Effects of Dietary Fat and Vitamin E on the Lipid Composition and Stability of Veal during Frozen Storage

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Calves were fed either coconut or corn oil as milk fat replacers with or without vitamin E as *d*-α-tocopheryl acetate until slaughtered at 9 weeks of age. The corn oil fed animals generally showed poor growth rates. The lipid composition of the depot fats was altered by the dietary oils; corn oil increased linoleic acid levels and coconut oil raised lauric and myristic acid contents. Vitamin E supplementation enhanced tissue levels and, consistent with chain extension, increased the stearic acid content at the expense of lauric, myristic, and palmitic acids. Supplemental vitamin E retarded lipid oxidation of longissimus dorsi (l. dorsi) tissues during frozen storage, but the reverse was generally true in omental and perinephric tissues. Thus, inhibition of autoxidation by vitamin E was not clearly evident in the more complicated intact tissue systems as has been demonstrated for extracted lipids.

Dietary fats fed to ruminants have little influence on the composition of the tissue fats (Beard et al., 1935), in contrast to the observations made with nonruminants (Hilditch and Williams, 1964). The explanation for the unresponsiveness of ruminant depot fats to dietary fats is the capacity of the rumen microorganisms to hydrogenate ingested fats, which was first demonstrated by Reiser (1951), showing that linolenic acid was hydrogenated to linoleic acid. Shorland et al. (1955) showed that even though linolenic acid, the main fatty acid of pasture lipids, is converted mainly to oleic and stearic acids, minor amounts of linoleic and trans and positional isomers of oleic and of linoleic acid are also formed. Alteration of ruminant depot fat necessitates either bypassing the rumen (Ogilvie et al., 1961), protecting the dietary fat against action of the rumen microorganisms by microencapsulation (Scott et al., 1971), or feeding the dietary fat prior to weaning before the rumen develops the capacity to hydrogenate dietary fat (Hoflund et al., 1956). In the present work, various dietary fats were fed in milk replacers to unweaned calves from 1 to 9 weeks of age, when the animals were slaughtered.

Deteriorative changes in frozen carcasses at −18 °C or below occur slowly. Wright (1921) stated that oxidative rancidity in lamb was not a problem even after 18 months of frozen storage. Subsequently, Privett et al. (1955) found that cuts from beef, lamb, pork, and turkey stored for up to 2.5 years at −15.5 to −17.8 °C showed the characteristic brown "met-pigment" and were unacceptable because of oxidative rancidity, but there was no evidence for loss of essential fatty acids. Interestingly, lamb showed relatively low peroxide values of 2.0–5.4. In contrast to fresh intact meat, cooked meat and uncooked ground meat becomes rancid within 48 h at 4 °C (Greene, 1969; Sato and Hegarty, 1971; Wilson et al., 1976). This phenomenon has been described by Tims and Watts (1958) as "warmed-over" flavor and was reviewed by Pearson et al. (1977).

The present work was designed to explore the stability of intact meat during frozen storage—a matter of particular relevance to long-distance transport by ship. There have been many attempts to improve the stability of the lipids of meat animals by inclusion of dietary vitamin E (Pearson et al., 1977). Perhaps the most notable success was reported by Ellis et al. (1974), in which eight Holstein bull calves were fed high levels of α-tocopheryl acetate. Four animals were fed normal and four were fed filled milk containing polyunsaturated fatty acids until weaned at 10 weeks. Thereafter until slaughter, the calves were fed a starter mixture, with two from each group receiving "protected" safflower oil and two "unprotected" safflower oil. The protected safflower oil resulted in up to 7-fold higher levels of 18:2 and vitamin E in the perinephric and round fats than was found in commercial beef fat. The increased tocopheryl acetate resulted in longer induction periods, even in the presence of elevated 18:2 levels, than was found for commercial veal fat.

Although Ellis et al. (1974) studied the stability of veal lipids containing high levels of linoleic acid, the present work was concerned mainly with stability of the lipids during frozen storage. Thus, the present investigation not only examined the effect of vitamin E on frozen storage stability but also examined the effect of saturated oil (coconut oil) in comparison to that of polyunsaturated oil (corn oil). As described later herein, both vitamin E supplementation and the type of dietary oil markedly influenced the stability of the lipids, but the effects differed in various depots. Two preliminary communications (Igene et al., 1976; Shorland, 1976) have reported some of the results described in greater detail herein.

