Food Science of Animal Products

Review Article

Co-oxidation behavior between lipid and protein in muscle food: a review

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Abstract: In recent decades, the oxidation of proteins and lipids in muscle meals has received increased study interest, as it is generally understood that such oxidation affects both the sensory quality and nutritional value of these goods. Lipids are essential components of muscle cell membranes, yet their presence in muscle tissue can result in lipid peroxidation and oxidative injury. Myoglobin, a heme protein found in muscle tissue, can also be oxidized under specific conditions. Myofibrillar protein is the primary protein found in muscle tissue, and its oxidation can cause irreversible damage to protein structure. About the effect of chemical reactions on the quality of muscle food, the oxidation of lipids and proteins does not occur independently. Understanding this interaction is critical in explaining the reasons for meat quality deterioration and selecting optimal antioxidants to preserve the quality of muscle food. This review thoroughly analyzes the oxidation processes and molecular mechanisms of lipids, myofibrillar proteins, and myoglobin in muscle foods. Additionally, it elucidates the intricate synergistic oxidation mechanisms among lipids-myofibrillar proteins, lipids-myoglobin, and myofibrillar proteins-myoglobin, along with their impact on product quality.

Keywords: muscle food; lipid; myofibrillar protein; myoglobin; co-oxidation

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1 Introduction

From a global perspective, meat ranks among the most essential food products. Muscle foods deliver high-quality animal protein and a broad spectrum of vital micronutrients to the vast majority of the world's population. However, the unsaturated lipids, myoglobin (Mb), metal catalysts, and oxidants found in muscle tissue readily promote oxidation, resulting in loss of meat quality[1]. The main causes of muscle food quality loss are lipid and protein oxidation reactions, which occur during practically all stages of meat production, including slaughtering, ripening, transportation, and storage[1]. In general, lipid oxidation is associated with changes in flavor, whereas protein oxidation can induce changes in texture and water retention[2]. Mb auto-oxidation results in variations in the color of red meat[3]. Flavor, texture, and appearance are three important factors affecting consumer purchase decisions and satisfaction with meat consumption. Therefore, unveiling and controlling the molecular oxidation process in muscle foods to enhance meat quality has garnered increasing attention.

Under external conditions (e.g., light, heating, initiators, and metal ions), lipid undergoes an auto-oxidation process. This reaction is a free-radical chain reaction with three main steps: chain initiation, chain propagation, and chain termination[4]. Protein oxidation is the direct or indirect alteration of a protein caused by reactive oxygen species (ROS) stress. Protein oxidation, like lipid oxidation, can be conveyed via free radical chain reactions[5]. Not only do the same reaction mechanisms exist, but oxidative reactions can be communicated between lipids and proteins. ROS, such as free radicals produced during oxidation, are important channels by which lipids and proteins interact and so mediate oxidation. Lipid oxidation produces free radicals and hydroperoxides, which enhance protein oxidation. This jointly mediated oxidation process is known as "co-oxidation behavior". However, because of the intricacy of oxidation mechanisms in muscle food, research progress has been limited, and the mechanism of co-oxidation remains unknown, limiting the development of antioxidant pathways. As a result, understanding the co-oxidation behavior of lipids and proteins in muscle food is critical for developing efficient meat oxidation inhibitors.

This review examines the mechanisms of lipid, protein, and Mb oxidation in muscle food. Furthermore, the mechanisms of co-oxidation of lipid-myofibrillar protein, lipid-Mb, and myofibrillar protein-Mb were thoroughly investigated, as well as their impact on meat quality alterations.

2 Oxidation mechanism

2.1 Lipid oxidation

Lipid oxidation is a complex process that can be classified into three types: enzyme-catalyzed oxidation, photo-oxidation, and autoxidation. Lipoxygenase is thought to be responsible for lipid enzyme-catalyzed oxidation, which is significantly dependent on the enzyme's concentration. In general, a higher enzyme concentration leads to a faster oxidation rate[6]. There are two types of factors that influence lipid photo-oxidation: direct and indirect. Direct factors include photosensitizer type, light conditions, and

lipid composition; indirect factors include packaging materials and procedures, temperature, moisture, enzyme, salinity, and so on. They interact with one another and perform critical roles in lipid photo-oxidation[7]. Among the lipid-oxidation pathways in muscle food, the most important one is autoxidation, which is a continuous free-radical chain reaction[6].

