17.85–28.46%) and stearic acid ($C_{18:0}$; 2.27–4.93%) of the total fatty acids. Lauric acid ($C_{12:0}$) and arachidic acid ($C_{20:0}$), which were present as minor fatty acids, have not been reported before. Linoleic acid ($C_{18:2}$), which was reported to represent 46.0% (Cornelius et al., 1970), 44.4% (Mohiuddin and Zaidi, 1975), 14.6% (Ahmad et al., 1979), and 30.1–37.5 (Ahmed and Hudson, 1982) of the total fatty acids, was found in this study to represent only about 1% of total recovered fatty acids. Cyclopropenoid fatty acids (CPFA), which are found in seed lipids of the order Malvales that comprises several important sources food for man and animals (Berry, 1980), were also reported to occur in roselle seed oil. Thus sterculic and malvalic acids were respectively 2.9% and 1.3% of total recovered fatty acids (Ahmad et al., 1979). However, recent investigation by Ahmed and Hudson (1982) on the fatty acid composition of seed oil from different seed collections (or cultivars) respresenting different growing areas reported that dihydrosterculic and malvalic acids were in the range of 1.0–1.6% and 0.4–2.0% of the total fatty acids, respectively. On the other hand, cotton seed, which represents one of the important sources of edible oil and feed for livestock, was reported to contain about 1% cyclopropenoid fatty acids (Berry, 1980); therefore, a comparison was made between roselle seed oil, okra seed oil, and cotton seed oil by using the Halphen test (Association of Official Analytical Chemistry, 1975. The data revealed that roselle seed oil contains cyclopropenoid materials half the amount present in okra seed and one-fifth that found in cotton seed. Gossypol, the phenolic compound found in cotton seed and known to cause undesirable physiological effects on nonruminants (Pons, 1977), was found in roselle seed only as traces.

Other Components. Crude fiber, ash, total carbohydrates, starch, and minerals were quantitated; but no data in the literature were available for the purpose of comparison. However, if the results are discussed in relation to okra, the results suggest that in roselle seed, crude fiber, total carbohydrates, and ash content were 16.30, 96.64, and 5.19%, respectively, in comparison to 27.3, 23.5, and 4.7% for okra seed. Both roselle and okra seeds were contained only traces of free and total gossypol, which is well below the tolerence level for this compound reported in the literature (Pons, 1977). Finally, animal feeding studies for the nutritional and safety of roselle seeds are in progress, and the preliminary results of this survey have been reported recently (Farjou et al., 1983).

Registry No. K, 7440-09-7; Na, 7440-23-5; Mg, 7439-95-4; Ca, 7440-70-2; Fe, 7439-89-6; Zn, 7440-66-6; Sr, 7440-24-6; Ni, 7440-02-0; Mn, 7439-96-5; starch, 9005-25-8; gossypol, 303-45-7.

LITERATURE CITED

Ahmad, M. V.; Hussain, S. K.; Ahmad, I.; Osman, S. M. *J. Sci. Food Agric.* **1979**, *30*, 424.
Ahmed, A. K.; Hudson, B. J. F. *J. Sci. Food Agric.* **1982**, *33*, 1305.
Al-Wandawi, H. *J. Agric. Food Chem.* **1983**, *31*, 1355.
Association of Official Analytical Chemists. "Methods of Analysis"; AOAC: Washington, DC, 1975; p 506.
Beattie, W. R. 1937, USDA Leaflet No. 139.
Berry, S. K. *J. Food Sci. Technol.* **1980**, *17*, 224.
Cornelius, J. A.; Hammonds, T. W.; Leicester, J. B.; Ndabahwiji, J. K.; Rosie, D. A.; Shone, G. G. *J. Sci. Food Agric.* **1970**, *21*, 49.
Esselen, W. B.; Sammy, G. H. *Food Prod. Dev.* **1973**, *7*, 80.
Farjou, I. B.; Al-Wandawi, H.; Karim, K. H.; Al-Jawad, F. H. *Nutr. Rep. Int.* **1983**, in press.
Karakoltsidis, P. A.; Constantinides, S. M. *J. Agric. Food Chem.* **1975**, *23*, 1204.
Koch, F. C.; McMeekin, T. L. *J. Am. Chem. Soc.* **1924**, *46*, 2066.
Mohiuddin, M. M.; Zaidi, H. R. *Fette, Seifen, Amstrichm.* **1975**, *77*, 488.
Osborne, D. R.; Voogt, P. "The Analysis of Nutrients in Foods"; Academic Press: London and New York, 1978.
Pons, W. A., J. J. *Assoc. Off. Anal. Chem.* **1977**, *60*, 252.
Wilson, F. D.; Menzel, M. Y. *Econ. Bot* **1964**, *18*, 80.

