OXIDATIVE PROCESSES IN MEAT AND MEAT PRODUCTS: QUALITY IMPLICATIONS

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Abstract

Lipid peroxidation is in most instances a free radical chain reaction that can be described in terms of initiation, propagation, branching and termination processes. With regard to lipid peroxidation, one of the most important questions concern the source of the primary catalysts that initiate peroxidation in situ muscle foods. When cells are injured, such as in muscle foods after slaughtering, lipid peroxidation is favored, and traces of $O_2^{-}\bullet$, H_2O_2 as traces of lipid peroxides are formed. The stability of a muscle food product will depend on the "tone" of these peroxides and especially from the involvement of metal ions in the process. The cytosol contains not only prooxidants but also antioxidants and the tone of both affect the overall oxidation. Lipid peroxidation is one of the primary mechanisms of quality deterioration in foods and especially in meat products. The changes in quality can be manifested by deterioration in flavor, color, texture, nutritive value and the production of toxic compounds.

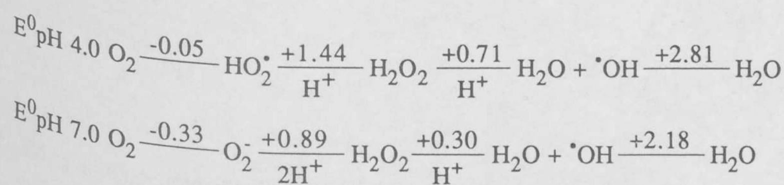
Introduction

Oxidation by free radicals, until the end of the sixties, was almost exclusively an area of research in radical chemistry, polymer and food sciences. During the last years this area was expanded and now is one of the most important areas of research in biology and medicine. When cells are injured, such as in muscle foods after slaughtering, oxidative processes are favored. These oxidative processes affected lipids, pigments, proteins, carbohydrates, vitamins and the overall quality of the foods.

The electronic structure of oxygen has two unpaired electrons at energy level of π antibonding, in triplet state, $^3\Sigma g$. The majority of organic biomolecules, including polyunsaturated fatty acids (PUFA) are in the singlet ground state. The reaction of triplet molecules, such as oxygen, with ground state singlet molecules are spin forbidden, because spin angular momentum must be conserved (KANNER et al., 1987). This barrier does not apply to reactions with single electrons, hydrogen atoms or other atoms or molecules containing unpaired electrons such as transition metals or free radicals.

Activation of Oxygen and Metal Compounds

The reduction of oxygen via one-electron reduction processes yields several products, such as superoxide anion radical ($O_2^{-}\bullet$), perhydroxyl radical ($HO_2^{\bullet}$), hydrogen peroxide (H_2O_2) and hydroxyl radical ($HO^{\bullet}$), all of which may participate directly or indirectly in oxidative processes in meat and meat products. The schematic one-electron reduction of oxygen and its redox potentials at pH 4.0 and 7.0 are presented:



$$E^0 \text{pH } 4.0 \quad O_2 \xrightarrow{-0.05} HO_2^{\bullet} \xrightarrow[H^+]{+1.44} H_2O_2 \xrightarrow[H^+]{+0.71} H_2O + {}^{\bullet}OH \xrightarrow{+2.81} H_2O$$

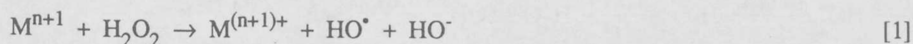
$$E^0 \text{pH } 7.0 \quad O_2 \xrightarrow{-0.33} O_2^{-} \xrightarrow[2H^+]{+0.89} H_2O_2 \xrightarrow[H^+]{+0.30} H_2O + {}^{\bullet}OH \xrightarrow{+2.18} H_2O$$

Thermodynamically, in order to predict or explain the feasibility of reactions which initiate oxidation, the relationship between the redox potential of the catalyst species and the substrate should be known. One electron oxidation of biomolecules such as hydrocarbons, PUFA, or aromatic compounds with a redox potential exceeding +1V can only be attacked by catalysts with redox potential over +1V. Such catalysts are, perhydroxyl radical, hydroxyl radical, ferryl ion and lipid free radicals (KANNER et al., 1987).

Superoxide and Perhydroxyl Radical. Under biological conditions, significant amount of O_2^- can be generated. In food muscle tis
don't have direct evidence that O_2^- is generated, however many studies done with other biological tissues support the presence o
muscle foods. The sources for O_2^- in muscle food could arise from membrane electron transfer systems, autoxidation of oxymyo
metmyoglobin, activation of several leukocytes presented in the vasculature of the muscle tissue, and oxidation of ascorbic acid a
reducing components by "free" iron (KANNER et al., 1987).
Perhydroxyl radical ($HO_2^{\bullet}$), whose pKa is 4.8 in water, is a much stronger oxidant than O_2^-, $HO_2^{\bullet}$, but not O_2^- could initi
peroxidation. (GEBICKI and BIELSKI, 1981). The loss of charge during formation of $HO_2^{\bullet}$ from O_2^- allows the radical to pene
the membrane lipid region more easily, where it could initiate lipid peroxidation (HALLIWELL and GUTTERIDGE, 1986
physiological conditions, nearly 0.3% of the O_2^- formed exists in the protonated form. However, near membrane the pH drop 3.
(ETHERINGTON et al., 1981), in muscle tissue the pH decreased from 6.5-7.0 to 5.5-6.0 and the amount of ($HO_2^{\bullet}$) could reach
of O_2^-. However $HO_2^{\bullet}$ has not yet been proved to initiate lipid peroxidation of cell membranes.

