# Lipid Oxidation in Red and Poultry Meats

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## Abstract

Lipid oxidation is one of the most important non-microbial causes of meat quality deterioration. However, there have been different/conflicting views concerning the primary catalysts of lipid oxidation in meat. This presentation provides brief overviews of lipid oxidation mechanism in general and catalysis of lipid oxidation in meat, and then focuses on inter-species differences in lipid oxidation potential, using results from our studies on meats (beef, pork and chicken) at retail and the respective meats of uniform postmortem history. The inter-species differences have highlighted the relative roles of meat pigment (myoglobin) content, catalase activity, and the concentration of oxidation substrates (particularly polyunsaturated fatty acids) in determining the lipid oxidation potential of raw meat versus cooked meat.

## Introduction

Lipid oxidation (peroxidation) is a leading cause of quality deterioration in meat products. The oxidative stability of skeletal muscle (meat animal muscle tissue) is dependent on the composition, concentration, and reactivity of three factors: oxidation substrates, oxidation catalysts, and antioxidants (Decker and Xu, 1998).

Lipid oxidation generally is a free radical chain reaction, which typically involves three stages: initiation, propagation, and termination. As for meat lipid oxidation, one of the most important questions is related to the nature of the initiation process or the source of the primary catalysts that initiate lipid oxidation in intact/actual meat, i.e., oxidation of meat lipids *in situ* rather than in model systems (Kanner, 1994; Gray et al., 1996).

Spontaneous lipid radical formation or direct reaction of unsaturated fatty acids with molecular oxygen is thermodynamically unfavorable (Gray et al., 1996). The electronic structure of oxygen has two unpaired electrons in triplet state, while the majority of organic biomolecules, including unsaturated fatty acids, are in singlet/ground state. The reaction of a triplet molecule with a singlet state molecule is spin-forbidden, as spin angular momentum must be conserved (Kanner, 1994). However, the spin restriction which prevents the direct addition of triplet state oxygen to singlet state unsaturated fatty acid molecules can be overcome by

photooxidation, activated oxygen species (such as hydrogen peroxide, superoxide anion, and hydroxyl radical), or active oxygen-iron complexes (ferryl radicals) (Kanner, 1994; Gray et al., 1996).

## Formation of activated oxygen species and ferryl radicals

Superoxide and hydroxyl radicals in meat may be generated from autoxidation of oxymyoglobin to metmyoglobin (MeMb), membrane electron transfer system, activation of several leukocytes present in the vasculature of the muscle tissue, and oxidation of various reducing components by "free" iron. Hydrogen peroxide ($H_2O_2$) is normally present as a metabolite at low concentration in aerobic cells. And a system (including meat animal tissue) generating superoxide radical would be expected to produce $H_2O_2$ by non-enzymatic dismutation or enzyme (superoxide dismutase)-catalyzed dismutation (Kanner, 1994). It has been suggested that a large part of $H_2O_2$ in meat may be generated by oxidation of heme pigments (Harel and Kanner, 1985a).

Most of the hydroxyl radical ($HO^{\bullet}$) generated *in vivo* or *in situ* may come from the metal-dependent breakdown of $H_2O_2$. In meats, only the iron(II)-dependent formation of $HO^{\bullet}$ would occur under normal conditions. Free iron in meat can decompose $H_2O_2$ or lipid hydroperoxides, producing free radicals. There is a small pool of non-protein non-heme iron ("free" iron) in animal tissues. The main source of such free iron may be ferritin (the iron-storage protein); during meat storage, some iron may be released from ferritin (Kanner, 1994). In a model system, ferritin was able to directly catalyze lipid oxidation (Seman et al., 1991).

Although singlet oxygen could initiate lipid oxidation, no strong evidence for the generation of this oxygen species in meat has been documented (Kanner, 1994). Whang and Peng (1988), nevertheless, reported that singlet oxygen quenchers inhibited the light-accelerated lipid oxidation in pork and turkey.