Received for review October 19, 1983. Accepted January 3, 1984.

Antioxidative Effect of Nitrite in Cured Meat Products: Nitric Oxide-Iron Complexes of Low Molecular Weight

Joseph Kanner,* Stela Harel, Joseph Shagalovich, and Shura Berman

The activity of iron–nitric oxide complexes of low molecular weight toward lipid peroxidation was studied in a linoleate–β-carotene model system. Cysteine, and especially cysteine in the presence of iron ions, caused a rapid increase in β-carotene destruction. Compared with cysteine, the complex of cysteine–Fe–NO had no prooxidant effect. Addition of the cysteine–Fe–NO complex to the model containing cysteine–Fe^{2+} or hemin inhibited β-carotene oxidation by 77% and 86%, respectively. The activity of hemin–NO on lipid oxidation was studied and compared under the same experimental conditions and concentrations as that of hemin. In the range of tested concentrations (1.8–9.0 μM), hemin acted as a prooxidant toward carotene oxidation and hemin–NO as an antioxidant. The antioxidative effect of hemin–NO was maintained even in the presence of lipoxygenase and myoglobin. The possible role of nitric oxide on the stabilization of lipid peroxidation in muscle foods is discussed.

Lipid peroxidation in raw muscle food is catalyzed by both a nonenzymatic and an enzymatic process. Heme proteins and non heme iron complexes have been implicated as prooxidants in meat lipid peroxidation (Tappel, 1962; Liu, 1970; Sato and Hegarty, 1971; Koizumi et al., 1976; Pearson et al., 1977; Igene et al., 1979). It was discovered more recently that muscle tissue of both warm- and cold-blooded animals contains a membrane-associated enzyme that catalyzed lipid peroxidation, the microsomal system required for the activation NADPH or NADH and iron complexed to ADP (McDonald et al., 1979). In cooked meat products non-heme iron of low molecular weight compounds are the main prooxidants (Sato and Hegarty, 1971; Igene et al., 1979).

Agricultural Research Organization, The Volcani Center, Bet Dagan 50250, Israel.

0021-8561/84/1432-0512$01.50/0

Inhibition of lipid oxidation and "warmed over" flavor in cured meat by nitrite was shown by many researchers. This effect was measured by carbonyl compounds by using gas chromatography (Cross and Ziegler, 1965; Bailey and Swain, 1973; Fooladi et al., 1979; MacDonald et al., 1980b), the thiobarbituric acid (TBA) value (Hadden et al., 1975; Greene and Price, 1975; Kanner and Juven, 1980), and panelist's perceptions (MacNeil and Mast, 1973; Skjelkvale et al., 1974; MacDonald et al., 1980c) and was reviewed by Gray et al. (1981). The TBA value (Hadden et al., 1975; Greene and Price, 1975; Kanner and Juven, 1980) and panelist's perceptions (MacNeil and Mast, 1973; Skjelkvale et al., 1974; MacDonald et al., 1980c) were reviewed by Gray et al. (1981).

