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Publication Date: October 10, 2002 | doi: 10.1021/bk-2003-0836.ch014

Chapter 14

Lipid Oxidation in Muscle Foods

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The main factors governing the eating quality of muscle foods are tenderness, color and flavor. Oxidation of lipids is a major cause of deterioration in the quality of muscle foods and can directly affect many quality characteristics such as flavor, color, texture, nutritive value and safety of the food. It is generally accepted that lipid oxidation in muscle foods is initiated in the highly unsaturated phospholipid fraction in subcellular membranes. Oxidative damage to lipids may occur in the living animal because of an imbalance between the production of reactive oxygen species and the animal's antioxidant defence mechanisms. This may be brought about by a high intake of polyunsaturated fatty acids, or by a deficiency of nutrients involved in the antioxidant defence system. Damage to lipids is accentuated in the immediate post slaughter period and, in particular, during handling, processing and storage. Dietary factors contribute to the antioxidant defence system and protect biological membranes against lipid oxidation. A variety of nutrients and non-nutrients, including vitamin E, have been shown to affect the prooxidant/antioxidant balance and ultimate quality of the food. This review focuses on the effects of vitamin E and other antioxidant micronutrients on lipid oxidation, color, water-holding capacity and cholesterol oxidation in muscle foods.

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In Freshness and Shelf Life of Foods; Cadwallader, Keith R., et al.;
ACS Symposium Series; American Chemical Society: Washington, DC, 2002.

Introduction

Meat plays an important role in the diet of humans by contributing quality protein, essential minerals and trace elements and a range of B vitamins. In addition to its nutritive value meat has other important attributes, including its attractive sensory properties. The main factors governing the acceptability and eating quality of meat are tenderness, color and flavor. However, despite these important and highly acceptable attributes, meat consumption has come under close scrutiny in recent years. Emphasis on the role of fat and saturated fatty acids in health and disease and the greater sensitivity of people to environmental and animal welfare issues has had an influence on animal production, animal nutrition, food processing and food consumption patterns. There is now a greater demand than ever for foods perceived as natural, fresh tasting and more nutritious. In addition, changing lifestyles have a major impact on food purchasing, preparation, convenience and consumption. A recent pan-European Union survey of consumer attitudes to food, nutrition and health showed that the most important factors influencing consumer food choice were 'quality/freshness', 'price', 'taste', 'trying to eat healthy' and 'family preference' *(1)*. One of the main factors limiting the quality and acceptability of meat and meat products is lipid oxidation, a process that leads to discoloration, drip loss, off-odor and off-flavor development and the production of potentially toxic compounds *(2)*. Preventing lipid oxidation during storage and retail display is, therefore, essential in order to maintain the quality, wholesomeness and safety of meats, and to ensure that customers will make repeat purchases.

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Lipid Oxidation *in vivo*

The production of free radicals *in vivo* is a critical determinant of animal health, and consequently food quality, wholesomeness and acceptability of muscle foods by the consumer. Lipid oxidation is a free radical-mediated chain reaction. The most important free radicals are reduced derivatives of oxygen called reactive oxygen species (ROS). These include free radicals having one or more unpaired electrons that can exist independently for a brief period *(3)*. Examples are the hydroxyl radical ($HO^{\bullet}$) (the most potent oxidant encountered in biological systems with an estimated half-life of about 10^{-9}s), superoxide anion ($O_2^{-\bullet}$) and the oxygen-centered radicals of organic compounds (peroxyl, $ROO^{\bullet}$, and alkoxyl, $RO^{\bullet}$). Other ROS include hydrogen peroxide (H_2O_2), hypochlorous acid (HOCl) and hydroperoxides and epoxide metabolites of endogenous lipids. These are not free radicals but contain chemically reactive oxygen-containing groups *(4)*.

ROS can be produced either accidentally or deliberately *(3)*. Small amounts of ROS, including $HO^{\bullet}$, $O_2^{-\bullet}$ and H_2O_2, are produced during normal

aerobic mitochondrial metabolism. Peroxisomes that include fatty acyl CoA oxidase, dopamine-β-hydroxylase and urate oxidase among others produce H_2O_2 as a by-product, which is then degraded by catalase. Some H_2O_2 molecules escape degradation, leaks into other compartments and induces oxidative damage. The cytochrome P450 mixed-function oxidase system in animals constitutes a primary defence against various xenobiotics and endogenous substances and enhances production of free radicals. On the other hand, phagocytic cells deliberately generate $HO^{\bullet}$, $O_2^{-\bullet}$ and HOCl and use them to inactivate bacteria and viruses. A number of exogenous factors may also increase the endogenous free radical load. High intakes of iron and copper or 'misplaced' iron as a result of tissue breakdown promote the generation of oxidizing radicals from peroxides *(5)*.

