Effect of Processing on Turkey Meat Quality and Proteolysis

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ABSTRACT Modern processing techniques for turkey involve rapid chilling to slow microbial growth and early deboning of the economically important breast meat. This paper shows that these 2 processes lead to significantly tougher meat with higher cooking losses. The toughening appears to be due to less extensive proteolysis and shortening of the sarcomeres. Calpains I and II and their inhibitor, calpastatin, were quantified in turkey breast. Calpain II was the more common isoform but showed no evidence of activation during aging. In contrast, calpain I and calpastatin activities declined rapidly and were no longer detected 24 h postslaughter. There was no evidence of an association between calpain activity and processing conditions.

(*Key words*: meat quality, turkey, breast meat, tenderness, calpain)

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INTRODUCTION

In recent years 2 factors have driven changes in the way turkeys are processed. First, there has been an increase in the use of processed turkey products (Owens and Sams, 1997). To meet this demand under the constraints of efficient plant operation requirements for reduced labor and energy, it has been more economical to remove the important retail cuts, such as breast meat, from the carcass as early as possible. Indeed, it is now common to debone turkey carcasses within 3 h of slaughter (Owens and Sams, 1997). A second related factor is the need to chill the carcasses rapidly to slow the growth of bacteria. The proliferation of bacteria determines the shelf life of poultry products (Zuckerman and Abraham, 2002). Rapid chilling also overcomes the potential problem of pale, soft, and exudative meat, which occurs in turkey breast and pork meat products (Briskey, 1964; Alvarado and Sams, 2002). Pale, soft, exudative is associated with the accelerated onset of rigor mortis in combination with a high carcass temperature. This quality defect is characterized by the pale color, soft texture, and poor water-holding capacity of the meat (Sosnicki et al., 1998).

However, previous research with broilers has shown that rapid chilling and early deboning of carcasses adversely affects other meat quality attributes (Wakefield et al., 1989; Lyons et al, 1992; Dunn et al., 1993). Anecdotal evidence has suggested that these processing practices have led to tougher turkey breast meat. It is well documented in other species that rapid cooling prior to the completion of rigor mortis can interfere with the tenderizing process (Bendall, 1978). In beef (Huff-Lonergann et al., 1996; Boehm et al., 1998), lamb (Koohmaraie, 1996; Morton et al, 1999; Geesink et al., 2000), and pork (Ouali, 1990; Claeys et al., 2001), tenderization has been associated with the breakdown of the myofibrillar proteins by calcium-dependent proteases or calpains. There is also evidence that rapid cooling of meat with a relatively high pH can directly toughen the meat by contraction of the sarcomeres or interfere with the tenderization process by altering the calpain activity (Hwang et al., 2003). As calpains are also present in chicken (Birkhold and Sams, 1994) and turkey muscles (Northcutt et al., 1998a) they may have a similar role in the tenderization process in these species.

The purpose of the first experiment in this report was to determine the relative importance of rapid cooling and early deboning in determining the tenderness of turkey breast meat. A second experiment investigated the mechanism of tenderization and the possible involvement of the calpains.

MATERIALS AND METHODS

Bird Processing

The effects of chilling and early deboning on meat quality were determined in 2 replicate experiments, the first with 12 turkeys and the second with 24 turkeys. As they

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Abbreviation Key: MFI = myofibrillar fragmentation index.

both gave similar results, only the second larger experiment is reported here. Twenty-four turkeys that were 17 wk old (9 to 10 kg) were electrically stunned and slaughtered by exsanguination at a local commercial processing plant. The carcasses were scalded, defeathered, washed, and manually eviscerated. The carcasses were cooled rapidly in a 4°C water bath followed by a 1°C ice-slush bath. After 30 to 40 min in the cooling baths, they were transferred to a 0°C chiller for 2 h more before deboning. To determine the effect of early deboning, the right breast muscle was removed from the bone, and the left breast was left intact on the carcass. For treatment 1, carcasses with the intact breast muscle and excised breast muscle from 12 birds were subjected to blast freezing (–24°C) for 2 h before being returned to the 0°C chiller. In treatment 2, the carcasses and excised breast muscles from the other 12 birds were placed directly in the chiller. At 24 h postmortem, all carcasses and muscles were removed from the 0°C chiller. The left breast muscles were removed from the carcasses. Subsamples were taken from all the breast muscles for pH, temperature, sarcomere length, myofibrillar fragmentation index (MFI), and shear force determinations. The remainder of each breast muscle was vacuum-packed and aged at 4°C for 6 d more. These samples were used to determine shear force and MFI values at 7 d postmortem.

