Breast meat quality of broiler chickens can be affected by managing the level of nitric oxide

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ABSTRACT The objective of this study was to investigate the effects of nitric oxide (NO) on the quality of broiler chicken meat during postmortem storage. Eighteen broiler chickens were slaughtered and breast meat was randomly assigned to 1 of 3 treatments including the control group, nitric oxide synthase (NOS) inhibitor group, and NO enhancer group. The breast samples were incubated with water, NO enhancer, and NO inhibitor for 1 d and then stored at 4°C under atmospheric conditions for 4 and 7 d. Left side of breast meat was used to determine protein oxidation, lipid oxidation, water-holding capacity, and pH, whereas the right side was used for the measurement of color and drip loss. Breast meat from NO enhancer group showed the lowest water-holding capacity during refrigerated storage, whereas drip losses were not significantly ($P > 0.05$) different among 3 treatments. Lipid oxidation showed a significant difference ($P < 0.05$) only at 7 d in which NOS inhibitor group showed the highest, whereas the NO enhancer group showed the lowest levels of lipid oxidation. Carbonyl content in NO enhancer group was significantly higher than the control and NOS inhibitor treatment at 1 and 4 d of refrigerated storage. Lightness of breast meat in the NOS inhibitor group was greater, whereas redness was lower than the control and NO enhancer group at 4 and 7 d of refrigerated storage. The ultimate pH of 3 treatments was not significantly ($P > 0.05$) different from one another. These data indicate that NO could play a significant role in modulating the quality of fresh broiler breast meat during refrigerated storage.

Key words: chicken breast, drip loss, lipid oxidation, nitric oxide, protein oxidation

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INTRODUCTION

Nitric oxide (**NO**)-induced protein nitrosylation has been gradually recognized as an important posttranslational modification of protein structure and function in biological systems (Stamler and Meissner, 2001). However, few studies have been conducted to investigate the possible roles of protein nitrosylation and oxidation in the area of meat science. Therefore, the effects of NO and NO-induced nitrosylation and oxidation on fresh meat quality are largely unknown.

Reactions between NO and sarcoplasmic reticulum Ca^{2+}-ATPase (**SERCA**) or ryanodine receptor (**RyR**) can nitrosylate some critical thiols in those proteins and cause leakage of calcium from the sarcoplasmic reticulum and increase of calcium content in muscle cells (Bellinger et al., 2008; Stamler et al., 2008). N-Nitro-L-arginine methyl ester injection [NO synthase (**NOS**) inhibitor] was shown to increase the rate of postmortem glycolysis, which was evidenced by reduced glycogen content and increased lactate concentration in the semimembranosus of preslaughtered exercised lambs. The proposed mechanism is that NO stimulates the activation of soluble guanylate cyclase to increase the production of cyclic guanosine momophosphate and improve glucose uptake in skeletal muscles (McConell and Kingwell, 2006). These modifications could possibly contribute to the variation of metabolism and the rate of pH decline in muscles during the early postmortem stage. In addition to nitrosylation, NO can react with superoxide to produce peroxynitrite, which is a strong oxidant for lipids and proteins (Rubbo et al., 1994). Protein oxidation and nitrosylation, especially calpain, a key enzyme of protein degradation during postmortem, are very likely to influence the aging of fresh meat, and thus regulate fresh meat quality (Rowe et al., 2004; Zhang et al., 2008, 2011).

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Nitric oxide is produced during the conversion of L-arginine to citrulline and is catalyzed by NOS. Previous studies indicated that NOS lost its activity after 28 h postslaughter in chicken dark muscle, whereas pork muscle lost its activity after 48 h at 4°C (Brannan and Decker, 2002). This indicated that there were high possibilities that endogenous NO can play a significant role in fresh meat quality during the early postmortem stage. The objective of this study was to test the hypothesis that NO and its products could be involved in the regulation of breast meat quality during postmortem refrigerated storage.