EXPERIMENTAL SECTION

Feeding Experiment. A total of 16 Holstein bull calves were allowed colostrum ad libitum for the first 3 days following birth and then given 1.8 kg of whole milk daily per calf through 7 days of age, at which time they were randomly alloted into four groups. They were then fed a milker replacer containing 15% fat and 25% protein (dry weight basis) by bucket–nipple for 57 days, at which time they were slaughtered. In groups 1 and 2, the source of fat in the milk replacer was coconut oil, and for groups 3

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0021-8561/81/1429-0863$01.25/0

and 4, corn oil. The calves in groups 1 and 3 were supplemented with 500 mg of d-α-tocopheryl acetate daily, which was dissolved in 250 mL of hydrogenated vegetable oil (containing negligible amounts of linoleic acid) and mixed with the diluted milk replacer once a day just prior to feeding. During the first week, the milk replacer (0.22 kg calf^{-1} day^{-1}) was diluted with 1.89 L of warm water and fed in two equal feedings at morning and night. The amount of milk replacer was increased by increments of 0.07, 0.11, 0.18, 0.18, 0.18, 0.18, and 0.22 kg/day at the beginning of weeks 2, 3, 4, 5, 6, 7, and 8, respectively. The amount of warm water used for diluting the milk replacer was increased weekly from 1.89 L calf^{-1} day during the first week to 2.17, 2.36, 3.31, 3.78, 4.25, 5.20, and 5.68 L calf^{-1} day^{-1}.

The milk replacer ration on a percentage of dry matter basis was comprised of the following: dried whey, 49.2; nonfat dried milk, 25.0; dried lactalbumin, 10.0; coconut or corn oil, 15.0; calcium carbonate, 0.625; a vitamin–mineral premixture, 0.175. The vitamin–mineral premixture provided the following nutrients per kilogram: vitamin A, 101 750 IU; vitamin D_3, 11 000 IU; riboflavin, 8.8 mg; pantothenic acid, 11 mg; niacin, 40 mg; vitamin B_{12}, 15.4 μg; thiamine, 4.4 mg; folic acid, 2.75 mg; choline, 187 mg; magnesium, 310 mg.

Slaughtering, Tissue Collection, and Storage. The calves were held overnight after the last feeding and slaughtered the following morning. Samples of the l. dorsi, omental, and perenephric tissues were removed immediately after slaughtering, wrapped in freezer paper, and either analyzed at once or frozen and stored at −18 °C. Samples were analyzed after 0, 1, 3, and 6 months of frozen storage.

Preparation of Lipids. The fatty tissue were rendered in a Thomas Teflon pestle tissue grinder (Thomas Co., Philadelphia, PA). The tissues (~10 g) were weighed into the grinder tube, heated on the steam bath (5 min), and then ground with the addition of several volumes of chloroform–methanol (2:1 v/v). The extract was decanted and the process repeated twice. The combined extracts were dried over anhydrous sodium sulfate and filtered through glass wool, and the precipitate was washed with chloroform to remove the last traces of lipid. The solvent was evaporated on the steam bath in a tared beaker and brought to a constant weight in a vacuum at 50 °C.

The total lipids from the muscle tissue (~100 g) were removed by the Folch et al. (1957) technique, three extractions being used. The chloroform layer from the combined extracts after washing twice with water (10% v/v) was evaporated in a rotary evaporator (Buchi, Switzerland) to small volume and transferred quantitatively to a tared 100-mL round-bottom flask for reevaporation to constant weight in vacuo at 40 °C. The methanol layers were also evaporated to check that there were no losses of methanol-soluble lipids. The muscle lipids were separated into neutral lipids (hereafter denoted triglycerides, though containing small amounts of unsaponifiable matter and free fatty acids) and phospholipids by the method of Choudhury et al. (1960).

Preparation and Analysis of Methyl Esters. Methyl esters were prepared by the method of Morrison and Smith (1964) using a boron trifluoride–methanol reagent. The chromatographic (GLC) analyses were performed on a Beckman GC-4 gas chromatograph equipped with a hydrogen flame detector. The glass column (180 × 0.2 cm i.d.) was packed with 10% (w/w) diethylene glycol succinate (DEGS) on 100–120-mesh Supelcoport (Supelco, Inc.). The column had previously been cleaned, silanized, and packed under suction. The helium carrier gas was adjusted to a 40 mL/min flow rate, the flow rates of oxygen and hydrogen being respectively 30 and 300 mL/min. The column temperature was 185 °C with an injection port temperature of 215 °C. Runs were made at two levels of sensitivity to include all components on the chart. At first, sensitivity was adjusted so that the highest peak occupied about the maximum height allowed on the chart. The second level of sensitivity was at 5 or 10 times the first to more effectively evaluate the minor components. Peaks were identified by comparing their retention times with those of standard mixtures and equivalent carbon numbers (ECN) with those of Hofstetter et al. (1965).

Table I. Means and Standard Deviations of Live Weights at Slaughter, Carcass Weights, and Percentage Yields of Veal Calves[a]

group	treatment	live wt, kg	carcass wt, kg	carcass yields, %
1	coconut oil + vitamin E	73.5 ± 12.2	45.4 ± 9.5	62 ± 3
2	coconut oil; no vitamin E[b]	73.0 ± 7.3	43.1 ± 6.8	59 ± 3
3	corn oil + vitamin E	63.5 ± 21.5	34.9 ± 17.7	55 ± 7
4	corn oil; no vitamin E[b]	65.3 ± 12.2	40.3 ± 10.0	62 ± 5

[a] Mean of four animals. [b] Vitamin E was not added, but the oils, especially corn oil, contained vitamin E.

