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Lipid Peroxidation in Muscle Foods - An Overview

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Abstract: Meat is considered a very nutritive food and has been evaluated and associated with a good health, by contributing quality protein, B vitamins, iron (a nutrient most often lacking in the diets of women and children) and zinc (a mineral that is essential for growth and metabolism). Meat fat is important in human nutrition with n-3 PUFA and CLAs playing a beneficial role. In addition to its nutritive value, meat has other important attributes, including its attractive sensory properties. Nowadays under a point of view, some kind of meat can be considered healthier of the past. The fatty acids composition of meat will affect the profile of compounds produced in lipid oxidation and the abundance of unsaturated fatty acids will favour the abstraction of a hydrogen atom and the start of the oxidation process. Lipid peroxidation is a main problem that reduces meat quality and this is a ubiquitous phenomenon that can lead to rancid odour and loss of product taste. Moreover, lipoperoxides and some cholesterol epoxides are considered in literature as atherogenic agents and are mutagenic, carcinogenic and cytotoxic.

Key words:

INTRODUCTION

Lipids are present in muscles as structural components of the muscle membranes, as storage droplets of triacylglycerol between muscle fibres and as adipose tissue (marbling fat). These lipids, or more precisely their fatty acids, contribute to a wide range of quality attributes like colour stability, drip loss and the development of oxidative rancidity. Finally, nutritional quality depends upon the fat content of the meat and its fatty acid composition.

The attractiveness of meat to the purchaser is mainly related to colour, after perceived economic value. As meat ages, it turns brown as the myoglobin is converted to oxidized metmyoglobin and is rejected by the consumer. Lipid peroxidation increases the rate of metmyoglobin formation and conversely metmyoglobin acts as a catalyst of lipid peroxidation so that in beef muscle displayed under oxygen permeable film, lipid oxidation and metmyoglobin levels were closely correlated. Lipid peroxidation depends upon the degree of unsaturation of the fatty acids and the levels of the antioxidant vitamin E (α-tocopherol) and prooxidants such as free iron. Increasing the degree of unsaturation of the fatty acids results in a decrease in colour and oxidative shelf-life.

However, in considering the possible role of meat lipids in disease it must be remembered that most diseases are of complex aetiology and that fat is only one risk factor. In fact muscle, if consumed without any adhering adipose tissue, can easily contribute to this aim since, as discussed above for texture, most muscles have less than 50g/kg lipid and hence can be classed as a low fat product. However, even a lamb or pork chop or sirloin steak can be included in meals that meet dietary guidelines. It is the consumption of "hidden" fat in burgers, pates, sausages and the like that contribute to the high fat intake from meat since a high proportion of people discard adipose tissue on the plate.

The saturated fatty acids, lauric (12:0), myristic (14:0) and palmitic (16:0) acid contribute to heart disease by raising plasma low density lipoprotein cholesterol whereas linoleic acid and α-linolenic acid lower it thereby decreasing the risk of heart disease. Stearic acid (18:0) does not affect plasma cholesterol concentrations. However, it may contribute to thrombosis, the final event in CHD that produces the heart attack. It is therefore included in the ratio of PUFA to saturated fatty acids (P:S) used to assess fat quality in terms of human nutrition and which has an acceptable value of 0.4 or above for the diet as a whole. The P: S ratio for pig muscle is generally above this value but for muscle from cattle and sheep is around 0.1 or less.

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Another consideration in addition to increasing the intake of total PUFA is the relative levels of those derived from linoleic acid, the n-6 or ω 6 series and those derived from α -linolenic acid, the n-3 or ω3 series. The longer-chain PUFA act as precursors for oxidized derivatives called eicosanoids and these function a regulator of many physiological processes. In heamostasis, thromboxane A2 produced from arachidonic acid (20:4 n-6) is a powerful clotting agent whereas thromboxane A3 from eicosapentaenoic acid (20:5 n-3) is much less active. The levels of the two eicosanoids depend on the quantities of their precursor fatty acids in the phospholipids of blood platelets. These amounts, in turn, depend upon the relative amounts of linoleic acid and α-linolenic acid in the diet since these are the precursors of the longer-chain PUFA. It is believed that primitive man evolved with a ratio of 18:2/18:3 of 1 in his diet but because we now consume large quantities of linoleic acid in vegetable oils the ratio ranges from 7-20. This contributes to heart disease by raising 20:4 n-6 and increasing the thrombotic tendency and contributes to autoimmune disease like arthritis because the leukotrienes produced from arachidonic acid stimulate the immune responses more than those from 20:5 n-3.

Because of these competitive metabolic effects between n-6 PUFA and n-3 PUFA the recommended level for the ratio of linoleic to α-linolenic acid is 4 or less. Muscle from forage finished cattle and sheep usually has an n-6: n-3 ratio below 2 whereas for pigs it is nearer 20. Fatty acids of considerable interest at present are the conjugated linoleic acids (CLA). One isomer, 9-cis, 11-trans is present in ruminant meats and milk. It is formed either in the rumen as the first step in biohydrogenation of linoleic acid or by n 9 desaturation in body tissues of transvaccenic acid, itself produced in the rumen. CLA isomers inhibit carcinogenesis, decrease atherosclerosis and modify the immune response and partition energy toward the growth of muscle rather than adipose tissue [1]. Amounts in ruminant meat and milk can be altered by dietary means [2,3] **and so on …..** However, the value of CLA in human nutrition and the amounts required for therapeutic effects remain to be established.