The initiation step in lipid oxidation involves the abstraction of a bis-allylic hydrogen from a polyunsaturated fatty acid (RH) by $HO^{\bullet}$ induced by ROS, heat, UV light or metal catalysts *(6)*. The resulting fatty acid acyl radical ($R^{\bullet}$) reacts rapidly with O_2 to form a fatty acid peroxyl radical ($ROO^{\bullet}$), with a reaction rate constant (k_1) of $3x10^8$ $mol^{-1}s^{-1}$. As $ROO^{\bullet}$ is more highly oxidized than $R^{\bullet}$, it will preferentially oxidize other unsaturated fatty acids, producing lipid hydroperoxides (ROOH) and $R^{\bullet}$, thereby propagating the chain reaction. The rate constant (k_2) for the reaction of $ROO^{\bullet}$ with an unsaturated fatty acid is relatively low ($10\text{-}10^2$ $mol^{-1}s^{-1}$). ROOH are both products of oxidation and substrates for further reactions with Fe^{2+} and Cu^{+}, yielding $ROO^{\bullet}$ and alkoxyl radicals *(7)*. Fe^{2+} reductively cleaves ROOH as follows:

$$Fe^{2+} + ROOH \rightarrow ROO^{\bullet} + Fe^{3+} + OH^{\bullet} \quad (1)$$

and Fe^{2+} can be regenerated as follows:

$$Fe^{3+} + ROOH \rightarrow ROO^{\bullet} + Fe^{2+} + H^{+} \quad (2)$$

Other strong reductants such as ascorbic acid also reduce Fe^{3+} to Fe^{2+} *(8)*. Both $ROO^{\bullet}$ and $RO^{\bullet}$ can initiate further reactions including:

$$RO^{\bullet} + RH \rightarrow ROH + R^{\bullet} \quad (3)$$

$ROO^{\bullet}$, $RO^{\bullet}$ and ROOH degrade to alkyl radicals ($R'CH_2^{\bullet}$), ethane, pentane and a range of aldehydes including hexanal, malondialdehyde and 4-hydroxynonanal, which can react readily with ε-amino groups of proteins to yield Maillard type complexes.

Lipid Oxidation in Meat and Meat Products

Phase two of oxidative damage occurs in the immediate post-slaughter stage. Biochemical changes during the conversion to muscle to meat result in conditions where oxidation in the highly unsaturated phospholipid fraction in

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subcellular membranes is no longer tightly controlled and the balance between prooxidative and antioxidative capacity favors oxidation. Post-slaughter factors such as early postmortem pH drop, carcass temperature and electrical stimulation are likely to disrupt cellular compartmentalization and release catalytic metal ions *(7)*.

The third phase of lipid peroxidation occurs during handling and processing and the mechanisms are likely to be similar to those that occur in stressed tissue *in vivo*. During handling, processing, cooking and storage, iron is released from high molecular weight molecules such as hemoglobin, myoglobin, ferritin and hemosiderin, resulting in the catalysis of lipid oxidation *(9)*. This may be compounded by dietary factors, particularly the degree of dietary fat unsaturation *(2)*.

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Antioxidants and stability of meat and meat products

Lipid oxidation may be controlled, or at least minimized, through the use of antioxidants. Dietary supplementation allows vitamin E to be incorporated directly into phospholipid membranes where lipid oxidation is initiated *(10)*. It has been calculated that vitamin E can scavenge $ROO^{\bullet}$ about 10^4 times faster than $ROO^{\bullet}$ can react with unsaturated fatty acids *(6)*, so that relatively small amounts of vitamin E are required for effective antioxidant protection *(2)*. Numerous studies have demonstrated that dietary vitamin E supplementation (in the form of *all-rac*-α-tocopheryl acetate) consistently increases α-tocopherol levels in muscle and improves oxidative stability in meat from a number of species including pigs, chickens, turkeys, cattle and lambs *(11, 12, 13, 14, 15)*. The majority of studies have focused on measuring chemical indices of oxidation, most notably thiobarbituric acid-reacting substances (TBARS). However, in recent years emphasis has begun to shift towards sensory evaluation and the measurement of secondary oxidation products with odor and flavor impact. Other naturally occurring antioxidants have also been examined. Some, such as ascorbic acid, have been shown to produce inconsistent effects and their practical value as antioxidants in meats has been questioned *(2)*. However, plant extracts such as tea catechins and rosemary extracts have produced more consistent antioxidant effects.

