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Improvement of Oxidative Stability of Myoglobin and Lipid with Vitamin E in Meat

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식육내 비타민 E에 의한 육색소와 지질의 산화 안정성 향상

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Abstract

Potential mechanisms by which vitamin E improves oxidative stability of myoglobin are documented. The basis by which this lipid-soluble antioxidant, α-tocopherol, protects water-soluble oxymyoglobin is beginning to be understood. Recent evidence suggests that α-tocopherol delays the release of prooxidative products of lipid oxidation from biomembranes, which in turn delays oxymyoglobin oxidation and the concomitant loss of desirable beef color. α, β-Unsaturated aldehydes are one class of lipid oxidation products that enhance oxymyoglobin oxidation in vitro and appear to act by covalently binding to the protein. If α-tocopherol delays the formation of these reactive aldehydes, then this could inhibit the prooxidative effect of these oxidation products toward oxymyoglobin. Additionally, α-tocopherol may exert part of its color-stabilizing effect in beef by enhancing the metmyoglobin reduction.

Key words : vitamin E, oxidative stability, myoglobin

Introduction

Meat color is the primary factor used by consumers to judge meat quality. The red color of meat is due to the pigment, myoglobin (Mb). Mb is the major oxygen storage and transport heme protein that gives meat its red color (Livingston et al., 1983). Myoglobin can exist in one of three pigment forms depending on the redox state of the heme iron and the molecule bound to the sixth coordination site of heme. The three principle states are deoxymyoglobin (DeoxyMb) which is purple, metmyoglobin (MetMb) which is brown, and oxymyoglobin (OxyMb) which is the cherry-red. Maintenance of desirable, fresh meat color is affected by many factors (Faustman and Cassens, 1990), and OxyMb may be maintained in meat by delaying its oxidation to MetMb, or through reduction of MetMb by reduced cytochrome b5 (Livingston et al., 1985).

Greene (1969) was one of the first investigators to report on the potential color-preserving effect of antioxidants in meat. The stability of Mb, specifically OxyMb, is enhanced in the presence of α-tocopherol *in vitro* (Chan et al., 1996), and is greater in beef that contains higher concentrations of this antioxidant (Faustman et al., 1989). A number of studies have demonstrated that supplementing the diets of cattle with α -tocopherol during the finishing period significantly delays lipid oxidation and color deterioration in beef, subsequently

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extending the color life in the display case by 2~5 days (Faustman et al., 1989; Arnold et al., 1993a,b). This is believed to be due to α-tocopherol's ability to act as an antioxidant in biological systems (McCay and King, 1980). α-tocopherol protects cell membranes from damage by neutralizing free radicals and terminating free radical chain propagation (Buttirss and Diplock, 1988). Schaefer et al. (1995) proposed that OxyMb stability increased in the presence of high concentrations of α-tocopherol due to slowing lipid oxidation directly via radical quenching. However, the mechanism by which lipid-soluble α-tocopherol maintains OxyMb, a water-soluble proteins remains unclear. Also the biochemical explanation for this interaction remain unresolved.

The purpose of this review article is to investigate the potential mechanisms by which α-tocopherol enhances the stability of OxyMb. A greater understanding of this effect would provide approaches for optimizing the meat color stability by vitamin E, and could provide a basis for studying other lipid:protein interactions which affect meat quality.

Myoglobin Chemistry, Oxidation and Reduction

Myoglobin can exist in one of three pigment forms (Fig. 1). The ferrous (+2) forms of Mb include DeoxyMb and OxyMb. DeoxyMb heme iron lacks a ligand at the sixth coordination site (Dickerson and Geis, 1983). DeoxyMb, purplished-red color, is typically viewed when the deep interior of a muscle is exposed during meat cutting, or when freshly cut meat is vacuum-packed. When meat is exposed to the atmosphere, oxygen binds to heme iron and forms OxyMb, the cherry-red pigment in beef. The process of Mb oxygenations is referred to as blooming and appears to differ in rate and extent among different species (Millar et al., 1994). Both of these ferrous forms may auto-oxidize to MetMb, which has an undesirable brownish-red color. The heme iron of MetMb is in the ferric state (+3) and is incapable of binding oxygen and instead binds water at the sixth coordination site (Romans et al., 1985; Allen and Hamilton, 1994). A ferryl (+4) form may result from hydrogen peroxide activation of the ferrous/ferric states, but this is short-lived and is more important for considerations of heme protein catalysis of lipid oxidation than for meat color

