The Effect of Seasonal Heat Stress on Rigor Development and the Incidence of Pale, Exudative Turkey Meat

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ABSTRACT

Heat stress is one of the prominent ante-mortem stressors that elicits pale, soft, and exudative meat characteristics in stress-susceptible pigs. Industry reports of exudative turkey meat increase in the early summer with the onset of prolonged high temperatures. To study the effect of seasonal heat exposure on turkeys, 122 17-wk-old Nicholas tom turkeys were subjected in January either to growth temperatures of 16/24 C (night/day) (control) or to elevated temperatures of 32/38 C (night/day) (heat-stressed, HS). Turkeys were processed at 21 wk of age in a manner simulating commercial conditions. Pectoralis muscle samples were taken at 15 min (prechill), 2 h (postchill), and 24 h and analyzed for R-value, pH, and color. At 2 h, the remaining intact Pectoralis muscle was harvested, aged on ice for 23 h, and analyzed for drip loss and cook loss. Percentage mortality and carcass weights were not significantly different between treatments. By 15 min post-mortem, the HS birds exhibited a faster pH decline and had higher R-values that persisted through 24 h. The HS birds were also paler in color and exhibited increased drip loss and cook loss when compared to controls; however, expressible moisture was not different between treatments. In addition, the HS birds had a higher frequency of abnormal birds than controls when birds were grouped as normal (L* < 53) or abnormal (L* > 53).

(Key words: heat stress, turkey meat, pale soft exudative meat, rigor mortis development)

INTRODUCTION

During the early summer season, the turkey industry reports substantial losses in yield due to formed turkey breast products with poor water-holding capacity, poor texture, and pale color. These meat characteristics are consistent with those observed in pale, soft, and exudative (PSE) pork. In pork, genetic selection for heavier, leaner muscling has resulted in animals that are genetically susceptible to various ante-mortem and post-mortem stressors, which induce physiological and biochemical changes in the muscle that result in the development of PSE meat characteristics (Cassens et al., 1975). More specifically, Briskey (1964) postulated that the low pH resulting from rapid metabolism early (prior to chilling) post-mortem when combined with high carcass temperatures caused extensive protein denaturation in the muscle. The loss of protein functionality due to extensive protein denaturation is considered to be the primary factor associated with the development of PSE meat characteristics (Warris and Brown 1987; Fernandez et al., 1994; Santos et al., 1994).

Heat stress has long been recognized as one of the prominent environmental elements influencing meat quality (Thomas et al., 1966; Howe et al., 1968; Aberle et al., 1969; Wood and Richards, 1975; Northcutt et al., 1994). Many studies have focused on resulting meat quality from heat stress applications just prior to slaughter. Sayre et al. (1963) reported stress-susceptible pigs that were subjected to heat stress (42 to 45 C for 20 to 60 min) prior to slaughter developed PSE meat characteristics. Northcutt et al. (1994) found that chicks that were subjected to heat (40 to 41 C for 1 h) and preconditioned (3 d at 35 to 36 C for 3 h) exhibited PSE meat characteristics. However, Northcutt (1994) found that turkeys subjected to 30 C for 1 h had lower initial pH after slaughter than controls, but they did not differ in lightness or water-holding capacity from the controls. In addition, Froning et al. (1978) found that turkeys exposed to immediate preslaughter heat stress did not exhibit PSE meat characteristics.

Researchers have not studied the effect of a sustained seasonal heat stress on turkey meat quality and the development of PSE meat characteristics; however, industry reports of PSE turkey meat increase in early summer with the onset of prolonged high temperatures. In addition, Santos et al. (1994) stated that the percentage of PSE pork carcasses doubles during the summer months and attributes this to higher environmental temperatures and relative humidity. Therefore, the objective of this study was to evaluate meat characteristics of turkeys that had been subjected to a sustained,
seasonal heat stress that was modeled after the normal temperature changes observed in the climate of the southern U.S. during the transition from spring to summer. The reports of turkey PSE meat diminish during the latter part of the summer as birds have become acclimated to the heat and are smaller in body size. Therefore, the timing of this heat application during the growth cycle was important, because it appears that heavier body weight is a factor in the way growing strains of birds respond to environmental heat stress (Bohren et al., 1982).

MATERIALS AND METHODS

A total of 132 Nicholas toms (12 wk of age) were obtained from a commercial grower. Birds were transported (<160 km) to the Texas A&M University Poultry Research Center, where they were equally divided into three replicate groups. The turkeys were housed in litter-covered floor pens and consumed a commercial ration (20% crude protein and 3,080 kcal ME/kg) ad libitum. The birds were provided a 5-wk period to acclimate to their new housing and recover from transportation. At 17 wk of age, the birds in each replicate were divided into equal groups and placed into either an environment that simulated seasonal heat exposure or a control environment (day/night temperature 24/16 C). These environments were created by partitioning the birds into separate growing areas having 12 3.05 × 1.83 m pens in each section with each area having its own heating controls and windows for exposure to the natural outside environment. The heat-stressed birds were exposed to increasing environmental temperatures over a 3-d period to a final day/night temperature of 38/32 C. To produce a stressful hot environment for the heat-stressed birds, as well as to avoid a cold-stress environment for the control birds (the study began in January), supplemental heating from a forced air natural gas heaters (47,445 kJ) was provided as needed to achieve and maintain the desired temperatures. Additional gas heaters were positioned throughout the elevated-temperature environment. Ceiling fans were positioned between pens to circulate air and minimize temperature differences within and between pens. To regulate the heat, temperature was monitored and recorded twice daily at the level of the birds’ head in the center of each growing area. Relative humidity was also monitored and recorded twice daily; however, no attempt was made to regulate or equalize humidity. It was determined that there was a much greater fluctuation in humidity from night to day and from day to day than between the heat stress and control environments. Furthermore, Reece et al. (1972) reported that birds that had been acclimated to heat stress were not affected by relative humidity. Mortality was monitored and recorded twice daily.