MATERIALS AND METHODS

Sample Preparation and Storage

Eighteen 6-wk-old broilers raised on a corn-soybean diet in the Department of Animal Science at Iowa State University were slaughtered following USDA (1982) guidelines. Immediately after slaughter, breast M. pectoralis was separated from carcasses and randomly assigned to 1 of 3 treatments: control (deionized distilled water), NOS inhibitor (0.06 *M* Nω-Nitro-L-arginine methyl ester hydrochloride, N5751, Sigma-Aldrich Corp., St. Louis, MO), and NO enhancer (0.1 m*M* S-nitrosoglutathione, N4148, Sigma-Aldrich Corp.). S-Nitrosoglutathione could decompose rapidly in solution to glutathione disulfide to release NO in water at room temperature (Noble et al., 1999). To help penetration of treatment solutions, breast muscles were punctured with 20 1/2 G needles uniformly and then individually placed into an oxygen-permeable zipper bag containing 200 mL of a treatment solution. After incubation for 24 h (1 d) at 4°C, breast samples were removed from original bags, towel dried, transferred to new bags, and then stored at 4°C for 4 and 7 d. Samples were stored for 7 d and were sampled for measurements of parameters at 4 and 7 d of storage. The left side of breast muscle from each broiler chicken was used to measure lipid oxidation, protein oxidation, and water-holding capacity (**WHC**). The samples for those analyses were frozen immediately in liquid nitrogen and then stored at −20°C until used. The other side of breast muscle from the remaining carcasses was used to determine color and drip loss.

pH Measurement

The pH was measured using a continuous method as described by Bendall (1973). Breast M. pectoralis muscle (3 g) was sampled in a test tube at 24 h postslaughter and homogenized with 27 mL of 150 m*M* KCl, 5 m*M* iodoacetate solutions using a polytron (type PT 10/35, Brinkman Instruments Inc., Westbury, NY) at maximum speed for 20 s (Brinkman Instruments Inc.). The pH of the solution was measured using a pH meter (Thermo Scientific, Beverly, MA).

Lipid Oxidation

Lipid oxidation was determined using a 2-TBA reactive substances (**TBARS**) method (Ahn et al., 1998). Five grams of samples from breast M. pectoralis were weighed into a 50-mL test tube and homogenized with 50 μL of butylated hydroxytoluene (7.2%) and 15 mL of deionized distilled water using a Polytron homogenizer (type PT 10/35, Brinkman Instruments Inc.) for 15 s at high speed. One milliliter of the meat homogenate was transferred to a disposable test tube (13 × 100 mm), and TBA/trichloroacetic acid (**TCA**; 15 m*M* TBA/15% TCA, 2 mL) was added. The mixture was vortex mixed and incubated in a boiling water bath for 15 min to develop color. Then samples were cooled in the ice water for 10 min, vortex mixed again, and centrifuged for 15 min at 2,500 × *g* at 4°C. The absorbance of the resulting supernatant solution was determined at 531 nm against a blank containing 1 mL of deionized distilled water and 2 mL of TBA/TCA solution. The amounts of TBARS were expressed as milligrams of malondialdehyde (**MDA**) per kilogram of meat.

Protein Oxidation

Protein carbonyl content was determined using the 2,4-dinitrophenylhydrazine (**DNPH**) derivatization method with minor modification (Lund et al., 2008). One gram of muscle from breast M. pectoralis was homogenized using a Polytron at maximum speed for 15 s in 10 mL of pyrophosphate butter (2.0 m*M* $Na_4P_2O_7$, 10 m*M* Trizma-maleate, 100 m*M* KCl, 2.0 m*M* $MgCl_2$, and 2.0 m*M* EGTA, pH 7.4). After homogenization, 2 equal aliquot of solutions (2 mL) were precipitated with 2 mL of 20% TCA and centrifuged at 12,000 × *g* for 5 min. The supernatant of sample was discarded and the precipitant was further washed with 2 mL of 10% TCA followed by centrifugation at 12,000 × *g* for 5 min at 4°C. After centrifugation, one pellet was treated with 2 mL of 10 m*M* DNPH dissolved in 2 *M* HCl and the other pellet was incubated with 2 *M* HCl as a blank. During 30 min of incubation, the sample was vortex mixed for 10 s every 10 min. The proteins were further precipitated with 2 mL of 20% TCA and centrifuged at 12,000 × *g* for 5 min at 4°C. The DNPH was removed by washing the sample 3 times with 5 mL of 10 m*M* HCl in 1:1 (vol/vol) ethanol-ethyl acetate followed by centrifugation at 12,000 × *g* for 5 min at 4°C after each wash. The pellet was finally solubilized in 2 mL of 6.0 *M* guanidine hydrochloride dissolved in 20 m*M* potassium dihydrogen phosphate (pH 2.3 adjusted with trifluoroacetic acid). The samples were kept at 4°C overnight and the final solution was centrifuged to remove insoluble materials the following day. The absorbance was measured using a spectrophotometer at 370 nm. The absorbance values of blank samples were subtracted from their corresponding sample values. The carbonyl content was calculated as nanomoles