Vitamin E Content of Tissues. The tocopheryl contents of the tissues were determined by the method of Erickson and Dunkley (1964).

Thiobarbituric Acid (TBA) Values. TBA values were determined by the procedure of Tarladgis et al. (1960), expressing results as milligrams of malonaldehyde per 1000 g of tissue.

RESULTS AND DISCUSSION

Details on the performance of the calves used in this work are given in Table I. The great variations in the live weights at slaughter, with means ranging from 63.5 kg in group 3 to 73.5 kg in group 1, appear to be related to the unthriftiness of some animals, especially those on the corn oil diet. Seven of sixteen original animals died during early phases of the experiment and had to be replaced. Similar results have been reported by Adams et al. (1959) for calves fed rations containing highly unsaturated oils and ascribed by them to the development of rancidity. In contrast, calves fed polyunsaturated milk from cows receiving protected polyunsaturated oils grew normally (Wrenn et al., 1973).

The relatively poor growth rates of the corn oil fed calves were accompanied by low fat levels in the perinephric and omental fatty tissues (Table II). Two perinephric and three omental tissues from the corn oil fed animals contained less than 6% fat, which can be compared to a mean fat content of 88% for the perinephric fats of three mature beef animals reported by Hilditch and Longenecker (1937).

In contrast to the fatty tissues, the mean lipid contents of the l. dorsi muscle were not significantly affected by the corn oil diet. Overall, the mean triglyceride contents of the l. dorsi tissues fell within the range of 0.63–1.01%, which is comparable to the mean of 0.67% quoted by Wrenn et al. (1973) for intramuscular tissue. The phospholipids (0.75–1.01%) in the l. dorsi tissues were relatively constant and similar to results of other workers (Link et al., 1967; Body and Shorland, 1974).

Table II. Mean Percent Lipid Levels[a] and Standard Deviations of Tissues of Veal Calves

group	treatment	l. dorsi		omental, total lipid	perinephric, total lipid
		total lipid	phospholipid		
1	coconut oil + vitamin E	1.66 ± 0.39	0.84 ± 0.09	66.0 ± 4.4	73.4 ± 17.4
2	coconut oil; no vitamin E[b]	1.44 ± 0.20	0.76 ± 0.09	56.9 ± 8.0	60.5 ± 14.7
3	corn oil + vitamin E	1.76 ± 1.10	0.75 ± 0.07	26.0 ± 30.8	21.4 ± 27.1
4	corn oil; no vitamin E[b]	1.45 ± 0.26	0.82 ± 0.08	42.6 ± 26.8	68.6 ± 18.7

[a] Mean of four animals. [b] Vitamin E was not added, but the dietary oils, especially corn oil, contained natural vitamin E.

Fatty Acid Composition: Triglycerides and Phospholipids. Triglyceride fatty acids ranged from 10:0 to 18:3 (Table III). The appearance of small amounts of unidentified material of equivalent carbon number (ECN) 21:0 in the l. dorsi of corn oil groups 3 and 4 is unexplained as are the variable but small amounts of linolenic acid in these groups. The absence of branched chain and of trans-unsaturated fatty acids, normally present in ruminant lipids (Shorland, 1962), is in accord with the nondevelopment of rumen function.

The phospholipid fatty acids (Table IV) ranged from 12:0 to 22:5ω6 and included traces of unidentified fatty acids with ECN's approximating 13:0 and 19:0, respectively. The presence in the dietary fats of no more than traces of linolenic acid (18:3ω3) precluded the occurrence of substantial levels of ω3 polyunsaturated fatty acids as found in pasture-fed ruminants (Body and Shorland, 1974); nevertheless, some 20:5ω3 was detected in the phospholipids in the present work.

The wide range of polyunsaturated C_{20} and C_{22} acids in the phospholipids and their absence in the triglycerides agrees with the results of other workers (Ansell and Hawthorne, 1964; Shorland, 1962). Preferential uptake of dietary linoleic acid by the phospholipids compared with the triglycerides agrees with previous work on horse (Shorland et al., 1952) and rabbit (Futter and Shorland, 1957) lipids.

Effect of Dietary Oils. The fatty acid composition of the triglycerides of depot fats of nonruminants (Shorland, 1962) and of calves prior to weaning (Hoflund et al., 1956) reflects that of the dietary fat. However, lower saturated fatty acids can be elongated prior to incorporation into the depot fat (Shorland and Czochanska, 1970). In the present work dietary coconut oil containing the following percentages of component fatty acids showed a ratio of lauric (12:0)/myristic (14:0) acids of 3.4:1 compared with 0.23–0.29:1 in the depot triglycerides (Table III): 8:00, 6.3; 10:0, 5.1; 12:0, 50.5; 14:0, 14.8; 16:0, 13.0; 18:0, 3.0; 18:1, 4.5; 18:2, 2.8. These observations are consistent with the endogenous conversion of lauric to myristic acid. Although chain extensions are characteristic of mammalian tissues, they are not found in birds (Shorland and Czochanska, 1970) since the specific enzymes for chain extension are not present (Stannard, 1975).

