Food Sci Biotechnol (2019) 28(5):1275–1285
https://doi.org/10.1007/s10068-019-00631-7

Lipid oxidation and its implications to meat quality and human health

Xi Huang[1] · **Dong Uk Ahn**[2]

Received: 28 March 2019 / Revised: 10 May 2019 / Accepted: 22 May 2019 / Published online: 7 June 2019

Abstract Lipid oxidation not only negatively influences the sensory characteristics but also the functional characteristics of meat. During the process, various primary and secondary by-products are formed depending upon the types of fatty acids, oxygen availability, and the presence of pro- and antioxidants. Some of the lipid oxidation products only influence the quality of meat but others are implicated to various diseases and human health. Therefore, prevention of lipid oxidation in meat is important for meat quality and for human health as well. The imbalance of oxidants and antioxidants that favors oxidants in the biological system is called oxidative stress in the body. Although the body is equipped with defense enzymes and antioxidant compounds, there are many sources of oxidants or free radicals that can destroy the oxidants/antioxidants balance. Therefore, supply of extra antioxidants through food can help maintaining the balance in favor of antioxidants and help preventing various diseases and malfunctions of our body.

Keywords Lipid oxidation · Meat quality · Antioxidants · Oxidative stress · Human health

✉ Dong Uk Ahn
duahn@iastate.edu

Xi Huang
huangxi@hzau.edu.cn

[1] College of Food Science and Technology, Huazhong Agricultural University, Egg Processing Technology Local Joint National Engineering Research Center, National R&D Center for Egg Processing, Wuhan 430070, Hubei, China

[2] Department of Animal Science, Iowa State University, Ames, IA 50011, USA

Introduction

Lipid oxidation is an oxidative deterioration of lipids containing any number of carbon–carbon double bonds. Lipid oxidation takes place through multiple stages that include initiation, propagation and termination stages, and produces various primary and secondary by-products that have implications to meat quality as well as human health (Frankel, 2005; Johnson and Decker, 2015; Sottero et al., 2019). Therefore, prevention of lipid oxidation in meat is important for meat quality and human health (Falowo et al., 2014; Jiang and Xiong, 2016; Sottero et al., 2019). The most important quality attributes that are influenced by lipid oxidation include sensory (flavor, color, texture), nutritive values and functional properties (water-holding capacity and emulsifying ability) of meat (Gray et al., 1996; Torrico et al., 2018). Among these, however, sensory properties—appearance/color, texture, and flavor—are the main factors that consumers use to judge meat quality (Liu et al., 1995). There are three types of lipid oxidation, which include photo-oxidation, enzyme-catalyzed oxidation and free radical oxidation (Frankel, 1984). The development of photoxidation requires light, photosensitizer and oxygen and important to food products containing photosensitizers (Lee and Min, 1990). Photoxidation can degrade unsaturated fatty acid as well as proteins and generate cabbage or burnt feather odor (Suscan, 2004). Enzyme-catalyzed oxidation is related to lipoxygenase and cyclooxygenase and is important in biological systems in the eicosanoids from the long-chain n-3 and n-6 fatty acids (EPA, DHA, and AA). Enzyme-catalyzed oxidation is also key to the production of eicosanoids (prostacyclins and leukotrienes) important for various biological activities such as vasoconstriction, vasodilation, pain, platelets aggregation, gastric production, hyperalgesia, and inflammatory reactions (Funk,

2001; Papuc et al., 2017). Free radical oxidation is also called autoxidation and is the most important form of lipid oxidation in meat and results in the production of off-odor and the formation of toxic compounds (e.g., carcinogens), causes loss of functional properties and nutritional value, and changes the color of meat (Soladoye et al., 2015). The current review discusses autoxidation and its implications for meat quality and human health.

Process of autoxidation

Many factors, including fatty acid composition, presence of free radical initiators/propagators, oxygen availability, the amounts of pro- and antioxidants, use of additives (such as spices, herbs, and salt), processing conditions (irradiation, cooking, grinding, cutting, mixing and restructuring), and packaging (oxygen permeable-, vacuum-, modified atmosphere- and active-packaging, etc.) and storage (temperature and time) conditions, are important for the rate and development of lipid oxidation in meat (Ahmed et al., 2017; Ahn et al., 2016; Mariutti and Bragagnolo, 2017; Min and Ahn, 2005). The autoxidation process and factors involved in the process are summarized in Fig. 1. The importance of fatty acid composition in the development of lipid oxidation is due to differences in bond energy of hydrogen atoms to carbon atoms in the fatty acid structure. Lipid peroxidation is initiated by a free radical strong enough to abstract a hydrogen atom from fatty acyl group ($–CH_2–$) of a fatty acid.