Ferryl radicals (heme iron radicals) can readily be generated in meat, especially red meat which is high in myoglobin content. Metmyoglobin (MetMb) or methemoglobin can be activated by $H_2O_2$, producing a short-lived intermediate of ferry ($Fe^{4+}$) radical, which may initiate oxidation of membrane lipids and lipid oxidation in meat (Kanner and Harel, 1985; Harel and Kanner, 1985b; Rhee et al., 1987). According to Harel and Kanner (1985b), heated (denatured) MetMb maintains its capacity to be activated by $H_2O_2$.

## Primary catalysts of lipid oxidation in meat

As for catalysis of lipid oxidation in meat, there have been different views or conflicting reports regarding the relative importance of non-heme iron and heme iron, as discussed in reviews (Rhee, 1988; Gray et al., 1996). In studies conducted in the 1970s, ferrous iron (ferrous sulfate) or purified MetMb was added to muscle fibers (washed meat) to construct a model meat system. Based on results from such studies, it was suggested that non-heme iron, rather than heme iron, would be the major catalyst of lipid oxidation in meat (particularly cooked meat). However, such model system may have been inadequate to represent actual/intact meat (i.e., meat *in situ*). Specifically, the early model system studies may have overlooked the importance of

other meat constituents that can be washed out along with non-heme iron and meat pigments (the primary heme iron in meat). Particularly, the water washing of ground muscles could also have extracted other prooxidative meat components (especially $H_2O_2$ and enzymes) that may affect lipid oxidation in actual meat. Thus, in our model system study (Rhee et al., 1987), we added MetMb-$H_2O_2$ (activated MetMb or ferrylmyoglobin), nonheme iron (ferrous sulfate) or the components of microsomal (or mitochondrial) enzymic lipid peroxidation system to water-extracted beef muscle residues. Our results indicated that the activated MetMb (activated heme iron), nonheme iron, and the enzymic lipid peroxidation systems might all play important roles in the catalysis of lipid oxidation in meat. The relative contributions of the three would be dependent on, among others, heat treatment/cooking, meat animal species, and meat handling, processing and packaging strategies.

## Inter-species differences in lipid oxidation

Since the mid-1980s, some of our lipid oxidation studies have focused on inter-species differences. I personally became very intrigued by what I had observed in the home when using aerobically-packaged, raw beef and chicken that had been stored in the freezer compartment of our refrigerator for a long time. According to my casual observations, there seemed to be notable inter-species differences in the rate of lipid oxidation. So we conducted a study to investigate animal species effects on lipid oxidation using meats at retail (Rhee and Ziprin, 1987). Beef (ribeye and top round steaks), chicken (breast halves and drumsticks), and pork (loin chops) were purchased from a local supermarket in the summer (July) for Experiment I and in the winter (December) of the same year for Experiment II. Beef *longissimus dorsi* (B/LD) and *semimembranosus* (B/SM), pork LD (P/LD), and chicken breast/white (C/W) and drumstick/dark (C/D) muscles were dissected from the retail cuts, and the lean pieces of the same species/muscle category were pooled, finely comminuted, and mixed with 30 ppm chlortetracycline (CTC) to inhibit microbial growth during subsequent refrigerated storage. These ground muscles were evaluated for lipid oxidation potential, concentrations of meat pigments (heme iron), nonheme iron, and microsomal enzymic lipid peroxidation activity. To determine lipid oxidation potential, 2-thiobarbituric acid-reactive substances (TBARS) content was determined on raw and cooked samples stored at 4℃ for varying lengths of time, using a distillation TBA assay procedure with antioxidant protection (Rhee, 1978). The TBARS values for raw chicken and pork muscles were low and changed little during 2~6 days of refrigeration, whereas those for raw beef muscles were higher and increased progressively (Fig. 1). In other words, the potential of retail raw meats to undergo lipid oxidation during refrigerated storage was greater for beef than pork and chicken muscles.

According to correlation analysis, heme pigment content seemed be more important variable for explaining the inter-species differences in the oxidative susceptibility of raw muscles than the nonheme iron content or microsomal enzymic lipid peroxidation activity (Table 1).

