Lipid Oxidation in Meat and Meat Products—A Review[1]

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ABSTRACT

Lipid oxidation is a major cause of deterioration in the quality of meat and meat products. Oxidation can occur in either the stored triglycerides or the tissue phospholipids. Ferric heme pigments have been implicated as the major prooxidants in tissue lipid oxidation. Pigment and lipid oxidation are interrelated, and ferric hemes are believed to promote lipid oxidation. The resulting oxidation destroys the hemes. Nonheme iron and ascorbic acid may also function as prooxidants in meat. Sodium chloride accelerates oxidation of the triglycerides, although the mechanism of salt catalysis is not completely known. Cooked meat undergoes rapid deterioration due to tissue lipid oxidation. The meat pigment in the cured pink ferrous form does not promote the rapid oxidation undergone by cooked uncured meat. Refrigerated and frozen fresh meats are also susceptible to lipid oxidation. Protein denaturation and cross-linking may result from lipid oxidation in stored freeze-dried meat. With increased consumption of prepackaged raw meat and precooked convenience meat items, control of oxidation has become increasingly important. Antioxidants and chelating agents are the most effective inhibitors of lipid oxidation.

INTRODUCTION

Lipid oxidation is a major cause of deterioration in the quality of meat and meat products. Undesirable changes in color, flavor and nutritive value occur as meat lipids oxidize and interact with other meat constituents, such as pigments and other proteins, carbohydrates and vitamins (1). Fresh meat and many processed meat products are susceptible to lipid oxidation. Oxidative deterioration occurs in raw meat stored at refrigerator temperatures (2) and in fresh meat in frozen storage (3). Stored cooked and cured meat (4), freeze-dried (5), and irradiated meat products (6) are all susceptible to lipid oxidation. In prepackaged fresh meat, the brown color and lipid oxidation due to ferric hemes are highly undesirable from the consumers' point of view. Control of lipid oxidation in meat and meat products has become increasingly important with greater production and consumption of precooked meat items for institutional and home use.

This paper on lipid oxidation in meat and meat products will review the composition of meat lipids, the nature of the oxidative reaction, the catalysts involved and outline some methods of controlling oxidation.

COMPOSITION OF MEAT LIPIDS

Lipids found in meat can be classified as depot or intermuscular and as intramuscular or tissue lipids. The depot or intermuscular lipids are generally stored in specialized connective tissues in relatively large deposits, whereas tissue lipids are integrated into and widely distributed throughout the muscle tissues (7). The intracellular lipids exist in close association with proteins and contain a large percentage of the total phospholipids (8). The phospholipids contribute about 1% of the tissue weight; the triglyceride fraction is about five times as large (9). Though the phospholipid content of meat is relatively small, the susceptibility of the phospholipids to oxidation makes them important in determining meat quality. The lability of the phospholipids is a result of their high unsaturated fatty acid content. For example, 19% of the fatty acids in beef phospholipids have four or more double bonds, while only 0.1% of the triglyceride fatty acids from beef show this degree of unsaturation (9). Particularly high levels of linoleic and arachidonic acids are found in the phospholipids (10). Phospholipids may also exist in closer contact with tissue catalysts of oxidation than do the triglycerides, thus increasing their tendency to oxidize (11).

The amount of phospholipid has been shown to be relatively constant in muscles from different animals or carcass locations (12,13), while the amounts of total lipid and neutral lipid are more variable. The fatty acid composition of phospholipids reportedly shows some variation with carcass location (12-14). Luddy et al. reported that phospholipid fatty acids from muscles classified as light-colored had a higher content of monoenes, while polyunsaturated fatty acids predominated in the phospholipids from dark muscle (15).

Kuchmak and Dugan (14) noted the variation in the composition of fatty acids from phospholipids from different muscle sources. Pork belly muscle phosphatidylethanolamine was observed to have elevated levels of linoleic and arachidonic acids. This factor may explain the tendency of pork bellies to undergo oxidative deterioration. It seems possible that compositional differences in fatty acids in the phospholipid fraction may result in varying susceptibilities to oxidative rancidity in cuts from different carcass locations.