The fatty acids of the dietary corn oil were made up largely (50.6%) of linoleic acid (18:2ω6) along with the following percentages of other fatty acids: 12:0, 0.4; 14:0, 0.7; 16:0, 14.7; 18:0, 3.6; 16:1, 0.4; 18:1, 29.4; 18:3, 0.2. As shown in Table III, the main dietary fatty acid (linoleic acid) was strongly reflected in the depot fat.

In response to the dietary oils (Table V), the triglycerides showed highly significant differences ($P < 0.01$) in the composition of the major fatty acids, including 12:0, 14:0, 16:0, 18:0, 18:1ω9, and 18:2ω6. The outstanding differences were the 4-fold increase in myristic acid in the coconut oil groups (1 and 2) compared with the corn oil groups (3 and 4) and the 5-fold increase in linoleic acid in the corn oil groups (3 and 4) compared with the coconut oil groups (1 and 2).

Wrenn et al. (1973) found that Holstein bull calves fed milk high in linoleic acid for 10 weeks followed by protected safflower oil until slaughter at 18 weeks produced similar levels of linoleic acid in different depot triglycerides. The present work also shows uniform but higher levels of linoleic acid (~28% compared with ~13%) in different depot triglycerides of the calves fed corn oil. These results differ from those of Cook et al. (1972) for Friesian steers, where 9-month-old animals were fed protected safflower oil for 2 months before slaughter. The linoleic acid content of the triglycerides decreased from 35% in perinephric fat to 30% for the omental fat and to 25% in the intramuscular fat. Duncan and Garton (1967) suggested that the internal tissues preferentially assimilate the fatty acids of chylomicron triglycerides, which in ruminants normally have a high content of stearic acid. Although the theory agrees with the high content of stearic acid in the perinephric fat compared with that of the external fat, it does not explain the present results for linoleic acid.

The extent to which dietary linoleic acid can affect its level in ruminant depot fats appears to diminish with age. Thus, Wrenn et al. (1976) fed protected safflower oil to Holstein cows at 1 month postcalving for 2 years at levels approaching those used for the bull calves mentioned earlier (Wrenn et al., 1973). The levels of linoleic acid in the various depot triglycerides ranged from 4.4 to 5.0% without showing preferential assimilation of linoleic acid by the perinephric fat. The rate of change in fatty acid composition for turkeys (Salmon, 1976) after changing the source of dietary fat also decreased with age.

The phospholipids (Table V) also responded strongly to the dietary oils, with the differences in levels of most fatty acids between the corn oil and coconut oil groups being significant ($P < 0.01$). However, the differences were generally less extreme than those observed for the triglycerides. A notable feature of the composition of phospholipids was the high level of linoleic acid (24%) in the coconut oil groups (1 and 2) despite the low level in the diet. The high dietary linoleic acid levels for groups 3 and 4 raised the linoleic acid content of the phospholipids to ~42% as compared to ~28% in the corresponding triglycerides. The linoleic acid levels corresponded closely to those found by Cook et al. (1972) for the muscle phospholipids of Friesian steers in the control and protected safflower oil fed groups, respectively. The tendency to maintain high levels of linoleic acid in the relative absence of dietary linoleic acid has been previously noted in cholesterol esters of plasma lipids of dairy cows (Cook et al., 1972) and of humans (Shorland et al., 1969). Although the levels of linoleic acid in the cow plasma cholesterol esters increased as in muscle phospholipids on feeding linoleic acid as protected safflower oil (Cook et al., 1972), the levels of linoleic acid in plasma cholesterol esters of humans were unaffected by dietary changes in this acid (Shorland et al., 1969).

No evidence was found for elevation of arachidonic (20:4ω6) or other C_{20} and C_{22} acids following increased levels of dietary linoleic acid, suggesting that modifications of C_{20} and C_{22} polyunsaturated fatty acids in muscle

Table III. Means[a] and Standard Deviations of the Fatty Acids of the Triglycerides, Expressed as Percent Area of Methyl Ester Peak of the Total Area of Methyl Ester Peaks