For the second experiment, 10 turkeys that were 10 wk old (mean weight of 6.2 kg) were used to study changes in calpain characteristics over the aging period. Four birds were removed from the processing line and at-death samples taken within 5 min postslaughter. The remaining 6 birds were processed as described for treatment 1 by using the blast freezing procedure.

Temperature and pH Measurements

The temperature and pH of turkey breast muscle were measured on the processing line at 30, 45, 70, and 200 min postmortem. A single incision was made in the middle of the breast muscle. The pH and temperature of the breast muscle were measured by inserting into the cut pH and temperature probes[2] attached to a portable pH meter[3] (Morton et al., 1999).

Shear Force

Breast meat samples for shear force measurement were cooked individually in plastic bags immersed in a water bath at 80°C until they reached an internal temperature of 75°C as measured by temperatures probes attached to a meter.[4] The cooked meat was cooled on ice, and at least 10 test pieces ($10 \times 10 \times 25$ mm) were cut from the samples and placed separately in a MIRINZ tenderometer[5] (Chrystall and Devine, 1985), and the shear force (N) to cut the fibers was determined. Mean values were calculated for each sample.

Cooking Loss

Cooking loss was determined by weighing breast muscle sample and then cooking in a plastic bag immersed in a water bath at 80°C until it reached an internal temperature of 75°C. The cooked meat was cooled rapidly in an ice-water bath to less than 3°C and reweighed. Cooking loss was determined as the loss in weight as a percentage of the initial weight.

MFI and Sarcomere Length

The MFI was determined by a modification of the method of Culler et al. (1978). A 2 g sample of muscle tissue was homogenized[6] in 20 mL of 4°C MFI buffer (20 m*M* phosphate buffer, pH 7.0 containing 100 m*M* KCl, 1 m*M* EDTA, and 1 m*M* sodium azide). The homogenate was centrifuged at $1{,}000 \times g$, the supernatant was discarded, and the pellet resuspended in 20 mL of MFI buffer and recentrifuged at $1{,}000 \times g$. The sediment was resuspended in 10 mL of MFI buffer and passed through a polyethylene strainer to remove connective tissue and debris. Protein concentration of these washed myofibrils was determined in triplicate using the biuret method (Gornall et al., 1949). The absorbance at 540 nm of the myofibrillar suspensions (0.5 mg/mL) was measured in duplicate with an ultraviolet/visible spectrometer[7] and multiplied by 200 to obtain the MFI values. Washed myofibrils were also used to determine the sarcomere length using the filar micrometer method as described by Cross et al. (1980). Sarcomere lengths were determined on at least 20 myofibrils per sample.

Calpain Separation and Assay

Calpain I and II and calpastatin were extracted using the method described by Koohmaraie (1990) with some modifications. Turkey breast samples (5 g) removed within 5 min of slaughter and at 3 and 24 h postslaughter were homogenized in 30 mL of precooled extraction buffer (100 m*M* Tris-HCl, 10 m*M* β-mercaptoethanol, and 10 m*M* EDTA, pH 8.3) with a Polytron. The homogenate was centrifuged at $27{,}000 \times g$ for 30 min at 4°C, and the supernatant was filtered through glass wool and cheesecloth. The filtrate was dialyzed (12,000 to 14,000 molecular weight cut-off) against 40 m*M* Tris, 3 m*M* EDTA, and 10 m*M* β-mercaptoethanol, pH 7.5, for 16 h and centrifuged at $27{,}000 \times g$ for 15 min at 4°C. Calpains I and II and calpastatin were separated on a 20×1.5 cm DEAE-Sepharose Fastflow column[8] (Morton et al., 1999). The dialyzed supernatant was applied to the column, which was then washed with 100 mL of elution buffer (40 m*M* Tris, 0.5

[2]Orion 8163 glass electrode, Orion Research Inc., Beverly, MA.
[3]Hanna HI 9025, Hanna Instruments Inc., Woonsocket, RI.
[4]Fluke Type K temperatures probe and Fluke 52 meter, Fluke Corp., Everett, WA.
[5]MIRINZ Tenderometer, Agresearch MIRINZ, Hamilton, New Zealand.
[6]Unicam 8625 UV/VIS spectrometer, ATI Unicam, Cambridge, UK.
[7]Polytron PT-3100, Kinematica AG, Littau, Switzerland.
[8]Pharmacia, Uppsala, Sweden.

