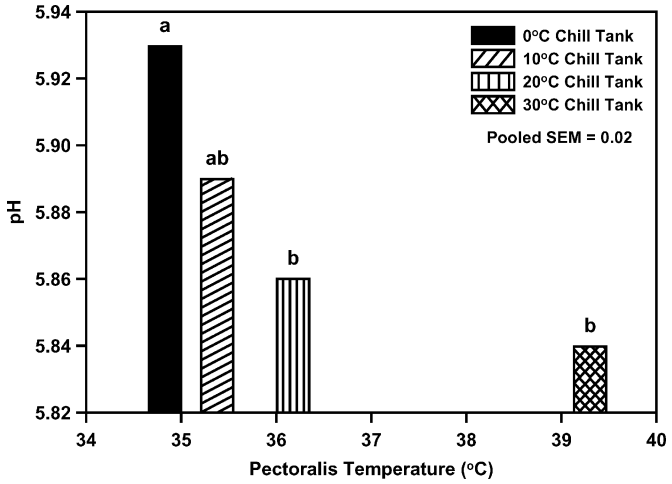

FIGURE 3. Pectoralis temperatures and postmortem (PM) pH decline in birds deboned at 60 min PM. [a,b]Means with different superscripts are different ($P < 0.05$).

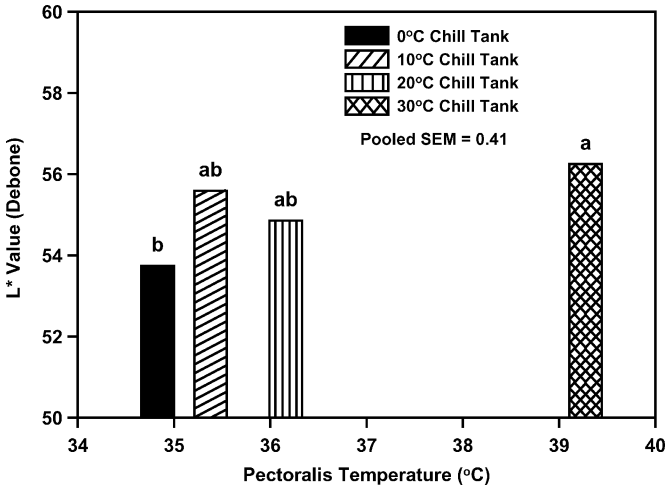

FIGURE 5.. Pectoralis temperatures and L* values from carcasses deboned at 60 min postmortem. [a,b]Means with different superscripts are different ($P < 0.05$). L* = degree of lightness (color).

1 and 2). These chilling temperatures may not be elevated sufficiently to cause irreversible denaturation. Therefore, even though differences can be observed at 60 and 105 min PM, once the fillets have been chilled on ice for 24 h following deboning, the denaturation may be reversed. The effect of carcass temperatures on L* value are given in Figures 5 and 6. At 60 min PM, there was an increase in L* value to greater than 54 between 35 and 36°C (Figure 5). Owens et al. (2000) suggested that at an L* value of 54 or greater there is reduced protein functionality, and that these turkey fillets may have reduced water-holding capacity. At 105 min PM, there was a significant increase in L* values of fillets having a muscle temperature of 28°C or higher, indicating lighter fillets (Figure 6).

Water-holding capacity of the meat is affected by PM pH decline (Hedrick et al., 1989). As pH declines, the proteins approach their isoelectric point where there is a net charge of zero and therefore a minimum number of charges available to bind water (Pearson and Young, 1989). Cook loss was measured in this study as an indirect measure of water-holding capacity, and the results are reported in Tables 1 and 2. Carcasses chilled at 30°C for 45 min had higher cook loss percentage at deboning compared with those chilled at 0°C. Santos et al. (1994) stated that the rapid rate of pH decline combined with high carcass temperatures resulted in a denaturation of proteins, which caused meat with increased drip loss and cook loss. Offer (1991) suggested that the shrinkage of the myosin head from 19 to 17 nm during low pH and high carcass temperatures results in more fluid being expelled into the extracellular space; this water can then be lost as drip or cook loss. At 24 h PM, the carcasses chilled at 0°C for 45 min were not different from the other treatment groups. Therefore, rapidly chilling carcasses can reduce cook loss.