The nitrite added to meat products was found as protein-bound nitrite (Cassens et al., 1977; Olsman, 1977), nitric oxide–myoglobin, nitrosothiols, and nitrite, nitrate, and gaseous nitrogen compounds (Emi-Miwa et al., 1976; Woolford et al., 1976).

S-Nitrosocysteine, a compound that is generated during the curing of meat, has been shown to act as an antioxidant in both an aqueous linoleate model system (Kanner, 1979) and in ground cooked turkey meat (Kanner and Juven, 1980). The antioxidant activity of nitric oxide–myoglobin (MbNO) was also demonstrated in a linoleate or β-carotene–linoleate model system. The specific antioxidative activity of MbNO was maintained in the presence of prooxidants such as heme proteins and lipoxygenase (Kanner et al., 1980).

Our work was extended and this study focused on the effect of a low molecular weight non heme iron–nitric oxide complex (cysteine–Fe–NO) and of a heme–nitric oxide complex (hemin–NO) on lipid oxidation in a model system.

MATERIALS AND METHODS

L-Cysteine, hemin, Tween-20, and ferrous sulfate were purchased from Sigma Chemical Co., St. Louis, MO; sodium nitrite, ascorbic acid, and butylated hydroxytoluene (BHT) were from British Drug Houses, Ltd., Poole, England; linoleic acid was obtained from Fluka AG, Buchs, SG, Switzerland.

The cysteine–Fe–NO complex was prepared according to the method of McDonald et al. (1965). The cysteine solution (10^{-2} M) in acetate buffer, 0.1 M, pH 6.0, was reacted with ferrous sulfate (10^{-3} M) in the presence of N_2 bubbling for 15 min. Nitric oxide was then bubbled through the solution of 5 min, followed by another 15 min of nitrogen flushing.

The same procedure was applied to distilled water and to a solution of ferrous sulfate alone. Nitric oxide–hemin was prepared as described previously for nitric oxide–myoglobin (Kanner et al., 1980).

The separation of the nitric oxide–hemin from the low molecular weight compounds was done by a Sephadex G-25 column (Pharmacia Fine Chemicals AB, 20–80 μm), as described previously (Kanner et al., 1980); however, the hemin was eluted from the column by 0.05% Tween-20 in distilled water.

The assay of β-carotene–linoleate oxidizing activity was carried out according to a colorimetric method (Ben-Aziz et al., 1971; Kanner et al., 1976). Briefly, the technique consists of following the decrease in absorbance at 460 nm in the cuvette of a double-beam recording spectrophotometer. The test sample contained 1.5 mL of buffered carotene–linoleate mixture at pH 5.7, 0.1–0.4-mL active fractions, and distilled water to a final volume of 2.0 mL. Concentrations in the initial reaction mixture were as follows: β-carotene, 1.4×10^{-5} M; linoleate, 2×10^{-3} M; Tween-20, 0.05%; buffer acetate, pH 5.6, 0.1 M. The blank sample contained all the reagents except β-carotene.

The initial rate of decrease in absorbance was computed from a recorded graph and converted into the rate of carotene decrease in concentration (μM) or in nmol/min.

Table I. Effect of Cysteine–Fe–NO Complex on Linoleate–β-Carotene Oxidation

treatment	β-carotene oxidation, nmol/min
cysteine (5×10^{-4} M)	3.1 ± 0.8
Fe^{2+} (5×10^{-5} M)	9.3 ± 0.8
cysteine–Fe^{2+}	13.4 ± 0.6
cysteine–Fe–NO	0