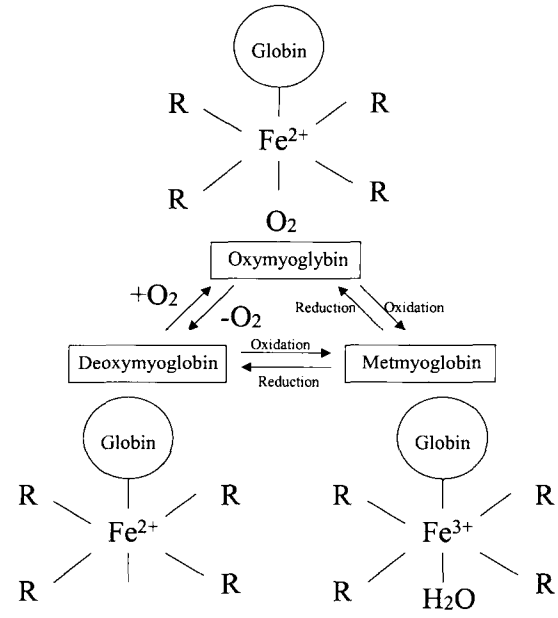

Fig. 1. Pathways of myoglobin oxidation and reduction.

(Kanner, 1994). In the retail setting, fresh meat is generally displayed in oxygen-permeable film and OxyMb is the desired pigment. Therefore maintenance of OxyMb is of primary concern. Though OxyMb can be readily oxidized to MetMb, a process which is thermodynamically favored (Shikama, 1985), reduction of MetMb to ferrous OxyMb may also occur. Once reducing equivalents in the meat are exhausted, complete MetMb formation will occur (Ledward, 1984). Thus, the color observed in meat represents the relative concentrations of ferrous and ferric pigments present at any time. Fig. 1 shows Mb oxidation and reduction pathways.

Reduction of MetMb to OxyMb by reduced cytochrome b_5 in meat has been reported (O'Keefe and Hood, 1982; Ledward, 1985; Faustman and Cassens, 1990). Livingston et al. (1985) first suggested that cytochrome b_5 was the likely mediator in skeletal muscle; this was confirmed by Arihara et al. (1990). Oxidized cytochrome b_5 can be reduced to an active form by cytochrome b_5 reductase, which utilize reducing equivalents from NADH (Strittmater, 1965). Cytochrome b_5 reductase has been identified in bovine cardiac (Livingston et al., 1985) and skeletal muscle (Arihara et al., 1989).

The potential practical implications of enzymatic MetMb reductions for improving meat color stability have been reported. Faustman et al. (1988) demonstrated that a crude liver containing cytochrome b_5 and cytochrome b_5 reductase

was capable of effecting MetMb reduction in the presence of NADH. Application of a similar liver extract with NADH to meat did not improve color stability (Mikkelsen and Skibsted, 1992). Reddy and Carpenter (1991) adapted the assay procedure originally proposed by Hagler et al. (1979) to measure MetMb reductase activity in beef. They found that this activity followed the order *tensor faciae latae* > *logissimus dorsi* > *gluteus medius* > *diaphragma medialis* > *semimembranosus* = *psoas majorc*. In subsequent work, Madhavi and Carpenter (1993) found that psoas steaks has greater MetMb accumulation, lower MetMb reductase activity, and greater oxygen consumption activity than *longissimus* steaks, which may in part explain the difference in color stability.

Lipid Chemistry and Oxidation

Triglycerides, phospholipids and cholesterol make up the three main forms of lipid in muscle. In fresh meat, lipid oxidation occurs primarily in membrane phospholipids (German and Kinsella, 1985) even though phospholipids represent only 0.5~1% of the total lipid content in meat (Igene and Pearson, 1979). Phospholipids are the major components constituting membrane bilayers (Karp, 1984). Both triglycerides and phospholipids have a glycerol backbone bound to fatty acids.