During lipid autoxidation, a free radical can engage in oxidation by reacting with the hydrocarbon chain of a polyunsaturated fatty acid in the lipid to produce peroxides, which then react with additional hydrocarbon chains to form hydroperoxides[8]. Thus, lipid autoxidation is known as a free-radical chain reaction[9], which is typically accomplished in three steps: initiation, propagation, and termination (Table 1). At the initial stage, the use of Fe^{4+} free radicals, lipoxygenase, metal catalysis, or ROS free radicals in unsaturated fatty acids causes the methylene (=CH_2) in the fatty acids' α-H position to be removed and form alkyl radical (L·). During the propagation stage, the reaction between L· and oxygen forms peroxyl radical (LOO·), and then a hydrogen atom is snatched from another LH to form the primary product of lipid oxidation, hydroperoxide (LOOH). LOOH is extremely unstable and decomposes to produce alkoxy radicals (LO·) and hydroxyl radicals (·OH). The new L· reacts again with molecular oxygen to form new LOO·, and the cycle is repeated. During the termination stage, the reaction between free and non-free radical chemicals (antioxidants) produces oxygen-containing or hydroxy fatty acids, lipid dimers, and other altered or damaged lipid molecules. These molecules disintegrate into tiny volatile compounds, such as aldehydes, ketones, and alcohols, which are considered secondary oxidation products of lipids[10–22].

Table 1 Process of lipid oxidation is as follows.

Stage	Process
Initiation	$LH + O_2 \rightarrow L\cdot + \cdot OOH$
	$L\cdot + O_2 \rightarrow LOO\cdot$
Propagation	$LH + LOO\cdot \rightarrow LOOH + L\cdot$
	$LOOH \rightarrow LO\cdot + \cdot OH$
	$L\cdot + L\cdot \rightarrow L\text{–}L$
Termination	$L\cdot + LOO\cdot \rightarrow LOOL$
	$LOO\cdot + LOO\cdot \rightarrow LOOL + O_2$

2.2 Protein oxidation

Protein oxidation is the covalent alteration of proteins that can be triggered directly by active substances (including free and non-free radicals) or indirectly by reacting with secondary products of oxidative stress[13]. Protein oxidation inflicts irreversible damage to the protein structure, including amino acid side-chain modification, protein main-chain breakage, protein crosslinking, and so on. Protein oxidation can also occur via a free-radical chain reaction. Similar to lipid oxidation, protein oxidation reaction is a chain reaction of free radicals. The involvement of free radicals and the creation of hydroperoxides begin at the early stage and progress to the reproductive period of free radical multiplication and transfer. Finally, it ends with the formation of nonreactive species[14]. For example, ROS and reactive nitrogen species (RNS) are reactive intermediates necessary for protein oxidation. In general, ROS predominantly comprises unstable molecules known as free radicals, including ·OH, superoxide anion radicals ($O_2^-\cdot$), and hydroperoxyl radicals (·OOH). Non-radical species such as hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$) are also classified as ROS[15].

In the process of protein oxidation, free radicals can directly act on proteins and directly induce protein oxidation through hydrogen capture, oxygenation, coupling, and cleavage reactions. These phenomena result in the oxidation of protein side-chain groups, the breakage of main peptide chains, and the formation of covalent crosslinks. Specifically, hydrogen atoms in proteins captured by free radicals are primarily derived from the α-carbonyl groups and side chains containing aliphatic and aromatic amino acids. The oxidized protein side chains can lead to the formation of protein carbonyls, mercaptan groups, and peroxides. Free radicals induce the oxidation of aliphatic amino acids in the main peptide chain and side chain of proteins through hydrogen abstraction, oxygenation, hydrogenation, and single electron reduction and then convert them into protein alkoxy radicals (PO·). PO· can result in the breakage of the polypeptide chain into small peptide segments or produce covalent crosslinks by α-amidation or diamide pathways. Aliphatic amino acid PO· can directly decompose into carbon-based compounds, thereby further oxidizing the proteins. ROS can induce lipid peroxidation and produce secondary metabolites (including alkyl radicals, hydroperoxide, active aldehydes, etc.), which can also cause protein oxidation to form carbonyl derivatives and covalent crosslinks[16].