fatty acids[c]	muscle				omental				perinephric			
	coconut oil		corn oil		coconut oil		corn oil		coconut oil		corn oil	
	+vitamin E (1)	no vitamin E[b] (2)	+vitamin E (3)	no vitamin E[b] (4)	+vitamin E (1)	no vitamin E[b] (2)	+vitamin E (3)	no vitamin E[b] (4)	+vitamin E	no vitamin E[b]	+vitamin E	no vitamin E[b]
10:0	trace	trace	1.6 ± 3.0	0.4 ± 0.72	0.1 ± 0.08	0.1 ± 0.14	trace	trace	0.1 ± 0.11	0.1 ± 0.10	trace	trace
12:0	5.1 ± 1.19	6.7 ± 1.74	1.0 ± 0.35	1.0 ± 0.45	6.2 ± 1.11	6.1 ± 1.65	1.2 ± 0.32	1.1 ± 0.51	5.6 ± 0.96	8.7 ± 0.90	0.8 ± 0.81	1.2 ± 0.24
14:0	18.8 ± 2.85	24.6 ± 1.70	5.0 ± 1.51	5.7 ± 1.80	22.1 ± 2.53	20.8 ± 1.38	5.0 ± 1.57	6.7 ± 2.14	21.8 ± 1.68	23.3 ± 1.27	4.4 ± 0.93	5.5 ± 0.57
15:0	1.8 ± 0.48	1.4 ± 0.49	0.2 ± 0.19	0.5 ± 0.35	1.1 ± 0.18	1.8 ± 0.42	0.3 ± 0.23	0.6 ± 0.28	1.0 ± 0.50	1.0 ± 0.06	0.1 ± 0.03	0.3 ± 0.23
15:1ω6	0.4 ± 0.46	0.6 ± 0.81	2.5 ± 2.64	2.2 ± 3.97								
16:0	27.9 ± 3.40	29.9 ± 0.48	16.8 ± 2.00	19.2 ± 2.73	30.3 ± 3.64	30.7 ± 3.71	20.1 ± 2.05	23.6 ± 2.98	28.2 ± 1.61	27.9 ± 1.46	19.2 ± 2.48	19.3 ± 3.40
16:1ω7	2.8 ± 0.77	2.1 ± 0.43	1.7 ± 0.36	2.5 ± 0.28	1.7 ± 0.45	2.8 ± 0.87	1.5 ± 0.55	2.0 ± 0.69	1.7 ± 0.20	1.8 ± 0.79	1.4 ± 0.35	1.6 ± 0.55
17:0	0.1 ± 0.08	trace	0.1 ± 0.17	0.2 ± 0.14	trace	trace	0.2 ± 0.14	0.1 ± 0.06	0.1 ± 0.10	0.1 ± 0.10	0.1 ± 0.08	0.1 ± 0.08
17:1ω8	trace	trace	0.4 ± 0.50	trace								
18:0	8.8 ± 0.44	7.3 ± 1.16	9.7 ± 0.32	8.8 ± 2.00	10.3 ± 0.82	9.6 ± 2.42	13.0 ± 2.30	10.3 ± 1.47	10.9 ± 1.14	9.7 ± 1.20	15.2 ± 2.29	11.9 ± 3.01
18:1ω9	26.8 ± 4.04	22.3 ± 1.62	29.3 ± 3.94	29.8 ± 2.52	22.5 ± 2.75	23.1 ± 2.60	31.2 ± 3.89	29.3 ± 2.20	24.3 ± 4.33	22.4 ± 1.90	33.4 ± 3.73	30.6 ± 1.23
18:2ω6	7.5 ± 2.43	5.1 ± 1.70	27.8 ± 5.78	28.2 ± 2.90	5.7 ± 2.00	5.0 ± 2.01	27.5 ± 4.39	26.1 ± 5.39	6.3 ± 2.29	5.0 ± 2.93	25.1 ± 3.52	28.8 ± 2.22
18:3ω3			2.9 ± 4.21	0.7 ± 1.13				0.2 ± 0.06			0.3 ± 0.48	0.7 ± 1.21
other			1.0 ± 2.00	0.8 ± 1.24								

[a] Means are values for four animals. [b] Vitamin E was not added to these diets, but the oils added, especially corn oil, had natural vitamin E. [c] Numbers before the colon indicate the number of carbons in the fatty acid; numbers after the colon indicate the number of double bonds. ω represents the commencement of the double bonds from the terminal methyl group of the molecule.

phospholipids are not readily achievable. Cook et al. (1972) also reported that linoleic acid supplementation by feeding safflower oil had little effect on arachidonic acid levels in muscle phospholipids. The higher levels ($P < 0.01$) of 20:3ω9 in phospholipids of the coconut oil groups are consistent with essential fatty acid deprivation as discussed by Noble et al. (1971) in relation to the phospholipids of newborn lambs. However, the theory that higher levels of 20:3ω9 are formed from oleic acid to replace a deficiency in the levels of other C_{20} and C_{22} polyenoic acids is not supported by the present work.

Effect of Dietary Vitamin E Levels. Dietary vitamin E supplementation (Table VI) was associated with increased levels of stearic acid ($P < 0.01$) in the triglyceride fractions and with reduced levels of lauric, myristic ($P < 0.01$), and hexadecenoic (16:1ω7) ($P < 0.05$) acids. The depression of linoleic acid in tissues of calves with lowered vitamin E intake reported by Poukka and Oksanen (1972) was not found in the present work nor by Bunyan et al. (1967), who concluded that vitamin E deficiency had little effect on polyunsaturated fatty acid levels in tissues of chickens and rats.