TABLE 1. Effect of changing processing conditions on turkey meat quality at 24 h postslaughter[1]

Time boned	Blast frozen	Shear force (N)	Cooking loss (%)	MFI[2]	Sarcomere length (μm)
3 h	No	84.4[a] ± 10.9	19.3[a] ± 1.61	54.8[b] ± 5.50	1.69[b] ± 0.20
3 h	Yes	100.9[a] ± 24.5	20.3[a] ± 1.98	51.3[b] ± 6.80	1.70[b] ± 0.18
24 h	No	45.1[b] ± 4.8	12.1[b] ± 0.42	83.9[a] ± 10.6	2.05[a] ± 0.14
24 h	Yes	53.4[b] ± 4.2	12.1[b] ± 0.32	74.0[ab] ± 18.2	1.92[a] ± 0.11

[a,b]Means within the same column with different superscripts are significantly different ($P < 0.05$).

[1]There were 12 replicate breast muscles for each treatment combination.

[2]MFI = myofibrillar fragmentation index.

m*M* EDTA, and 10 m*M* β-mercaptoethanol, pH 7.5) to remove unbound proteins. Subsequently, the calpains and calpastatin were eluted at 2 mL/min by a 2-stage procedure consisting of a 200-mL linear 0 to 175 m*M* NaCl gradient followed by a steeper 100-mL linear gradient of 175 to 500 m*M* NaCl. Both gradients were in elution buffer. The active fractions were identified using a BODIPY fluorescent microplate assay[9] (Thompson et al., 2000) and pooled.

The pooled fractions were reassayed quantitatively with casein as a substrate (Dayton et al., 1976). Aliquots (1 mL) of column fractions were incubated at 25°C with 1 mL of 0.7% casein in 100 m*M* Tris-HCl, 10 m*M* mercaptoethanol, and 1 m*M* NaN3 containing 100 μL of 0.1 *M* $CaCl_2$ or 0.2 *M* EDTA. After 30 min, 2 mL of 5% trichloroacetic acid was added and centrifuged at 3,000 × *g* for 15 min. The absorbance of the supernatant was read at 278 nm (1-cm light path). A unit of calpain was defined as the amount that gave a calcium-dependent increase of 1.0 unit of absorption in 1 h. Calpastatin was determined and expressed as inhibitory equivalents of calpain II.

Statistical Analysis

All data were analyzed using analyses of variance.[10] Means were separated using Student's *t*-test at a significance level of $P < 0.05$, and mean results are followed by the standard deviation in brackets.

RESULTS AND DISCUSSION

Experiment 1: Effects of Early Deboning, Blast Freezing, and Postmortem Aging on Meat Quality

The combination of the 2 cooling baths lowered the mean internal breast temperature from 36.5 ± 2.35°C at 30 min to 11.0 ± 1.01°C) by 70 min postslaughter. Two hours in the chiller after the cooling baths reduced the mean temperature to 7.8 ± 0.64°C. The application of blast freezing had a significant effect on the average temperature of the breast muscles at 6 h postslaughter. The temperature was 3.3 ± 0.14°C compared with 7.8 ± 0.64°C for those muscles that remained in the chiller. There were no significant differences between the mean temperatures of muscles deboned and those muscles remaining on the carcass.

The mean pH of the carcasses was 6.69 (± 0.13) at 30 min after slaughter. The pH declined steadily to 6.0 (± 0.17) at the time of deboning. Neither early deboning nor blast freezing had any significant effect on pH. The mean pH of all breast muscles at 24 h postmortem was 5.93 (± 0.09). The rate of pH decline in the turkey breast muscle was most rapid over the first 30 min of the postslaughter period. This result is consistent with previous studies that show the breast muscle of turkey exhibits accelerated rigor mortis compared with beef or lamb muscles (Sosnicki, 1993; Wynveen et al., 1999).

Early deboning resulted in tougher meat compared with the breast muscle that remained attached to the carcass, which was significantly more tender at 1 and 7 d postslaughter (Tables 1 and 2). The use of blast freezing increased the mean shear force of the meat by 20 to 40%, irrespective of whether the muscle was attached or detached from the carcass. However, the toughening effect was only significant at 7 d in the muscles that had been left on the bone for 24 h. Early boning caused significant differences in cooking losses at 24 h postslaughter, but the difference had disappeared by 7 d. These results do not support those reported by Alvarado and Sams (2000) who suggested that deboning turkey breast at 2 h postmortem or later will not significantly impair meat tenderness.