The bond-energy between hydrogen and carbon atoms depending upon the carbon–carbon double-bond status and the number and the position of double bonds in a fatty acid are shown in Fig. 2. The hydrogen atoms of methylene groups located in between double bonds have the weakest bond energy (Buettner, 1993; Koppenol, 1990). Thus, these hydrogens are the easiest target for free radical attack. Therefore, the greater number of double bonds in a fatty acid, the greater number of methylene groups between double bonds. Thus, the multiple double bonds in a fatty acid makes it easier for initiation as well as propagation of lipid oxidation. The hydrogen atoms attached to the carbons adjacent to a double bond are the next susceptible ones to the free radical attacks. The difference in the bond strengths in the fatty acyl groups with or without double bonds is due to the formation of dipole moments formed between the carbon–carbon atoms due to the shift of electrons involved in the covalent bonding (orbiting the outermost orbital) towards the carbons with the double bonds. The relative susceptibility of a fatty acid to lipid oxidation increases exponentially as the number of double-bond increases. Figure 2 explains why the susceptibility of

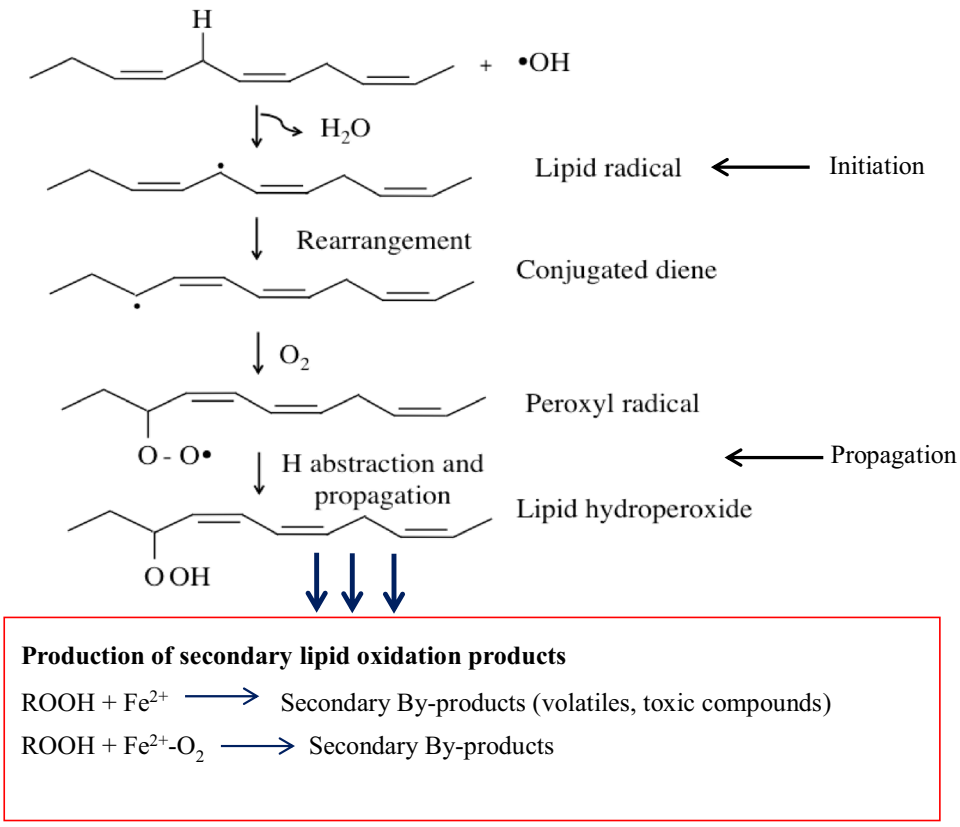
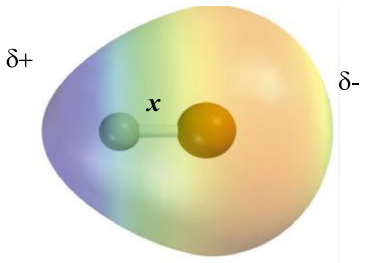


Fig. 1 Autoxidation process—initiation and propagation

A Dipole moment formation (μ)

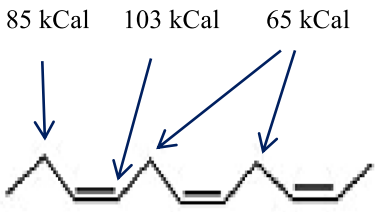


$\mu = qx$

μ, dipole moment
q, charge (or fractional charges)
x, distance between charges

*The bonding electrons are attracted to more strongly by bigger atom than by smaller atom, generating a polar covalent bond.