Lipid Oxidation Process in Meat: The oxidation of lipids is commonly described as an oxidative, oxygen dependent, deterioration of fats, notably the unsaturated fatty acids. This modification of fatty acids is principally carried out by an autocatalytic mechanism of 'free radicals', called auto oxidation, consisting of three phases [4].

1. Initiation:

(a) $RH + O_2 \rightarrow R.\bullet + \bullet OOH$

2. Propagation:

(b) $R\bullet + O_2$ $ROO\bullet$
(c) $RH + ROO\bullet$ $ROOH + R\bullet$
(d) $ROOH$ $RO\bullet + \bullet OH$

3. Termination:

(e) $R\bullet + R\bullet$ $R\text{-}R$
(f) $R\bullet + ROO\bullet$ $ROOR$
(g) $ROO\bullet + ROO\bullet$ $ROOR + O_2$

Lipids hydroperoxides (ROOH) are the primary lipid oxidation products and once formed, they are relatively stable at moderate reaction conditions, such as low temperature and absence of metal ions. However, these conditions are never found in muscle foods and hydroperoxides become susceptible to further free radical chain reaction such as isomerization and decomposition. Their breakage causes secondary products such as pentanal, hexanal, 4-hydro-xynonenal and malondialdehyde (MDA).

Meat is rich in heme proteins such as oxymyoglobin and oxyhemoglobin, that are particularly susceptible to oxidation and are affected by transition metals, lipoxy radicals and active oxygen species such as H_2O_2, $OH\bullet$, HOCl and $NO_2\bullet$. Several investigations have shown that the free iron redox cycle, provided by ascorbic acid, is the main initiator of lipid peroxidation in fresh muscle foods and that such a system could greatly affect the oxidation of oxymyoglobin. Haemoglobin and myoglobin as inhibitors of hydroxyl radical generation in a model system of "Iron Redox" cycle. [5]. It was postulated that there is interrelation between lipid peroxidation in muscle tissue and oxymyoglobin [6, 7] and recent works have shown that there are two pathways that affect oxymyoglobin: a) oxygen active species (H2O2, OH•, $O2^{\bullet-}$ and perferryl), generated during autoxidation of myoglobin and oxidation of ferrous ions; b) lipid radicals ($ROO^{\bullet}$, $RO^{\bullet}$) and hydroperoxides generated during lipid peroxidation [8].

During oxidation processes it is necessary to consider also the effects of enzymatic component (autocatalyzed oxidation) that operate after slaughtering. In the post mortem step, endogenous antioxidant systems (for example superoxide dismutase and glutathione peroxidase) available in vivo in the cell are not active and this doesn't permit to balance free radical production.

Factors Influencing Lipid Peroxidation: Many factors can influence fat composition of meat, i.e. diet, race, weight, age, tissue of deposit, sex, hormones etc. [9-12]. Meat industry has worked for reduction of fat in meat, reaching important results, but the problem of lipid peroxidation remains still open.

The fatty acids composition of meat will affect the profile of compounds produced in lipid oxidation and the abundance of unsaturated fatty acids will favour the abstraction of a hydrogen atom and the start of the oxidation process. Dietary supplementation to animal foods and the tendency of the species to accumulate certain fatty acids in the membrane phospholipids, affect the lipid composition of the membrane and, consequently, its susceptibility to peroxidation. Polyunsaturated fatty acids (PUFA) of muscle membrane cells are particularly susceptible during storage to peroxidation when the degree of unsaturation of membrane lipids is increased with reduction of oxidative stability of muscle [13]. Furthermore, other factors that will affect the lipid oxidation of muscle foods are exposure to light, oxygen availability, temperature conditions and microbial growth [14]. Cooking process can affect lipid compounds in meat, especially the fatty acids component, changing the nutritional value of cooked products respect to raw sample [15]. These factors can make the quality of the foodstuff not acceptable for human consumption, but before these conditions take place, lipid oxidation could generate toxic molecules with possible hazards for human health.

Effects of Oxidized Lipids in Nutrition: The toxicity of dietary oxidized lipids was studied in various animal species and involved highly oxidized fats, particularly thermoxidized frying fats. Among oxidation products contained in such fats, polymers appear to be harmless because they are minimally absorbed and are removed with feces. Nonpolar dimer FA showed digestibility below 5% [16]. Noncyclic dimeric FA were also poorly absorbed (10%). On the other hand, as much as 50% of the total oxidized monomeric acids and 95% of the total cyclic monomeric acids from thermoxidized fats and TAG were recovered in the lymphatic lipids [17]. Rats fed mackerel fried in coconut oil over the course of 4 days showed initial stages of cell damage in the liver and kidney as well as an increase in total lipids and cholesterol in heart and serum, compared with the control group fed steamed mackerel [18].