Hydrogen Peroxide. Hydrogen peroxide is normally present as a metabolite at low concentration in aerobic cells. A system gene
would be expected to produce H_2O_2 by non-enzymatic dismutation or by superoxide dismutase (SOD) catalyzed dismutation.
Mitochondria, microsomes, peroxisomes and cytosolic enzymes have all been recognized as effective H_2O_2 generators when fully p
with their substrates. H_2O_2 can be generated directly by several enzymes, such as aldehyde oxidase or glucose oxidase (KANNER
1987). H_2O_2 is produced from autoxidation of flavins, thiols, phenoleates, or ascorbic acid, by O_2^- generation and spontaneous dism
to H_2O_2, or by the interaction of the O_2^- with the semiquinone radical, such as that of ascorbic acid (KANNER et al., 1986). The
these reactions was found to be $2.6.10^8$ M^{-1} sec^1 (BIELSKI et al., 1983), the overall rate of O_2^- dismutation is only 5.10^5 M
(HALLIWELL and GUTTERIDGE, 1986). H_2O_2 has limited reactivity and has not been shown to react directly with polyunsaturate
acids, however it can cross biological membranes (HALLIWELL and GUTTERIDGE, 1986).
The generation of H_2O_2 in turkey muscle tissues was determined. Incubation of muscle tissues at 37°C shows H_2O_2 genera
1 nmole/min/g of fresh weight. Muscle tissues aging at 4°C increase the generation of H_2O_2 (HAREL and KANNER, 1985).

Hydroxyl Radical. Hydroxyl radical, ($HO^{\bullet}$), is produced when water is exposed to high-energy ionizing radiation, and its propertie
been documented (SIMIC and KAREL, 1980). One electron reduction of H_2O_2 decompose it to HO^- and $HO^{\bullet}$, a highly reactive (E
+2.18) capable to oxidize lipids and any other biological molecules. Hydroxyl radical produces in vivo or in situ would react at or c
its site of formation. Most of the $HO^{\bullet}$ generated in vivo or in situ comes from the metal-dependent breakdown of H_2O_2, according
following reaction:

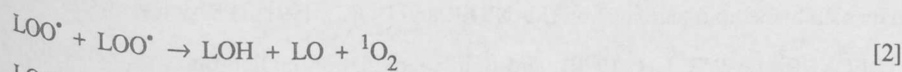


$$M^{n+1} + H_2O_2 \rightarrow M^{(n+1)+} + HO^{\bullet} + HO^- \qquad [1]$$

in which M^{n+1} is a transition metal. Fe+2 is known to form the same reaction, which is also called Fenton reaction (ANBAR and N
1967). In vivo or in situ muscle tissues, only the iron(II)-dependent formation of $HO^{\bullet}$ actually happens under normal co
(KANNER et al., 1987). It may be possible that in several muscle foods and especially in fish, Cu(I) also play an important role i
generation (DECKER and HULTIN, 1990). An interesting calculation of $HO^{\bullet}$ formed in one cell if free iron and H_2O_2 is in the ran
µM, was presented to be 46/sec (HALLIWELL and GUTTERIDGE, 1990). Such an amount could produce an enormous effect o
and especially to biological systems. Hydroxyl radical was detected in beer (KANEDA et al., 1988). We have adopted a me

ermine $HO^•$ in muscle homogenate and cytosol using benzoate as a scavenger of the radical. During this reaction, benzoate hydroxylate monohydroxy compounds, mostly to salicylic acid, the compounds are separated by HPLC and the hydroxylated compounds detected orometrically (KANNER et al., 1991). Our study demonstrated that $HO^•$ radicals are formed especially during heating. The addition of DTA, which is known to increase the yield of $HO^•$ produced by iron-ascorbate (KANNER et al., 1986) was found to also enhance the eration of $HO^•$ in muscle food. Hydroxyl radicals attack every biological molecule, the homogenate, which contains more proteins and er compounds, because competition with benzoate to $HO^•$ shows lower results than the cytosol. The same differences were obtained if homogenate and the cytosol were gamma-irradiated. The results demonstrated that the potential of the cytosol and homogenate to erate $HO^•$ is 3-fold less than samples gamma-irradiated by 100 krad (KANNER, 1992). Hydroxyl radicals could be determined in ogical tissues and food by the accumulation of o-tyrosine, a non-metabolite product of phenylalanine (KARAM and SIMIC, 1988).

glet Oxygen. Singlet oxygen could be generated by microwave discharges (ARNOL et al., 1964), chemicals (KHAN and KASHA, 64) and photochemical reactions (FOOTE and WEXLER, 1964). During propagation of lipid oxidation, hemeproteins could accelerate generation of peroxyl radicals, which by disproportionation from singlet oxygen and electronically excited states of carbonyl (KANNER al., 1987), by the following reaction:



$$LOO^• + LOO^• \rightarrow LOH + LO + {}^1O_2 \quad [2]$$

$$LOO^• + LOO^• \rightarrow LOH + LO^* + O_2 \quad [3]$$

inglet oxygen could initiate lipid peroxidation (KANNER et al., 1987), however no strong evidence of this pathway was found in muscle oods. Most recently, it was published that light may accelerate lipid peroxidation in pork and turkey, which was inhibited by ${}^1O_2$ uenchers (WHANG and PENG, 1988).