B C-H bond strength (kCal)



*The presence of double bonds pulls the sharing electron (C-H) toward the carbon atoms with the double bond and lowers the electron density in the carbons without double bonds due to the dipole moment formation. This weakens the C-H bond energy in the carbon atoms without double bonds and makes that H atom susceptible to abstraction by free radicals.

C Bond characteristics of methane, ethane, ethylene, and acetylene

Molecule	Bond	Bond strength (kJ/mol)	Bond strength (kcal/mol)	Bond length (pm)
Methane, CH_4	(sp^3) C—H	439	105	109
Ethane, CH_3CH_3	(sp^3) C—C (sp^3)	377	90	154
	(sp^3) C—H	421	101	109
Ethylene, $H_2C{=}CH_2$	(sp^2) C=C (sp^2)	728	174	134
	(sp^2) C—H	464	111	109
Acetylene, HC≡CH	(sp) C≡C (sp)	965	231	120
	(sp) C—H	558	133	106

Fig. 2 Dipole moment formation, the bond strength (C–H), and the bond characteristics*

lipids to oxidation depends upon the degree of unsaturation of fatty acids.

Once a hydrogen atom from a fatty acid is abstracted by a free radical, the free radical becomes a non-radical and the fatty acid becomes a free radical, called a lipid radical ($L^{\cdot}$). The lipid radical is unstable and thus tries to stabilize itself through a rearrangement of the double bond position, called diene conjugation (making a conjugated double bond system). In the presence of oxygen, the lipid radical accepts oxygen and becomes lipid peroxyl radical ($LOO^{\cdot}$), which is strong enough to abstract a hydrogen atom from an adjacent lipid with double bonds, and itself becomes a hydroperoxide (LOOH). The newly formed lipid radical ($L^{\cdot}$) binds with oxygen and propagates free radical formation of a new fatty acid and this process continues (Halliwell and Gutteridge, 1989). This stage is called the propagation. Both initiation and propagation involve abstraction of hydrogen by reactive oxygen species and are difficult to differentiate. The only difference is that initiation is started with free radicals such as hydroxyl radical ($^{\cdot}OH$) without lipid components while propagation is done by lipid-containing radicals such as lipid, alkoxyl and peroxyl radicals (Aikens and Dix, 1991; 1992). However, propagation does not occur in the absence of oxygen, which makes the presence of oxygen very important for the process of lipid oxidation (Ahn et al., 1992; 1993a; Smiddy et al., 2002). Ahn et al. (1993d) clearly showed that blocking oxygen from meat prevents the progress of lipid

oxidation even in the presence of prooxidants. Ground state oxygen (O_2) cannot oxidize polyunsaturated fatty acids (PUFA) because it does not have strong enough reactivity (Halliwell, 1991). However, it can be converted to various reactive oxygen species (ROS) such as hydroxyl radical ($^{\cdot}OH$), superoxide anion ($O_2^{\cdot-}$), hydrogen peroxide (H_2O_2), hydroperoxyl radical ($HO_2^{\cdot}$), lipid peroxyl radical ($LOO^{\cdot}$), alkoxyl radical ($LO^{\cdot}$), iron–oxygen complexes (ferryl- and perferryl radicals), nitric oxide radical ($NO^{\cdot}$), hypochlorous acid (HOCl) and singlet oxygen ($^1O_2$) (Frijhoff et al., 2015; Pisoschi and Pop, 2015; Radi, 2018). Some of the ROS such as $^{\cdot}OH$, $LOO^{\cdot}$, $LO^{\cdot}$, ferryl- and perferryl radicals, and $^1O_2$ can be directly involved in the initiation or propagation stages of lipid oxidation, while $O_2^{\cdot-}$, H_2O_2, and hydroperoxyl radical $HO_2^{\cdot}$ can be converted to more reactive oxygen species with the help of enzymes and transition metals and can then initiate or propagate lipid oxidation (Droge, 2002; Pisoschi and Pop, 2015).