Fatty acids can be either saturated or unsaturated. Saturated fatty acids have hydrocarbon chains that do not contain any double bonds whereas unsaturated fatty acids contain varying numbers of double bonds located throughout the hydrocarbon chain. The degree of unsaturation determines membrane fluidity. Unsaturation can cause bending of fatty acid tails thus decreasing membrane rigidity (Baenziger et al., 1991). A greater number of double bond (i.e. greater unsaturation) increases the susceptibility of fatty acid to lipid oxidation (Gosgrove et al., 1987; Ledward et al., 1992). Sinclair et al. (1982) reported that muscle membrane phospholipids contained high concentration of C18:2, C18:3, C20:4, C22:4, C22:5 and C22:6.

The susceptibility of muscle tissue to lipid oxidation differs among species (Siu and Draper, 1978) and may also differ between the separate muscles of a single species (Rhee et al., 1986). The process of lipid oxidation involves a free radical chain reaction divided into three steps: initiation, propagation and termination (Halliwell and Gutteridge, 1990). The initiation step involves the production of a radical from some molecular precursor. Free radicals are generated most commonly through bond cleavages and the transfer of an electron from one molecule to another. Several free radical species such as hydroxyl radicals ($OH^{\cdot}$), peroxyl radicals ($LOO^{\cdot}$), alkoxyl radicals ($LO^{\cdot}$) and alkyl radicals ($L^{\cdot}$) are created during the initiation phase through direct thermal dissociation, metal catalysts, or exposure to light (Frankel, 1984; Nawar, 1985). Free radicals are extremely reactive due to the unpaired electron of the free radical not being spin paired with a second electron in a chemical bond.

A major oxidation initiator is singlet oxygen. Singlet oxygen is created when natural pigments like chlorophyll, riboflavin or myoglobin act as triplet sensitizer (S^3) in the presence of light, thereby exciting the ground triplet state molecular oxygen (O_2^3) to singlet oxygen state (O_2^1) (Frankel, 1984). The stable form of molecular oxygen (O_2^3) cannot attack the singlet state double bonds of fatty acids directly because this reaction does not obey the rule of spin conservation. The rule of spin conservation states that the magnetic charge of electrons allow them to exist in two different spin orientation, +1 and −1. In the singlet oxygen state, the two electrons have opposite spin orientations and great electrostatic repulsion causing the excited state. Singlet oxygen is more electrophilic than triplet oxygen and can react rapidly to initiate oxidation (Nawar, 1985).

Heme pigments, non heme iron and enzymatic lipid oxidation systems are thought to be the most critical catalysts of lipid oxidation in red meat and poultry (Love, 1983; Harel and Kanner, 1985; Rhee et al., 1986; Decker and Hultin, 1990; Anton et al., 1991; Chan et al., 1997). O'Brien (1969) reported that heme pigments act as catalysts of lipid oxidation primarily through their sensitizing action in the initiation step, while Harel and Kanner (1985) suggested that both ferrous and ferric forms of meat pigments may actually catalyze lipid oxidation. Non-heme iron may also accelerate lipid oxidation in cooked meats because its transition metal properties enable it to extract electrons to produce radicals (Love and Pearson, 1974).

The propagation step is characterized by rapid reaction of radicals with oxygen and formation of peroxyl radicalss ($LOC^{\cdot}$) (Burton and Ingold, 1986). The second half of propagation is the much slower reaction of the peroxy radical with a lipid molecule. During this reaction, hydroperoxide is formed, as

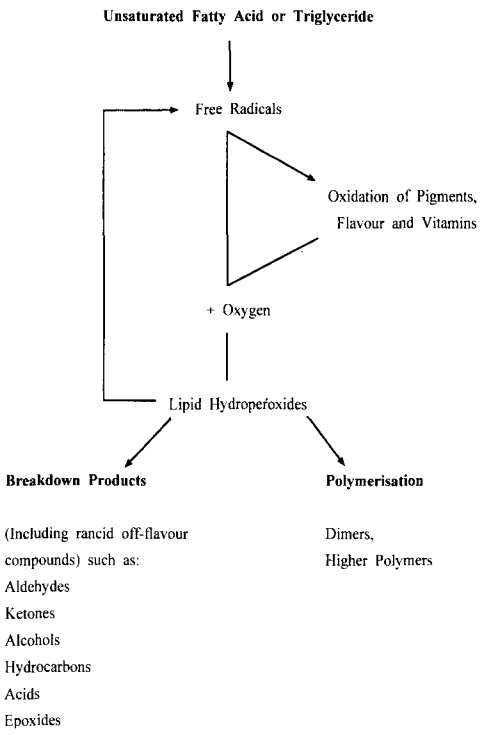

Fig. 2. Overall mechanism of lipid oxidation.

well as other radicals, through abstraction of hydrogen atoms from adjacent fatty acid side chains. The propagation step continues to cycle until two peroxy and/or carbon radicals come together to form molecular products.