Table II. Effect of Cysteine–Fe–NO Complex on Linoleate–β-Carotene Oxidation by Cysteine–Fe

treatment	β-carotene oxidation, nmol/min	rel act.
control (cysteine, 5×10^{-4} M, + Fe^{2+}, 5×10^{-5} M)	13.4 ± 0.6	1.00
cysteine (5×10^{-4} M) + cysteine (5×10^{-4} M) + Fe (5×10^{-5} M)	10.2 ± 0.7	0.76
Fe^{2+} (5×10^{-5} M) + cysteine (5×10^{-4} M) + Fe (5×10^{-5} M)	15.0 ± 0.9	1.11
Fe^{2+} + NO[a] + cysteine (5×10^{-4} M) + Fe (5×10^{-5} M)	14.3 ± 0.9	1.06
H_2O + NO[a] + cysteine (5×10^{-4} M) + Fe (5×10^{-5} M)	13.5 ± 0.4	1.07
cysteine–Fe–NO + cysteine (5×10^{-4} M) + Fe (5×10^{-5} M)	3.2 ± 0.5	0.23

[a] Prepared as cysteine–Fe–NO.

RESULTS AND DISCUSSION

Peroxidation of polyunsaturated fatty acids by transition metals and heme compounds have been studied extensively. The acceleration of lipid peroxidation due to a variety of metal ions and heme compounds is now a generally accepted phenomenon (Ingold, 1962; Tapel, 1962; Frankel, 1981).

The acceleration of lipid peroxidation by those compounds was described by a mechanism of hydroperoxide decomposition (Uri, 1956). According to O'Brien and Little (1969), Fe^{2+} catalyzed the decomposition of linoleic acid hydroperoxides (ROOH) about 10-fold faster than did Fe^{3+}. Several investigators (Wills, 1965; Barber, 1966; O'Brien and Little, 1969; Kanner et al., 1977) recognized that certain reductants, such as cysteine and ascorbate, especially at low concentration, induced an accelerated rate of ROOH decomposition, presumably by regenerating the active ferrous ion. More recently Gardner and Jursinic (1981) offered the cystein–$FeCl_3$ complex as a catalyst model for hydroperoxide degradation in biological tissues.

In our model system, cysteine at a concentration of 0.5 mM acts as a prooxidant, presumably by reducing contaminating transition metals to their lower valency state. Ferrous ions (50 μM), especially in the presence of cysteine (0.5 mM), increased significantly β-carotene-coupled oxidation. Compared with cysteine–Fe, the complex of cysteine–Fe–NO has no prooxidative effect (Table I). Addition of the cysteine–Fe–NO complex to the model containing the cysteine–ferrous ion inhibited β-carotene oxidation measured after 1 min by 77% (Table II).

The cysteine–Fe–NO complex activity also was tested in the presence of hemin. Cysteine alone inhibits hemin

Table III. Effect of Cysteine-Fe-NO Complex on Linoleate-β-Carotene Oxidation by Hemin

treatment	β-carotene oxidation, nmol/min	rel act.
control (hemin, 2×10^{-6} M)	6.2 ± 0.5	1.00
cysteine (5×10^{-4} M) + hemin (2×10^{-6} M)	4.0 ± 0.7	0.64
Fe^{2+} (5×10^{-5} M) + hemin (2×10^{-6} M)	6.8 ± 0.8	1.09
cysteine-Fe + hemin (2×10^{-6} M)	15.5 ± 0.8	2.50
cysteine-Fe-NO + hemin (2×10^{-6} M)	0.9 ± 0.7	0.14