per milligram of protein using an absorption coefficient of 22,000 $M^{-1} \cdot cm^{-1}$ (Levine et al., 1994).

Drip Loss

Drip loss was measured following the method of Zhang et al. (2006). Whole breast samples were placed in individual plastic bags under atmospheric conditions at 4°C. Immediately before being placed in bags, breast samples were dried by towel and the initial weights were recorded. One day after storage, samples were removed from their individual bags and were towel-dried and weighed again. The breasts were then placed in new bags, stored for an additional 3 d, towel dried, and weighed. Drip loss after 1 d or 4 d of storage was calculated as a difference between final and initial weight expressed as a percentage of the initial weight: [(initial weight – final weight)/initial weight] × 100%.

Water Holding Capacity

A centrifugal method was used to measure WHC as reviewed by Trout (1988). Fifteen grams of minced breast M. pectoralis samples were placed in a 35-mL test tube and centrifuged for 10 min at 40,000 × *g* at 4°C. The liquid in the tube was removed and the meat sample was weighed. The percentage of water lost was recorded as drip loss.

Color Measurement

Color measurements for breast muscles were conducted at 4 and 7 d after slaughter using a HunterLab MiniScan XE colorimeter (Hunter Laboratory Inc., Reston, VA) with D65 illuminant and 10° standard observer. The instrument was calibrated against blank and white references before use. Four random readings per sample were taken and averaged for Commission Internationale d'Eclairage L* (lightness), a* (redness), and b* (yellowness) values.

Statistical Analysis

All data were analyzed using the SAS version 9.1 (SAS Institute Inc., Cary, NC), and significance was reported at the $P < 0.05$ level. General linear procedure and ANOVA were used to determine the significance of the effects of treatment.

RESULTS AND DISCUSSION

pH

No significant differences in pH values of chicken breast meat were found after incubating with 3 different solutions for 24 h ($P > 0.05$, Figure 1). The SERCA and RyR are known to be susceptible to NO-induced nitrosylation and oxidation due to their high amounts of cysteine (Zhao et al., 1996; Stamler et al., 2008). Chronic low-frequency stimulation in fast-twitch muscles of rabbit inactivated SERCA due to protein oxidation and peroxynitrite-mediated tyrosine nitration (Klebl et al., 1998). In addition, protein oxidation and nitrosylation in RyR led to leaky channels for calcium and a rapid rise of calcium concentration in sarcoplasm of muscle cells (Bellinger et al., 2008). In addition, NO may be involved in regulation of glycogen metabolism of skeletal muscle by influencing the formation of cyclic guanosine monophosphate and GMP-dependent protein kinase activity through the inhibition of guanylate cyclase (Young et al., 1997; Merry et al., 2010). These regulations may possibly influence the rate of metabolism in postmortem muscle and thus regulate pH changes. These regulations could lead to increased rate of glycolysis in postmortem muscle and thus result in accelerated pH decline or lower ultimate pH. However, there were no significant differences in pH values among muscles with 3 different treatments in the current study ($P > 0.05$). Injection of NO inhibitor (L-NG-nitroarginine methyl ester hydrochloride) at 135 min preslaughter in ovine reduced the ultimate pH in the longissimus thoracis et lumborum (**LTL**), but not semimembranosous (**SM**; Cottrell et al., 2008). Exercise, as a treatment to release NO, significantly reduced pH at 24 h in both LTL and SM without affecting the rate of pH decline (Cottrell et al., 2008). The authors indicated that pH differences could be attributed to the changes in glucose uptake and glycolysis in postmortem muscle induced by NO. The inconsistence between the current study and previous study may be due to the differences in species, the composition of muscle fiber type, and the different time point for managing the level of NO in muscle.