At 21 wk of age (after 4 wk in the temperature environments), the turkeys in the three replicated groups were processed on 3 separate processing days with a total of 41, 44, and 37 turkeys being killed on Days 1, 2, and 3, respectively. Following a 12-h period of feed withdrawal on each processing day, turkeys were hung on shackles and killed by bleeding for 90 s from a single neck cut severing the right carotid artery and jugular vein. Birds were not electrically stunned prior to exsanguination because this type of stunning has been reported to retard rigor mortis development, and this study required rigor to develop without any conflicting factors (Papinaho and Fletcher, 1995). After bleeding, birds were subscalded2 at 63 C for 45 s, defeathered in a rotary drum3 picker for 35 s, and manually eviscerated. At 15 min post-mortem, birds were weighed and banded and tissue samples were taken for biochemical analyses. A lengthwise incision was made in the skin covering the right breast muscle in preparation for sampling. Muscle tissue samples were cut (20 × 30 × 30 mm) parallel to the muscle fiber from the right Pectoralis of each carcass at 15 min (prechill), 2 h (postchill), and 24 h post-mortem. Pectoralis muscle samples were cut at least 30 mm apart from the previous sample area. Muscle samples for biochemical analyses were placed in labeled plastic bags, placed directly into liquid nitrogen, and stored at −75 C until analyzed (<1 mo). Pectoralis muscle samples for determination of expressible moisture were placed in a plastic bag, aged on ice until 24 h post-mortem, and then analyzed using the press method described by Urbin et al. (1962). Immediately after the samples were removed, lightness (L* value) was evaluated4 in triplicate on the cut surface of the remaining breast muscle. Location of sampling in the Pectoralis muscle was randomized between carcasses and sampling times to account for any variation within the muscle.

Carcasses were placed in a manually agitated tap water-ice slush after the 15 min sampling period. The skin covering the breast was clamped5 after sampling to minimize water contact with the muscle. After sampling at 2 h post-mortem for biochemical analyses, both breast muscles were removed. The left, undisturbed breast muscle was weighed and both the left and right breast muscles were placed into 8.46 L zip seal, perforated-plastic bags and then stored on ice for an additional 22 h. Following the 22 h storage, right fillets were sampled for biochemical analyses and left fillets were reweighed in order to determine drip loss. Left fillets were baked on racks in foil covered aluminum pans in an air convection oven6 at 177 C to an internal temperature of 76 C. Cooked fillets were reweighed for cook loss determination.

Tissue samples at 15 min, 2 h, and 24 h post-mortem were used for the measurement of R-value and pH. The
R-value, the ratio of inosine to adenosine, is an indication of adenosine triphosphate (ATP) depletion in the muscle and was determined using the method described by Thompson et al. (1987). The post-mortem pH of samples was determined using the iodoacetate method described by Sams and Janky (1986).

Mortality and carcass weight data was subjected to chi-square analysis. The rest of the data were classed by treatment and replication and the remaining parameters were subjected to ANOVA using the General Linear Models procedure (SAS Institute, 1985). Because no interaction was detected between treatment and replication, the data from all replicates were pooled into a completely randomized block design with post-mortem time as the blocking factor. The residual mean square was used as the error term and treatment means were compared utilizing the F test (SAS Institute, 1985).

RESULTS AND DISCUSSION

There was no difference in mortality (7.14% for heat stressed vs 4.29% for controls) or carcass weights (13.06 kg for heat-stressed vs 16.27 kg for controls) between the two treatment groups. Heat stress has been associated with increased mortality (Phelps, 1990); however, the birds in the current study were acclimated to the elevated temperatures over a 3-d period. Acclimation has been previously shown to reduce mortality related to heat stress (Reece et al., 1972). Although bird weight was not affected by the applied heat stress in the current study, chronic heat stress has been shown to result in a 20% reduction in growth rate in fast-growing strains of poultry (Bohren et al., 1981). Bohren et al. (1982) reported that although preconditioning or acclimation increased the survival rate of fast-growing strains of heat stressed poultry, it did not improve growth performance.