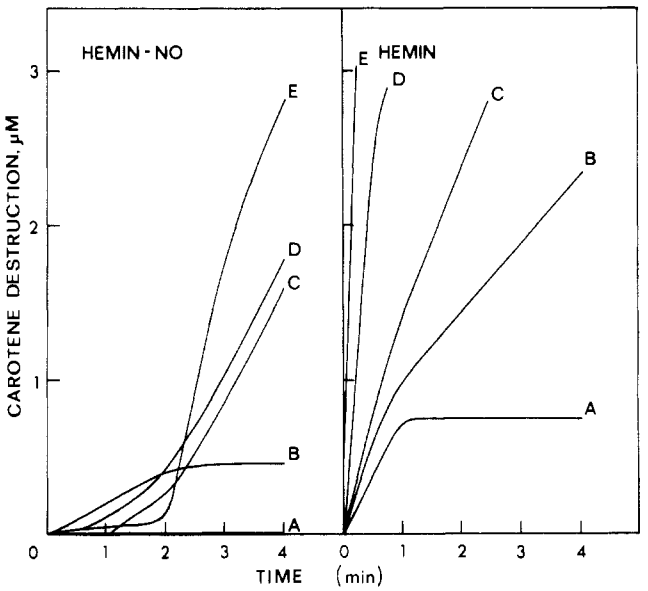

Figure 1. Effect of hemin and hemin-NO on carotene destruction: (A) 36 μM; (B) 9 μM; (C) 4.5 μM; (D) 3.6 μM; (E) 1.8 μM.

prooxidant activity only at a high concentration (0.5 mM). Nevertheless, compared with cysteine-Fe, which increases the prooxidative activity of hemin, the cysteine-Fe-NO complex decreases lipid peroxidation significantly (Table III).

The activity of hemin-NO on lipid peroxidation was studied and compared under the same experimental conditions and concentration as that of hemin, since it is known that heme compounds can act both as catalysts and as inhibitors of lipid oxidation (Kendrick and Watts, 1969; Hirano and Olcott, 1971; Kanner, 1974; Kanner et al., 1980).

In the range of tested concentrations (1.8–9.0 μM), hemin acted as a prooxidant toward carotene; however, at 36 μM, its activity is antioxidative after a short prooxidative effect. When compared with the activity of hemin, hemin-NO acts as an antioxidant (Figure 1). Hemin-NO activity also was tested in the presence of lipoxygenase (Figure 2). Its antioxidative activity was maintained even after this strong prooxidant was added, and it was highly effective even at 9 μM.

The activities of cysteine-Fe-NO and hemin-NO toward lipid peroxidation seem to be similar to those of *S*-nitrosocysteine and nitric oxide-myoglobin, which were previously reported (Kanner, 1979; Kanner et al., 1980).

It was proposed by several researchers (Zipser et al., 1964; Brown et al., 1963) that nitrite forms a complex with iron porphyrin in heat-denatured meat, thus preventing its prooxidative effect. This inactivation was attributed to the valence state (+2) of the iron. The stabilizing effect

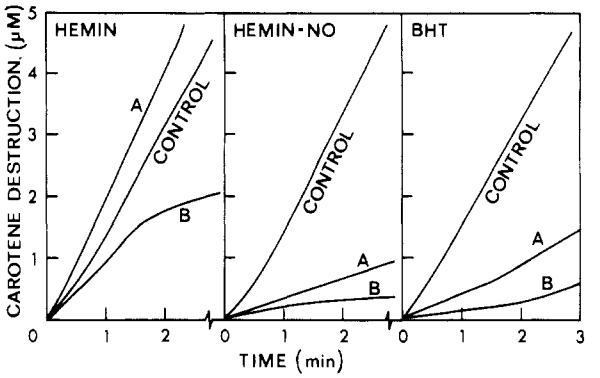

Figure 2. Inhibitory effect of hemin, hemin-NO, and BHT on carotene destruction by soybean lipoxygenase. Control: lipoxygenase alone. Hemin: (A) 9 μM; (B) 35 μM. Hemin-NO: (A) 9 μM; (B) 36 μM. BHT: (A) 5 μM; (B) 10 μM.

of cysteine-Fe-NO and hemin-NO may be attributed to the fact that Fe^{2+} is complexed by nitric oxide. A similar explanation was given by MacDonald et al. (1980A) to explain the inhibitory effect of nitrite toward the activity of iron ions and the Fe^{2+}-EDTA complex on lipid oxidation. However, this cannot explain the antioxidant effect of cysteine-Fe-NO and hemin-NO complexes in the presence of prooxidants.