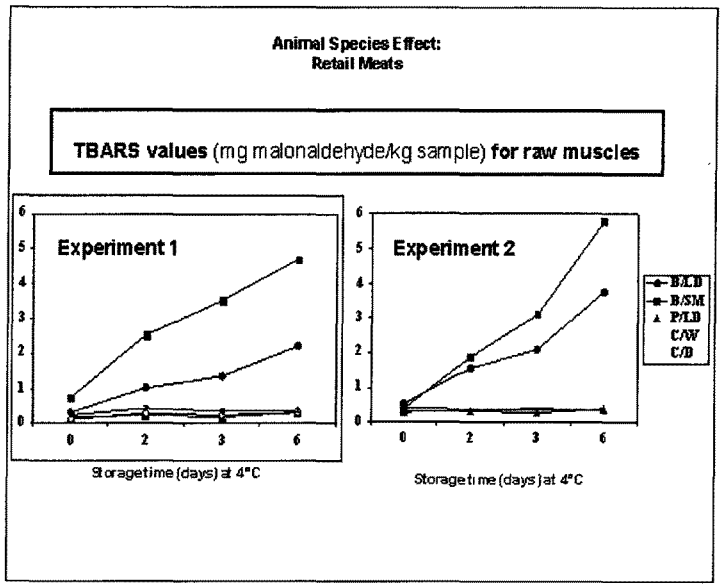


Figure 1. TBARS values for raw beef, pork and chicken muscles from meats at retail when stored at 4° C. [Data from Rhee et al. (1987)]

Table 1. Correlation coefficients between TBARS values and other variables for raw muscles from meats at retail. [Data from Rhee et al. (1987)]

Animal Species Effect:
Retail Meats

**Correlation coefficients for raw muscles**

| | TBARS values at days | | |
|---|---|---|---|
| | 2 | 4 | 6 |
| Heme pigment content* | 0.75 | 0.77 | 0.84 |
| Non-heme iron content | 0.51 | 0.49 | 0.44 |
| Microsomal enz. Lipid perox. activity | 0.45 | 0.41 | 0.31 |

* Correlation coefficients are significant ($P < 0.05$).

Unlike the case for raw muscles, TBARS values for cooked muscles, regardless of species, all increased during 2~4 days of storage with no marked differences among species (Fig. 2).

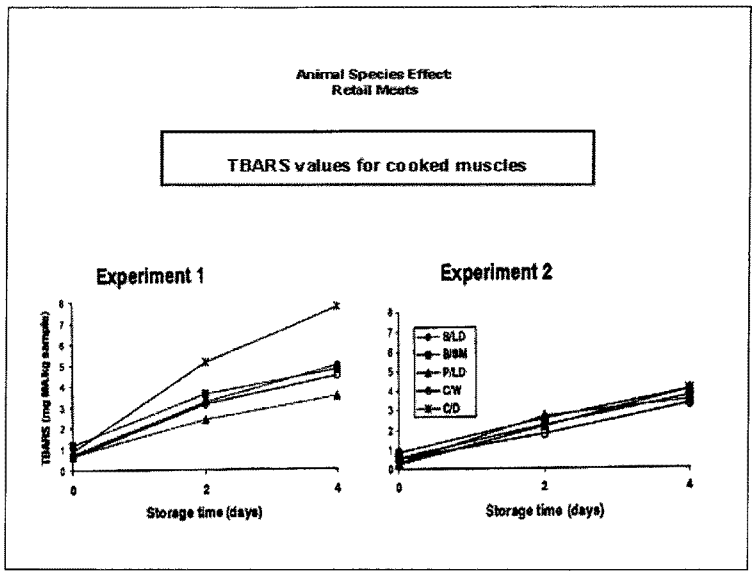


Figure 2. TBARS values for cooked beef, pork and chicken muscles stored at 4℃.
[Data from Rhee et al. (1987)]

When we analyzed muscles from retail meats (beef LD and SM, pork LD, and chicken breast and thigh muscles) for catalase activity in subsequent studies (Rhee et al., 1996; Pradhan et al., 2000), pork LD exhibited the highest activity and chicken breast meat showed the lowest activity. The beef and chicken dark muscles were intermediate in catalase activity. Mei et al. (1994), who studied development of lipid oxidation and inactivation of antioxidant enzymes in cooked beef and pork, also observed a higher catalase activity in raw pork (ground composite of muscles from the pork boston butt at retail) compared to raw beef (ground composite of muscles from the beef chuck at retail). Impacts of the inter-species differences in catalase activity on lipid oxidation during raw meat storage will be delineated in later discussions.