Lipid Oxidation

Lipid oxidation, measured by TBARS after 1 and 4 d of refrigerated storage, was not significantly different among control, NOS inhibitor-, and NO enhancer-treated groups ($P > 0.05$, Table 1). The control group showed no significant difference for TBARS with other 2 treatments after 7 d of storage ($P > 0.05$). However, the TBARS values of samples treated with NOS inhibitor was significantly higher than that of NO enhancer-treated group at d 7 ($P < 0.05$). Nitric oxide and the products derived from NO-enhancer can both accelerate or delay lipid oxidation reaction. Postmortem muscles have decreased antioxidant ability, which leads to increased accumulation of reactive oxygen species (Renerre et al., 1996). Nitric oxide can react with superoxide ($\cdot O_2$) to form peroxynitrite, which can be decomposed to nitrogen dioxide and hydroxyl radicals. These radicals can initiate lipid oxidation and result in the formation of nitrated products (Botti et al., 2004). However, NO can also act as a potent terminator of radical chain propagation reactions catalyzed by alkoxyl and hydroperoxyl radical intermediates of lipid peroxidation. In a lipoxygenase-dependent liposome

Table 1. Effects of managing level of nitric oxide (NO) on TBA reactive substances [TBARS; mg of malondialdehyde (MDA)/kg of meat] in breast meat samples

TBARS (mg of MDA/kg of meat)	Control	NO synthase inhibitor	NO enhancer
Day 1	0.021 ± 0.002	0.024 ± 0.002	0.022 ± 0.001
Day 4	0.031 ± 0.001	0.031 ± 0.002	0.030 ± 0.002
Day 7	0.056 ± 0.005[ab]	0.062 ± 0.007[a]	0.039 ± 0.003[b]

[a,b]Means within the same row with different superscripts are significantly different ($P < 0.05$). n = 6.

and low-density lipoprotein system, NO reacted with LO· and LOO· at near diffusion-limited rates. The inhibition of lipoxygenase-dependent liposome and lipoprotein oxidation was dose-dependent. The NO reacted with LO· and LOO· to form nitrito-, nitro-, nitrosoperoxo-, and nitrated lipid oxidation adducts to terminate the radical chain propagation reactions (Rubbo et al., 1995). High and low levels of TBARS in NOS inhibitor- and NO enhancer-treated groups in the current study, respectively, indicated a possible inhibitory role of NO in lipid oxidation in postmortem muscles. However, whether the differences were attributed to the direct effect of NO toward lipid oxidation or due to the indirect effect of NO by regulating the antioxidant system of postmortem muscle are not known.

Protein Oxidation

The carbonyl content in meat samples from the NOS inhibitor-treated group was significantly lower than those from control and NO enhancer-treated group after 1 d of incubation ($P < 0.05$, Figure 1A). The levels of carbonyl compounds in control samples tended to be lower than those from NO enhancer-treated ones ($P = 0.06$). This trend was also shown after 4 d of refrigerated storage in which carbonyl content in the NO enhancer-treated group was 48% higher than that of the NOS inhibitor-treated group ($P < 0.05$, Figure 1B). These results indicated that high levels of NO in postmortem muscle may be associated with an increased degree of protein oxidation. Because there is no significant difference about TBARS among 3 treatments (Table 1), the high amount of carbonyl in NO enhancer-treated group should not be from the reactions between protein- and lipid oxidation-derived products (Faustman et al., 2010). Peroxynitrite produced from NO with superoxide can nitrosylate cysteine, nitrify tyrosine, and oxidize methionine, lysine, tryptophan, and phenylalanine (Viner et al., 1999). Among the polymonopeptides including poly-Glu, poly-Pro, poly-His, poly-Arg, poly-Lys, and poly-Lys-Arg, only poly-Arg-Lys and poly-Lys can be oxidized to carbonyl groups after incubation with peroxynitrite, indicating that lysine residues could be the candidates for the formation of carbonyl compounds (Tien et al., 1999). However, oxidation induced by peroxynitrite cannot lead to the formation of carbonyl derivatives (Tien et al., 1999). In addition, high levels of NO and NO-derived reactive nitrogen species could decrease antioxidant ability of postmortem muscles. Decreased antioxidant system could be associated with increased accumulation of reactive oxygen and nitrogen species, leading to high levels of protein oxidation in meat samples during refrigerated storage (Astruc et al., 2007; Zhao et al., 2010). Therefore, the high levels of carbonyl content in samples from NO enhancer indicates the possible involvement of NO and other nitrogen species on regulation of protein function in postmortem muscle.