Turkey shear force values from the MIRINZ tenderometer have been shown to be highly correlated with Warner-Bratzler shear values (Bekhit et al., 2003). With the conversion factor from this experiment, the turkey aged on the carcass would be moderately tender at 24 h, whereas the meat that was boned at 3 h would be tough at 1 d and only slightly tender by 7 d (Lyon and Lyon, 1990).

The tenderness of meat depends on the extent that sarcomeres have shortened during the rigor process and the amount of postmortem proteolysis. Measurements were taken to determine the contribution of each of these 2 processes to the toughening effects of early boning and blast freezing (Tables 1 and 2). Removal of breast muscle from the carcass at 3 h postmortem resulted in a significant shortening of the sarcomeres and would have con-

[9]BODIPY fluorescent microplate assay, Molecular Probes, Inc., Eugene, OR.

[10]Minitab Statistical Software Package, Version 13.1, 2000, Minitab Inc., State College, PA.

TABLE 2. Effect of changing processing conditions on turkey meat quality at 7 d postslaughter[1]

Time boned	Blast frozen	Shear force (N)	Cooking loss (%)	MFI[2]	Sarcomere length (μm)
3 h	No	62.6[a] ± 11.1	11.9[a] ± 0.32	100.4[a] ± 17.7	1.77[b] ± 0.14
3 h	Yes	80.4[a] ± 17.4	12.0[a] ± 0.26	93.4[a] ± 18.9	1.73[b] ± 0.09
24 h	No	31.9[c] ± 8.7	11.7[a] ± 0.45	117.8[a] ± 14.3	1.96[a] ± 0.07
24 h	Yes	42.6[b] ± 10.9	11.8[a] ± 0.27	112.2[a] ± 10.5	1.89[a] ± 0.06

[a–c]Means within the same column with different superscripts are significantly different ($P < 0.05$).

[1]There were 12 replicate breast muscles for each treatment combination.

[2]MFI = myofibrillar fragmentation index.

tributed to the toughening of the meat. The shortening of the sarcomere length was due to rigor mortis not being complete before muscle excision. Hence, the muscles experienced cold shortening due to the loss of skeletal restraint (Sams et al., 1990). However, the corresponding muscles left on the bone for 24 h before deboning did not cold-shorten due to the restraint provided by the skeletal framework. A previous report has shown that rapid chilling of turkey carcasses produced meat with shorter sarcomeres and higher, more variable, toughness (Wakefield et al., 1989). The combination of the temperature of the muscle being below 10°C while the pH is above 6 are the conditions whereby meat could undergo cold shortening (Dunn et al., 1993).

The meat that was boned out at 3 h postslaughter also showed significantly less proteolysis compared with that left on the carcass for the first 24 h as measured by MFI at 24 h postmortem (Table 1). At 7 d postmortem the MFI values were still lower in the hot-boned breast meat, but the difference was not significant. The reduced proteolysis may be associated with the shorter sarcomeres in that the proteases may be unable to access their substrates in contracted muscles. Blast freezing the muscles caused no significant differences in sarcomere length or MFI.

The effect of extended aging on the tenderness of turkey breast meat was also evaluated in this study. The results showed that tenderness improved by 20 to 30% after 7 d of aging. Postmortem aging of muscles improves red meat tenderness (Goll et al., 1992; Taylor et al., 1995; Geesink and Koohmaraie, 1999). There is, however, insufficient data in the literature on the effects of extended aging on the tenderness of turkey breast meat. Numerous studies have evaluated the tenderness of chicken fillets deboned prerigor and subsequently aged for 20 to 72 h after deboning (Sams et al., 1990; Lyon et al., 1992; Smith et al., 1992; McKee et al., 1997). Lyon et al. (1992) observed that increasing the aging time after prerigor boning had no significant effects on meat tenderness. In contrast, Hirschler and Sams (1994) observed a significant decrease in shear force values between 24 and 72 h of post-excision refrigerated aging.

The insignificant changes in sarcomere lengths of the turkey breast muscle during aging does not explain the significant improvement in the tenderness of the muscles originating from carcasses deboned at 3 h postslaughter and 24 h postmortem, respectively. Thus, the improvement in the tenderness over the 6 d extended aging period must have been due to other mechanisms involved in the postmortem tenderization such as proteolysis (Koohmaraie, 1992) or ionic strength differences (Ouali, 1992).

Experiment 2: Calpain Levels in Turkey and Changes with Aging

The effects of early deboning and blast freezing on the biochemical parameters and meat quality parameters were similar in the second experiment to those in the first experiment. The mean pH of the breast muscle was 6.02 ± 0.14 at 3 h postmortem and declined to an ultimate pH of 5.84 ± 0.07 by 24 h. There was no difference in pH between deboned breast muscle and muscle still on the carcass. However, early boning significantly toughened the breast meat and this was associated with significantly higher MFI and shorter sarcomeres (Table 3).