The mechanism proposed by us for the antioxidative effect of cysteine-Fe-NO and hemin-NO fits our explanation on the activities of nitric oxide-myoglobin on lipid oxidation and is based on the quenching effect of nitroxide radicals to free radicals involved in lipid autooxidation (Harris and Olcott, 1966; Weill et al., 1968; Lin et al., 1974: Lin and Olcott, 1975).

Cysteine-Fe-NO and porphyrin-NO compounds gave electron spin resonance spectra similar to those of organic nitroxide free radicals, the unpaired electron being associated with the NO group (McDonald et al., 1965; Woolum et al., 1968; Yonetani et al., 1972). The nitroxide radical acts as an antioxidant by direct coupling with radicals (Thomas and Tolman, 1965; Brownlie and Ingold, 1967). Weill et al. (1968) showed that some stable synthetic nitroxides were quite effective antioxidants. Lin et al. (1974) and Lin and Olcott (1975) showed a similar effect with proline nitroxide and ethoxyquin-NO.

We believe that cysteine-Fe-NO and hemin-NO act in the early stages of the reaction to neutralize substrate-free radicals and thus inhibit oxidation. During this reaction, the molecule of NO interacts with free radicals and the hemin or cysteine-Fe remains in the system. The concentration of the lipid free radicals and the hydroperoxides that are then left in the system dictates its subsequent behavior. We found that nitric oxide-myoglobin at a low concentration results in a brief induction period, whereas metmyoglobin, which remains in the system, acts as a hydroperoxide decomposer and a prooxidant (Kanner et al., 1980). A similar effect was found by us when hemin interacted at a low concentration in the lipid model system.

The rapid termination in lipid peroxidation achieved by cysteine-Fe-NO and hemin-NO above a critical concentration fits the activities of nitric oxide-myoglobin (Kanner et al., 1980) and could be explained as the effect of simultaneous quenching of free radicals (eq 1) and decom-

$$\text{Cys-Fe}^{2+}\text{-NO}\cdot + \text{R}\cdot \rightarrow \text{RNO} + \text{Cys-Fe}^{2+} \quad (1)$$

$$\text{Cys-Fe}^{2+} + \text{ROOH} \rightarrow \text{RO}\cdot + {}^{-}\text{OH} + \text{Cys-Fe}^{3+} \quad (2)$$

$$\text{Cys-Fe}^{2+}\text{-NO}\cdot + \text{RO}\cdot \rightarrow \text{Cys-Fe}^{2+} + \text{RONO} \quad (3)$$

position of hydroperoxides (eq 2). The free radicals that

are formed during the decomposition of the hydroperoxides by the cysteine–Fe complex or heme compounds are quenched by nitrc oxide complexes that are in the system (eq 3) and thus inhibit the initiation of a new chain reaction. (In eq 1–3, R = alkyl radical, RO = alkoxy radical, and ROOH = hydroperoxide.)

Most recently it was found that nitrite inhbition of clostridium botulinum seems to occur by the inactivation of iron–sulfur enzymes. The iron–sulfur proteins that react wih added nitrite form iron–nitric oxide complexes. These complexes exhibited an electron spin resonance signal characteristic to those of nitric oxide–iron containing proteins, peptides, and chelate complexes (Reddy et al., 1983).

Non heme iron and low molecular weight iron compounds were implicated as the main prooxidants in cooked meat products (Sato and Hegarty 1971; Igene et al., 1979).

Most of the iron in animal tissue occurs in heme proteins, but a number of substances contain non-heme iron (Aisen, 1977). Iron protein complexes as ferritin and transferrin, and many enzymes formed most of those non heme iron compounds. Wills (1966) has demonstrated that when non-heme iron is released from ferritin, it becomes an active catalyst of lipid peroxidation. Iron overload, which can be due to a number of causes, was reported to increase in vivo lipid peroxidation (Graziano, 1976).