The R-value is an indirect measure of ATP depletion in the muscle. During rigor mortis development, ATP in the muscle is depleted and R-value increases (Calkins et al., 1982). The R-value data presented in Figure 1 indicate that by 15 min post-mortem, the heat-stressed birds had significantly higher R-values than controls; this trend persisted through 24 h post-mortem. These results indicated that the seasonal heat stress application accelerated ATP depletion, thereby hastening the onset of rigor mortis. Moreover, Pietrzak et al. (1994) reported that turkeys characterized as PSE due to rapid post-mortem metabolism, pale color, and poor water-holding capacity exhibited lower ATP levels by 20 min post-mortem than the group with slower post-mortem metabolism.

The effects of seasonal heat stress on post-mortem pH decline are shown in Figure 2. The decline in pH is a result of the breakdown of glycogen to lactic acid during post-mortem glycolysis, thereby resulting in the accumulation of lactic acid in the muscle (Lawrie, 1991). Results in Figure 2 illustrate that as early as 15 min post-mortem, the heat-stressed birds exhibit a significantly lower pH than the controls. The accelerated pH decline in the heat stress birds persisted through 24 h post-mortem. Ma and Addis (1973) and Vanderstoep and Richards (1974) have demonstrated that maximum differences in post-mortem pH decline between fast and slow glycolyzing turkeys occurred within 1 h post-mortem, decreasing thereafter during post-mortem aging. Although the current study did not measure the pH
heat stress treatment significantly (previously described in pork. In the current study, the poultry is susceptible to a PSE condition similar to that meat is associated with low pH and concluded that van Hoof (1979) reported that the pale color in poultry values than the slower glycolyzing group. In addition, exhibiting a rapid pH decline had significantly higher L* and Pietrzak controls. These results agree with those of Barbut (1993) stressed birds exhibited greater L*values than the Figure 3. At 15 min, 2 h, and 24 h, heat-fillets of both heat-stressed and control birds are Accelerated rigor mortis development occurring with poor water-holding capacity and light color is indicative of PSE meat (Cassens et al., 1975; Fox et al., 1980; Warris and Brown, 1987). Lawrie (1991) attributed the pale color meat to the denaturation of sarcoplasmic proteins, which causes increased light scattering in the muscle. The lightness (as indicated by L*values) of turkey breast fillets of both heat-stressed and control birds are illustrated in Figure 3. At 15 min, 2 h, and 24 h, heat-stressed birds exhibited greater L*values than the controls. These results agree with those of Barbut (1993) and Pietrzak et al. (1994), who reported that turkeys exhibiting a rapid pH decline had significantly higher L* values than the slower glycolyzing group. In addition, van Hoof (1979) reported that the pale color in poultry meat is associated with low pH and concluded that poultry is susceptible to a PSE condition similar to that previously described in pork. In the current study, the heat stress treatment significantly (P < 0.01) increased the percentage of birds that would be classified as PSE-like (36.9 vs 10.5%) according to the criterion of 24 h L*values greater than 53 being PSE-like. This criterion was selected to be more conservative than the L*value criteria used by Barbut (1993) in identifying abnormally pale meat. This result suggests that the heat may have caused sufficient stress to trigger the development of the PSE-like condition in the muscle. Ante-mortem stressors such as mixing, breeding, and heat have previously been reported to trigger the PSE condition in swine (Louis et al., 1993).

Data in Table 1 indicates the extent to which heat stress influenced drip loss, cook loss, and expressible moisture of turkey breast fillets. Heat stress significantly increased drip loss and cook loss but appeared to have no effect on expressible moisture. The increased drip loss and cook loss were expected because they are directly associated with protein functionality in the muscle.

Santos et al. (1994) postulated that the early development of rigor mortis in PSE pork meat combined with high carcass temperatures caused the denaturation of muscle sarcoplasmic and contractile proteins, which resulted in meat with poor water-holding capacity, which was reflected by higher drip loss and cooking losses. Moreover, Warris and Brown (1987) found that the early post-mortem pH decline from 30 min to 1 h was determined to be the most important factor in determining the degree of protein denaturation and resulting exudate in meat. However, the fact that there was no difference in expressible moisture between treatments was not expected because changes in expressible moisture usually follow a similar trend to changes observed in drip loss and cook loss. In this study, samples for expressible moisture were collected and analyzed at 24 h post-mortem possibly after much of the expressible moisture had been lost to drip. Earlier (15 min and 2 h) samples may have been more meaningful in determining changes in expressible moisture between treatments over time.

In conclusion, the current study suggested that the seasonal heat stress accelerated post-mortem metabolism and biochemical changes in the muscle, which produced pale, exudative meat characteristics in tom turkeys.

**ACKNOWLEDGMENTS**

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**TABLE 1. The effect of seasonal heat stress on drip loss, cook loss, and expressible moisture in turkey Pectoralis**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pooled SEM</th>
<th>Environmental treatments</th>
</tr>
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<tbody>
<tr>
<td>Drip loss, %</td>
<td>0.08</td>
<td>0.44*</td>
</tr>
<tr>
<td>Cook loss, %</td>
<td>0.51</td>
<td>24.56*</td>
</tr>
<tr>
<td>Expressible moisture, %</td>
<td>0.62</td>
<td>22.02</td>
</tr>
</tbody>
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*Means (n = 61 per mean) within each row with no common superscript differ significantly (P < 0.05).
REFERENCES