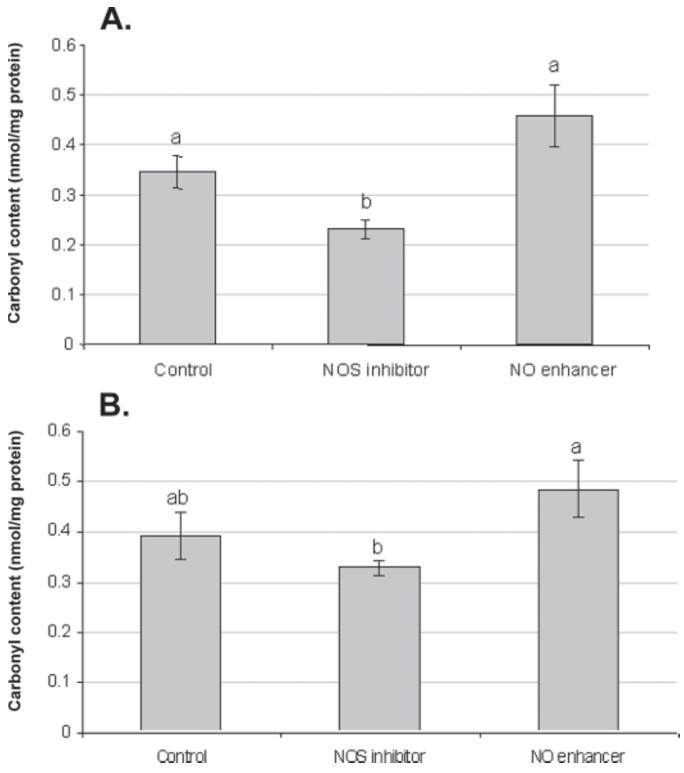

Figure 1. Effects of managing level of nitric oxide (NO) on carbonyl content (nmol/mg of protein) at A) d 1 and B) d 4 in breast meat. NOS = nitric oxide synthase. Different letters (a,b) within a figure differ significantly ($P < 0.05$). n = 6.

WHC and Drip Loss

The drip loss among 3 treatments was not significantly different from each other after 4 and 7 d of refrigerated storage under atmospheric conditions ($P > 0.05$, Table 2). This was consistent with the study of Cottrell et al. (2008) who reported that infusion of NO enhancer or inhibitor at 135 min preslaughter in ovine or exercise preslaughter did not influence drip loss in both LTL and SM of ovine significantly. After

Table 2. Effects of managing level of nitric oxide (NO) on drip loss and water-holding capacity (WHC) in broiler breast meat

Item	Control	NO synthase inhibitor	NO enhancer
Day 1 to 4 drip loss (%)	6.24 ± 0.32	5.47 ± 0.36	6.12 ± 0.31
Day 4 to 7 drip loss (%)	6.36 ± 0.82	7.69 ± 1.23	7.58 ± 0.92
Day 1 WHC (%)	78.60 ± 0.62^{b}	82.08 ± 1.13^{a}	75.25 ± 0.18^{c}
Day 4 WHC (%)	83.06 ± 0.67^{a}	81.35 ± 0.65^{ab}	80.01 ± 0.82^{b}

$^{a–c}$Means within the same row with different superscripts are significantly different ($P < 0.05$). n = 6.