Since beef, chicken and pork at retail may vary widely in terms of postmortem history (particularly the time elapsed between animal slaughter and consumer meat purchase), we evaluated inter-species differences in lipid oxidation potential using meats of the same/uniform postmortem history. Animals of each species had been on a typical commercial finishing diet for the species and the postmortem muscle excision time was early and uniform for all three species. Also, muscles were selected from the same anatomical locations for beef and pork (i.e., LD and SM muscles), while light and dark muscles were used for chicken. All samples were handled in the same manner and potential further lipid oxidation during the TBA test was minimized by adding antioxidants at the beginning step (Rhee, 1978). Beef LD and SM muscles were excised 24 hours postmortem from carcasses of 3 market-weight crossbred feedlot steers shipped to the Texas A&M University Rosenthal Meat Science and Technology Center. Samples of each muscle from the 3 carcasses were used to conduct 3 replications of a treatment (species/muscle group), with the muscle sample from one carcass

representing a replication. From each carcass, ~5.5 kg of each muscle was used to provide enough sample for all analyses. Pork LD and SM muscles were removed 24 hours postmortem from carcasses of 6 marketweight castrated animals (finished on a sorghum and soybean-based diet) from the Texas A&M University Swine Center. To obtain sufficient meat sample for 3 replications (~5.5 kg/muscle/replication), 6 animals were used, with the composite of each muscle from 2 carcasses constituting a replication. Chicken breast (B) and thigh (T) parts were obtained 24 hours postmortem from male broilers raised on a corn and soybean-based diet at the Texas A&M University Poultry Center. About 50 birds (~8 weeks of age) were used to provide enough meat (free from skin and separable fat) for 3 replications of B and T muscles. Breast and T muscles removed from all the birds were divided into 3 batches, each batch representing a replication. Each muscle/species was immediately ground finely and formed into patties. Patties to be stored/analyzed raw were frozen immediately, whereas samples to be cooked were held at 4°C for ~8 hours before cooking (pan-frying to an internal temperature of ~74°C). Raw and cooked patties for storage experiments were placed on polyfoam trays and packaged in Ziploc® freezer bags. Cooked samples were stored for 0, 3, or 6 days at 4°C and for 0, 75, or 150 days at -20°C. Raw patties were stored only at -20°C, to avoid effects of microbial growth on meat lipid oxidation potential.

Table 2. Fat content for raw and cooked beef, pork and chicken muscle patties of uniform postmortem history. [Data from Rhee et al. (1996)]

Animal Species Effect
Meats with Similar Postmortem History

| | Fat content (%) | |
|---|---|---|
| | Raw | Cooked |
| B/LD | 4.4 $^{b}$ | 5.3 $^{bc}$ |
| B/SM | 3.5 $^{c}$ | 4.2 $^{d}$ |
| P/LD | 4.5 $^{b}$ | 5.9 $^{b}$ |
| P/SM | 3.3 $^{c}$ | 4.8 $^{cd}$ |
| C/B (breast) | 1.4 $^{d}$ | 1.9 $^{e}$ |
| C/T (thigh) | 6.0 $^{a}$ | 7.5 $^{a}$ |

Compositional analyses showed that chicken B had the lowest fat content and chicken T had the highest, for both raw and cooked samples (Table 2). Likewise, cooked chicken B had the lowest fat content among cooked samples. Chicken T contained more total polyunsaturated fatty acids (PUFAs) per 100g sample than other

species/muscle categories. However, as a percentage of total fatty acids, PUFAs level was highest in chicken B (Table 3). Beef muscles, raw or cooked, were distinctly higher in heme iron content compared to pork and chicken muscles (Table 4). Such was not the case for non-heme iron content.