erryl Ion. Myoglobin and hemoglobin, in the ferrous or ferric states of these proteins, are activated by H_2O_2 producing a short-lived ntermediate of ferryl (Fe^{+4}) or oxo-ferryl radical (KANNER and HAREL, 1985a). Hydrogen peroxide-activated metmyoglobin and emoglobin were found to oxidize a series of compounds (KANNER and HAREL, 1985b) and to cause protein cross-linking (RICE et al., 983). We have reported that H_2O_2-activated myoglobin and hemoglobin could initiate membrane lipid peroxidation (KANNER and HAREL, 1985a). Activation of myoglobin and hemoglobin by H_2O_2 producing a ferryl compound which initiate membrane and non-membrane lipid peroxidation were demonstrated during the last years by many researchers (ASGHAR et al., 1988; RHEE, 1988; ARDUINO et al., 1990).

In order to determine if ferryl myoglobin is formed in muscle tissue, we performed spectrophotometric studies in the presence of sodium ulfide (Na_2S). The results indicated that ferryl ion is generated in meat and could be reduced by Na_2S to a chlorin-type structure or, in its absence, by muscle cytosol reducing agents (KANNER, 1992).

"Free" Metal Ions. Transition metals, e.g. iron and copper, with their labile d-electron system, are well-suited to catalyze redox reactions. Iron is an important catalyst in biological systems (KANNER et al., 1987). About two-thirds of body iron is found in hemoglobin, and smaller amounts in myoglobin. A very small amount of prosthetic components in various iron-containing enzymes, and in the transport protein transferrin. The remainder is present in intracellular storage proteins ferritin and hemosiderine (AISEN and LISKOWSKY, 1980). A small pool of non-protein non-heme iron provides "free" iron at micromolar concentration in tissues.

The small "transit pool" of iron seems to be chelated to small molecules. The exact chemical nature of this pool is not clear, but represent iron ions attached to phosphate esters (ATP ADP), organic acids (citrate), and perhaps to membrane lipids, and (CRICHTON and CHARLOTEAUX-WALTER, 1987). All these iron compounds are capable of decomposing H_2O_2 or ROOH free radicals (KARAM and SIMIC, 1988). GUTTERIDGE et al. (1981) developed the bleomycin assay to measure the availability in biological tissues for radical reaction. Using this method and others which were developed, we found that turkey muscle tissue co between 0.5-2.0 μg/g F.W. of "free" chelatable iron ions (KANNER et al., 1988; KANNER et al., 1991). Compared with the non iron found in beef, the amount of "free" iron in dark turkey muscle is higher than that found by IGENE et al. (1979) or SCHRINKE MILLER (1983) and lower than that reported by SKLAN et al. (1983) for turkey muscles.

The main source of free iron in cells seems to be ferritin. Ferritins are the main-molecule-containing proteins that store iron in cells and LISKOWSKY, 1980). Iron can be released from ferritin and utilized by mitochondria for the synthesis of hemoproteins. In cells, mitochondria synthesize myoglobin (FLATMARK and ROMSLO, 1975). Ferrous ions can be released from ferritin by red small enough to pass through channels in the protein shell (ULVIK, 1982). Recently, it was found that O_2^- releases iron from (BOYER and MCCLEARY, 1987) and O_2^- is the primary reductant in ascorbate mediated ferritin iron release (BOYER and MCCL 1987). Recently, ferritins were separated from turkey muscle tissue. During storage of muscle food, ferritin lost iron at a significan and this amount was found to initiate membrane lipid peroxidation (KANNER and DOLL, 1991). Physiological concentration of was found also in beef muscle (DECKER and WELCH, 1990), and lipid peroxidation of liposomes by horse spleen ferriti demonstrated. SEMAN et al. (1991) provided data which also suggested that ferritin may be responsible for catalyzing lipid peroxida muscle foods. The amount of free copper in muscle foods seems to be very low and most of it chelated to histidine-di-peptides su carnosine, which stop them from catalysis of lipid peroxidation (KOHEN et al., 1988).

Catalysis of Lipid Peroxidation

Non-enzymic Catalysis. Lipid peroxidation is one of the primary mechanisms of quality deterioration in stored foods, especially in tissues (SIMIC and KAREL, 1980; KANNER et al., 1987; ST. ANGELO and BAILEY, 1987; ASGHAR et al., 1988; RHEE, Many studies have been directed toward the identification of the catalysts that promote the oxidation of muscle lipids. In the beginning peroxidation was attributed to heme catalysts (WATTS and PENG, 1947; TAPPEL, 1953). The involvement of heme proteins as ca of lipid peroxidation was first described by ROBINSON (1924). Studies employing model systems of linoleate emulsions (L WATTS, 1970) showed that both heme and non-heme iron are important catalysts of muscle lipid peroxidation. Model system studies water-extracted muscle residues implied that myoglobin was not the principal prooxidant in meat, and non-heme iron are the main ca (SATO and HEGARTY, 1971; LOVE and PEARSON, 1974; TICHIVANGANA and MORRISSEY, 1985).

