

Oxidation-Induced Color and Flavor Changes in Meat

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Studies demonstrating evidence of heme and nonheme iron catalysis of lipid oxidation in model systems and in meats are reviewed along with evidence for oxidation and destruction of hemes by oxidizing lipids. Additional factors that may accelerate or retard oxidation of lipids and heme pigments are also mentioned. Current research by the authors on the relationship of sodium nitrite to flavor and retardation of lipid ox-

idation in cured meats is presented. General conclusions on the studies reviewed are: (1) heme pigments may be more active lipid oxidation catalysts when iron is in the 3+ state; (2) nonheme iron may be a more active catalyst in the 2+ state; and (3) sodium chloride appears to be the major factor responsible for cured meat flavor rather than sodium nitrite or an absence of lipid oxidation.

Heme pigments have been found to catalyze oxidation of muscle tissue lipids. This results in a stale or rancid odor and flavor in the meat, sometimes referred to as warmed-over flavor (WOF). Free radicals produced in oxidizing lipids can oxidize and decompose heme pigments. Meat in which this reaction has occurred is brown in color and likely to be rejected by the consumer. These two reactions appear to be interdependent in that inhibition of one reaction will often result in inhibition of the other—and *vice versa*.

The lipids involved are the unsaturated fatty acids of the tissue lipids which exist primarily as phospholipids (Younathan and Watts, 1959). Adipose tissue of red meats, which contains a high percentage of saturated fatty acids and is physically less accessible to muscle catalysts, is of less significance to this reaction.

The pigments involved are the heme pigments and mainly the muscle pigment, myoglobin. The main forms in which myoglobin may exist in meats are: oxymyoglobin (MbO₂), the bright red pigment associated with fresh raw meat; reduced myoglobin (Mb), the purple-red pigment seen when raw meat is first sliced, before the surface oxygenates to MbO₂; and nitric oxide ferrohemeochrome (NOH), the pink pigment of cured meat. In all of the above iron is in the Fe²⁺ state. Metmyoglobin (MetMb), seen in raw meat that has been stored, and denatured globin ferrihemeochrome, the gray-brown pigment of cooked meat, both contain iron in the Fe³⁺ state.

Considerable differences of opinion have been expressed among researchers regarding the importance of the state of iron to the catalytic activity of the heme pigments. There are also those who question whether the heme pigment is a catalyst at all. This is the main theme around which this paper will be developed.

PREVIOUS STUDIES ON THE INVOLVEMENT OF HEME PIGMENTS IN LIPID OXIDATION

Haurowitz *et al.* (1941) demonstrated in model systems that oxidizing lipids could destroy heme pigments. Younathan and Watts (1960), from their studies with meat (pork), proposed that the Fe³⁺ hemes were the active lipid oxidation catalysts. Evidence for this was demonstrated in that cooked meat developed WOF more rapidly than did raw meat during refrigerator storage. This was measured by sensory evaluation and the 2-thiobarbituric acid (TBA) test for malonaldehyde, a lipid oxidation product. Smith and Dunkley (1962) presented evidence for the reverse when this was tested in model systems. These workers reacted unsaturated fatty acids with FeSO₄ and Fe₂(SO₄)₃ and found Fe₂(SO₄)₃ to be the active catalyst. Brown *et al.* (1963) reacted unsaturated fatty acids

with heme compounds, as opposed to the iron salts of Smith and Dunkley. They found no difference in the rate of catalysis between the Fe²⁺ and Fe³⁺ compounds. However, the Fe²⁺ hemes had an induction period. When removed from the reaction medium for pigment analysis, the pigment was in the 3+ form. This suggested that oxidation to the ferric state took place before catalysis began. In a later paper (Koizumi *et al.*, 1973) MbO₂ was allowed to react with arginine linoleate to determine the effect of oxidizing lipids on Mb oxidation. It was found that increased concentrations of linoleate resulted in increased oxidation of MbO₂ to MetMb. Pigment destruction was also noted as well as was evidence of lipid oxidation. Furthermore, a time lapse was observed between initiation of pigment oxidation and evidence of lipid oxidation. This could again suggest that conversion to MetMb is necessary for accelerated lipid oxidation catalysis. Hirano and Olcott (1971), in a study of the effect of heme concentration on linoleate oxidation, found no difference in rates of oxidation catalyzed by Fe²⁺ and Fe³⁺ hemes. Data for this are not presented and these workers do not mention whether or not a lag was observed with Fe²⁺.