We concluded that the antioxidative effect generated by nitrite in cured muscle foods arises by the formation of nitric oxide. Nitric oxide, a well-known complexant, interacts with metals and especially with iron heme and non-heme compounds. The complexing of iron by NO, as was shown in model systems, not only eliminates its prooxidative effect toward lipid peroxidation but also converts its activities from catalytic to antioxidative.

This inversion of catalysts to inhibitors of lipid peroxidation seems to contribute greatly to the preventive effect of nitrite on the development of off-flavors in cured muscle foods.

Registry No. Linoleic acid, 60-33-3; β-carotene, 7235-40-7; cysteine, 52-90-4; hemin, 16009-13-5; hemin–NO, 54854-55-6; lipoxygenase, 9029-60-1; nitric oxide, 10102-43-9; iron(II), 15438-31-0.

LITERATURE CITED

Aisen, P. *Ciba Found. Symp.* **1977**, *51*, 1–14.
Bailey, M. E.; Swain, J. W. *Proc. Meat Ind. Res. Conf.* **1973**, 29.
Barber, A. A. *Lipids* **1966**, *1*, 146.
Ben-Aziz, A.; Grossman, S.; Ascarelli, I.; Budowski, P. *Phytochemistry* **1971**, *10*, 1445.
Brown, W. D.; Harris, L. S.; Olcott, H. S. *Arch. Biochem. Biophys.* **1963**, *101*, 14.
Brownlie, J. T.; Ingold, K. C. *Can. J. Chem.* **1967**, *45*, 2427.
Cassens, R. G.; Woolford, G.; Lee, S. H.; Goutefongea, R. *Proc. Int. Symp. Nitrite Meat Prod. 2nd, 1976* **1977**, 95.
Cross, C. K.; Ziegler, J. *J. Food Sci.* **1965**, *30*, 610.
Emi-Miwa, M.; Okitani, A.; Fujimaki, M. *Agric. Biol. Chem.* **1976**, *40*, 1387.
Fooladi, M. H.; Pearson, A. M.; Coleman, T. H.; Merkel, R. A. *Food Chem.* **1979**, *4*, 283.
Frankel, E. M. *Prog. Lipid Res.* **1981**, *19*, 1.
Gardner, H. W.; Jursinic, P. A. *Biochim. Biophys. Acta* **1981**, *665*, 100.
Gary, J. I.; MacDonald, B.; Pearson, A. M.; Morton, I. D. *J. Food Prot.* **1981**, *44*, 302.
Graziano, J. H. In "Iron Metabolism and Thalassemia"; Bergsma, D.; Cerami, A.; Peterson, C. M.; Graziano, J. H., Eds.; Alan R. Liss: New York, 1976; Vol. XII, p 135.
Greene, B. E.; Price, L. G. *J. Agric. Food Chem.* **1975**, *23*, 164.
Hadden, J. P.; Ockerman, H. W.; Cahill, V. R.; Parret, N. A.; Borton, B. J. *J. Food Sci.* **1975**, *40*, 626.
Harris, L. A.; Olcott, H. S. *J. Am. Oil Chem. Soc.* **1966**, *43*, 11.
Hirano, Y.; Olcott, H. S. *J. Am. Oil Chem. Soc.* **1971**, *48*, 523.
Igene, J. O.; King, J. A.; Pearson, A. M.; Gray, J. I. *J. Agric. Food Chem.* **1979**, *27*, 838.
Ingold, K. U. In "Symposium on Foods: Lipids and their Oxidation"; Schultz, H. W.; Day, E. A.; Sinnhuber, R. O., Eds.; Avi Publishing Co.: Westport, CT, 1962; p 93.
Kanner, J. Ph.D. Thesis, Hebrew University of Jerusalem, Israel (Hebrew, with English summary), 1974.