Vitamin E also raised the stearic acid content of the phospholipids ($P < 0.10$) with no other apparent changes. In view of the conversion of stearic to oleic acid in mammals formally established by Glascock and Renius (1956), the highly significant elevation of stearic acid levels by vitamin E supplementation might be due to the lowered activity of Δ^9-desaturase. However, there was no evidence of an interrelationship between the levels of stearic and oleic acid, metabolism of which may follow largely different routes (Gerson et al., 1968). On the other hand, cumulative reduction in the levels of 12:0, 14:0, and 16:0 of 3.7% (±SE 0.82) with vitamin E supplementation was real ($P < 0.01$). The evidence is consistent with the hypothesis that vitamin E, aside from its already known functions, promotes chain elongation of 12:0, 14:0, and 16:0 acids to stearic acid. Such chain elongation may occur in mitochondria upon addition of acetyl-CoA or in microsomes where malonyl-CoA serves as a source of acetyl groups (Lehninger, 1975).

Effect of Depot Fat Position. As shown in Table VII and in accord with observations of Hilditch and Williams (1964), the external l. dorsi triglycerides contained less stearic acid as compared with the internal omental and perinephric fatty tissues ($P < 0.01$). Results are also in agreement with those of Cook et al. (1972) for Friesian steers. In contrast, however, the present work showed that depot position affected palmitic acid levels, which were greater ($P < 0.01$) in the omental than in the perinephric and l. dorsi triglycerides. Likewise, levels of hexadecenoic acid in the l. dorsi triglycerides were greater ($P < 0.01$) than in omental or perinephric triglycerides.

Analysis of variance revealed that changes in the fatty acid composition occurred independently of changes in dietary fat composition, regardless of vitamin E supplementation or location of the depot fat (Table VII). Differences caused by the above three factors were consistent within themselves (Figure 1). Thus, the corn oil diet produced a higher percentage of stearic acid in perinephric triglycerides than the coconut oil diet. This difference was also consistently found in omental and l. dorsi triglycerides.

Levels of Vitamin E in the Tissues at Slaughter and during Frozen Storage. Results on the levels of vitamin E in the tissues at slaughter and during frozen storage are summarized in Table VIII. Analysis of variance showed that vitamin E levels (micrograms per gram of tissue) were influenced ($P < 0.01$) by location (P) in the body (l. dorsi, omental, and perinephric tissues), the kind of oils (O) in

Table IV. Means[a] and Standard Deviations of the Fatty Acids of the L. Dorsi Phospholipid, Expressed as Percent Area of Methyl Ester Peak of the Total Area of Methyl Ester Peaks

	coconut oil		corn oil	
fatty acid[c]	+vitamin E (1)	no vitamin E[b] (2)	+vitamin E (3)	no vitamin E[b] (4)
12:0	0.7	0.5	trace	0.1
?			0.2	0.1
14:0	3.5 ± 2.38	2.2 ± 0.63	0.5 ± 0.30	0.9 ± 0.52
15:0	0.3	0.1		
15:1	2.9 ± 0.65	3.6 ± 0.91	3.8 ± 0.87	3.5 ± 0.83
16:0	19.1 ± 3.86	18.6 ± 0.89	13.2 ± 2.16	14.2 ± 3.36
16:1	1.1 ± 0.28	1.3 ± 0.35	0.3 ± 0.08	0.6 ± 0.31
17:0	0.1	trace	0.1	trace
17:1	0.2	0.2	0.3	0.2
18:0	11.4 ± 0.93	11.0 ± 1.31	15.0 ± 0.79	11.9 ± 2.96
18:1	24.5 ± 4.99	26.0 ± 5.43	14.2 ± 0.93	15.6 ± 2.35
18:2	23.8 ± 3.01	24.1 ± 6.59	42.4 ± 1.92	41.7 ± 2.83
?			0.1	0.1
18:3	0.4	0.4	trace	0.1
20:2ω6	0.2	0.3	0.6	0.8
20:3ω9	1.6 ± 0.22	1.5 ± 0.32	0.5 ± 0.34	0.6 ± 0.37
20:4ω6	8.2 ± 2.38	8.3 ± 0.73	7.2 ± 0.62	7.6 ± 2.45
20:5ω3	0.4	0.3	0.1	0.1
22:4ω6	0.8	0.8	0.4	0.8
22:5ω6	0.8	0.8	1.1	0.9

[a] Means are values from four animals. [b] Vitamin E was not added to the diet, but the oils, especially corn oil, had natural vitamin E. [c] ? = unidentified component of the approximate chain length indicated by their position in the list of fatty acids. The number before the colon indicates the number of carbons in the fatty acid; the number after the colon indicates the number of double bonds; ω indicates the carbon number where the double bond commences, counting from the terminal methyl group of the molecule.