Evidence to this point suggests that when iron is liganded to porphyrin, as it is in meat pigments, both Fe²⁺ and Fe³⁺ pigments may catalyze lipid oxidation, but conversion to the Fe³⁺ state may be necessary for rapid catalysis.

Sato and Hegarty (1971) extracted cooked meat samples with water and estimated development of WOF in the water-extracted meat and in various fractions of the extract by means of the TBA test. The water-extracted meat did not develop a significant TBA value during storage. TBA values increased when the extract was recombined with the meat. This suggested that the component responsible for initiating lipid oxidation was water soluble. Various substances believed to be responsible for catalyzing lipid oxidation in meat were added to the extracted meat and TBA development was followed. Hb and Mb were among the substances added. Neither of these was found to catalyze lipid oxidation in this system. When FeCl₂ and FeCl₃ were added to the water-extracted meat, FeCl₂ produced a TBA value twice that of the FeCl₃ sample. This agrees with the work of Smith and Dunkley (1962) in that when iron salts are used, the Fe²⁺ form is the active catalyst. When these iron salts were added to plain cooked ground beef they did not produce an increase in TBA. However, when iron powder was added, the TBA was almost double that of the control containing no additives. The authors concluded that the catalytic activity might be related to the ratio of Fe²⁺ to Fe³⁺ in meat. These data also suggest that nonheme iron may be the catalytic agent rather than heme iron. Liu (1970a,b) and Liu and Watts (1970) found both heme and nonheme iron to be catalysts of lipid oxidation in beef.

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Greene *et al.* (1971) added phenolic antioxidants (butylated hydroxyanisole [BHA] or propyl gallate) and/or ascorbic acid (AH₂) to raw ground beef and measured pigment and lipid oxidations after several days' storage by means of reflectance spectrophotometry, TBA test, and organoleptic evaluation. While either BHA or propyl gallate alone afforded complete protection to lipids and good protection to pigments, the inclusion of AH₂ with either of these generally resulted in an even lower percentage of MetMb and a more favorable panel rating. Ascorbic acid alone provided good protection to the pigment but only moderate protection to the lipids. Samples containing no additives were very high in MetMb, had high TBA numbers, and were given very unfavorable sensory ratings. The synergistic effect of AH₂ with phenolic antioxidants is well established in the case of lipid oxidation. The authors suggested that AH₂ alone afforded protection by being preferentially oxidized. Sato and Hegarty (1971) studied the effect of AH₂ on iron catalysis of lipid oxidation. One part per million of Fe²⁺ plus increasing concentrations of AH₂ were added to the water-extracted beef. Concentrations up to 500 ppm exhibited prooxidant properties but at 10,000 ppm AH₂ acted as an antioxidant. The authors concluded that AH₂ acted as a prooxidant by keeping some of the iron in the Fe²⁺ state (which they found to be catalytic). At the higher inhibitor concentration, it may either act as an oxygen scavenger or it may upset the Fe²⁺/Fe³⁺ ratio. The AH₂ concentration used by Greene *et al.* (1971) in raw meat was within the range demonstrated to be prooxidative by Sato and Hegarty (500 ppm). Nonheme iron may be catalytic in the Fe²⁺ state while heme pigments may be more active catalysts when the iron is in the Fe³⁺ state. AH₂ may have maintained both heme and nonheme iron as Fe²⁺.

CURRENT WORK AT THE UNIVERSITY OF GEORGIA RELATING NOH TO WOF AND CURED MEAT FLAVOR

Currently, workers in our laboratory are engaged in a study of the relation of lipid and heme oxidations to flavor of cured meat. Since the discovery of the *in vivo* and sometimes *in vitro* production of the carcinogens, nitrosamines, from cured meats, this problem has become a source of concern to the meat industry and the consumer. In nearly all reported studies on the effect of NaNO₂ on flavor of cured meats, NaNO₂, or an additional flavor ingredient, was found to be necessary to produce the typical cured meat flavor (Cho and Bratzler, 1970; Simon *et al.*, 1973; Hustad *et al.*, 1973; Wasserman and Talley, 1972, except with smoked frankfurters; MacNeil and Mast, 1973, unless NaNO₂ was replaced with a spice extractive). The present study was undertaken as a result of comments made by Bailey and Swain (1973). These workers postulated that the reason nitrite is responsible for cured meat flavor is not that nitrite imparts special flavor qualities itself but that by maintaining the pigment in the Fe²⁺ state, lipid oxidation is prevented and therefore WOF is prevented as well. Cured meat flavor is simply a lack of WOF. It must be remembered that meat is not cured with NaNO₂ alone. Sodium chloride is the other standard ingredient of a cure. Sugar and various spices may then be added to produce the specific flavor characteristics of individual products. If the main contribution that nitrite makes to cured meat flavor is that of preventing WOF, then the common phenolic antioxidants should work as well.