Among the transition metals, iron is the major catalyst and plays a critical role in lipid oxidation because of its involvement in the production of reactive oxygen species (Halliwell and Gutteridge, 1990). The production of hydroxyl radicals from hydrogen peroxide by the Fenton reaction and the metal catalyzed Haber–Weiss reaction, the main pathway of .OH radical production are shown in Fig. 3. Ionizing radiation also can produce .OH radicals by breaking water molecules. Heme-pigments are also considered as strong prooxidants. The catalysis of lipid oxidation by heme pigments are related to the formation of the highly reactive heme-pigment derivatives such as non-protein bound heme iron (hemin, hematin and heme) and hypervalent heme pigments [ferryl- or perferryl-heme pigments: heme-Fe(IV)=O] (Carlsen et al., 2005; Jongberg et al., 2016; Lee et al., 2015; Yin et al., 2017). However, their prooxidant activity in meat can vary depending upon the conditions of meat or food systems. Myoglobin and hemoglobin had strong prooxidant effects in oil emulsion, washed muscle and cooked-meat homogenates, but had little prooxidant effect in raw-meat or raw-meat homogenates (Ahn and Kim, 1998). The reason for the different catalytic activities of heme pigments between raw- and cooked- (or washed) meat systems is not well known, but could be related to the reducing power (e.g., ascorbate and reducing enzymes) of the system.

Effect of lipid oxidation on meat quality

The major effects that lipid oxidation have on meat quality include the production of odor, color change, changes in taste and texture, negatively influencing functional properties such as protein solubility and water holding capacity and lowering the bioavailability of some nutrients (Falowo et al., 2014). Some of the quality parameters are important for raw meat, while others are for cooked meat. Lipid oxidation of raw meat generates warmed-over flavor and rancidity in cooked meat (Cross et al., 1987) (Tables 1, 2). The off-odors in oxidized meat are mainly caused by aldehydes, especially hexanal. However, other aldehydes such as pentanal, heptanal, 2,6-nonadienal, 3,6-nonadienal, 3-methyl butanal and 4,5-epoxy-2-decenal depending upon the composition of fatty acids and the reactions involved and ketones such as 1-octen-3-one and 2,3-octanedione are also important for the off-odor generation in raw and cooked meat (Mottram, 1998; Suscan, 2004). Lipid oxidation discolors meat due to the oxidation of heme pigments and lowers protein solubility and water holding capacity because lipid oxidation leads to protein oxidation (Lund et al., 2011; Wu et al., 2017). The oxidation of proteins significantly lowers protein solubility and water holding capacity, which are important for meat processing. Lower water holding capacity of proteins makes the meat dry due to higher moisture loss during cooking and lower protein extractability leads to lower emulsion capacity and binding ability of the meat when used in further processed

1. Fe-dependent decomposition of HO (Fenton reaction)

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{+3} + \cdot OH + OH^-$$

$$Fe^{2+} + H_2O_2 \rightarrow \text{ferryl?} \rightarrow Fe^{+3} + \cdot OH + OH^-$$

2. Metal-catalyzed Haber-Weiss reaction or superoxide-driven Fenton reaction

$$Fe^{+3} + O_2^- \rightarrow Fe^{2+} + O_2$$

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + \cdot OH + OH^-$$

$$\text{Net} \quad O_2^- + H_2O_2 \rightarrow O_2 + \cdot OH + OH^-$$

3. High-energy ionizing radiation

$$H_2O \rightarrow \cdot OH + \cdot H + H_2 + H_2O_2 + H_3O^+ + e^{-aq} \text{ (ionizing radiation)}$$

Fig. 3 Production of hydroxyl radicals ($^{\cdot}OH$)

Table 1 Number of double bonds in a fatty acid and the relative oxidation rates

Type of fatty acid	Rate of reaction relative to stearic acid
18:0	1
18:1Δ9	100
18:2Δ9,12	1200
18:3Δ9,12,15	2500

meat (Lund et al., 2011). The color changes in raw meat makes the meat less attractive for consumers and can lead to downgrading of meat (Mancini and Hunt, 2005). The production of off-odor (rancid flavor) in cooked products can lead to taste changes of meat. Cooked meat is more susceptible to oxidative changes because of the inactivation of antioxidant enzymes and exposure of lipids to prooxidants and outer environment (oxygen) by heat denaturation of the muscle fiber structure (Ahn et al., 1993d). However, the conditions of raw meat are very important for oxidative changes of meat after cooking because the primary oxidation products or oxidized lipids from the raw meat can continue the oxidation process after cooking (Du et al., 2001). Therefore, preventing lipid oxidation of raw meat is as important as in cooked meat.