ROS, such as ·OH, can oxidize a protein's hydrogen atom (PH), resulting in the creation of a carbon-centered protein radical (P·). The initial P· then undergoes successive transformations. In the presence of oxygen, it can convert into a protein peroxyl radical (POO·) or form a crosslinked derivative (P–P) through C–C with another protein radical (P·) even without O_2. Subsequently, the POO· can abstract a hydrogen atom from another protein molecule, giving rise to an alkyl peroxide (POOH)[17]. The POOH can react with ROS, such as the ·OOH, or with reduced forms of transition-metal ions (M^{n+}), leading to the formation of alkoxyl radicals (PO·). These PO· can further generate hydroxyl derivatives (POH)[14,18]. General mechanism of protein oxidation is shown as follows.

$$PH + \cdot OH \rightarrow P\cdot + H_2O \quad (1)$$

$$P\cdot + O_2 \rightarrow POO\cdot \quad (2)$$

$$P\cdot + P\cdot \rightarrow P - P \quad (3)$$

$$POO\cdot + PH \rightarrow POOH + P\cdot \quad (4)$$

$$POOH + \cdot OOH \rightarrow PO\cdot + O_2 + H_2O \quad (5)$$

$$POOH + M^{n+} \rightarrow PO\cdot + OH^- + M^{(n+1)+} \quad (6)$$

$$PO\cdot + \cdot OOH \rightarrow POH + O_2 \quad (7)$$

$$PO\cdot + H^+ + M^{n+} \rightarrow POH + M^{(n+1)+} \quad (8)$$

2.3 Mb oxidation

Mb appears principally in 3 forms: deoxymyoglobin (DeoMb), oxymyoglobin (OxyMb), and metmyoglobin (MetMb), which correlate to the purple-red, bright-red, and brown colors of meat, respectively. Mb in meat typically occurs in three different states at the same time, rather than just one. Mb oxidation can be affected

by a variety of both internal and external variables. The former consists mostly of MetMb reductase, pH, microorganisms, and lipid oxidation, whereas the latter comprises oxygen partial pressure, temperature, light, and so on.

Mb oxidation is most commonly associated with the oxidation of iron in heme, also known as Mb autoxidation. During oxidation, ferrous states of Mb including DeoMb and OxyMb can be converted into their ferric state (MetMb). The conversion of different Mb states is a proton-mediated reaction. In this process (Eqs. (9)–(13)), protons protonate liganded O_2 after entering the heme pocket, resulting in a positive charge on O_2 and leading to the removal of one electron from the iron atom of ferroprotoporphyrin IX[10]. Afterward, the neutral superoxide radical dissociates, resulting in the formation of MetMb, whereas the oxygenated form of Mb initially releases the dioxygen ligand, followed by the oxidation of the resulting deoxy species. Notably, MetMb is an intermediate product of Mb oxidation and not the final form. As the $O_2^-\cdot$ undergoes dismutation, H_2O_2 is generated. H_2O_2 then interacts with MetMb, forming a heme radical cation ($Mb^+\cdot(Fe^{IV})=O$). Subsequently, the $Mb^+\cdot(Fe^{IV})=O$ species can then be transformed into perferrylmyoglobin ($Mb\cdot(Fe^{IV})=O$) by a fast electron-transfer reaction with adjacent globin molecules. The resulting $Mb\cdot(Fe^{IV})=O$ can further react with H_2O_2 to form ferrylmyoglobin ($Mb(Fe^{IV})=O$)[10].