Table 3. Percentages of total polyunsaturated fatty acids for raw beef, pork and chicken muscle patties of uniform postmortem history. [Data from Rhee et al. (1996)]

Animal Species Effect
Meats with Similar Postmortem History

**Polyunsaturated fatty acids as % of total fatty acids**

| | Total PUFA (%) | |
|---|---|---|
| | Raw | Cooked |
| B/LD | 4.91 $^{d}$ | 5.50 $^{d}$ |
| B/SM | 6.57 $^{cd}$ | 6.92 $^{d}$ |
| P/LD | 5.26 $^{d}$ | 5.17 $^{d}$ |
| P/SM | 9.21 $^{c}$ | 10.02 $^{c}$ |
| C/B | 18.87 $^{a}$ | 20.53 $^{a}$ |
| C/T | 15.51 $^{b}$ | 16.89 $^{b}$ |

Table 4. Heme iron values for raw and cooked beef, pork and chicken muscle patties of uniform postmortem history. [Data from Rhee et al. (1996)]

Animal Species Effect
Meats with Similar Postmortem History

**Heme iron (µg/g)**

| | Raw | Cooked |
|---|---|---|
| B/LD | 18.1 $^{a}$ | 21.4 $^{a}$ |
| B/SM | 20.3 $^{a}$ | 21.9 $^{a}$ |
| P/LD | 9.6 $^{b}$ | 7.3 $^{b}$ |
| P/SM | 6.5 $^{b}$ | 6.2 $^{b}$ |
| C/B | 5.3 $^{b}$ | 5.5 $^{b}$ |
| C/T | 7.5 $^{b}$ | 6.2 $^{b}$ |

TBARS values on sample weight basis (TBARSs) for raw patties stored at -20°C were lowest for chicken muscles (Fig. 3). Even on a fat-weight basis (TBARSf), chicken muscles tended to have the lowest values. Initial (0-day) TBARSs values for chicken and pork samples were low (< 0.2 mg malonaldehyde equivalent/kg sample), compared to mean values of 0.37 for beef sm and 0.63 for beef LD. However, pork TBARSs values increased rapidly when patties were stored frozen. In our previous study with retail meats (Rhee and Ziprin, 1987), we did not find such TBARSs increases in refrigerated, microbial growth-controlled raw pork samples (Rhee and Ziprin, 1987). Specifically, when finely-chopped raw pork LD (treated with CTC) was stored at 4°C for up to 6 days, TBARSs values remained during the entire 6-day period.

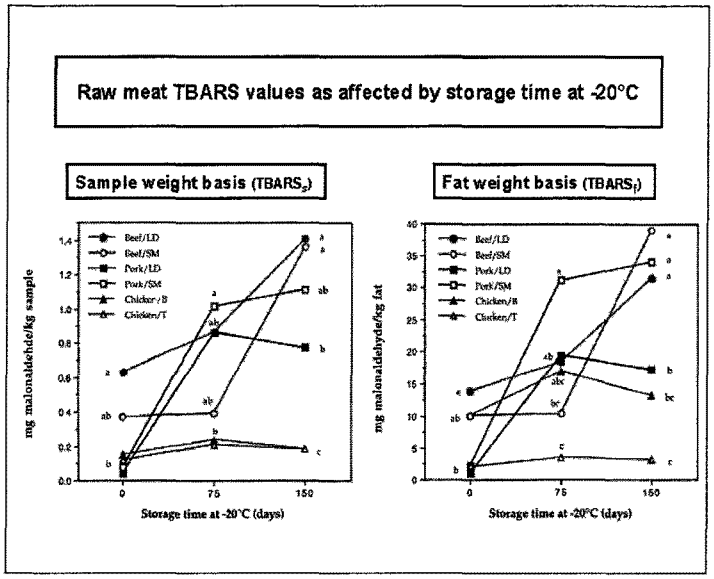


Figure 3. TBARS values for raw beef, pork and chicken muscle patties of uniform postmortem history when stored at -20°C. [Data from Rhee et al. (1996)]