Prevention of lipid oxidation

The prevention or retardation of oxidative process in meat can be accomplished by several approaches: prevention or minimizing oxygen contact with meat, chelation of transitional metals, inactivation of free radicals, or changing the composition of fatty acids in meat (Wood et al., 2008). Access of oxygen to meat during storage can be influenced by the packaging methods and materials used. Vacuum packaging, modified atmosphere packaging and oxygen permeable packaging are common methods used with raw and cooked meat, but vacuum packaging is the preferred method for cooked meat because cooked meat is more susceptible to oxidative changes during storage. However, even vacuum packaging cannot completely block oxygen from meat because packaging materials are also permeable to oxygen and some residual oxygen remains in the packaged-meat. Also, the exposure of meat to oxygen after cooking inevitably allows the initiation and progress of oxidative changes. Ahn et al. (1992) showed vacuum-packaging meat immediately after cooking while the meat is still hot minimized oxidative changes during storage by cutting the time for oxygen contact with meat after cooking. Insertion of oxygen scavengers inside the bag can remove the residual oxygen and extend the shelf-life of the meat. Oxygen contact with meat during processing is also important for oxidative changes during storage. Recently, various active packaging technologies impregnated with antioxidants, antimicrobial or oxygen scavengers in the packaging materials or packaging bags have been used to protect meat from oxidative changes or improve the safety of meat during storage (Fang et al., 2017). Processing including cutting, grinding and chopping increase the surface area of meat. Cooking inactivates many antioxidant enzymes. Also, all these processes leave meat lipids exposed to oxygen and catalysts and accelerate oxidative changes. Addition of antioxidants can stop or slow down oxidative processes in meat. Many synthetic antioxidants have been used but demands for natural antioxidants are increasing rapidly (Karre et al., 2013). Although most natural antioxidants are phenolic compounds from plant sources, search for antioxidant proteins and peptides from animal sources are also under way (Shahidi and Ambigaipalan, 2015; Sohaib et al., 2017). Common phenolic antioxidants from plant sources include tocopherols, gallic acid, flavonoids, sesamol, ferulic acid and epicatechin although other plant compounds such as essential oils, tannic acid and carotenoids also are known to have strong antioxidant effects (Al-Hijazeen et al., 2016; 2018; Hać-Szymańczuk et al., 2019; Lee et al., 2017; Maqsood et al., 2014; Tang et al., 2002). Spices and herbs used in processed meat contain various antioxidant compounds and also impart flavor to meat products (Embuscado, 2015; Xie et al., 2017). The main mechanism of free radical scavenging antioxidants, mainly phenolic antioxidants, is

Table 2 Odor compounds produced by lipid and protein oxidation and their odor characteristics

Chemical processes	Volatile compounds	Odor characteristics
Lipid oxidation	1-Octen-3-one, hexanal, (z)-3-hexenal, 2,6-nonadienal, 3,6-nonadienal, 3-methyl butanal	Metallic, taint, grassy, WOF, beany, rancid, cow house-like odor
Lipid oxidation of pre-cooked meat	Pentanal, hexanal, 2,3-octanedione 4,5-epoxy-2-decenal	Warmed-over-flavor: grassy, old, stale, rancid, metallic, painty
Proteins oxidation by photooxidation	Thiols, sulfides, 3-methylpropanal	Cabbage, burnt protein, burnt feather