Lipid oxidation

Dietary vitamin E supplementation inhibits lipid oxidation in pork chops *(16)* and ground pork *(17)*. Supplemental vitamin E also improves oxidative stability in cooked chops packed in modified atmospheres *(18)* and vacuum packaged precooked chops and roasts *(19, 20)*. The majority of studies have examined the effects of supplementation in the range of 100-200mg α-tocopheryl kg^{-1} feed. Based on the rate of uptake of dietary α-tocopherol by various tissues and the time required to achieve tissue saturation and optimal

resistance to oxidation, Morrissey *et al. (11)* proposed that pigs should be fed 200mg kg^{-1} feed for about 90 days.

Supplementation of broiler diets with 200mg α-tocopheryl acetate kg^{-1} feed for at least 4 weeks prior to slaughter has been recommended in order to provide adequate protection against lipid oxidation in broiler breast and thigh meat *(21)*. Turkey tissues accumulate α-tocopherol more slowly than other species such as chicken. Wen *et al. (22)* observed that at supplemental intakes (300 or 600 mg α-tocopheryl acetate kg^{-1} feed) 13 weeks were required for muscle α-tocopherol to reach its highest level. At these levels of supplementation, lipid oxidation was inhibited in raw and cooked ground breast meat during refrigerated and frozen storage. Higgins *et al. (23)* supplemented turkey poults with 600mg α-tocopheryl acetate kg^{-1} feed for 21 weeks. Supplementation inhibited lipid oxidation in vacuum-packaged and aerobic-packaged raw ground breast and thigh meat, with the inhibitory effect being greater in aerobic-packaged meat. Supplementation with 300 and 600mg kg^{-1} feed also reduced lipid oxidation in previously frozen turkey breast which was cooked, sliced and refrigerated in aerobic packaging *(24)*. Mercier *et al. (25)* supplemented turkey diets with 400mg kg^{-1} for 16 weeks, and reported inhibition of lipid oxidation in refrigerated raw meat, when diets contained saturated (tallow) or unsaturated (rapeseed and soya) fats.

Refined rosemary extract has been proposed as a natural antioxidant in several food systems, especially those containing animal fats and vegetable oils *(26)*. The addition of rosemary extracts has been shown to inhibit lipid oxidation in pork fat *(27)* and fresh and precooked, minced meat products stored under refrigerated and frozen conditions *(28)*. Recently, the optimum concentration of rosemary required to effectively inhibit oxidation in fresh and previously frozen pork patties was determined as 0.1% *(29)*. In addition, Murphy *et al. (30)* observed that rosemary oleoresin extract inhibited lipid oxidation in the presence of salt in pre-cooked roast beef slices during refrigerated, but not frozen, storage. Mixtures of α-tocopherol and rosemary extract have been shown to exert a stronger protective effect than either antioxidant alone *(31)*. The addition of antioxidants (Duralox, Herbalox and BHA/BHT) during processing was found to be more effective in preventing lipid oxidation than dietary α-tocopherol supplementation alone in modified atmosphere packaged (MAP) and aerobically packaged beef patties. Furthermore, the combination of dietary α-tocopherol and rosemary extracts (Duralox and Herbalox) was found to be as effective in inhibiting lipid oxidation as the combination of dietary α-tocopherol and BHA/BHT during processing *(32)*.

Tea catechins are a group of polyphenols present mainly in green tea (*Camellia sinensis*). They are reported to be efficient scavengers of the superoxide anion and hydrogen peroxide *(33)*, and singlet oxygen *(34)*. Tang *et al. (35)* supplemented broiler diets with 50, 100, 200 and 300mg tea catechins kg^{-1}. Tea catechins exhibited an antioxidant effect in breast and thigh meat that had been ground and refrigerated following frozen storage for up to 9 months,

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with the greatest protection seen at the 200 and 300 level. Tea catechins offered a similar level of protection as an equivalent level of dietary vitamin E up to 3 months storage, while higher levels of tea catechins were required for longer storage periods. The antioxidant effects of added tea catechins in raw minced red meat (beef and pork), poultry (chicken, duck and ostrich) and fish (whiting and mackerel) muscle on susceptibility to lipid oxidation were compared to that of α-tocopherol during 10 days of refrigerated display *(36)*. The antioxidant potential of tea catechins was 2-4 fold greater than that of α-tocopherol at the same concentration and this potential was species dependent.