Our studies have utilized a simple system of ground fresh pork + NaCl + NaNO₂ in the standard approved amounts (unless otherwise specified) of 0.02% NaNO₂ and 2.0% NaCl. Antioxidants when added were either BHA, propyl gallate, AH₂, or a combination of these. A stable cure was established by packing the ground meat tightly into a loaf shape, wrapping in double aluminum foil, plac-

Table I. Pooled Data Results: Effect of Curing Agents and Antioxidants on Cured Meat Flavor Scores and TBA Numbers of Ground Pork Stored Aerobically 2 Days at 6°

Additive	Sample no.	Sensory evaluation		TBA no.
		Mean score ^a	Std dev.	
2.0% NaCl	11	2.7 ^a	0.29	5.0
0.02% NaNO ₂	7	1.5 ^{bc}	0.35	0.5
0.001 or 0.01% BHA	4	1.1 ^c	0.10	0.6
2.0% NaCl + 0.02% NaNO ₂	12	2.9 ^a	0.36	0.4
2.0% NaCl + antioxidant ^b	19	2.7 ^a	0.25	1.4
Untreated control	16	1.4 ^b	0.21	5.0

^a Based on 1-4 intensity scale (*i.e.*, 1 = no cured meat flavor; 4 = strong cured meat flavor). Means bearing different roman superscripts are significantly different at the $P = 0.001$ level except for untreated controls *vs.* BHA samples (significantly different at the $P = 0.02$ level). ^b Antioxidants include: 0.001% BHA (butylated hydroxyanisole); 0.005% BHA; 0.01% BHA; 0.05% AH₂ (ascorbic acid); 0.001% BHA + 0.05% AH₂; 0.01% BHA + 0.05% AH₂; 0.01% PG (propyl gallate); 0.005% PG + 0.0005% BHA.

Table II. Effect of NaCl Concentration on Cured Meat Flavor and TBA Numbers of Ground Pork Stored Aerobically 2 Days at 6°

[NaCl], %	Mean panel score ^a	TBA no.
0.0	1.4	5.0
0.5	1.7	6.0
1.0	1.8	5.0
1.5	2.7	2.9
2.0	2.7	4.2

^a 1 to 4 scale; 1 = no cured meat flavor; 4 = strong cured meat flavor.

Table III. Effect of NaCl Concentration on Cured Meat Flavor and TBA Numbers of NaNO₂-Treated (0.02%) Ground Pork Stored Aerobically 2 Days at 6°

[NaCl], %	Mean panel score ^a	TBA no.
0.0	1.5	0.5
0.5	2.7	0.8
1.0	2.9	0.8
1.5	2.7	0.2
2.0	2.9	0.4

^a 1 to 4 scale; 1 = no cured meat flavor; 4 = strong cured meat flavor.

ing it into a loaf baking pan, and heating in a 163° oven to an internal temperature of 85°. Temperature was recorded on a potentiometer *via* thermocouples inserted into the meat and oven. To test the effect of lipid oxidation on cured meat flavor, the cooked loaves were cut into 3/8 in. slices, spread out on plates, covered with oxygen-permeable meat film, and stored at 6°. After 2 days, the samples were analyzed by the TBA test (Tarladgis *et al.*, 1960; Zipser and Watts, 1962) and sensory evaluations. Panelists were asked to rate samples on intensity of cured meat flavor.