$$DeoMb(Fe^{II}) + O_2 \rightarrow MetMb(Fe^{III}) + O_2^-\cdot \quad (9)$$

$$OxyMb(Fe^{II}) \rightarrow DeoMb(Fe^{II}) + O_2 \quad (10)$$

$$2O_2^-\cdot + 2H^+ \rightarrow H_2O_2 + O_2 \quad (11)$$

$$Mb(Fe^{III}) + H_2O_2 \rightarrow Mb\cdot(Fe^{IV}) = O_2 + H_2O \quad (12)$$

$$2Mb\cdot(Fe^{IV}) = O_2 + H_2O_2 \rightarrow 2Mb(Fe^{IV}) = O_2 \quad (13)$$

3 Co-oxidation behavior of lipid, myofibril protein and Mb

3.1 Co-oxidation of lipid and protein

According to the free-radical chain reaction of lipid and protein oxidations, the oxidation reaction can apparently be transferred between two fractions. In reality, free radicals and other ROS produced during lipid and protein oxidation play a crucial role in the coupling oxidation reaction that occurs between the two oxidation phenomena. For protein, the peroxyl radical, ·OH, alkoxyl radical, and active aldehydes produced by lipid oxidation are all powerful ROS that cause oxidation reactions. These free radicals are extremely oxidizing and can react with functional groups in other organic compounds in redox reactions. Aldehydes differ from the strong oxidizing activity of free radicals in that they can react with many biological macromolecules, including DNA and proteins, to form a variety of adducts (Figure 1)[19–20].

The peroxyl radical, a medium-reactivity ROS, can specifically damage protein cysteine, methionine, tryptophan, and tyrosine residues. In comparison to the peroxyl radical, the alkoxyl radical is more reactive with protein and can damage the majority of amino acid residues. Peroxyl radicals and alkoxyl radicals are electrophilic radicals that may easily steal hydrogen from the C–H bond (or S–H bond of cysteine), resulting in the free-radical chain reaction of proteins. The ·OH has extraordinarily high oxidation activity and can react with functional groups in other organic compounds in an oxidation-reduction reaction. ·OH in solution can quickly oxidize the side chain groups of surface-active amino acid residues in proteins due to their comparable chemical characteristics to H_2O. The reaction between ·OH and protein is affected by the specific chemical properties of proteins and the accessibility of solvents. The oxidation of proteins by ·OH easily generates the modifications of amino acid residue side chains, which can change the protein conformation. Aromatic amino acids and sulfur-containing amino acids are the most sensitive to ·OH, followed by aliphatic amino acids and arginine, whereas other charged amino acids and hydrophilic amino acids are the least sensitive. These radicals can directly trigger the free-radical chain reaction of protein or participate in protein oxidation at certain processes during chain propagation. As the lipid radicals intervene, many different protein radicals are produced because 20 common amino acid side chains easily participate in the reaction[16]. Furthermore, Meissner et al.[21] discovered that lipid oxidation could act as a mediator for protein skeleton breakdown, taking place on the protein surface. In a related study, utilizing an excitation-emission matrix fluorescence spectrum, Meissner et al.[22] demonstrated that lipid oxidation enhanced the fluorescence of protein-lipid adducts, especially under conditions with high water levels. These findings establish a cohesive understanding of the interplay between lipid oxidation and protein oxidation. Lipid radicals can induce protein oxidation, whereas the breakdown of the protein skeleton and the production of protein-lipid adducts demonstrate the impact of lipid oxidation on protein structure. The majority of these protein radicals can actively engage in the free-radical chain reaction of proteins. As a result, lipid radicals can mediate protein oxidation by initiating and speeding the free-radical chain reaction of protein.