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Flavor

Secondary oxidation products (volatiles) contribute significantly to the flavor, and hence acceptability of meat. However, uncontrolled oxidation results in the formation of off-odors and off-flavors and the phenomenon of warmed over flavor (WOF). Dirinck *et al. (37)* found that supplementing the finishing diet with 200mg α-tocopheryl acetate kg^{-1} resulted in fresher flavor in refrigerated, cooked pork compared to meat from animals fed 60mg kg^{-1}. Cava *et al. (38)* reported that supplementation of pig diets with 100mg α-tocopheryl acetate kg^{-1} feed reduced levels of saturated aldehydes (hexanal, pentanal and heptanal) in raw muscle.

Blum *et al. (39)* observed flavor deterioration in refrigerated meat from broilers fed 20mg α-tocopheryl acetate kg^{-1} compared to those fed 160mg kg^{-1}. However, no effect was observed in frozen samples. De Winne and Dirinck *(40)* found that vitamin E supplementation (200mg kg^{-1} feed) inhibited the formation of saturated and unsaturated aldehydes in raw chicken breast and thigh meat and reduced off-flavor in cooked meat. Vitamin E supplementation also reduced WOF development in cooked ground chicken thigh meat following refrigerated storage for up to 5 days, and in previously frozen meat (up to 10 weeks*) (41)*.

Wen *et al. (42)* reported lower hexanal levels in ground cooked turkey following storage for 7 days from birds supplemented with 600mg α-tocopheryl acetate kg^{-1} feed compared to an unsupplemented group, and Higgins *et al. (43)* reported that the same level of supplementation resulted in less WOF development in refrigerated ground cooked meat. Sheldon *et al. (44)* investigated the effect of supplementation with up to 25 times the normal vitamin E requirement on flavor and headspace volatiles in turkey. This level consistently produced higher turkey meat flavor and aftertaste scores and lower oxidized meat flavor and aftertaste scores in cooked ground meat during refrigerated storage for up to 8 days. Similar effects were observed in samples which had been previously frozen for up to 150 days. Supplementation with over 10 times the normal requirement reduced total headspace aldehydes (which contribute to flavor) in raw samples refrigerated for 7 days. Recent data from our laboratory also indicate that supplemental dietary vitamin E reduces the formation of aldehydes in cooked ground duck meat (unpublished data).

Irradiation is known to induce off-odors in meats. Patterson and Stevenson *(45)* reported that dietary vitamin E supplementation (800mg α-tocopheryl acetate kg^{-1}) resulted in a 39 and 44% reduction in total volatiles at doses of 2.5 and 10.0 kGy, respectively, in thigh meat.

Cholesterol oxidation

Cholesterol oxidation products (COPs) may be involved in atherogenesis, and their control is, therefore, of interest in muscle foods. Dietary α-tocopherol supplementation has been shown to reduce COPs in refrigerated (2 and 4 days) cooked ground pork *(46)*. COPs represented 1.6% of total cholesterol in pork from pigs supplemented with 200mg α-tocopheryl acetate kg^{-1} diet compared to 2.1% in pigs fed a basal diet (10mg α-tocopheryl acetate kg^{-1} feed). Recently supplementation of pig diets with 200mg α-tocopheryl acetate kg^{-1} feed was shown to reduce total COPs in cooked pork chops after 0 and 9 days refrigerated storage, when diets contained sunflower oil, olive oil or mixtures of both with linseed oil *(47)*. Supplementation with 500mg α-tocopheryl acetate kg^{-1} feed reduced COPs by 65% in cooked veal following refrigerated storage for 4 days *(48)*. In ground, cooked chicken breast and thigh, 25-hydroxycholesterol concentrations were reduced by vitamin E supplementation (200 or 800mg kg^{-1} feed) after 12 days refrigerated storage *(49)*. Similarly, supplementation with 200mg kg^{-1} reduced total COPs by approximately 60% in cooked ground chicken refrigerated for 4 days *(50)*. Grau *et al. (51)* reported that vitamin E supplementation (225 kg^{-1} feed) inhibited COPs formation, regardless of dietary fat source, (saturated or unsaturated) in raw and cooked vacuum packaged ground dark chicken meat following storage at -20°C for 7 months. In beef, Galvin *et al. (52)* found that supplementation with 3000mg α-tocopheryl acetate/head/day reduced 7-ketocholesterol concentrations in vacuum packaged cooked *M. psoas major* steaks during refrigerated and frozen storage, but not in *M. longissimus dorsi*. This was attributed to the greater lipid stability of *M. longissimus dorsi*.

Irradiation increases COPs in meat at doses permitted for food use *(53)*. Supplementation of broiler diets with 400mg α-tocopheryl acetate kg^{-1} feed reduced total COPs in irradiated ground breast and thigh meat after 5 days refrigerated storage, compared to supplementation with 100 and 200 kg^{-1} feed *(54)*.