OXIDATION OF MEAT LIPIDS

Early studies of rancidity in meat were concerned with oxidation of the adipose tissue and have been reviewed by Watts (16). More recent reviews of the oxidation of tissue lipids are also available (8,17). The role of tissue lipids in rancidity was postulated by Tims and Watts (18). They noted rapid flavor deterioration in cooked meats during storage and proposed that this change in flavor resulted from the oxidation of highly unsaturated protein-bound phospholipids. Later work (19) showed that less oxidation occurred in the neutral lipid fractions separated from rancid cooked pork than in the total lipid extract or in the fraction referred to as phospho- or proteolipids. Hornstein et al. (9) also observed that the phospholipid fraction and the total lipids from pork and beef became rancid quickly when exposed to air. The neutral fat fraction developed off flavors less readily, leading to the conclusion that phospholipids contribute to poor flavor, particularly in excessively lean meat. Neutral fat may trap volatile decomposition products of polar lipids and thus reduce their effect on flavor.

In addition to the rapid lipid oxidation observed in cooked meat, lipid oxidation occurs in raw meat (2,3) with adverse changes in flavor and color resulting. Although frozen raw meat is generally fairly resistant to oxidation, rancidity can develop during freezing and thawing (17). Wide fluctuations in temperature and inadequate protection from oxygen can accelerate the development of rancidity. Under good conditions, however, lean raw meat is quite

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stable for periods of several months to a year, depending on the species from which it originated and storage conditions.

Lower levels of lipid oxidation have been observed in cooked, cured meat than in uncured samples. As long as the meat pigment is in the pink, cured, ferrous form, lipid oxidation occurs very slowly. On storage of cured meat, however, the pigment is converted to the brown ferric form, which accelerates oxidation and results in increased TBA values for the stored product (4).

Treatments which retard lipid oxidation are effective in preventing pigment breakdown. Frozen, cured, cooked pork samples may be protected from salt-catalyzed lipid oxidation with sodium tripolyphosphate and 0.108% sodium ascorbate (20). Increases in lipid oxidation are related to the loss of ascorbate in the stored samples (20).

Many researchers have noted adverse changes occurring in freeze-dried foods as a consequence of oxygen uptake. Harper and Tappel (21) have reviewed earlier work on oxidative deterioration in freeze-dried meats. Oxidation of hematin compounds, lipids and proteins occurs during the storage of freeze-dried meat. Oxidation of tissue lipids has been reported to occur in two steps, with the phospholipids becoming oxidized first and the neutral fats later (11). Protein denaturation, resulting in a decrease in solubility, may also result from pronounced oxidation of the polyunsaturated fatty acids. Deteriorative reactions occurring in freeze-dried meats may not be the oxidative rancidity reactions typical of fresh and frozen meats (5). Interactions between proteins and cross-linking of autoxidizing lipids and proteins may take place. Tuomy et al. (5) found highly significant positive correlation coefficients between oxygen uptake and taste panel results for a variety of meat products. Presumably oxidation of meat lipids contributed to the undesirable flavor and odor. Certain products were also observed to be less susceptible to oxygen uptake and subsequent deterioration, indicating that some natural antioxidant activity was present. Certainly the correlations between oxygen uptake and flavor and odor deterioration indicate the desirability of low levels of oxygen in the storage atmosphere of freeze-dried meat items.

Lipid oxidation is an important cause of flavor deterioration in cooked meat irradiated at pasteurizing levels and refrigerated (17). Chang et al. showed that cooked, irradiation-sterilized beef did not develop oxidative rancidity when stored in air tight containers, and postulated that the pigments were converted to a catalytically inactive form during radiation treatment. Greene and Watts (6) later indicated that the low TBA values observed for stored, cooked, irradiated meat were due to a combination of antioxidant development and further reactions undergone by lipid oxidation products.