Eventually, termination results when these radicals combine or are quenched by antioxidants to produce nonradical species which can on longer attack lipid components (Allen and Hamilton, 1994). This entire lipid oxidation cascade leads to the production of short chain fatty acid, aldehydes, alcohols and ketones which cause off-flavor, rancid odor and discoloration in meat (Moerck an Ball, 1974; Fogerty et al., 1989). Fig. 2 shows overall mechanism of lipid oxidation.

Interaction of Myoglobin and Lipid Oxidation

The complexity of muscle foods makes it extremely difficult to study myoglobin and lipid oxidation interactions. The redox chemistry of hemoglobin is similar to that of myoglobin and results from experiments with hemoglobin can provide insight into similar reactions involving myoglobin. Haurowitz et al. (1941) were among the first investigators to report the prooxidative effect of oxidizing fatty acids toward heme proteins. They demonstrated that hemoglobin was destroyed and heme iron released when incubated in the presence of unsaturated fatty acids and oxygen at 38℃. Gotz et al. (1994) reported that myoglobin extracted from chicken gizzard could bind fatty acids. Interestingly, OxyMb had a higher binding affinity than MetMb. Koizumac et al. (1973) showed that when arginine linoleate was mixed with OxyMb, the pigment was oxidized rapidly to MetMb with some degradation of the heme moiety.

The oxidation of OxyMb to MetMb appears related to lipid oxidation and dependent on antioxidant status (Yin et al., 1993). Myoglobin and lipid oxidation have been reported to be interrelated in liposomes (Szebeni et al., 1984; Yin and Faustman, 1993) and muscle foods (Hutchins et al., 1967; Rhee et al., 1986; Faustman et al., 1989). This relationship remains unclear but it may be that the process of lipid oxidation yields products which are prooxidative towards OxyMb. Heme iron is an effective catalyst of lipid oxidation (Love, 1983; Decker and Hultin, 1990; Anton et al., 1991; Chan et al., 1997). The oxidation of OxyMb produces superoxide anion (O_2^-) which can dismutate to hydrogen peroxide (H_2O_2) and can be subsequently converted to OH (Little and O'Brian, 1968; Kanner, 1994). MetMb can also react with H_2O_2 to form ferrylMb which is also prooxidative. Similarly, free radicals produced during lipid oxidation can alter heme chemistry and initiate pigment oxidation causing loss of desirable color (Greene, 1969; Faustman et al., 1992; Chan et al., 1997).

Renerre et al. (1992) demonstrated that the auto-oxidation rate of OxyMb extracted 2 hours postmortem from *psoas*, a color labile muscle, and *longissimus*, a color stable muscle, was not different. However, at 192 hours postmortem, OxyMb obtained from *psoas* oxidized more rapidly than that from *longissmus*. Foucat et al. (1994) repeated this work utilizing ^{1}H-NMR. These results supported the concept that oxidative processes occurring during extended storage of the two muscles led to a decreased stability of OxyMb after extraction from *psoas* relative to *longissimus*.

Effect of Aldehyde Lipid Oxidation Products on Protein

A variety of secondary aldehyde products such as n-alkanals,

trans-2-alkenals, 4-hydroxy-trans-2-alkenals and malondialdehyde are generated from lipid oxidation (Esterbauer et al., 1982; Esterbauer et al., 1991). Aldehyde products are more stable than free radical species and readily diffuse into the cellular media where they may exert toxicological effects by reacting with critical biomolecules *in vivo* (Esterbauer et al., 1991; Szweda et al., 1993; Loidl-Stahlhofen and Spiteller, 1994). Protein modification by aldehydes is believed to play a central role in many pathophysiological conditions (Szweda et al., 1993). 4-Hydroxynonenal (4-HNE) is an α,β-unsaturated aldehyde formed by oxidation of ω-6 unsaturated fatty acids (Esterbauer et al., 1985; Esterbauer and Cheeseman, 1990; Sakai et al., 1995) and is thought to be an important indicator of free radical stimulated lipid oxidation.