Table V. Effect of Dietary Oils on the Percent Fatty Acid Composition[a] of Lipids from Veal Calves

	mean % fatty acids ± SE		significance of
fatty acid[b]	coconut oil	corn oil	differences[c]
	Triglycerides		
12:0	6.4 ± 0.21	1.0 ± 0.21	****
14:0	21.9 ± 0.37	5.3 ± 0.37	****
16:0	29.1 ± 0.40	19.6 ± 0.40	****
16:1ω7	2.1 ± 0.08	1.7 ± 0.08	**
18:0	9.4 ± 0.37	11.5 ± 0.37	***
18:1ω9	23.5 ± 0.49	30.5 ± 0.49	****
18:2ω6	5.7 ± 0.43	27.2 ± 0.43	****
	Phospholipids		
14:0	2.8 ± 0.44	0.7 ± 0.44	***
16:0	18.8 ± 0.99	13.7 ± 0.99	***
16:1ω7	1.2 ± 0.09	0.4 ± 0.09	****
18:0	11.1 ± 0.61	13.4 ± 0.61	**
18:1ω9	25.2 ± 1.38	14.8 ± 1.38	****
18:2ω6	23.9 ± 1.41	42.1 ± 1.41	****
20:3ω9	1.5 ± 0.11	0.5 ± 0.11	****
20:4ω6	8.2 ± 0.62	7.4 ± 0.62	NS

[a] Mean of eight different animals. [b] The number before the colon indicates the number of carbon atoms in the fatty acid; the number after the colon indicates the number of double bonds; ω indicates the carbon number counting from the terminal methyl group on the molecule where the double bond commences. [c] (****) Significant at $P < 0.001$; (***) significant at $P < 0.01$; (**) significant at $P < 0.05$; NS = nonsignificant.

Table VI. Effect of Dietary Vitamin E on the Fatty Acid Composition[a] of Lipids

	mean % fatty acids ± SE		signifi-
fatty acid[b]	added vitamin E	no vitamin E[c]	cance of differences[d]
	Triglycerides		
12:0	3.3 ± 0.22	4.1 ± 0.22	**
14:0	12.8 ± 0.37	14.4 ± 0.37	**
16:0	23.7 ± 0.40	25.0 ± 0.40	NS
16:1ω7	1.7 ± 0.08	2.2 ± 0.08	*
18:0	11.3 ± 0.37	9.6 ± 0.37	**
18:1ω9	27.8 ± 0.49	26.2 ± 0.49	NS
18:2ω6	16.6 ± 0.43	16.3 ± 0.43	NS
	Phospholipids		
14:0	1.9 ± 0.44	1.5 ± 0.44	NS
16:0	16.1 ± 0.99	16.4 ± 0.99	NS
16:1ω7	0.7 ± 0.09	0.9 ± 0.09	NS
18:0	13.1 ± 0.61	11.4 ± 0.61	*
18:1ω9	19.3 ± 1.38	20.7 ± 1.38	NS
18:2ω6	33.1 ± 1.41	32.9 ± 1.41	NS
20:4ω6	7.6 ± 0.62	7.9 ± 0.62	NS

[a] Mean of eight different animals. [b] Numbers before the colon indicate the number of double bonds. ω indicates the carbon number where the double bond commences counting from the terminal methyl group of the molecule. [c] Vitamin E was not added to the diet, but the oils, especially corn oil, contained natural vitamin E. [d] (*) Significant at $P < 0.1$; (**) significant at $P < 0.05$; NS = nonsignificant.

the diet (coconut and corn oil), vitamin E (E) in the diet (supplemented or unsupplemented), and storage time (S). However, significant interactions ($P < 0.01$) were observed between P and O and between O and E. The interaction between P and E was also significant at the 10% level. On expressing vitamin E content as micrograms per gram of lipid, O, E, and S continued to affect vitamin E levels ($P < 0.01$) significantly, but the effect of P was reduced ($P < 0.05$) and the strong interactions were reduced so only that between P and O was significant at the 5% level. Thus, the results for vitamin E levels were calculated on both a total tissue and lipid basis. As percentage lipid in the tissues varied widely, the vitamin E levels also differed greatly, depending on whether expressed on the total tissue or lipid basis. Thus, mean vitamin E value (micrograms per gram of tissue) for the l. dorsi, omental, and the perinephric tissues fell within the limits 3.3–5.2, 15.0–41.1, and 16.4–43.7, respectively, as compared with 189–363, 27–188, and 29–170 expressed as micrograms of vitamin E per gram of lipid. On a total tissue basis, omental and perinephric tissues were richer in vitamin E than the l. dorsi, but on a lipid basis the reverse was true. Some 20–30% of the variability in vitamin E content was explainable by tissue lipid levels, with a positive linear correlation coefficient

Table VII. Effect of the Position of the Depot Fat of Veal Calves on Its Fatty Acid Composition[a]

fatty acids[b]	mean % fatty acids ± SE			significance of differences[c]
	l. dorsi	omental	perinephric	
12:0	3.5 ± 0.26	3.7 ± 0.26	4.1 ± 0.26	NS
14:0	13.5 ± 0.45	13.6 ± 0.45	13.7 ± 0.45	NS
16:0	23.4 ± 0.50	26.1 ± 0.50	23.6 ± 0.50	***
16:1ω7	2.2 ± 0.10	1.9 ± 0.10	1.5 ± 0.10	***
18:0	8.6 ± 0.46	10.8 ± 0.46	11.9 ± 0.46	****
18:1ω9	27.0 ± 0.60	26.4 ± 0.60	27.6 ± 0.60	NS
18:2ω6	17.1 ± 0.53	16.0 ± 0.53	16.3 ± 0.53	NS

[a] Mean of 16 different animals. [b] Numbers before the colon indicate the number of carbons in the fatty acids; numbers following the colon show the number of double bonds; ω indicates the carbon number where the double bond commences, counting from the terminal methyl group. [c] (***) Significant at $P < 0.01$; (****) significant at $P < 0.0001$; NS = nonsignificant.