Aside from free radicals, some secondary oxidation products formed by lipid oxidation can interact with the amino side chain groups of protein molecules. These reactions are primarily involved in the formation of protein crosslinking via Michael addition reaction with nucleophilic amino acid residues, such as the sulfhydryl group of cysteine and the imidazole group of histidine. As electrophilic compounds, α,β-unsaturated[19–20] aldehydes (malondialdehyde, acrolein, 4-hydroxynonenal, etc.) are prone to add to the nucleophilic sites of proteins[23]. The sulfhydryl group of cysteine, the imidazole group of histidine, and the ε-amino group of lysine are relatively active nucleophilic sites in protein[24]. Malondialdehyde can react with histidine residue, glutamine side chain, and peptide N-terminal to form Schiff base adduct[25]. It has electrophilicity with peptide N-terminal and glutamic acid ε-amino reaction, forming a dihydropyridine adduct[25] covalently bonded with lysine to produce N^{ε}-[2-propenal]-lysine[26]. Acrolein can react

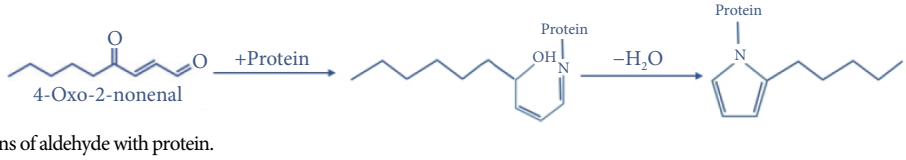

Figure 1 Addition reactions of aldehyde with protein.

with protein histidine residues to form N^{τ}-[3-popanal]-histidine[27]. α,β-Unsaturated aldehydes retain aldehyde functional groups on modified protein molecules after addition with protein, which is an important way for lipids to initiate protein oxidation.

Researchers have investigated the interaction between protein and lipid oxidation, highlighting their interplay in oxidative processes. For instance, Li et al.[28] discovered that beef and pork carboxy Mb were vulnerable to lipid oxidation-induced browning. Lipid oxidation is also more critical to color in beef than in pork when retailing fresh red meats in modified atmosphere packaging containing carbon monoxide. Moreover, Wang et al.[9] discovered that protein-free radicals could move to the lipid component in rabbit meat, boosting lipid oxidation. This study lends weight to the idea that protein and lipid oxidation are inextricably linked. Hajieva et al.[29] discovered that the amino acid radicals might reinitiated lipid peroxidation by attacking an unsaturated fatty acid. Thereby, they would have acted as potentially prooxidative chain-transfer agents. Additionally, Utrera et al.[30] suggested a possible connection between the oxidation of lipids and proteins, both of which can be initiated by ROS attack. Furthermore, Meissner et al.[31] revealed that proteins played an important role in the breakdown of lipid oxidation products in oleogels. This finding illustrates the active involvement of proteins in the degradation and modification of lipid oxidation products. Considering determinants in the development of oxidative processes, Domínguez et al.[6] emphasized the significance of pro-oxidant compounds, such as heme-proteins, as well as pro-oxidant enzymes and antioxidant compounds, including antioxidant enzymes or peptides. As a result, there is a contact between proteins and lipids that mediates their respective oxidative processes. Protein free radicals can be transported to the lipid component, enhancing lipid oxidation. At the same time, lipid oxidation influences the oxidation state of proteins.

3.2 Co-oxidation of lipid and Mb

Similarly, lipid oxidation can spread to Mb components. Electrophilic free radicals formed from lipid oxidation can remove hydrogen from Mb globin, facilitating the free-radical chain process. Furthermore, the addition reaction between electrophilic α,β-unsaturated aldehydes and nucleophilic sites in globin results in direct lipid oxidation. Aldehydes with high reactivity are often polar, allowing them to quickly diffuse into the sarcoplasm fraction and subsequently react with Mb via covalent addition[9]. OxyMb covalently modifies α,β-unsaturated aldehydes, altering their tertiary structure and making the protein more susceptible to subsequent oxidation. In Mb molecules, heme exists in the hydrophobic "heme pocket" comprising relatively dense globulin, which is almost completely embedded. Thus, globin can shield heme from the effects of an external oxidative environment. The reaction between lipid oxidation products and Mb globin causes globin to unfold, destroying the "heme pocket". Under this situation, the heme molecule is in direct touch with the external oxidation environment and is susceptible to oxidative destruction. Notably, as an amino acid residue maintaining the stability of heme in Mb, histidine residues are vulnerable to attack by α,β-unsaturated aldehydes. Gürbüz et al.[25] demonstrated that malondialdehyde can react with histidine residues to form Schiff base adducts, exposing the heme group to the oxidation environment and accelerating the oxidation of Mb. Manat[32] noted that covalent addition of α,β-unsaturated aldehydes to key amino acid residues of Mb could alter the tertiary structure of Mb and improve its oxidative sensitivity. In a related study, Naveena et al.[33] discovered that the Michael addition reaction between 4-hydroxy-2-nonenal and chicken Mb accelerated the oxidation of Mb. The findings indicate that lipids play an important intermediary role in Mb oxidation, speeding the process through direct transfer of oxidation products and change of Mb structure.