Lopez-Bote *et al. (55)* reported that supplementation of diets with rosemary oleoresin (500mg kg^{-1} feed) reduced total COPs in white and dark cooked ground chicken meat following refrigerated storage for up to 4 days. Supplementation with α-tocopheryl acetate (200mg kg^{-1} feed) reduced cops to a greater extent.

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Color

At the point of sale, color and color stability are the most important attributes of fresh meat quality and various approaches have been used to meet consumer expectation that an attractive bright-red color is compatible with long shelf-life and good eating quality *(56)*. There is no doubt that the willingness of consumers to purchase fresh meats is strongly influenced by the appearance of the meat on display. In red meats, particularly beef, a bright cherry-red color (bloom) is perceived by consumers as being indicative of freshness, while they discriminate against beef which has turned brown *(57)*. It is the oxidation of the fresh muscle pigment oxymyoglobin to the brown pigment metmyoglobin that leads to the discoloration of red meats. There is a general consensus that the processes of oxymyoglobin and lipid oxidation in muscle foods, while independent of each other, can be inter-related. However, the exact nature of this inter-relationship has not been established. For instance, one hypothesis is that oxymyoglobin oxidation initiates the first step in a sequence of chemical reactions leading to the production of radicals (porphyrin cation radicals) that lead to the initiation of lipid oxidation *(58)*. Conversely, another hypothesis is that muscle lipids and liposomes can catalyze oxymyoglobin oxidation *(59, 60)*. Irrespective of the process and inter-relationship between oxymyoglobin and lipid oxidation, dietary supplementation of α-tocopheryl acetate in bovine and ovine animals clearly stabilizes and extends the color shelf-life of meat cuts taken from these species. A comprehensive review of this area has been provided by Kerry *et al. (62)*. The majority of studies carried out on dietary supplementation with α-tocopheryl acetate to cattle have consistently shown improved color stability on subsequent retail storage of all meat cuts *(62, 63, 64, 65, 66, 67, 68, 69, 70)*. With the exception of the report by Strohecker *et al. (71)*, similar observations have been made for lamb meat color (*13, 14, 15)*. Little has been reported on the effects of tea catechins and rosemary extracts on meat color stability. However, research carried out in our laboratory has shown that their direct addition to red meat during processing has improved and extended fresh meat color. Conversely, addition of tea catechins, in particular, to white processed meat systems can cause problematic discolorations in the final products.

Drip loss

Excessive drip loss from fresh meat signifies not only financial losses associated with such meat but losses in valuable vitamins, minerals, flavor compounds and water. Loss of the latter component can affect overall eating quality, producing meat that can be described as tough and having poor mouthfeel characteristics. Vitamin E may have beneficial effects on drip loss, as it is involved in stabilizing lipid membranes *(72)*. One of the first reports on the effect of vitamin E on drip loss in meat was provided by Asghar *et al. (73)*. They reported that pork chops from pigs receiving a control diet had significantly higher drip loss than pork chops from pigs which had received a

diet supplemented with 200 mg α-tocopheryl acetate kg^{-1}. In addition, pork chops with higher drip loss were found to be more favorable to the growth of spoilage microorganisms. Den Hertog-Meischke *et al. (74)* showed that vitamin E supplementation affected drip loss in various bovine muscles. However, the authors demonstrated that this effect was muscle dependent. They showed that while α-tocopheryl acetate supplementation reduced drip loss in *M. semitendinosus*, it increased drip loss in *M. psoas major*. This contrast in terms of vitamin E effects on drip loss from muscles, as well as the methodologies used to carry out drip loss analysis, may help explain the wide range of views and data that researchers have generated in relation to this particular meat quality parameter.

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Conclusions

Lipid oxidation is one of the main factors limiting the quality and ultimately the acceptability of meat and meat products. There is considerable evidence that natural antioxidants, particularly vitamin E, are effective in reducing the extent of lipid oxidation. Dietary supplementation with vitamin E reduces lipid and cholesterol oxidation, oxymyoglobin oxidation, and, in some situations, drip loss. Supplementation also inhibits off-flavor formation. Based on current evidence, optimum levels of dietary vitamin E to effectively inhibit lipid oxidation can be suggested, as discussed previously *(11)*. However, the optimum levels of supplementation required to effectively maintain sensory quality are not clear. There is growing evidence to show that rosemary extracts and tea catechins are effective inhibitors of lipid oxidation. However, their impact on sensory quality also needs to be clarified.

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