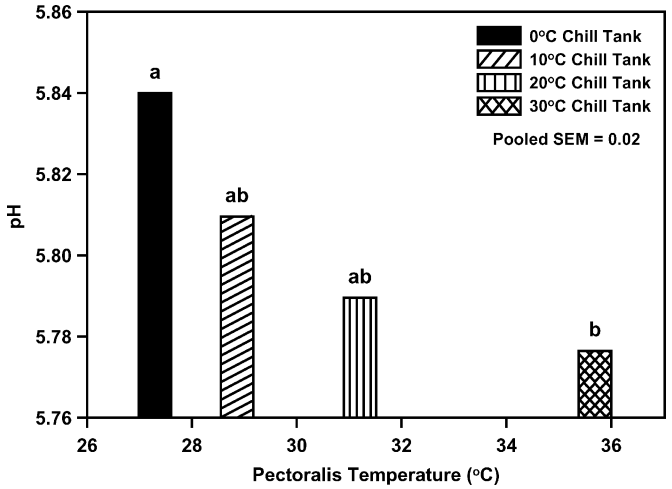

FIGURE 4. Pectoralis temperatures and postmortem (PM) pH decline in birds deboned at 105 min PM. [a,b]Means with different superscripts are different ($P < 0.05$).

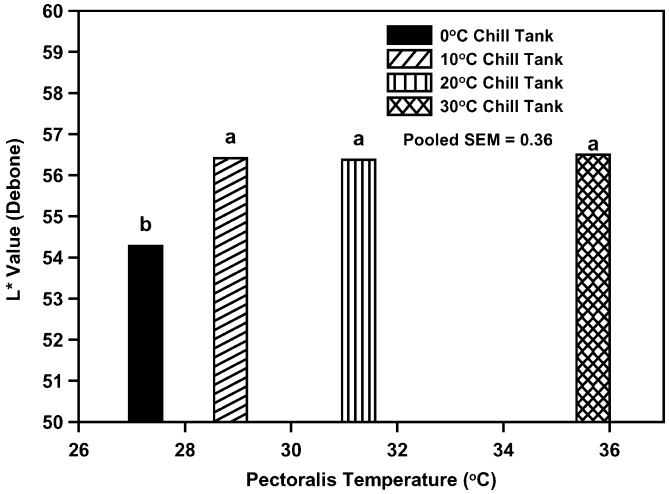

FIGURE 6. Pectoralis temperatures and L* values from carcasses deboned at 105 min postmortem. [a,b] Means with different superscripts are different ($P < 0.05$). L* = degree of lightness (color).

Pale meat has been negatively correlated with gel strength, indicating that as the meat becomes lighter, a softer texture is obtained (Barbut, 1996). The effects of chilling rate on gel strength are shown in Tables 1 and 2. For carcasses immersed in the tanks for 45 min, gel strength significantly decreased as the temperature of the chilling solution increased. This was true when gel strength was measured at deboning or at 24 h PM. The carcasses immersed in the 20°C chill tank for 90 min had significantly lower gel strength compared with those immersed at 0°C, but were not significantly different from 10 and 30°C chill water immersion. Because myosin is the most abundant and most functional muscle protein, any alterations in solubility can affect texture and water-holding capacity. According to Samejima et al. (1969), damage to myosin can decrease gel strength. Bendall and Wismer-Pedersen (1962) reported that sarcoplasmic proteins precipitated onto myofibrils, mainly myosin, resulting in a decrease of protein solubility. Because low pH and high carcass temperatures early PM can lead to increased protein denaturation, a decrease in myosin solubility will decrease the strength of heat-induced gels, which can cause sliceability problems in cooked whole muscle products. At 24 h PM, the gel strength from the carcasses chilled at 0°C and deboned at 60 min PM was significantly higher than that of carcasses chilled at 30°C. This difference in gel strength could have resulted from a decrease in the solubility of specific proteins in the 30°C treatment compared with the 0°C treatment. However, there were no differences in gel strength in any of the chilled carcasses deboned at 105 min PM. Once again, this lack of difference may be due to the renaturation of the proteins once the samples were properly chilled following deboning.