However, all these model systems suffer from many errors. Model systems using linoleate contain preformed hydroperoxides (KA 1974). They could not simulate lipid peroxidation in muscle cells, which are mostly of membranal nature, containing uno phospholipids. Most researchers using washed muscle tissues had omitted from the system several important compounds which, the exhaustive washing or dialysis were removed (SATO and HEGARTY, 1971; RHEE et al., 1987; ASGHAR et al., 1988; JOHNS 1989). During incubation of minced muscle tissue H_2O_2 is generated (HAREL and KANNER, 1985) and this could activate myogl hemoglobin to ferryl compound, which initiate membrane lipid peroxidation (HAREL and KANNER, 1985a,b; KANNER and H 1985). H_2O_2 is only one of the cytosolic compounds that could affect lipid peroxidation in muscle foods. One should know cytosol contains pro- and antioxidants, all these compounds will affect in situ lipid peroxidation.

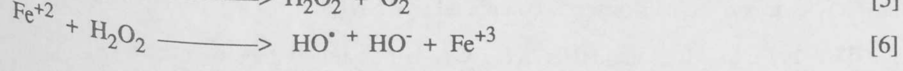
The cytosolic extract from turkey muscles totally inhibited membranal lipid peroxidation, catalyzed by ferryl ion. Turkey muscle cytosol contains reducing compounds at a level equivalent to ~3 mg of ascorbic acid equivalent/100 g of fresh weight, or a concentration of 150 μM. The amount of ascorbic acid is 80% of the reducing compounds (KANNER et al., 1991). Most recent results indicate that destruction of ascorbic acid by ascorbate oxidase does not prevent the inhibitory effect of the cytosol toward ferryl (results not shown). Carnosine present in the muscle tissue at high concentration was also found by us to reduce ferryl to ion (KANNER, 1992).

In spite of recent studies, we do not know which are the main inhibitors in the cytosol that prevent ferryl oxidation of membrane lipids. Our data indicate that <u>in situ</u> turkey muscle, the iron-redox cycle-dependent lipid peroxidation, and not ferryl, may play a major role in the first phase of catalysis of lipid peroxidation in muscle foods.

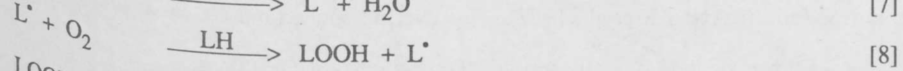
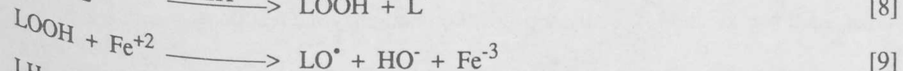
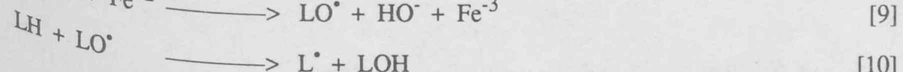
<u>Iron Redox Cycle-Dependent Lipid Peroxidation</u>. Biological oxidation is due almost exclusively to metal ion-promoted reactions, of which iron is the most abundant (AISEN and LISKOWSKY, 1980). It has long been known that iron can catalyze peroxidation and that this can be stimulated by the presence or addition of thiols and ascorbic acid (OTTOLELENGHI, 1959; WILLS, 1965; KANNER, 1974). However, only recently has the involvement of hydroxyl radicals in these reactions been postulated along with the contribution to the initiation of lipid peroxidation (BORG et al., 1978; TIEN et al., 1982).

Ferrous ions in aerobic aqueous solution produce superoxide, hydrogen peroxide and hydroxyl radical (KANNER et al., 1987) by the following reactions:

$$Fe^{+2} + O_2^- \longrightarrow Fe^{+3} + O_2 \quad [4]$$

$$2O_2^- + 2H^+ \longrightarrow H_2O_2 + O_2 \quad [5]$$

$$Fe^{+2} + H_2O_2 \longrightarrow HO^{\bullet} + HO^- + Fe^{+3} \quad [6]$$

This reaction is cycled by O_2^- (so-called Haber-Weiss), or by reducing agents, e.g. ascorbic acid or thiols (so called Redox-Cycle). Ascorbic acid NAD(P)H, glutathione and cysteine are known reducing compounds present in biological tissues. They very rapidly reduce ferric to ferrous ions, producing a redox cycle (KANNER et al., 1986). In fact, reaction [6] describes Fenton's reagent (FENTON and JACKSON, 1898), and the first redox cycling Fenton's reagent was developed by UDENFRIEND et al. (1954) using ascorbic acid, Fe-EDTA, oxygen and H_2O_2 for hydroxylation of aromatic compounds. Ferrous ions can stimulate lipid peroxidation by generating the $^{\bullet}OH$ from H_2O_2 but also by the breakdown of performed lipid peroxides (LOOH) to form the alkoxyl ($LO^{\bullet}$) radical (KANNER, 1974) (Equation 9).

$$LH + HO^{\bullet} \longrightarrow L^{\bullet} + H_2O \quad [7]$$

$$L^{\bullet} + O_2 \xrightarrow{LH} LOOH + L^{\bullet} \quad [8]$$

$$LOOH + Fe^{+2} \longrightarrow LO^{\bullet} + HO^- + Fe^{-3} \quad [9]$$

$$LH + LO^{\bullet} \longrightarrow L^{\bullet} + LOH \quad [10]$$

The reaction of ferrous with LOOH are an order of magnitude faster than their reaction with H_2O_2, 10^3 M and 76 $M^{-1}s^{-1}$, respectively (GARNIER-SUILLERO et al., 1984).

Iron redox-cycle-dependent membrane lipid peroxidation needs to attack unsaturated fatty acids only by a few HO$^{\bullet}$ radicals, once th hydroperoxides are formed, propagation of lipid peroxidation will be catalyzed by the breakdown of LOOH to free radicals, and reactions are not affected by SOD, catalase or HO$^{\bullet}$ scavengers.