Previous work has shown covalent attachment of malondialdehyde to LDL (Haberland et al., 1988) and 4-HNE to glucose-6-phosphate dehydrogenase, hemoglobin and LDL (Esterbauer et al., 1991; Szweda et al., 1993; Bruenner et al., 1995; Uchida et al., 1994). Investigators have also shown modification of LDL by hexanal (Chen et al., 1992), modification of hemoglobin by pentanal, hexanal, hexenal, heptenal, octenal and nonenal (Kautiainen, 1992), modification of BSA by octenal (Alaiz and Giron, 1997) and modification of myglobin by 4-HNE (Faustman et al., 1999b). 4-HNE has also been shown to be highly cytotoxic to Ehrlich ascites tumor cells and *Salmonella typhimurium* and leads to lysis of erythrocytes (Esterbauer et al., 1985). 4-HNE has an inhibitory effect on glucose-6-phosphatase, Cyt P450, aminopyrine demethylase and adenylate cyclase (Esterbauer et al., 1985). Benedetti et al. (1984) proposed that α,β-unsaturated aldehydes can induce cytopathological effects in liver in the course of lipid peroxidation *in vivo*.

It has been shown that α,β-unsaturated aldehydes target sulfhydryl groups, primary amino groups, histidine, cysteine and lysine residues for modification (Uchida and Stadtman, 1992). Uchida and Stadtman (1992) have shown that these adducts can be formed through a Michael-type addition reaction. However, Szweda et al. (1993) also proposed the possibility of modification through a Schiff base bond. Chemical modification of amino acids of proteins during lipid oxidation is thought to be the basis for the role of prooxidative damage in the pathogenesis of disease (Requena et al., 1996). Faustman et al. (1999) showed increased oxymyoglobin oxidation in the presence of lipid oxidation products of aldehydes (Table 1). It could be possible that the presence of these lipid oxidation products may alter Mb redox stability through covalent modification (Faustman et al., 1998; Faustman et al., 1999). Covalent modification of equine, bovine and porcine Mb by 4-HNE has been demonstrated (Phillips et al., 2001; Lee et al., 2003) and Phillips et al. (2001) suggested that 4-HNE-induced alteration in bovine Mb structure might enhance susceptibility of the heme iron to oxidation.

Table 1. Initial rates of oxymyoglobin oxidation in the Presence of Selected Aldehydes at pH 7.4, 37℃

Treatment	Oxidation Rate (%MetMb/min)
Control	0.10
Hexanal	0.11
2-Heptenal	0.21
4-Hydroxynonenal	0.21
2-Nonenal	0.26

Vitamin E Incorporation into Muscle

Vitamin E is a fat soluble vitamin synthesized by plants and associated with subcellular membranes. Vitamin E is the generic term used to identify a family of compounds consisting of α-, β-, δ-, and γ-tocopherols (Toc) and their corresponding tocotrienols. The component structure has a 6 chromanal ring structure with a 13 carbon atom side chain. Methyl groups are attached to the side chain at the 4', 8' and 12' carbons. At the 3', 7' and 11' positions, tocopherols have saturated side chains whereas tocotrienols have unsaturated side chains. α-, β-, δ-, and γ-tocopherols differ from one another by the position, placement and number of methyl groups on the benzene ring. α-Toc accounts for 75% of the total vitamin E activity while the other forms of vitamin E are present in smaller concentrations (Hunt and Groff, 1990).

α-Toc is easily oxidized in the presence of oxygen and its oxidation is accelerated by light, heat, alkali and trace minerals such as ferric and cupric ions (Johnson, 1995). Therefore, a more stable form, α-tocopherol acetate is added to food systems rather than the free alcohol form. α-Toc can protect fatty acids by neutralizing free radicals and terminating free radical chain propagation (Buttirss and Diplock, 1988). In this function, vitamin E is itself consumed and becomes a stable tocopherol radical by donating its phenolic

hydrogen atom to peroxyl radicals formed by the oxidation of membrane phospholipids (Koshas et al. 1984).