Pietrzak et al. (1997) reported that precipitation of the sarcoplasmic protein phosphorylase onto the myofibrillar proteins was responsible for the decreased solubility of myosin. If such precipitation results in decreased myofibrillar protein solubility, then it could result in decreased water-holding capacity and gel strength. Therefore, phosphorylase precipitation was measured in this study to determine the effect of chilling rates on protein denaturation. The results of the Western blot are in shown Figure 7 and confirm that phosphorylase is located just below the 100 kD molecular weight standard. The results of the phosphorylase precipitation are shown in Figure 8 and Table 3. There were no significant differences in phosphorylase suspended in the myofibrillar fraction from carcasses deboned at 60 or 105 min PM. However, the pellet fraction from the carcasses deboned at 60 min and chilled at 30°C had significantly higher amounts of bound phosphorylase compared with the remaining treatment groups, possibly indicating a decrease in protein functionality. These results are consistent with the 30°C treatment group having a lower gel strength (Table 1). Also, the carcasses chilled at 30°C had a more rapid pH decline resulting in a lower ultimate pH reached at an earlier time PM. This could have resulted in an increase in phosphorylase precipitation onto the myofibrils. At 105 min PM, carcasses chilled at 30°C had significantly higher phosphorylase in the pellet fraction compared with the 0 and 10°C treatments. These results indicate that chilling carcasses at high temperatures and for longer periods causes an increased precipitation of phosphorylase.

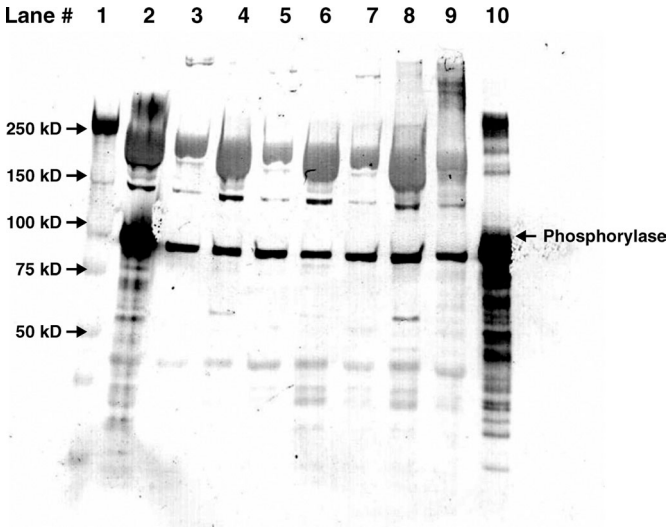

FIGURE 7. Western blot of turkey myofibrils using anti-phosphorylase. Lane identification is as follows (left to right): lane 1 = molecular weight standard; lanes 2 and 10 = phosphorylase; lanes 3 and 4 = myofibrillar and pellet fractions from carcasses chilled at 0°C; lane 5 = myofibrillar fraction from carcasses chilled at 10°C; lanes 6 and 7 = myofibrillar and pellet fractions from carcasses chilled at 20°C; lanes 8 and 9 = myofibrillar and pellet fractions from carcasses chilled at 30°C. All samples were chilled for 90 min.

Total protein solubility in the myofibrillar fraction is reported in Table 3. There were no significant differences in protein solubility in any of the myofibrillar fractions. The lack of differences noted in the myofibrillar fraction used for determination of total solubility as well as concentration of phosphorylase could be due to such a small proportion of the protein present being precipitated at

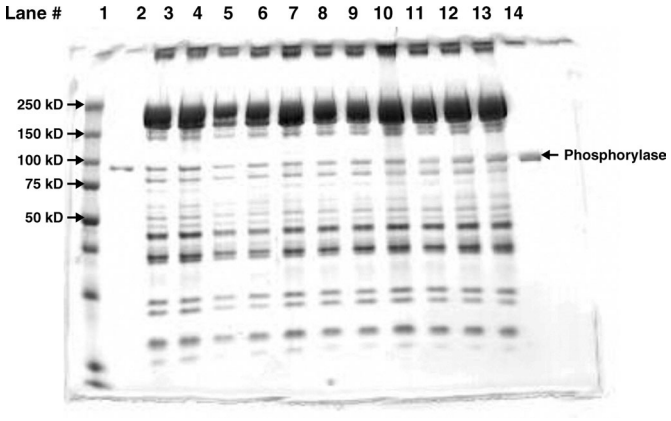

FIGURE 8. SDS-PAGE analysis of myofibrillar proteins. Lane identification is as follows (left to right): lane 1= molecular weight standard; lanes 2 and 14 = phosphorylase standards; lanes 3 and 4 = samples from 0°C carcasses; lanes 5 to 7 = samples from 10°C carcasses; lanes 8 to 10 = samples from 20°C carcasses; and lanes 11 to 13 = samples from 30°C carcasses.