The importance of "free" iron ions in the catalysis of turkey muscle lipid peroxidation was demonstrated by us using ceruloplasm specific inhibitor of ferrous-dependent lipid peroxidation (KANNER et al., 1988). Our results were also confirmed by others (DE and HULTIN, 1990).

Reducing compounds are the driving forces for the catalysis of lipid peroxidation by metal ions. We found that in turkey muscle, as acid is the main reducing compound which affects iron-redox cycle. Destruction of the cytosolic ascorbic acid by ascorbic acid ox prior to the addition of the cytosol to the membrane washed model system, totally inhibited lipid peroxidation (KANNER, 1992). Ho in fish, other compounds, and not ascorbic acid, seem to affect the iron-redox cycle (DECKER and HULTIN, 1990).

The cytosol contains compounds acting to stimulate, but also to inhibit, the process of lipid peroxidation. Turkey muscle cytosol co compounds which totally inhibit membrane lipid peroxidation by ferryl ion, however only partially-inhibited lipid peroxidation by an redox cycle (KANNER et.al., 1991). The inhibitory effect of the cytosolic extract was also shown by other researchers (SLAB HULTIN, 1983; HAN and LISTON, 1989).

Enzymic Catalysis. Lipid peroxidation in isolated microsomes requires NADPH or NADH, cytochrome P450 reductase, met oxygen, and is markedly stimulated by ADP-Fe^{+3} (HOCHSTEIN and ERNSTER, 1963; PEDERSON et al., 1973). The enzyme tr an electron to oxygen molecule producing $O_2^{\bullet-}$ which dismutate to H_2O_2. Superoxide reduce also ferric-ADP to ferrous-ADP which like Fenton reaction generate from H_2O_2 hydroxyl radicals or ferryl compound (KANNER, 1987). HULTIN and his associates (LI HULTIN, 1976; PLAYER and HULTIN, 1977; SLABYJ and HULTIN, 1982) demonstrated the presence, in microsomal fraction chicken and fish skeletal muscles an enzymic system dependent on NAD(P)H, ADP-Fe^{+3} and O_2 for lipid peroxidation. The prese such a system was also demonstrated in beef, pork (RHEE et al., 1984, 1985; RHEE and ZIPRIN, 1987) and turkey (KANNER 1986).

In addition to microsomal lipid peroxidation systems, mitochondrial enzymic lipid peroxidation was demonstrated in fish by LU HULTIN (1986), and found to be dependent on the same co-factors such as the microsomes. It is important to note that the microsomal and mitochondrial lipid peroxidation is not a direct reaction of an enzyme toward unsaturated fatty acid, but only a gener $O_2^{\bullet-}$, and lipid oxidation is merely a non-enzymic process.

Lipid peroxidation in raw muscle of fish and chicken may be stimulated also by the enzymes lipoxygenase (GERMAN and KINS 1985; SKLAN et al., 1988; HSIEH and KINSELLA, 1989) or cyclooxygenase, but only if the enzymes are activated by pre peroxides and the fatty acids are in free form (KANNER et al., 1987). However, in cooked muscle foods, during refrigerated s which produce the warmed-over flavor, lipid peroxidation is totally dependent on non-enzymic reactions.

Lipid Peroxidation in Meat Affected by Biological and Technological Factors

Many factors seem to affect lipid peroxidation in animal tissues after slaughtering, thus include a) species, b) anatomical lo c) diet, d) environmental temperature, e) sex and age, f) phospholipid composition and content (GRAY and PEARSON During processing several other factors influence the rate of lipid peroxidation such as g) composition and freshness of ra components, h) cooking or heating, i) chopping, flaking, emulsification, deboning, and j) adding exogenous compounds such nitrite, spices and antioxidants. The discussion and evaluation of all these factors are very important, however this would go bey

goal of this review. We will address a very brief discussion on several topics such as: lipid composition, heating and adding exogenous compounds. Reviews on diet and technological factors were published (PEARSON et al., 1977) and more recently by several authors (ILLARD, 1987; ASGHAR et al., 1988; BAILEY, 1988; RHEE, 1988; HSIEH and KINSELLA, 1989).

Lipid Composition. Dietary fat supplements to feedstuffs of animals and the trend of the species to accumulate fatty acids in the membranal phospholipids, affect the lipid composition of the membrane and its susceptibility to peroxidation. Studies on individual phospholipids have demonstrated that phosphatidyl ethanolamine is the major phospholipid involved in lipid peroxidation in cooked meat (KELLER and KINSELLA, 1973; PEARSON et al., 1977; IGENE and PEARSON, 1979; PEARSON and GRAY, 1983; ASGHAR et al., 1988). Phospholipids are the major contributors to oxidative off flavor in several animal muscles and its susceptibility and severity of oxidation depend on the degree and the amounts of the unsaturated fatty acids, being high in fish > turkey, chicken, pork, beef lamb (CRAWFORD et al., 1975; WILSON et al., 1976; MELTON, 1983).

Temperature of Processing and Storage. The term of "warmed-over flavor" was first introduced by TIMS and WATTS (1958) to describe the rapid onset of rancidity in cooked meat during refrigerated storage. Oxidized flavors are developed after 2 days, in contrast to more slowly developing of rancidity in freezer storage (PEARSON and GRAY, 1983). Oxidized flavors could be developed in raw meat after grinding (GREENE, 1969; SATO and HEGARTY, 1971), however, lean raw meat is quite stable for long periods of several months, depending on species from which it originated.