Table I gives composite results of the studies to date. Pooled data were analyzed by student's *t* test (Steel and Torrie, 1960) and data for each individual experiment by the Wilcoxon Matched-Pairs Signed-Ranks Test (Siegal,

Table IV. Comparison of Conditions Used in Effecting Cure on TBA Numbers of Ground Pork Stored 2 Days at 6°

Time of addition	Simulated anaerobic storage		Aerobic storage	
	TBA no.	Color devel.	TBA no.	Color devel.
Cooked, curing agents added, reheated until pink	0.2	Slight	0.4	Typical
Cooked, curing agents added, reheated to 85°	0.1	Typical	0.4	Typical
Curing agents added, then cooked	0.1	Typical	0.1	Typical
Cooked, curing agent added	0.2	Slight	2.2	None
Cooked without curing agent	1.3	None	3.1	None

1956). Standard deviations were computed on the pooled data. In all cases samples containing salt were rated significantly higher (more cured meat flavor) than samples without salt. $\text{NaNO}_2 + \text{NaCl}$ samples gave a slightly higher mean panel score but this was never statistically significant. Samples containing NaCl alone resulted in lower mean panel scores than samples with NaCl plus NaNO_2 or antioxidants, but these differences were seldom statistically significant. NaNO_2 alone produced no cured meat flavor. TBA numbers were low in the antioxidant and in the NaNO_2 -treated samples and were high for samples with NaCl alone and for samples with no additives. High TBA numbers, indicating increased lipid oxidation, did not affect cured meat flavor. Tables II and III show the effect of NaCl concentration without and with NaNO_2 , respectively, on cured meat flavor and TBA number. Again, TBA number was not significantly related to intensity of cured flavor. When nitrite was not present (Table II) concentrations of salt below 1.5% produced samples with less cured meat flavor. In the presence of nitrite (Table III) decreasing salt concentration did not decrease intensity of cured flavor. NaNO_2 may contribute to flavor perception at lower NaCl levels. Additional work is needed to confirm these data. Other variations (*e.g.*, constant NaCl , varying NaNO_2 ; constant BHA, varying NaCl ; etc.) remain to be tested. Increasing the concentration of nitrite alone (data not presented) produced no difference in panel scores. Nitrite alone does not produce a cured meat flavor.

The main reasons that the above data differ from those of other workers are: either a variable with NaCl alone or NaNO_2 alone was not reported; panelists were not asked to rate samples on intensity of cured meat flavor, but were asked to rate degree of preference; color was not masked during sensory testing (our samples were presented under green lights so that all samples appeared brown); or complete formulations such as hot dogs or hams were prepared and presented to panelists.

The preceding tables reported data that are significant from the practical standpoint. To ascertain whether nitrite actually prevents lipid oxidation by retaining the pigment in the Fe^{2+} state, nitrite was added to ground pork at various times before and after cooking to determine the importance of establishing a cure. Samples were heated in test tubes with thermocouples inserted into the center of each. After treating the samples the meat was either stored in test tubes (simulated anaerobic storage) or spread on plates in $\frac{3}{8}$ in. thickness and covered with oxygen-permeable meat film (aerobic storage). The samples were then stored at 6° for 2 days and analyzed by the TBA test. Observations on color were recorded. Table IV presents these results. The main point to be made here is that it was necessary to establish a cured meat color, or the Fe^{2+} pigment, in order to obtain a low TBA number under conditions conducive to lipid oxidation (aerobic

storage). Sensory data were not obtained on this set of variables so relation to cured flavor is not known at this point. However, the present data suggest again that Fe^{3+} is the active lipid oxidation catalyst.

Younathan and Watts (1959) added NaNO_2 to nonheme model systems. Nitrite did not act as an antioxidant in their systems. Brown *et al.* (1963) found that nitric oxide hemoglobin (NOHb) catalyzed oxidation of unsaturated fatty acids at a rate similar to other Fe^{2+} hemes, but at lower concentrations it also had an induction period as did the other Fe^{2+} hemes. During the induction period NOHb was in the Fe^{2+} state, but after lipid oxidation had started, NOHb was in the Fe^{3+} state, again suggesting that the Fe^{3+} heme may be the more active catalyst.

OTHER FACTORS RELATIVE TO OXIDATION-INDUCED COLOR AND FLAVOR CHANGES IN MEAT

In studying lipid and pigment oxidations in muscle tissue, consideration must be given to a number of other factors as well as those mentioned so far. One of these is the physical association between the tissue lipids and the catalyst. In model systems catalyst and substrate are usually intimately associated. In muscle the chemical components are compartmentalized and the conditions that might bring the reactants together must be considered. For example, the work of Eriksson *et al.* (1970, 1971) demonstrated that denaturation of heme proteins resulted in greater catalytic activity. The reason given for this was that the unfolding of the protein exposed more heme to the unsaturated fatty acids. This could be another explanation for the more rapid lipid oxidation observed in cooked meat.