TABLE 3. Means of total protein solubility and phosphorylase for turkey pectoralis fillets deboned at 60 and 105 min postmortem (PM)

Parameter	Chiller temperature (°C)				Pooled SEM
	0	10	20	30	
Myofibrillar phosphorylase[1] (60 min PM)	25.00	18.36	23.08	13.64	1.14
Myofibrillar phosphorylase (105 min PM)	26.27	19.36	24.82	26.64	1.01
Precipitated phosphorylase (60 min PM)	52.36[b]	45.50[b]	53.92[b]	90.27[a]	0.05
Precipitated phosphorylase (105 min PM)	33.90[b]	36.08[b]	51.91[ab]	73.42[a]	0.05
Total protein solubility[2] (60 min PM)	23.52	24.69	23.34	25.13	0.64
Total protein solubility (105 min PM)	23.50	25.17	26.14	24.48	0.86

[a–c]Means with no common superscript within each row differ ($P < 0.05$).

[1]Phosphorylase is reported as nanograms of phosphorylase per five micrograms of protein applied per lane.

[2]Total protein solubility is reported as milligrams of protein per milliliter of extract.

the chilling temperatures used in this study. If the solubility of only a small fraction of the proteins was affected by the pH and temperature conditions, the treatment effects on protein solubility may have been overshadowed by the remaining unaffected proteins. Additionally, solubility changes in specific proteins may be more relevant to cooking loss and gel strength changes than a total myofibrillar protein measurement. This could explain a difference in gel strength in the absence of a difference in a measurement of general myofibrillar protein solubility.

The results of this study indicate that there is a relationship between chilling time and temperature, with 30°C chilling temperatures for 45 and 90 min resulting in PSE meat. Furthermore, these quality effects seem to be related to the reduction in phosphorylase solubility. Therefore, processors should chill turkey carcasses rapidly in the early PM period to prevent poor meat quality problems.

REFERENCES

Alvarado, C. Z., and A. R. Sams. 2002. The role of carcass chilling rate in the development of pale, exudative turkey pectoralis. Poult. Sci. 81:1365–1370.

Barbut, S. 1996. Estimates and detection of the PSE problem in young turkey breast. Can. J. Anim. Sci. 76:455–457.

Bee, G., M. B. Solomon, S. M. Czerwinski, C. Long, and V. G. Pursel. 1999. Correlation between histochemically assessed fiber type distribution and isomyosin and myosin heave chain content in porcine skeletal muscles. J. Anim. Sci. 77:2104–2111.

Bendall, J. R., and J. Wismer-Pedersen. 1962. Some properties of the fibrillar proteins of normal and watery pork muscle. J. Food Sci. 27:144–159.

Bilgili, S. F. 1992. Electrical stunning of broilers-basic concepts and carcass quality implications: A review. J. Appl. Poult. Res. 1:135–146.

Bradford, M. 1976. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein-dye binding. Anal. Biochem. 72:248–254.

Briskey, E. J., R. W. Bray, W. G. Hoekstra, P. H. Phillips, and R. H. Grummer. 1959. The chemical and physical characterization of various pork ham muscle classes. J. Anim. Sci. 18:146–152.

Briskey, E. J., and J. Wismer-Pedersen. 1961. Biochemistry of pork muscle structure. I. Rate of anaerobic glycolysis and temperature change versus the apparent structure of muscle tissue. J. Food Sci. 26:297–305.

Dransfield, E., and A. A. Sosnicki. 1999. Relationship between muscle growth and poultry meat quality. Poult. Sci. 78:743–746.

Ferket, P. R., and E. A. Foegeding. 1994. How nutrition and management influence PSE in poultry meat. Pages 64–78 in Proceedings from BASF Technical Symposium, Multi-State Poultry Feeding and Nutrition Conference, Indianapolis, IN.

Fernandez, X., A. Forslid, and E. Tornberg. 1994. The effect of high postmortem temperature on the development of pale, soft, and exudative pork: Interaction with ultimate pH. Meat Sci. 37:133–147.

Fischer, C., C. R. Hamm, and K. O. Honikel. 1979. Changes in solubility and enzymatic activity of muscle glycogen phosphorylase in PSE muscle. Meat Sci. 3:11–15.

Goodwin, T. L., W. C. Mickelberry, and W. J. Stadelman. 1960. The influence of humane slaughter on the tenderness of turkey meat. Poult. Sci. 39:921–924.

Harlow, E., and D. Lane. 1998. Using Antibodies. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY.

Hedrick, H. B., E. D. Aberle, J. C. Forrest, M. D. Judge, and R. A. Merkel. 1989. Principle of Meat Science. 3rd ed. Kendall/Hunt Publishing Co., Dubuque, IA.