Heating could affect many factors involved in lipid peroxidation. Heat disrupts muscle cell structure, inactivates enzymes and releases oxygen from oxymyoglobin. The release of oxygen from oxymyoglobin produce H_2O_2 and this reaction increased at 60°C (HAREL and KANNER, 1985). The level of free iron greatly increased during cooking (IGENE et al., 1979; RHEE et al., 1987), more in low temperature and during slow heating than in high temperature. We assume that the reason for these results are connected with the higher production in H_2O_2 which activate but also destroy the porphyrin structure releasing free iron (RHEE et al., 1987; HAREL et al., 1988). At high temperature it seems that more O_2 escape from the meat tissue without oxidizing the pigment producing rapidly an environment very low in oxygen. High temperature decreases the activation energy for oxidation and especially breaks down preformed hydroperoxides to free radicals which propagate lipid peroxidation and the development of off-flavors.

During freezing we slow down the oxidation but not stop the process. Lipid free radicals are soluble in the oil fraction and are more stable at low temperature, which allow them to diffuse to longer distances and to spread the reaction. Water works to inhibit this reaction. It is well known that meat dehydrated in the frozen cabinet as a result of poor packaging, oxidized faster than samples with full water content.

The Effect of NaCl on Lipid Peroxidation. Sodium chloride, which is an important additive in the meat industry, has been reported to act as a prooxidant (LEA, 1937, CHANG and WATTS, 1950; TAPPEL, 1952; BANKS, 1961; POWERS and MAST, 1980; KANNER and KINSELLA, 1983; ELLIS et al., 1986; KANNER et al., 1991; OSINCHAK et al., 1992) or as an antioxidant (CHANG and WATTS, 1950; MABROUK and DUGAN, 1960).

It is well recognized that sodium chloride may accelerate muscle lipid peroxidation, but its action is poorly understood. In the beginning it was assumed that metal impurities in salt enhance lipid peroxidation (CHANG and WATTS, 1950) or it may enhance the activity of oxidases (LEA, 1937). More recently it was found by us that NaCl enhance the activities of iron ions. The prooxidant effect of NaCl was inhibited by EDTA and ceruloplasmin. This effect of NaCl seems in part to be attributed to the capability of NaCl to displace iron ions from binding macromolecules for oxidative reactions. OSINCHAK et al. (1992) demonstrated the stimulation of lipid peroxidation in liposomes

by NaCl, and attributed its effect to the Cl^- anion, which may solubilize iron ions. We believe that the effect of salt catalysis o peroxidation needs further clarification.

The Effect of Nitrite and the Curing Process on Lipid Peroxidation. Nitrite plays an important role both in color development a preservative exerting an anticlostridial effect in cured meat. It was also recognized that the addition of nitrite during the curing p decrease lipid peroxidation (CROSS and ZIEGLER, 1965; SATO and HEGARTY, 1971; GREEN and PRICE, 1975; HADDEN 1975; LOVE and PEARSON, 1976). As almost all of the added nitrite in cured meat was found as nitrosothiols and nitric oxide myo (EMI-MIWA et al., 1976) we have conducted several studies on the effect of both compounds on lipid peroxidation, in model syste minced turkey meat. The results demonstrated that nitric oxide myoglobin, nitric oxide ferrous complexes and S-nitrosocysteine antioxidants (KANNER, 1979, KANNER et al., 1980; KANNER and JUVEN, 1980; KANNER et al., 1984). More recently it was that nitric oxide could inhibit Fenton reaction, the generation of ferryl (KANNER et al., 1991) and to inhibit lipoxygena cyclooxygenase activities (KANNER, 1992). The antioxidative effects of nitric oxide seem to be derived from its capability to be li to ferrous ion and to work as an electron donor and a scavenger of free radicals. The addition of the cured meat pigment, di ferrohemochrome to meat has an antioxidant effect, and was proposed to be used to replace nitrite (SHAHIDI et al., 1987). The nitrite in lipid peroxidation and in the flavor of cured meat was studied by many researchers. MACDONALD et al. (1980) assu nitrite and not nitric oxide may function as a metal chelator. IGENE et al. (1985) showed that nitrite decreases the release of non-hem during heating, and hypothesize that this effect decreases the catalysis of lipid oxidation. More recently we found that nitric oxide p the release of iron from nitric oxide myoglobin by H_2O_2 by a mechanism connected to its antioxidative effect (KANNER et al., 19 addition to these effects, during the curing process nitrite and by-products from nitrite seems to interact also with lipids, changing susceptibility to oxidize (WALTERS et al., 1979; ZUBILLAGA et al., 1984). Several reviews were published on the role of nitrite curing process, which include also its antioxidative effects (CASSEUS et al., 1979; GRAY et al., 1981; GRAY and PEARSON O'BYLE et al., 1990). Summing up, it seems that nitrite, during the curing process by forming nitric oxide, induce an antioxidative by: a) Interacting with iron non-heme and iron heme proteins prevent metal catalysis, b) Nitric oxide, nitric oxide complex S-nitrosothiols works as radical scavengers, c) Nitric oxide complexed to heme proteins prevent iron release from the porphyrin d attack of H_2O_2 or hydroperoxides, d) Stabilization of the unsaturated lipids within the membrane (but mostly formed by other ni oxides generated during the curing process.