Upon evaluating the aforementioned raw meat data (i.e., data on meats of uniform postmortem history) in conjunction with our research data on retail meats (including data on TBARS and heme pigment contents as well as catalase activity), a hypothesis emerged. That is, heme level in conjunction with another endogenous factor may be responsible for inter-species differences in lipid oxidation potential of raw meats. As mentioned earlier, ferrylmyoglobin radicals generated from the reaction of MetMb with $H_2O_2$ can initiate or catalyze lipid oxidation. When considering the effect of $H_2O_2$ (i.e., causing MetMb to become an active catalyst of oxidation), lipid oxidation potential of raw samples may be determined, in large part, by a combination of heme pigment content and catalase activity. Observations or explanations supporting such hypothesis follow. (1) Beef has the most myoglobin, followed by pork and chicken in a decreasing order (Rhee and Ziprin, 1987). (2) More $H_2O_2$ may be formed in the meat of high pigment content (e.g., beef) than in less pigmented meats (e.g., chicken and pork), because the autoxidation of oxymyoglobin (or oxyhemoglobin) reportedly lead to

formation of MetMb (or methemoglobin) and $O_2^-$ (which could dismutate to $H_2O_2$). [As mentioned previously, a large part of $H_2O_2$ in meat may come from the oxidation of heme pigments (Harel and Kanner, 1985a).] (3) Catalase activity which may be important in decreasing the amount of $H_2O_2$ remaining in meat was higher in pork LD than in beef LD or chicken T, and chicken B had the lowest activity (our data on retail meats). [It should be mentioned that catalase was stable during normal refrigerated storage of red and poultry meats, and lipid oxidation in the refrigerated raw meat increased markedly when its catalase was inhibited by sodium azide treatment (Pradhan et al., 2000).] (4) When stored at -20°C where catalase activity would be limited, raw pork TBARSs values were initially low as were raw chicken values, but increased sharply in 75 days unlike raw chicken TBARSs (Rhee et al., 1996). However, at 4°C where catalase would be fairly active, TBARSs values for raw pork (treated with CTC) remained low throughout 6 days of refrigeration (Rhee and Ziprin, 1987; a study with meats at retail). In the latter study, raw beef TBARSs increased steadily during 4°C storage while raw chicken TBARSs remained low as did raw pork TBARSs. Since chicken B is the least pigmented, the ultimate MetMb level and the amount of $H_2O_2$ from heme pigment oxidation could also be least in chicken B. Although chicken T is somewhat more pigmented than chicken B, it may have a higher catalase activity as well (according to our retail meat sample data). Thus, chicken muscles (B and T) should be least susceptible to lipid oxidation in the raw state. Beef muscles - highest in heme pigment content and moderate in catalase activity - would make much $H_2O_2$ generated from heme pigment oxidation available to activate MetMb. Thus, it should have the greatest lipid oxidation potential among raw-stored muscles of the 3 meat animal species.

TBARSs values for cooked samples stored at 4°C were far higher for chicken T than other species/muscle categories, after either 3 or 6 days of storage (Fig. 4) (Rhee et al., 1996). When TBARS data for refrigerated

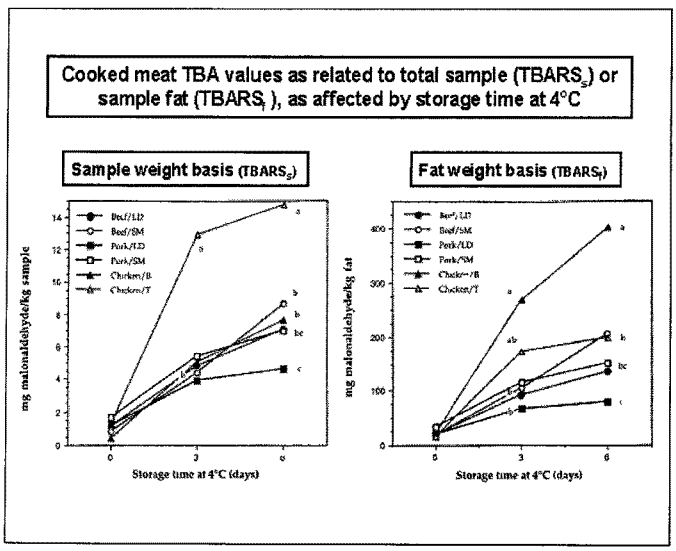


Figure 4. TBARS values for cooked beef, pork and chicken muscle patties of uniform postmortem history when stored at 4°C. [Data from Rhee et al. (1996)]

cooked samples were expressed on a fat weight basis (TBARSf), chicken B had the highest values because of its lower fat content. TBARSs values for cooked samples stored (up to 150 days) at -20°C were highest for chicken T. However, TBARSf values were similar for cooked-frozen chicken B and T, exceeding those of beef and pork.