Eder (1999) [19] showed that the consumption of oxidized lipids (thermally, at 150°C, 6 days) caused a reduction in the desaturation rate of linoleic acid and O-linolenic acid by microsomal P 4, P 5 and P 6desaturases and also a reduction in P 9 desaturation. On the other hand, recent research on humans demonstrated the conversion of trans- O -linolenic acid isomers into the following long-chain PUFA: 19-trans-C22:5n-3, 19-trans-C22:6 n-3 and trans- C24:5. This trans-PUFA can interfere with platelet and endothelium metabolism. The trans- O -linolenic acid isomer-rich diet raised the LDL- to HDL-cholesterol and total cholesterol ratio, which would increase the risk of cardiovascular disease by 8% [20]. Moderately thermally oxidized soybean oil (130°C, air flow-through) of peroxide value (PV) = 75 mEq O2/kg diet (compared with the control of 9.5 Eq O2/kg diet) was fed to rats for 40 days [21]. The study showed no adverse effects on liver, heart, kidney, or adipose tissue FA composition and even a reduction in the osmotic fragility of erythrocytes and hepatic lipogenesis. However, the moderately oxidized oil slightly reduced the vitamin E status in the tissues. A slightly increased susceptibility of LDL to lipid peroxidation and an increased concentration of thiobarbituric acid reactive substances (TBARS) in LDL, was also observed.

The harmful effects in animal fats are closely related to the potential activity of cholesterol oxidation products, particularly in the presence of unsaturated FA and thermal treatment at temperatures exceeding 100°C. UV-oxidized fish lipids and a threefold increase in PV and TBA compared to controls, resulted in weight loss, lower body weight increments, increased liver weight, reduced hemoglobin and hematocrit in guinea pigs fed a diet with a 14% addition of those lipids for 12 weeks [22].

The presence of oxidized lipids in the diet of humans and animals results in an increase in TBARS in plasma and tissues. An increase in the peroxide value could not be detected. There was a dose-dependent increase in conjugated dienes in chylomicrons of rats and humans given oxidized lipids [23]. The research on animals conducted so far, including experiments on mammals shows aldehydes in free form or conjugated with amino acids to be absorbed from the gastrointestinal tract to plasma, muscles and liver. The absorbable aldehydes adducts with protein from the diet are less toxic than free aldehydes. With regard to hydroperoxides, they are generally thought to be decomposed in the stomach, from where they are not transported any farther. It is possible that at low doses, FA hydroperoxides are converted to the corresponding hydroxy FA in the mucosal membrane before being transported to the blood. A study on structurized lipids confirmed that hydroperoxides were absorbed as monohydroxy and monoepoxy FA. They can influence endothelial dysfunction, promote thrombosis and induce atherosclerosis [24].

Apart from the antioxidant system, an additional mechanism of protection is furnished by diarrhea, induced in rats by high doses of lipid hydroperoxide [25]. The LD50 of the hydroperoxide of highly unsaturated FA methyl ester was between 285 and 545 mg active hydroperoxide oxygen per kilogram body weight [26]. Maximum hydroperoxide content in fatty fish products does not exceed 50 mg/100 g. This hydroperoxide level is accompanied by a 20% decrease in EPA and DHA content. Thus, the effects of oxidized lipids in human diet are rather long-lasting.

Antioxidant Addition to Prevent Lipid Oxidation: Antioxidants are added to fresh and further processed meat to prevent oxidative rancidity, retard development of off-flavours and improve colour stability. In food industry they can be grouped into natural antioxidants and synthetic antioxidants, with the latter including, for example, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and tertiary butylhydroquinone (THBQ). Both types of antioxidants play a very important role in the food market. However, because of toxicological concerns of synthetic antioxidants, nowadays the new trend in food industry includes an enhanced concern for the quality and safety of food products, increased preference for natural products over synthetic ones and broadened regulations related to nutritional and toxicity levels of active ingredients. Consequently food market is demanding for natural food ingredients free of chemical additives orientated to promote the use of natural products.

The research for natural additives has notably increased in recent years; compounds obtained from natural sources such as grains, oilseeds, spices, fruit and vegetables have been investigated and the effect of antioxidants on controlling oxidative reactions in meat has been well documented.

Moreover another strategy to prevent lipid oxidation is feeding animals with antioxidats. Addition of vitamin E to the feed increased O-tocopherol concentration in sarcosomes and thus significantly increased the stability of the lipids and Mb-Fe2+-O2 against oxidation. Vitamin E is the primary lipid-soluble antioxidant in biological systems and breaks the chain of lipid peroxidation in cell membranes and prevents the formation of lipid hydroperoxides [27, 28] for this reason it has been found to improve the quality of farm animal products. Feeding with vitamin E-supplemented diets reduced lipid peroxidation in turkey muscle [29-31] in chicken meat [32-34] in pork [35], in fish [36, 37] and in beef [38, 39].

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