α-Toc may be incorporated into muscle foods endogenously or exogenously. Vitamin E has been supplemented in the diets of livestock species to reduce lipid oxidation of meat from poultry (Marusich et al., 1975; Klopfenstein and Clegg, 1980), pig (Monahan et al., 1994), cattle (Faustman et al., 1989; Lynch et al., 1999) and trout (Boggio et al., 1985). α-Toc acetate, the form used in dietary supplementation, has the acetate group cleaved during digestion, regenerating the free alcohol form of vitamin E which can be absorbed. Although the exact site of cleavage is unknown, Hunt and Groff (1990) suggested that it may occur in the lumen of the intestine, in the brush border, or within the enterocytes. Diplock (1984), suggested that the uptake of tocopherol by the gut proceeds by a non-saturable passive diffusion process without a carrier. Machlin (1991) reported a dependence of α-Toc absorption on the presence of bile salts and pancreatic enzymes. The efficiency of absorption decreased as large amounts of tocopherols were consumed.

Vitamin E can also be incorporated into muscle foods exogenously. The most common way of adding tocopherols to a ground product is by incorporation into the meat after the first grinding and before the last grinding (Mitsumoto et al., 1991a). On whole muscle cuts, a dipping or spraying procedure can be used (Mitsumoto et al., 1991b). With exogenous addition, Mitsumoto et al. (1991a) hypothesized that lipid soluble tocopherols would associate with adipose tissue as well as membrane lipids but would not be incorporated into the membranes as in endogenous addition. Mitsumoto et al. (1993) reported that exogenous addition significantly improved ground beef pigment and lipid stability, but was much less efficient than equal concentration of vitamin E achieved by dietary supplementation.

As further evidence that α-Toc can exert its antioxidant function in postmortem muscle, Faustman et al. (1999a) investigated the occurrence of α-Toc oxidation products in beef. They characterized α-Toc oxidation using a stable isotope dilution capillary gas chromatography-mass spectrometry assay for α-Toc and its principal oxidation products (Fig. 3). The concentration of α-Toc (TH) and three major oxidation products, α-tocopherolquinone (TQ), 5,6-epoxy-α-tocopherolquinone (TQE1), and 2,3-epoxy-α-tocopherolquinone (TQE2), found in ground psoas muscle at 0 and 4 days of 4℃ storage are presented in Table 2. During storage, TBARS and % MetMb values increased, indicating that oxidation occurred. The concentration of α-Toc decreased while that of α-TQ, α-TQE1, and α-TQE2 increased, a result consistent with the peroxy radical scavenging activity of α-Toc (Liebler et al., 1996).

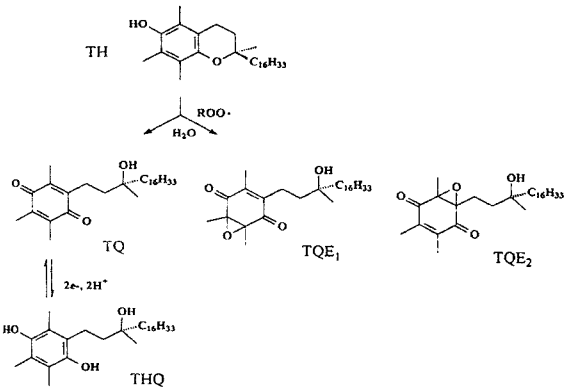

Fig. 3. Oxidation pathway for α-tocopherol in peroxyl radical scavenging reactions. TH, α-tocopherol; TQ, α-tocopherolquinone; THQ, α-tocopherolhydroquinone; TQE1, 5,6-epoxy-α-tocopherolquinone; TQE_2, 2,3-epoxy-α-tocopherolquinone.