Meat Quality Affected by the Process of Lipid Peroxidation

Flavors and Off-Flavors. The storage of precooked meat for a short period results in the development of a characteristic off-flavor by catalytic peroxidation of unsaturated fatty acids. The involvement of the lipid fraction on the chemistry of meat flavors produced heating and storage is very complexed. The data presented in this field support the belief that multiple cascade of free-radicals, hy and condensation reactions are occurring, each contributing to the development of the overall flavor both on-flavor and off (SPANIER et al., 1988). It seems that thermally induced oxidation of lipid, during the first period of cooking, produce a range of products which contribute to the desired flavor of meat. This area was most recently summarized concluding that reactions of lipid carbonyl products interact with amino groups, the amino group of phosphatidyl ethanolamine interact with sugar derived carbon free radicals from peroxidized lipids interact with Maillard reaction compounds, producing a range of desirable profile of v

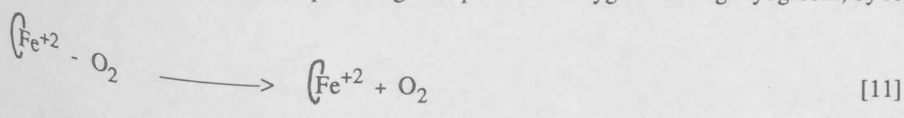
ARMER and MOTTRAM, 1990). These special flavors are not developed in cured meat because oxidative processes are prevented AILEY, 1988).

e general pattern of warmed-over flavor involves the disappearance of the fresh flavors, appearance of cardboard like flavor and further vors associated with rancid fats and oils. Lipid peroxidation generates a great range of hydroperoxides. Decomposition of these droperoxides creates a wide range of carbonyl compounds, hydrocarbons, furans and other materials that contribute to flavor terioration of foods and muscle products especially. The main cleavage mechanisms recognized for 13-linoleate hydroperoxide 8:2 LOOH) are hexanal and pentane produced and from the 9-hydroperoxide methyl 9-oxononanoate, 2.4 decadienal and methyl tanoate. Linolenate hydroperoxides (18:3 LOOH) mainly decomposed to propanal, metyl 9-oxononanoate, methyl octanoate and 4,7 decatrienal (FRANKEL, 1985). The propagation step in the process of lipid peroxidation induce a cascade of chain reactions which oduce a large range of mono and dimer hydroperoxides which could breakdown catalytically (homolytically or heterolytically) or by acid d thermal decomposition forming the secondary compounds and flavor deterioration (FRANKEL, 1991).

rect GC and GC/MS analysis of volatiles developed to follow the onset of off flavor in cooked meats identify changes within hours after orage (DUPUY et al., 1987). During storage at 4°C of roast beef a significant accumulation of pentanal, hexanal, 2,3-octanedione, nanal and 2,4-decadienal were demonstrated by the same authors. A good correlation was obtained between sensory score and total olatiles, TBA hexanal and 2-3 octanedione (ST. ANGELO et al., 1987). The examination of the effluent from the column of a gas romatograph by sniffing is commonly used in aroma analysis. By using a similar method it was shown by GROSH group (1992) that 5 octadien-3-one is the most important compound which affect the off flavor during lipid oxidation of boiled trout fish. This compound as a threshold of 0.001 ppb (HSIEH and KINSELLA 1989). CROSS And ZIEGLER (1965) found that certain aldehydes and especially exanal were present to a much greater extent in uncured than in cured meat, as a result of major lipid peroxidation. The use of hexanal to etermine the course of lipid peroxidation was used by several researchers (BAILEY et al., 1987; ST. ANGELO, 1987) and was proposed y SHAHIDI et al. (1987) as an indicator of oxidative stability and flavour acceptability in cooked ground pork. A rapid headspace GC of exanal as a measure of lipid peroxidation in biological samples and foods was developed more recently by FRANKEL and TAPPEL 1989).

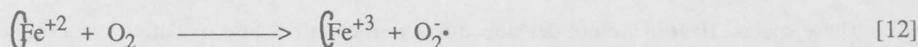
olor Deterioration by Oxidative Reactions. The color of fresh meat is perhaps the most important characteristic by which consumers judge reshness and quality of raw meat. The appeal of fresh meat decreases as the cherry red oxymyoglobin is oxidized to the red-brown metmyoglobin (GIDDINGS, 1974; GIDDINGS, 1977; LEDWARD, 1985; LANARI and CASSENS, 1991). Oxymyoglobin, the main pigment in muscle foods, absorb light in the soret range (380-440 nm) and between 480-650 nm. The loss of oxygen from oxymyoglobin and an electron from ferrous ion, producing the metmyoglobin, are changes which change the absorption properties of the molecule and the complimentary color which turn from bright-red to dark-red and further to brown.