The levels of calpains I and II and their inhibitor, calpastatin, in breast muscle were determined in 4 carcasses immediately postmortem. Samples were also taken for analysis of the calpains and their inhibitor from deboned

TABLE 3. Changes in physical and biochemical parameters associated with postmortem storage in experiment 2[1]

	Shear force (N)	MFI[2]	Sarcomere length (μm)
Off bone[3]	126.4[a] ± 35.3	27.0[b] ± 4.83	1.78[b] ± 0.09
On bone[4]	47.3[b] ± 13.7	50.2[a] ± 5.69	1.98[a] ± 1.01

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$).

[1]There were 6 replicates for each treatment.

[2]MFI = myofibrillar fragmentation index.

[3]Breast muscles that were removed from the carcass 3 h postslaughter.

[4]Breast muscles that remained on the carcass for 24 h.

TABLE 4. Changes in the calpains and their inhibitor associated with postmortem storage

	Postslaughter time			
Measurement	At death[1]	3 h[2]	Off bone (24 h)[2]	On bone (24 h)[2]
Calpain I activity	0.46[a] ± 0.14	0.08[b] ± 0.06	ND[3]	ND
Calpain II activity	2.81 ± 0.39	2.70 ± 0.21	2.50 ± 0.27	2.57 ± 0.19
Calpastatin activity	0.64[a] ± 0.09	0.23[b] ± 0.09	ND	ND

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$).

[1]Measurement was on 4 birds.

[2]Measurement was on 6 birds.

[3]ND = not detected.

breasts and breasts still on the carcass of 6 more carcasses at 3 and 24 h postmortem. The calpain I and calpastatin levels declined significantly over the first 3 h and were not detected 24 h postmortem. There were no significant changes in the levels of calpain II. Although the MFI values indicated that some of the differences in shear force were due to reduced proteolysis, there was no evidence that early deboning had any effect on the calpain system (Table 4).

The activity of calpain I in the breast muscle at death was 0.46 units/g of muscle, which was higher than that reported by Northcutt et al. (1998b), although the birds were of similar weights in both experiments. The differences in calpain activity may be due to the lower ionic strength of the buffer used by Northcutt et al. (1998b) to extract the enzyme since Veiseth and Koohmaraie (2001) demonstrated that extractable calpain and calpastatin activity in ovine muscle depended on the buffer used. The loss in calpain I activity during aging was expected due to its activation and subsequent autolysis (Koohmaraie, 1990; Morton et al., 1999). McKee et al. (1997) and Walker et al. (1995) have reported that calpain I activity is absent from pectoralis muscles of broilers at 24 h postmortem.

Residual activity of calpain II in turkey breast muscle at death was 2.81 units/g of muscle. Northcutt et al (1998b) reported an activity of between 1.2 and 1.8 units/g of muscle in a 5-wk-old turkey breast, whereas McKee et al (1997) reported 5.99 units/g of muscle in a 48-d-old broilers. The variations in calpain II activity in these reports and the results of this experiment may be related to differences in the age of the birds and extraction buffer. There was no significant difference among calpain II activities at death, 3 h postmortem, and 24 h postmortem. These results confirm those reported by Ducastaing et al. (1985), Etherington et al. (1987), Koohmaraie et al. (1987), and McKee et al. (1997) who demonstrated that normal postmortem aging had little effect on calpain II across species. In contrast Walker et al. (1995) observed a substantial loss in calpain II activity in broiler pectoralis muscles during postmortem aging; however, they did not specify the extraction buffer used in the experiment.

Calpastatin activities at death and 3 h postslaughter were 0.64 and 0.23 units/g of muscle, respectively. Calpastatin was reported to retain 20% of its at-death activity in beef longissimus muscle at 24 h postmortem (Koohmaraie et al., 1987). Although calpastatin was detected in turkey breast muscle at 3 h postslaughter, the levels were no longer detectable at 24 h. The loss in calpastatin activity during postmortem storage is probably a result of its degradation by calpain I or other endogenous proteases (Vidalenc et al., 1983; Koohmaraie et al., 1988).

In summary, this study has shown that the introduction of rapid cooling and early deboning of carcasses during turkey processing have the potential to cause significant deterioration in the quality of the breast meat. Removal of breast meat from the carcass at 3 h led to a marked toughening of the meat. However, there was no evidence that the toughening was related to the activity of the calpain proteases.

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