CATALYSTS OF LIPID OXIDATION IN MEAT

Much effort has been devoted to identification of catalysts in animal tissues responsible for the oxidation of unsaturated lipids. The accelerating effect of hemoglobin and other iron porphyrins on the oxidation of lipids is a generally accepted phenomenon, and hemoproteins have been implicated as the major prooxidants in meat and meat products (1,4,23). Early work, reviewed by Watts (16), demonstrated that heme catalyzed lipid oxidation results in destruction of the pigments, as well as oxidation of the fatty tissue.

Ferric hemochromogen is postulated to be the active catalytic form of the muscle pigments (4,23). In cooked meat, the pigment is in the active denatured ferric hemochromogen form, accounting for the rapid initiation of lipid oxidation. The lower level of oxidation in cured, stored meat results from the conversion of the pigments to the catalytically inactive ferrous nitric oxide hemochromogens (20).

In fresh meat the pigments exist in three forms: purple reduced myoglobin, red oxymyoglobin and brown metmyoglobin. Metmyoglobin is undesirable from the standpoint of meat color and also because of the catalytic effect of ferric hemes on the oxidation of unsaturated lipids. Free radical intermediates from this reaction can decompose hemes, resulting in loss of color. Thus, pigment and lipid oxidation are interrelated in fresh meat, and of crucial importance from the standpoint of consumer acceptability (2).

Some of the work on iron porphyrins as biocatalysts has been reviewed by Tappel (25). A mechanism proposed for the prooxidant activity of hemes is based on their known ability to decompose lipid peroxides. In this theory, free radicals resulting from the peroxide scission initiate new reaction chains. Heme compounds can also act as antioxidants rather than prooxidants (26). Kendrick and Watts (27) reported that no oxidation occurred at heme concentrations of three to four times the concentration required for optimum catalytic activity.

While studies with model systems (27) are of great value, the implications for complex systems, such as meat and meat products, are of a speculative nature. Treatments, such as cooking and freezing, may alter the influence of cellular hemes on the oxidation of tissue lipids.

Other meat components have been attributed catalytic roles in lipid oxidation. Some metals, especially ferrous iron, occurring in meat in trace amounts, are efficient lipid oxidation catalysts. Iron, in combination with ascorbic acid, has been implicated in lipid oxidation in meat (28), also as the major catalyst of lipid oxidation in tissue homogenates and fractions (29).

Sodium chloride, a common meat additive, has a puzzling effect on oxidative changes in meat. The role of NaCl in initiating color and flavor changes in cured meat is well recognized, but poorly understood.

Although some evidence indicates that trace metal impurities present in salt account for its effects on lipid oxidation (31), there is evidence for a direct role of sodium chloride in initiating fat oxidation (32). Salt is believed to catalyze the oxidation of the stored triglycerides (8), and the effect of sodium chloride on fat oxidation depends on the level of free moisture in the system (30). The effect of NaCl on oxidation has been attributed to the action of the reactive chloride ion on lipids, or to a modification of the hemoprotein catalysts of lipid oxidation (32).

CONTROL OF LIPID OXIDATION IN MEAT

Lipid oxidation and the related deterioration in color and flavor, can be inhibited by using antioxidants and chelating agents, especially polyphosphates (17,18). In raw meat, propyl gallate and butylated hydroxyanisole protect meat pigments and inhibit lipid oxidation (2). Polyphosphates are ineffective in raw meat, presumably due to hydrolysis of the phosphate group by muscle phosphatases (2).

There is also evidence to indicate that natural antioxidants may be produced in meat under certain conditions, such as prolonged heating at high temperatures and by sterilizing doses of ionizing radiation (8).

If sufficient reducing activity is present in meat, wrapping fresh meat in an oxygen impermeable wrap should result in reduction of myoglobin, anaerobiosis and retarded lipid oxidation (2). Control of lipid oxidation by removal of oxygen is difficult to achieve, however. Attempts to control oxidation of canned, refrigerated raw meat by evacuation have been reported to be less successful than treatment with antioxidants (17).

In view of the importance of meat color and flavor to the consumer, efforts to find acceptable ways of limiting lipid oxidation are of great importance.

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