Another factor is the metmyoglobin-reducing activity in raw meat demonstrated by Stewart *et al.* (1965b), Watts *et al.* (1966), and Saleh and Watts (1968). These workers presented evidence for a biological MetMb-reducing system in post-mortem muscle. The specific enzyme(s) and substrate(s) were not determined but a number of compounds were found to increase this activity. The amount of metmyoglobin-reducing activity naturally present in a meat sample may have a significant effect on the Mb and unsaturated fatty acid stability of that meat.

The idea that a high pH provides greater color retention in raw meats is well established (for example, see Watts, 1954). The relation to its concurrent retardation of lipid oxidation has not been clearly established. A beginning study in our laboratory compared ground beef samples treated with sodium bicarbonate to raise the pH with actual dark-cutting beef. Percentage of MetMb was measured by reflectance spectrophotometry (Stewart *et al.*, 1965a); lipid oxidation, by the TBA test. In both sets of high pH samples, considerable pigment protection was afforded but TBA numbers were intermediate (*i.e.*, smaller than the lower pH, higher MetMb-containing controls, but larger than antioxidant-treated samples). These data re-

semble those of the AH₂-treated samples of Greene *et al.* (1971). Again, nonheme iron may be the lipid oxidation catalyst in this case. When either NaCO₃-treated or dark-cutting samples were cooked or when cooked samples were treated with NaCO₃, TBA numbers were as high as in untreated controls.

CONCLUSION

General conclusions of the authors are: (1) heme pigments may be more active catalysts when iron is in the 3+ state; (2) nonheme iron may be a more active catalyst in the 2+ state; and (3) sodium chloride appears to be the major factor responsible for cured meat flavor rather than sodium nitrite or the absence of lipid oxidation.

Clearly, a number of factors must be taken into account when studying the cause and prevention of lipid and pigment oxidations in meat. The answer no doubt lies in a combination of these factors as well as others not presented in this paper. Further studies on the role of nonheme iron and the Fe²⁺/Fe³⁺ ratio might be profitable. These areas have been studied less extensively and may hold a part of the answer.

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Effect of Lipid Antioxidants on the Stability of Meat during Storage

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Deteriorative changes in meat may occur from heme and lipid oxidations, producing alterations in color, flavor, and odor. Samples of ground beef with either low levels (*ca.* 3%) or high levels (*ca.* 10%) of polyunsaturation in the added fat were examined for storage produced changes. High polyunsaturation levels increased meat deterioration. The antioxidant effectiveness of five additives (0.005% level) derived from natural sources (α -tocopherol, ascorbic acid, 1-ascorbyl stearate, citric acid, and ascorbic acid with sodium bicar-

bonate) was examined during 10 days storage. Samples were adjudged to be commercially unacceptable after 1-4 days storage but monitoring was continued to determine differences in the additive's antioxidant action. Ascorbic acid exerted a definite prooxidant action. The other additives showed only a slight effect in decreasing the rate of lipid and heme oxidations compared to untreated samples. A hypothesis of coupled heme-lipid oxidation is presented.

Generally, the oxidation of lipids in foods is undesired as it may lead to alterations in flavor, odor, and color. The reactivity of fats and other lipids with oxidants within a biological system can vary markedly from the reactivity of extracted and purified lipids. Such factors as the degree of lipid unsaturation, the content of prooxidants and antioxidants, and the cellular and tissue structure all contribute to these differences in oxidation rate (Ledward and MacFarlane, 1971; Love and Pearson, 1971; Kwoh, 1971; Sato and Herring, 1973). With fresh meats, deteriorative changes resulting from oxidation of lipids have not

been a major problem to date. Most spoilage in fresh meat occurs as a result of bacterial action, producing an acid odor and flavor and a brown color. The aerobic bacteria on the meat surface and the endogenous reductants within the tissue act to diminish the oxygen available for tissue lipid oxidations (DeVore and Solberg, 1974). In addition, beef fat is not highly polyunsaturated, with beef adipose tissue lipid containing only about 2.5% polyunsaturates (Swern, 1964). In the more unsaturated pork fat (10-12% polyunsaturates, Swern, 1964), the lipid oxidation is limited somewhat by the lower levels of the prooxidant heme pigments, myoglobin and hemoglobin. Fresh pork, however, does have a limited shelf and storage life, even in frozen storage, because of the development of oxidative rancidity and off-flavors.

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