Total PUFA level differences seem to best explain the high lipid oxidation potential of cooked chicken muscles, compared to cooked beef and pork. Cooking inactivates catalase. Cooking also would denature the membrane and release phospholipids (the major source of PUFAs), bringing reactants and catalysts closer (Asghar et al., 1988; Rhee, 1988). Thus, amounts of oxidation substrates (particularly PUFAs), rather than catalysts, would become a more important determinant of inter-species differences in lipid oxidation rate in cooked meat.

## Salt effect on lipid oxidation meat

For lipid oxidation in processed meats, one cannot overlook the effect of salt (NaCl), a universal seasoning in prepared foods. Salt has multiple functions in meat products. It tenderizes meat by increasing ionic strength, enhances flavor and increases water-holding capacity. At high concentrations, it also inhibits microbial growth, functioning as a preservative. However, salt has some undesirable side effects, even at concentrations normally used in meat products. It promotes lipid oxidation in raw and cooked meat and accelerates metmyoglobin formation and discoloration in raw meat (Rhee, 1999; Rhee and Ziprin, 2000). At the salt levels commonly used in processed meats (0.5~2.5% in most products), lipid oxidation would increase progressively with increasing amounts of salt and such potential would be greater for red meat than for white/poultry meat (Rhee and Ziprin, 2000). Stronger or stepped-up antioxidative measures will be needed for distribution and merchandising of uncured meat products or marinated meat items with substantial amounts of salts, to fully counteract the salts prooxidant effect. This also would be true when meats salted or marinated in the home are refrigerated for cooking/consumption on later days.

## "Warmed-over" flavor of cooked-stored meat

Cooked meat undergoes lipid oxidation more readily during storage when compared to the uncooked counterpart, because the cooking process promotes lipid oxidation. The following are various hypotheses on how cooking may cause the acceleration of lipid oxidation in meat: (1) cooking increases "free" iron concentration due to heat-induced release of protein-bound iron; (2) cooking increases amounts of $H_2O_2$ (from oxidation of oxymyoglobin) and MetMb, thus resulting in more $H_2O_2$-activated MetMb (ferrylmyoglobin radicals); (3) cooking disrupts muscle membrane systems, thus resulting in mixing of oxidation substrates with oxidation catalysts; and (4) cooking inactivates antioxidant enzymes in meat (Asghar et al., 1988; Rhee 1988; Harel and Kanner 1985; Mei et al., 1994).

When uncured cooked meat is stored, it develops a characteristic objectionable odor and flavor. The term

"warmed-over "flavor (WOF)-introduced by the research group led by Dr. Betty M. Watts (the author's major professor) in 1958-has been used most commonly to describe this off-flavor. Other terms used include rancid, oxidized, stale or cardboard flavors (Johnson and Civille, 1986). WOF develops in a few hours when cooked meat is held at room temperature, and becomes apparent within 24 to 48 hours when cooked meat is stored at refrigeration temperatures. Although WOF develops more slowly in cooked meat stored at freezing temperatures, it can be a major factor influencing the quality of frozen precooked meat unless antioxidant measures are taken.

### Antioxidant strategies

Lipid oxidation and accompanying oxidative quality deterioration in meat products can be minimized by several different means. These include: (1) using meat handling and processing techniques/technologies that do not increase prooxidants and decrease endogenous antioxidants; (2) manipulating animal diets to increase amounts of antioxidants and decrease oxidation-susceptible fatty acids, especially PUFAs, in meat (as discussed extensively in the authors June 11, 2003, presentation at the National Livestock Research Institute, Suwon, Korea, which was entitled "Quality Traits of Meats and Meat Products as Affected by Animal Production System/Nutritional Background and Product Formulation Strategy/Design"); and (3) utilizing antioxidative nonmeat additives (Rhee,1999; a review)]; and (4) vacuum/anoxic packaging of products. Decker and Xu (1998) also have provided detailed discussion of strategies to be taken to minimize oxidative rancidity in muscle foods.

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