1 d of postmortem storage, meat samples from NOS inhibitor-treated group showed highest ability of retaining water, whereas the NO enhancer-treated group presented the lowest levels of WHC ($P < 0.05$). After 4 d of refrigerated storage, the NO enhancer-treated group still showed lower levels of WHC compared with the control ($P < 0.05$), whereas the WHC in the NOS inhibitor-treated group was not significantly different from the other 2 groups ($P > 0.05$). Barbut et al. (2008) reported that pale, soft, and exudative (**PSE**)-like conditions in poultry cost the poultry industry millions of dollars per year. The fundamental biochemical events for PSE in poultry have not yet been well understood, although similar mechanisms to pork PSE are expected to be involved (Solommon et al., 1998). Protein denaturation due to rapid pH decline or low ultimate pH is the major reason for the high level of drip loss in postmortem muscles (Offer and Cousins, 1992). However, no significant difference was found in the ultimate pH among breast muscles with 3 treatments, indicating that pH may be not the major factor involved in WHC difference. A possible explanation could be that higher levels of protein oxidation in the NO enhancer group could modulate the ability of the meat sample to retain water during centrifuge. Protein oxidation can lead to the production of intermolecular bonds including disulfide, dityrosine, and other intermolecular bridges to form protein aggregation and polymerization (Morzel et al., 2006). Physical and chemical properties of proteins including solubility, hydrophobicity, WHC, and even the nutritional value can be modified by protein oxidation (Srinivasan and Hultin, 1997; Liu and Xiong, 2000). Therefore, low WHC in the enhancer-treated group may be due to the high level of protein oxidation evidenced by the great amount of carbonyl compounds.

Color

The breast meat in NOS inhibitor-treated group showed higher L* and lower a* values compared with control and NO enhancer-treated group after 4 and 7 d of refrigerated storage ($P < 0.05$, Table 3). However, the b* of breast meat among the 3 treatments was not different during the 7 d postmortem storage period ($P > 0.05$). No significant differences in L*, a*, and b* values between control and NO enhancer-treated group at both 4 and 7 d of storage were found ($P > 0.05$). In ovine LTL and SM, preslaughter exercise increased a* values at 1 and 4 d postmortem, indicating that NO plays a role in regulating meat color development (Cottrell et al., 2008). The pH is known to play key roles in meat color. Swatland (2008) reported that low pH is associated with pale color, whereas high pH is associated with dark color. No significant difference was found in pH among the 3 treatments in the current study (pH range of 5.95–5.98), indicating that other factors might have involved in the L* and a* differences in breast meat. Low a* in the NOS inhibitor-treated group could be related to the low levels of NO, which was known to react with myoglobin to produce pink color in meat products (Møller and Skibsted, 2002).

In conclusion, this is a preliminary study to investigate the possible regulation of breast meat quality during refrigerated storage by managing levels of NO. Breast meat treated with NOS inhibitor showed lower levels of protein oxidation and higher levels of lipid oxidation during refrigerated storage. Breast meat incubated with NO enhancer presented decreased ability to retain water probably due to increased protein modification. Thus, NO could play a role in regulating biochemical changes and thus influences breast meat quality of broiler chickens. However, more information

Table 3. Effects of managing level of nitric oxide (NO) on broiler breast meat color

Day	Color value[1]	Control	NO synthase inhibitor	NO enhancer
4	L*	61.47 ± 0.75^{a}	59.48 ± 0.62^{b}	58.07 ± 0.91^{b}
	a*	1.071 ± 0.110^{b}	1.595 ± 0.244^{a}	1.831 ± 0.181^{a}
	b*	12.23 ± 0.27	11.56 ± 0.63	12.49 ± 0.21
7	L*	59.55 ± 1.16^{a}	57.19 ± 0.44^{b}	56.64 ± 1.09^{b}
	a*	1.454 ± 0.119^{b}	1.898 ± 0.179^{a}	2.271 ± 0.103^{a}
	b*	12.23 ± 0.32	12.08 ± 0.72	12.81 ± 0.33

^{a,b}Means within the same row with different superscripts are significantly different ($P < 0.05$). n = 6.

[1]L* = lightness; a* = redness; b* = yellowness.

is needed to elucidate the mechanisms behind how NO regulates fresh meat quality.

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