Conversely, Mb oxidation can affect lipid oxidation. Proteins can inhibit lipid oxidation in meat and its products through biologically designed mechanisms (e.g., iron-binding proteins and antioxidant enzymes) as well as nonspecific mechanisms such as hydroperoxide reduction, free radical scavenging, ROS inactivation, pro-oxidative transition-metal sequestration, and alteration of meat product properties[34–35]. Proteins not only have antioxidant properties, but their autoxidized by-products, such as $O_2^-\cdot$, are powerful ROS that can directly cause lipid oxidation. Mb·(Fe^{IV})=O can extract a hydrogen atom from the diallyl position of polyunsaturated fatty acids, thereby triggering lipid oxidation[15]. Mb·(Fe^{IV})=O also has a strong ability to promote lipid oxidation (Figure 2)[36].

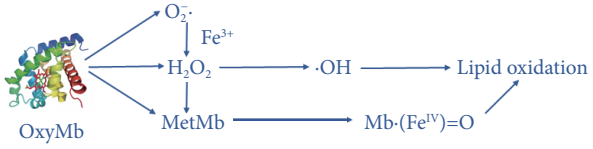

Figure 2 Effect of Mb on the process of lipid oxidation.

The free iron released from heme is also a main catalyst for lipid oxidation in the muscle food system. Non-heme iron reacts with molecular oxygen to produce superoxide radicals, and with H_2O_2 to produce ·OH. These free radicals are the precursors to lipid oxidation. Furthermore, as a unique protein, globin in Mb undergoes free-radical chain reactions, generating C-centered radicals that may act as a starter or enhancer in lipid oxidation. Soyer et al.[37] studied the relationship between Mb and lipid oxidation during the ice storage of *Lates calcarifer* and red tilapia. They found that the oxidation of Mb and the release of non-heme iron were related to lipid oxidation. In a separate study, Lee et al.[38] discovered that Mb released a variety of compounds following dissociation, including heme sulfide, MetMb, Mb(Fe^{IV})=O, Mb crosslinking, and iron atoms released during porphyrin group disintegration. These substances can play a role in mediating lipid oxidation through distinct mechanisms. Furthermore, Zheng et al.[39] found that Mb participated in lipid oxidation without acting as a catalyst. The key process in Mb-induced lipid oxidation involves the liberation of hemin from Mb, and it plays a significant role in promoting lipid oxidation. While the auto-oxidation of OxyMb can also contribute to lipid oxidation, the formation of Fe^{2+} during the cracking process of Mb has minimal impact on lipid oxidation. These investigations shed light on the link between Mb and lipid oxidation. The numerous mechanisms involved in mediating lipid oxidation include Mb oxidation, non-heme iron release, and the creation of particular chemicals following Mb dissociation.

Obviously, lipids and Mb have a close interaction that influences their oxidation processes. Understanding the interaction of lipids and Mb is crucial for enhancing our understanding of oxidation mechanisms and developing effective muscle meal preservation and quality control strategies.

3.3 Co-oxidation of protein and Mb

Similar to the influence of Mb on lipid oxidation, the ROS (e.g., ·OH, $O_2^-\cdot$, H_2O_2, heme cationic free radical, Mb·(Fe^{IV})=O, and Mb(Fe^{IV})=O) generated during the cascade reaction of Mb autoxidation and degradation can trigger protein oxidation (Figure 3)[15].