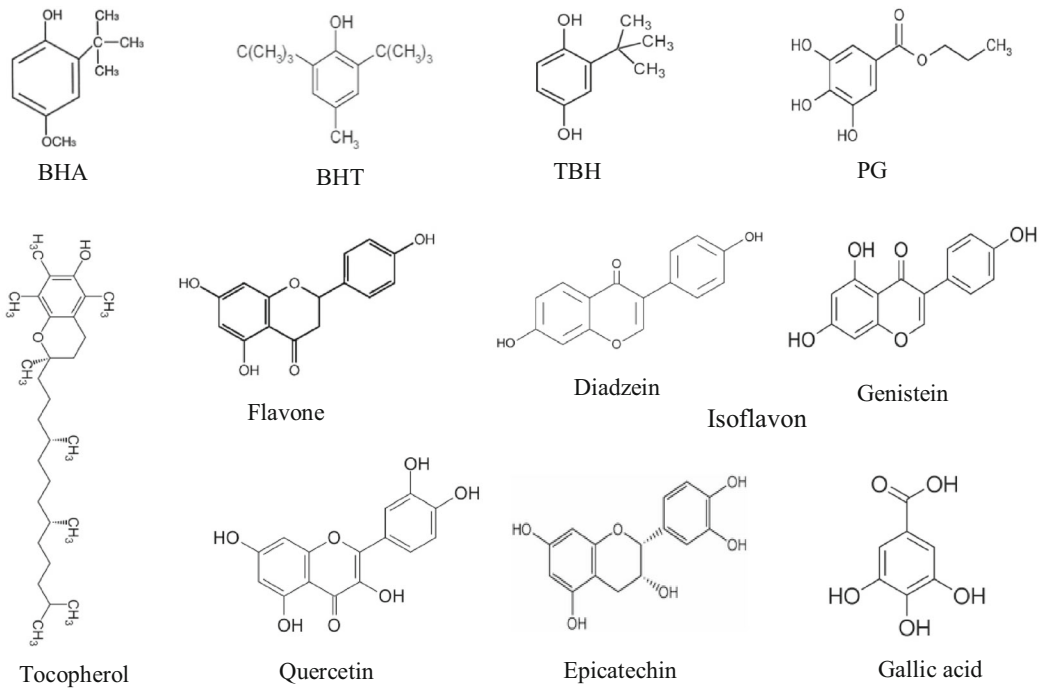
Suscan (2004). Food Technol. 58:36–40

through the redistribution of reaction potential within the antioxidant structure (Figs. 4, 5). All the free radical scavenging phenolic antioxidants, regardless synthetic or natural, contain a phenol ring with resonating double bonds, which redistributes the reaction potential throughout the ring structure (Shahidi et al., 1992; Shahidi and Ambigaipalan, 2015). The reaction potential of lipid radicals or reactive oxygen species is very high (usually high than 1000 mV) but once the free radical is transferred to a phenolic antioxidant (antioxidant radical), its reaction potential level drops down to levels that the antioxidant radical can no longer be involved in the initiation or propagation of lipid oxidation, and the autoxidation process stops there (Lu et al., 2010).

Free and loosely bound iron are the main forms of iron involved in lipid oxidation. Only the ferrous form of iron can catalyze lipid oxidation, but ferric iron is also important because it can be easily reduced in the presence of reducing agents, reducing environments or reducing enzymes (Ahn et al., 1993b). Usually, ferric iron cannot catalyze lipid oxidation in cooked meat, cooked meat homogenate or oil emulsions unless reducing agents are present. However, both forms of iron can catalyze lipid oxidation in raw meat because of the presence of reducing enzymes and reducing agents such as ascorbic acid. Some bound or stored forms of iron such as in ovotransferrin or ferritin are also reported to catalyze lipid oxidation but their catalytic activities are much weaker than the free forms of iron (Ahn et al., 1993b). Structural irons (e.g., heme pigments) are not involved in the catalysis of lipid oxidation unless they are released from the structure. Chelated iron (e.g., EDTA-Fe, DTPA-Fe, desferrioxamine-Fe etc.) is not involved in the catalysis of lipid oxidation (Ahn et al., 1993c; Ahn and Kim, 1998). Modification of fat by enriching with saturated fatty acids or vitamin E through dietary or adding at the time of processing can increase meat oxidative stability (Bellés et al., 2018), but their effects are limited (Ahn et al., 1995). Creating a reducing environment can also retard or prevent oxidative changes but it is not as effective as using iron chelators or free radical scavenging antioxidants. The ideal requirements for antioxidants to be used in meat or other foods include easy to get, work under lipid as well as water conditions, not be absorbed into body system, high potency, cheap, and stability under processing conditions.

Implications of lipid oxidation to human health

Although a major concern for lipid oxidation has been its detrimental effects on sensory and functional qualities of



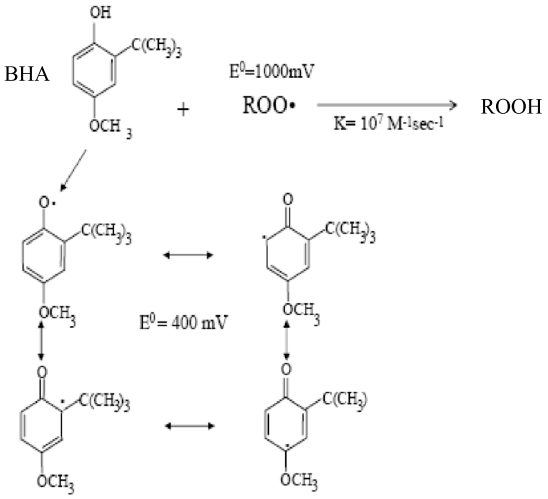
*All the synthetic and natural chain-breaking and free radical scavengers have at least one phenol ring with -OH group attached to the phenol ring, which is essential for the stabilization of the free radicals.