Vitamin E and Meat Quality

The strategy for supplementing the diets of beef cattle is to

Table 2. Oxidation of α-tocopherol[a] to its quinone and quinon epoxide products and formation of TBARS and metmyoglobin(MetMb) in ground beef *Psoas*(n=2) stored at 4℃

	TH	μg/g	TQ	μg/g	TQE1	μg/g	TQE2	μg/g	TBA		Met	%
Day	0	4	0	4	0	4	0	4	0	4	0	4
Sample												
A	3.28	2.59	0.31	0.45	0.09	0.10	0.20	0.16	0.12	0.22	27	42
B	3.36	2.59	0.34	0.50	0.06	0.13	0.14	0.19	0.18	0.36	32	46

[a] Recovery of total tocopherol was 86% for sample A and 88% for sample B.

achieve sufficient muscle concentration of α-Toc to maximize the antioxidant effect. Faustman et al. (1989) proposed 3μg of α-Toc/g of fresh *gluteus medius* (GM) as the ideal concentration. Arnold et al. (1993a) analyzed a larger data set and proposed critical concentrations for *longissimus lumborum* (LL) and GM of 3.3 and 3.8 μg/g of fresh muscle, respectively. To achieve an ideal concentration of 3.5μg/g, Arnold et al. (1993b) suggested 1,300 IU/steer daily for 44 days. Vitamin E supplementation was first shown to delay the discoloration of Holstein loin steaks. However, in a subsequent study, Arnold et al. (1993b) found that the color-stabilizing influence of vitamin E was not restricted to Holsteins. The authors fed Holstein, Angus, Hereford, and Charolais steers with diets including 0, 360, and 1,290 IU α -tocopheryl acetate/steer daily for 252 days. Concentrations of α-Toc in LL and GM were increased by each increment of supplementation. MetMb formation in LL and GM was delayed by vitamin E supplementation. Supplementation of 1,290 IU daily did not give additional benefit compared with 360 IU/day for meat aged 7 days, but resulted in the most color stable meat when an ageing period was extended up to 21 days. Increased color stability of vitamin E supplemented beef was further supported by results from other studies and several reviews have been published on dietary supplementation of vitamin E for improving beef color stability (Faustman, 1993; Schaefer et al., 1995; Liu et al., 1995). Improved color stability has been observed in fresh (Faustman et al., 1989; Arnold et al., 1993a; Sherbeck et al., 1995; Chan et al., 1996; Liu et al., 1996; Lynch et al., 1999) and frozen (Lanari et al., 1995) beef, fresh pork (Ashgar et al., 1991), lamb (Wulf et al., 1995; Guidera et al., 1995) and turkey (Sante and Lacourt, 1994).

It is important to note that the relative color stability of different muscles (O'Keefe and Hood, 1982) is not changed as a result of vitamin E supplementation. Chan et al. (1996) demonstrated that the order of color stability of beef *psoas* < GM < LL within control or vitamin E-supplemented treatments even though within each treatment, *psoas* contained the highest concentration of α-Toc. Thus, while vitamin E has profound effects on OxyMb stability, there remain other factors equally or more critical to meat color stability. Schaefer et al. (1995) proposed that the enhanced OxyMb stability is due to α-Toc slowing the process of lipid oxidation directly via radical quenching. This delays production of lipid oxidation breakdown products (e.g. peroxides) which can accelerate OxyMb oxidation. Lynch et al. (1998) proposed a potential mechanism through which α-Toc reduced cytochrome b_5 which in turn reduced MetMb to OxyMb *in vitro*. Thus, the protection of OxyMb by α-Toc appears to be indirect. However, further work is needed to determine if a direct interaction between α- Toc and OxyMb exists, and whether or not α-Toc improves meat color stability by enhancing MetMb reduction.

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요 약

비타민 E에 의한 육색소의 산화 안정성 향상 기작을 정리하였다. 지용성 산화제인 α-토코페롤이 수용성 단백질인 옥시마이오글로빈을 보호하는 원리가 밝혀지고 있다. 최근의 연구들에서 α-토코페롤이 세포막 지방산화의 2차 산화물들의 방출을 지연시켜 옥시마이오글로빈 산화를 억제시키고 식육의 바람직한 육색을 유지시킨다는 증거가 제시되고 있다. 지방산화물의 한 그룹인 α,β-불포화 알데하이드들은 단백질과 서로 결합하는 역할을 하여 옥시마이오글로빈의 산화를 증진시키는 것으로 밝혀졌다. 만약 α-토코페롤이 이런 활동적인 알데하이드들의 발생을 지연시킨다면 이런 지방산화물들이 옥시마이오글로빈의 산화에 미치는 영향도 억제될 것이다. 또한 α-토코페롤은 메트마이오글로빈의 환원에 작용하여 쇠고기의 육색 안정성 유지에 일정부분 역할을 담당하는 것으로 사료된다.

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