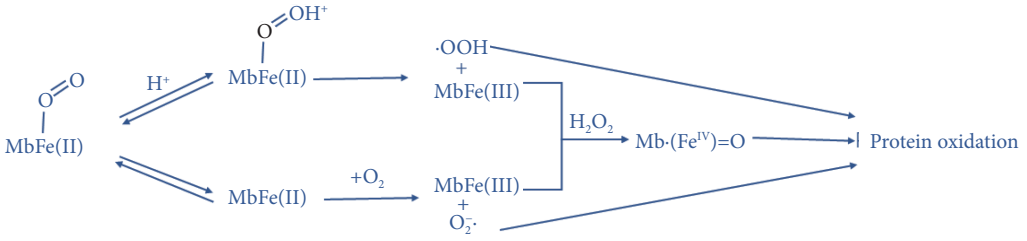

Figure 3 Effect of Mb on the process of protein oxidation.

As a type of free radical, heme cationic free radical and Mb·(Fe^{IV})=O can absorb hydrogen atoms from protein molecules and trigger the free-radical chain reaction of proteins[40]. In fact, Mb(Fe^{IV})=O is a strong oxidant that can directly extract hydrogen atoms from protein and be protonated, thereby exhibiting stronger oxidation-promoting activity[32]. Mb(Fe^{IV})=O and its precursors can induce protein to produce stable protein free radicals. The most potent oxygen free radicals are ·OH, which can damage any amino acid residue in a protein molecule and trigger a protein oxidation reaction[40–41]. Li et al.[42] found that ·OH can induce myofibril protein to produce carbonyl, dimer tyrosine, disulfide bond crosslinking, and non-disulfide bond crosslinking. Based on proteomic technology, Fu et al.[43] reported that myosin, actin, calpain, and cysteine protease with high sulfhydryl content in myofibril can easily form disulfide bond crosslinking.

In addition, the oxidation of Mb is influenced by the active compounds generated by protein autoxidation. Lund et al.[44] discovered that protein oxidation can propagate through a free-radical chain reaction in meat, suggesting a potential link with Mb oxidation. Furthermore, Wang et al.[9] discovered that a variety of radicals, including thiyl and alkyl peroxyl radicals, might alter the heme protein in the muscle protein's radical cascade reaction, resulting in redox instability of Mb. Another study found that carbonyl chemicals produced by protein oxidation could function as promoters of Mb autoxidation, thus linking protein oxidation and Mb oxidation[30]. Free radicals have been observed to denaturize this heme protein, which can impact Mb's redox stability[9,45–46].

These results demonstrate the connection between Mb oxidation and protein oxidation. The redox stability of Mb can be impacted by the presence of carbonyl chemicals, heme protein alteration, and the spread of oxidation via free radicals.

4 Conclusion

There are interactions among lipid, Mb, and nonheme protein oxidation in meat. Thus, variations in meat quality are the result of interaction oxidation, rather than a single incident of lipid or protein oxidation. Mb's redox stability can be influenced by aldehydes produced during lipid oxidation. Mb oxidation produces ROS, and chemicals like Mb(Fe^{IV})=O can accelerate lipid oxidation. Free radicals and other chemicals created by lipid oxidation can directly engage in protein oxidation, whereas lipid oxidation products can react with proteins to facilitate protein oxidation. Despite some studies into the impact of lipid and protein oxidation on meat quality, as well as the interaction between lipid and protein interactive oxidation, this field continues to have significant flaws. More research is needed to identify how to use meat oxidation to increase its quality and what the optimum quality of meat during oxidation is. It is vital to research the interactions of oxidation between different livestock and poultry products, particularly the mediating influence of nonheme proteins on lipid oxidation. between, especially on the mediating effect of nonheme proteins on lipid oxidation, is necessary. Although the association between lipid oxidation and Mb oxidation has been thoroughly researched, more theoretical research is needed to better understand the interaction of these two factors in meat. Furthermore, relatively little research has been conducted on the interaction between Mb and nonheme proteins, and their theoretical foundation needs further development.

Conflict of interest

The authors have no competing interests to declare.

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