Oxymyoglobin in the presence of anions, low pH or high temp. loss the oxygen forming myoglobin, by reaction [11],



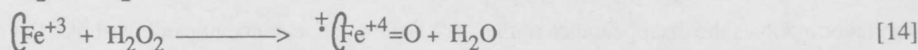
$$Fe^{+2} - O_2 \longrightarrow Fe^{+2} + O_2 \qquad [11]$$

SATO and SHIKAMO, 1981; WALLACE et al., 1982; KANNER et al., 1987). Myoglobin could autoxidize producing O_2^-, H_2O_2, metmyoglobin and ferryl compounds by the following reactions:



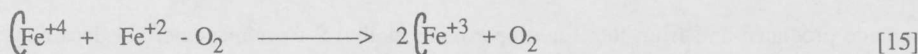
$$(Fe^{+2} + O_2 \longrightarrow (Fe^{+3} + O_2^{-}\bullet \quad [12]$$

$$O_2^{-}\bullet + O_2^{-}\bullet \longrightarrow H_2O_2 + O_2 \quad [13]$$



$$(Fe^{+3} + H_2O_2 \longrightarrow {}^{+}\bullet(Fe^{+4}{=}O + H_2O \quad [14]$$

GIULIVI and DAVIS (1990) demonstrated that ferryl ion could interact with oxy-heme producing met-heme by the following reactio



$$(Fe^{+4} + Fe^{+2} - O_2 \longrightarrow 2(Fe^{+3} + O_2 \quad [15]$$

Muscle tissues contain also an enzymatic system which could reduce metmyoglobin to oxymyoglobin. These reactions were intensively by several groups (RENERRE and LABAS, 1987; FAUSTMAN et al., 1989; FAUSTMAN and CASSENS, 1991; LA and CASSENS, 1991). More recently it was found that feeding cattle with vitamin E improve pigment and lipid stability of th (FAUSTMAN et al., 1989). These results emphasize the importance of the lipid peroxidation process not only concerning flavor o but also in the color stability of raw meat.

Nitric oxide myoglobin which is the main pigment of cured meat, is sensitive to direct oxidation by oxygen, the reaction is enha light, and form NO_3^- and metmylglobin. Replacement of the atmosphere with carbon dioxide, protection from light and low temp protect the pigment from oxidation (ANDERSEN et al., 1988; ANDERSEN et al., 1990; ANDERSEN and SKIBSTED, 1992).

<u>Texture of Meat Affected by Lipid Peroxidation</u>. Reaction between peroxidizing lipids and amino acids and proteins are another in aspect of lipid peroxidation in meat, however this area should be explored more in the future. The interaction between oxidized lip proteins can be placed into three categories: 1) formation of non-covalent complexes, 2) radical type reaction producing c complexes, and 3) reactions with secondary oxidation products. NARAYAN et al. (1964) reacted egg albumin with lipids and fo the resulting physical complexes occurred due to hydrogen bonding. Covalent bonding and protein polymerization was achiev radical mechanism using oxidized linoleic acid and lysozyme (KANNER and KAREL, 1976). SCHAICH and KAREL (1976) i that radical transfer occurs through complexes between the lipid and sulfhydryl or nitrogen centers of reactive proteins. Radical between peroxidized lipids and proteins led to protein protein crosslinks, protein to lipid crosslinks and protein scission (KAREL Secondary compounds such as aldehydes by reacting with amino groups form Schiff base compounds which emitted fluorescent lig the basis of this reaction a method to identify lipid peroxidation was developed (CHIO and TAPPEL, 1969). The results of protein linking leading to polymerization decrease solubility, partially denaturation and inhibition of enzymes (KANNER and KAREL GARDNER, 1979; POKORNY, 1981).

Lipid-protein interaction during lipid peroxidation in muscle foods was studied by several researchers (CASTELL, 1971; JAREN and LILJEMERK, 1975; ANDOU et al., 1980; NAKHOST and KAREL, 1983; KAMAREI and KAREL, 1984) and was revie LILLARD (1987).

<u>Nutritional Value of Meat Affected by Lipid Peroxidation</u>. In addition to the potential implication of lipid peroxidation to change flavors, color and texture, the autoxidation of unsaturated lipids and cholesterol results in a significant generation of toxicants (AD PARK, 1989). As it is well known the process of lipid peroxidation which generate free radicals by coordination oxidize several such as vitamin A and carotenoids, vitamin C and vitamin E, but by the same process they could co-oxidize many other molec especially cholesterol, which is in the lipid fraction and membranes. This cooxidation could lead to cholesterol oxidation and form

ass of compounds which induce atherogenicity (ADDIS and PARK, 1989). Pure cholesterol is not atherogenic, even in a sensitive animal ch as the rabbit (TAYLOR et al., 1979). The atherogenecity of cholesterol is thought to be to contaminating cholesterol oxidation oducts. Recent studies on the atherogenicity of cholesterol show that cholestanetriol and 25-hydroxycholesterol, which are oxysterol, ems to be the most atherogenic formed during oxidation (SMITH and JOHNSON, 1989). Several reports of oxysterols in human blood mples have appeared in the literature (GRAY et al., 1981; BROOKS et al., 1983; ADDIS et al., 1989). Recent research has clearly monstrated the chylomicron-associated absorption of cholesterol oxidation products in human. However, the cholesterol oxides are also pidly cleared from the plasma by a mechanism which is not fully understood (EMANUEL, 1989).

wdered eggs, heated fats and oils and precooked meat products are among the problematic foods which produce oxidized cholesterol oducts in significant amounts. Precooked intact beef muscle contain little or no cholesterol oxides, but approximately 2% of the olesterol in comminuted precooked beef has been noted to be oxidized (PARK and ADDIS, 1987). In comminuted precooked turkey, hich oxidize more rapidly than beef, the amount of cholesterol that had undergone oxidation was approximately 3%. Storage at 4°C of the eat increase oxycholesterol products (DE VORE, 1988).

here is growing evidence that lipid oxidation by products could affect our health. Some of these aspects are very briefly outlined in this ction. The reader is referred for further information to ADDIS and PARK (1989) and KUBOWS (1992) reviews.

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