Kanner, J. *J. Am. Oil Chem. Soc.* **1979**, *56*, 74.
Kanner, J.; Ben-Gera, I.; Berman, S. *Lipids* **1980**, *15*, 944.
Kanner, J.; Juven, B. J. *J. Food Sci.* **1980**, *45*, 1105.
Kanner, J.; Mendel, H.; Budowski, P. *J. Food Sci.* **1976**, *41*, 183.
Kanner, J.; Mendel, H.; Budowski, P. *J. Food Sci.* **1977**, *42*, 60.
Kendrick, J.; Watts, B. M. *Lipids* **1969**, *4*, 454.
Koizumi, C.; Wada, S.; Nonaka, J. *Bull. Jpn. Soc. Sci. Fish.* **1976**, *42*, 497.
Lin, J. S.; Olcott, H. S. *J. Agric. Food Chem.* **1975**, *23*, 798.
Lin, J. S.; Smith, V.; Olcott, H. S. *J. Agric. Food Chem.* **1974**, *22*, 682.
Liu, H. P. *J. Food Sci.* **1970**, *35*, 593.
MacDonald, B.; Gray, J. I.; Gibbons, L. N. *J. Food Sci.* **1980a**, *45*, 893.
MacDonald, B.; Gray, J. I.; Kakuda, Y.; Lee, M. L. *J. Food Sci.* **1980b**, *45*, 889.
MacDonald, B.; Gray, J. I.; Stanley, D. W.; Usborne, W. R. *J. Food Sci.* **1980c**, *45*, 885.
MacNeil, J. H.; Mast, M. G. *Food Prod. Dev.* **1973**, *7*, 36.
McDonald, C. C.; Phillips, W. D.; Mower, H. F. *J. Am. Chem. Soc.* **1965**, *87*, 3319.
McDonald, R. E.; Kelleher, S. O.; Hultin, H. O. *J. Food Biochem.* **1979**, *3*, 125.
O'Brien, P. J.; Little, C. *Can. J. Biochem.* **1969**, *47*, 485.
Olsman, W. J. *Proc. Int. Symp. Nitrite Meat Prod., 2nd, 1976* **1977**, 101.
Pearson, A. M.; Love, J. D.; Shorland, F. B. *Adv. Food Res.* **1977**, *23*, 1.
Reddy, D.; Lancaster, J. R., Jr.; Cornforth, D. P. *Science (Washington, D.C.)* **1983**, *221*, 769.
Sato, K.; Hegarty, G. R. *J. Food Sci.* **1971**, *36*, 1096.
Skjelkvale, R.; Tjaberg, T. B.; Valland, M. *J. Food Sci.* **1974**, *39*, 520.
Tappel, A. L. In "Symposium on Foods: Lipids and their Oxidation"; Schultz, H. W.; Day, E. A.; Sinnhuber, R. O.; Avi Publishing Co.: Westport, CT, 1962; p 122.
Thomas, J. R.; Tolman, C. A. *J. Am. Oil Chem. Soc.* **1962**, *84*, 2930.
Uri, N. *Nature (London)* **1956**, *177*, 1177.
Weill, J. T.; Vender Veen, J.; Olcott, H. S. *Nature (London)* **1968**, *219*, 168.
Wills, E. D. *Biochim. Biophys. Acta* **1965**, *98*, 238.
Wills, E. D. *Biochem. J.* **1966**, *99*, 667.
Woolford, G.; Cassens, R. G.; Greaser, M. L.; Sebranek, J. G. *J. Food Sci.* **1976**, *41*, 585.
Woolum, J. C.; Tiezzi, E.; Commoner, B. *Biochim. Biophys. Acta* **1968**, *160*, 311.
Yonetani, T. H.; Yamamoto, J. E.; Erman, J. S.; Leigh, J.; Reed, G. H. *J. Biol. Chem.* **1972**, *247*, 2447.
Zipser, M. W.; Kwon, T. W.; Watts, B. M. *J. Agric. Food Chem.* **1964**, *12*, 105.

Received for review February 24, 1983. Revised manuscript received October 3, 1983. Accepted January 5, 1984. Contribution No. 590-E, 1982 series, from the Agricultural Research Organization.