Honikel, K. O., and C. Fischer. 1977. A rapid method for detection of PSA and DFD porcine muscles. J. Food Sci. 42:1633–1636.

Khan, A. W., and A. R. Frey. 1971. A simple method for following rigor mortis development in beef and poultry. Can. Inst. Food Technol. 4:139–142.

Lawrie, R. A. 1991. Pages 56–60 in Meat Science. 5th ed. Pergamon Press, New York.

Lawrie, R. A. 1998. Lawrie's Meat Science. 6th ed. Woodhead Publishing, Cambridge, UK.

Lee, Y. B., and Y. I. Choi. 1999. PSE (pale, soft, exudative) pork: The causes and solutions— Review. Asian-australas. J. Anim. Sci. 12:244–252.

McKee, S. R., and A. R. Sams. 1998. Rigor mortis development at elevated temperatures induces pale exudative turkey meat characteristics. Poult. Sci. 77:169–174.

Murphy, B. S., R. J. Hasiak, and J. G. Sebranek. 1988. Effect of antemortem electrical stunning on functional properties of turkey muscle. Poult. Sci. 67:1062–1068.

Offer, G. 1991. Modeling of the formation of pale, soft, and exudative meat: Effects of chilling regime and rate and extent of glycolysis. Meat Sci. 30:157–184.

Owens, C. M., E. M. Hirschler, S. R. McKee, R. Martinez-Dawson, and A. R. Sams. 2000. The characterization and incidence of pale, soft, exudative turkey meat in a commercial plant. Poult. Sci. 79:553–558.

Papinaho, P. A., and D. L. Fletcher. 1995. Effect of stunning amperage on broiler breast muscle rigor development and meat quality. Poult. Sci. 74:1527–1532.

Pearson, A. M., and R. B. Young. 1989. Muscle and Meat Biochemistry. Academic Press, Inc. San Diego.

Penny, I. F. 1969. Protein denaturation and water-holding capacity in pork muscle. J. Food Technol. 4:269–273.

Pietrzak, M., M. L. Greaser, and A. A. Sosnicki. 1997. Effect of rapid rigor mortis processes on protein functionality on

Pectoralis major muscle of domestic turkeys. J. Anim. Sci. 75:2106–2116.

Poole, G. H., and D. L. Fletcher. 1998. Comparison of a modified atmosphere stunning-killing system to conventional electrical stunning and killing on selected broiler breast muscle rigor development and meat quality attributes. Poult. Sci. 74:342–347.

Samejima, K., Y. Hishimoto, and T. Fukazawa. 1969. Heat gelling properties of myosin, actin, actomyosin, and myosin subunits in a saline model system. J. Food Sci. 34:242–248.

Sante, V., A. A. Sosnicki, M. L. Greaser, M. Pietrzak, E. Pospiech, and O. Ouali. 1995. Impact of turkey breeding and production on breast meat quality. Pages 151–56 in Proceedings of XII European Symposium on the Quality of Poultry Meat. Zaragoza, Spain.

Santos, C., L. C. Roserio, H. Goncalves, and R. S. Melo. 1994. Incidence of different pork quality categories in a Portuguese slaughterhouse: A survey. Meat Sci. 38:279–287.

SAS Institute. 1990. SAS/STAT User's Guide: Statistics. Version 6. 1st ed. SAS Institute Inc., Cary, NC.

Scopes, R. K. 1970. Characterization and study of sarcoplasmic proteins. Pages 471–492 in The Physiology and Biochemistry of Muscle as a Food. 2nd ed. Briskey, E. J., R. G. Cassens, B. B. Marsh, ed. University of Wisconsin Press, Madison, WI.

Swatland, H. J. 1993. Paleness, softness, and exudation in pork-Review. Pages 273–286 in Pork Quality: Genetic and Metabolic Factors. E. Poulanne and D. I. Demeyer, eds. C. A. B. International, Wallingford, UK.

United States Department of Agriculture. 1997. Code of Federal Regulations, Poultry Products Inspection Regulations. Title 9, Vol. 1, Part 381.65c. United States Government Printing Office, Washington, DC.

Warner, R. D., R. G. Kauffman, and M. L. Greaser. 1997. Muscle protein changes postmortem in relation to pork quality traits. Meat Sci. 45:339–352.

Warris, P. D., and S. N. Brown. 1987. The relationship between initial pH, reflectance and exudation on pig muscle. Meat Sci. 20:65–74.