Fig. 4 Synthetic and natural chain-breaking and free radical scavengers*

A Free radical reaction potentials

Radical	mv
•OH (hydroxyl)	+2300
LO• (alkoxyl)	+1600
LOO• (peroxyl)	+1000
GS• (glutathione)	+920
•HU⁻ (urate)	+590
•Toc (tocopherol)	+480
•Asc⁻ (ascorbate)	+282
Fe^{3+}-EDTA	+120

B Resonance stabilization of antioxidant*



*Antioxidants such as BHA accepts the free radical reaction energy and spreads the reaction potential within the phenol ring structure by the resonating double bonds. This dramatically lowers the free radical reaction potential of the antioxidant. So, the BHA radical cannot be involved in the propagation process and the lipid oxidation process stops there.

Fig. 5 Free radical reaction potentials and resonance stabilization

meat, lipid oxidation also produces free radicals and toxic compounds that can cause diseases, or have negative impacts on the health of humans who consume the products. Many primary and secondary by-products of lipid oxidation such as 4-hydroxynonenal, malonaldehyde are known as potential carcinogens (Csala et al., 2015). Carbonyl compounds can affect cellular signal transduction, hydroperoxides are known to damage DNA, and epoxides and hydrogen peroxide by-products are known carcinogens. Free radicals including lipid radicals, reactive oxygen species, reactive nitrogen species, and reactive sulfur species generated during oxidative processes can increase oxidative stress in human bodies. Reactive oxygen species are produced during aerobic metabolism in mitochondria, reactive nitrogen species such as peroxynitrite ($ONOO^-$) can be formed via the reaction of nitric oxide with $O_2^{-\cdot}$, and reactive sulfur species (thiyl radicals) can be formed from the reactions of thiols with reactive oxygen species (Giles and Jacob, 2002; Li et al., 2014). The reactive oxygen species produced during cellular metabolism also have important roles in killing pathogens, cell signaling, apoptosis, gene expression and ion transportation. Short-term oxidative stress may also be important in the prevention of aging (Yan, 2017). However, excessive amounts of reactive oxygen species are directly or indirectly involved in various diseases such as inflammatory diseases, cancer, diabetes, autism, Alzeheimer's disease, Pakinson's disease, atherosclerosis, heart failure, fatty liver, chronic fatigue syndrome, obesity, and depression (Chen et al., 2018; Fernandez-Sanchez et al., 2011; Furukawa et al., 2017; Polimeni et al., 2015; Siti et al., 2015; Wang et al., 2014).

Oxidative stress

Oxidative stress is defined as "an imbalance between oxidants and antioxidants, which favors prooxidants and/or disfavors antioxidants, potentially leading to damage" (Sies, 1997). There are many sources of oxidants. Some of them are generated from the endogenous sources and others are from the exogenous sources. The oxidants or reactive oxygen species are produced from mitochondria as a part of normal metabolic process and as microbicidal products by macrophages. Under normal conditions, about 3–4% of the oxygen used in aerobic metabolism of mitochondria are converted to superoxide but the amount can be elevated up to about 20% under severe exercise or pathophysiological conditions (Davies, 2000). In addition to reactive oxygen species, reactive nitrogen species such as nitric oxide radical, nitrogen dioxide, peroxynitrite and peroxynitrous acid can also be produced endogenously (Biswas, 2016; Li et al., 2014). The external sources of oxidants or reactive oxygen species include ionizing radiation, smoking, polluted air, and foods such as oxidized oils and oxidized meat (Droge, 2002; Sies, 2018; Xiao et al., 2011a; 2011b). The effect of oxidative stress depends upon the degree of the stresses. Even mild oxidative stress conditions can trigger apoptosis, but cells usually can

overcome the perturbations using antioxidant enzymes as well as small antioxidant compounds in the body. Under the severe oxidative stress conditions in which the defense mechanisms of the body cannot regain prooxidants/antioxidants balance, however, the oxidants and free radicals can cause extensive cellular damages and even cause cell death (Davies, 2000; Gaschler and Stockwell, 2017). Severe oxidative stress changes the structural integrity of membrane components, including lipids as well as proteins (in lipid raft), and alter membrane fluidity, membrane permeability and membrane-bound signaling proteins (Gaschler and Stockwell, 2017). Oxidants and free radicals can also induce oxidation of proteins (mainly enzymes). Some amino acids in the proteins are more susceptible to oxidation than others and the oxidation of amino acids can induce changes in the secondary and tertiary structure of the proteins and cause loss or abnormal function (Davies, 2005). Changes in structure increases susceptibility of proteins to proteolysis. Damages of DNA by oxidants can block transcription and cause mis-replication that leads to erroneous protein structure and functions. Unless the damage to DNA is repaired, it can cause long-term effects leading to various pathological conditions including inflammation, atherosclerosis, aging and cancer (Valle, 2011; Mikhed et al., 2015; Vieira et al., 2017).

Antioxidant defenses in biological systems

The body tries to maintain oxidative balance using antioxidant enzymes as well as small, non-enzymatic antioxidant compounds in the body. The defense against oxidants in the body includes prevention of prooxidant formation, interception of prooxidants, breaking the chain of radical reactions, and repair of damages caused by prooxidants. The antioxidant defense enzymes in the body include superoxide dismutase, catalase, peroxidase, glutathione peroxidase and glutathione reductase. Superoxide dismutase converts superoxide anion ($O_2^{\cdot-}$) into hydrogen peroxide hydrogen peroxide (H_2O_2), and then catalase degrades the H_2O_2 to water and oxygen (Halliwell and Gutteridge, 1990; Robinett et al., 2018). Hydrogen peroxide can also be removed by GSH with the help of glutathione peroxidase that produces oxidized glutathione (GSSG) and water molecules. The oxidized glutathione is converted back to the reduced form (GSH) using NADPH, which is catalyzed by glutathione reductase, and then involved in the further removal of H_2O_2 (Droge, 2002; Gaucher et al., 2018). Co-factors of enzymes such as selenium are important for the activity of glutathione peroxidase and glutathione reductase enzymes (Cossu et al., 1997; Zoidis et al., 2018). Although the oxidative reactivity of $O_2^{\cdot-}$ and H_2O_2 are much lower than other reactive oxygen species such as hydroxyl, peroxyl and alkoxyl radicals the body is equipped with multiple enzyme systems for their removal because $O_2^{\cdot-}$ and H_2O_2 can be converted to hydroxyl radical in the presence of oxygen and/or iron (Frankel, 2005). The antioxidant enzymes and reducing agents in biological systems are working in coordination instead of independently in eliminating $O_2^{\cdot-}$ and H_2O_2 and other oxidants. The non-enzymatic, low-molecular weight antioxidants include tocopherols, carotenoids, ubiquinones, ascorbate, glutathione, phosphate, uric acid and citric acid (Gaucher et al., 2018). Some of the low-molecular weight antioxidants such as vitamin E, beta-carotene and coenzyme Q are fat-soluble and present as a part of cell membrane, while others like ascorbate and citric acid are water-soluble and mainly work in the cytoplasm (Gutteridge and Halliwell, 2018; Halliwell et al., 1992).

Although the body is equipped with defense enzymes and antioxidant compounds, the supply of antioxidants through food is very important. As discussed above, there are many sources of oxidants or free radicals and the balance between prooxidants and antioxidants in the body can be easily destroyed. Supplying extra antioxidants through food can help maintain the balance in favor of antioxidants, and help preventing various diseases and malfunctions of our body (Jacob and Burri, 1996; Poprac et al., 2017; Sindhi et al., 2013).

In summary, lipid oxidation is an important factor that can influence the quality and the functionality of meat. Lipid oxidation products in meat and other foods increase the oxidative stress that can be directly or indirectly related to various diseases of humans. Although oxidants can be generated from normal metabolic processes in the body, many can be derived from foods, including meat products, and various environmental sources. Therefore, preventing lipid oxidation of foods as well as other sources is important to avoid oxidative stress in the body. Prevention of lipid oxidation in meat products using antioxidants or other prevention measures not only improves the quality of the products but are also important for the health of humans who consume the products. Although human bodies are equipped with various defense mechanisms, including antioxidant enzymes and antioxidant compounds, maintaining oxidant/antioxidant balance in favor of antioxidants by consuming more antioxidants through foods, and avoiding oxidized food or prooxidants are the best methods for reducing oxidative stress in the body.

Acknowledgements This work was supported by the National Key Research and Development Program of China (2018YFD0400302), and the Iowa Agriculture and Home Economics Experiment Station, Ames, Iowa. Project No. IOW03721 is sponsored by Hatch Act and State of Iowa funds, and by the National Natural Science Foundation of China 31471602.

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