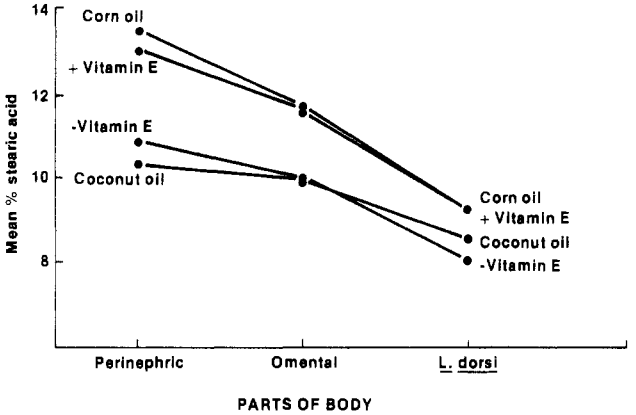

Figure 1. Changes in percent stearic acid in different fat depots as a result of feeding corn or coconut oils with and without supplemental vitamin E.

between vitamin E levels and lipid content being significant at the 10% level.

Vitamin E at Slaughter. The vitamin E supplemented groups (1 and 3) received some 28 g of *d*-α-tocopheryl acetate per animal over the experimental period, which plus that in the diet made a total of 28.9 g in coconut oil group 1 and 38.0 g in corn oil group 3. The level of vitamin E in l. dorsi muscle on a total tissue basis as well as on a lipid basis was significantly raised in the corn oil supplemented group (3) but not in the coconut oil fed group (Table VIII).

In contrast to l. dorsi muscle, supplemental vitamin E significantly raised the levels of vitamin E on a total tissue basis in omental and perinephric fatty tissues of group 1 but not for group 3. However, when expressed on a lipid basis, vitamin E levels of the omental and perinephric tissues were elevated in both groups 1 and 3 ($P < 0.05$). There was no evidence to suggest that conclusions relating to the effects of vitamin E supplementation would change with storage time.

Ellis et al. (1974) showed that round and perinephric fats of calves supplemented with vitamin E had as much as 7-fold more vitamin E than commercial samples from unsupplemented animals. In the present work group 3, which received corn oil supplemented with vitamin E, showed means of 170 and 188 μg of vitamin E/g of lipid in the perinephric and omental tissues, respectively. These values are several times higher than those reported by Ellis et al. (1974) for the extracted fat of veal calves supplemented with vitamin E. These high levels of vitamin E may be associated with very low fat contents, since the mean lipid content for the omental and perinephric tissues of group 3 were 26.0 and 21.4%, respectively. At more typical lipid levels (~70%) as found in the omental and perinephric tissues of group 1, which received supplemental vitamin E, the vitamin E contents of 63 and 65 μg/g of lipid were similar to those reported by Ellis et al. (1974) for perinephric fat.

In the present study vitamin E supplementation increased the vitamin E levels (micrograms per gram of lipid) in the omental tissues of the coconut oil and corn oil fed animals (groups 1 and 3) by 233 and 186%, respectively, with the corresponding values for perinephric tissues being 196 and 586%. In the l. dorsi of coconut oil group 1, no increase in vitamin E occurred upon vitamin E supplementation, but in corn oil group 3 an increase of 140% was observed. Although the lack of responsiveness of l. dorsi muscle to vitamin E supplementation is unexplained, the high levels on a lipid basis were consistent with ready transport of ingested vitamin E to these tissues. Incorporation of vitamin E into muscle by chickens fed low levels (40 mg vitamin E/kg of feed) increased the amounts in breast muscle from 2.0 to 6.2 μg/g of tissue during 8 weeks (Marusich et al., 1975). Breast muscle of the turkey, however, was much less responsive.

Loss of Vitamin E during Frozen Storage. Changes in vitamin E levels during frozen storage expressed as a percentage of the initial level are presented in Table VIII. On a total tissue basis, the mean percent of vitamin E retained was higher at the end of the first month in perinephric (81) and omental (80) than in the l. dorsi (74). At the end of 3 months, however, when observations on l. dorsi were discontinued, differences between tissues were more apparent. At this stage the l. dorsi of group 1 showed the highest mean retention of vitamin E (77.5) and l. dorsi of group 4 the least (25.0). Values of the omental tissues (62.1–74.3), except from group 3, were higher than those of the perinephric tissues (57.9–67.2), which in turn exceeded those of the l. dorsi (25.0–53.7) for groups 2, 3, and 4. At 6 months, the percentage of vitamin E retained in the omental tissues of all groups (46.8–58.8) substantially exceeded that of the perinephric tissues (29.6–42.0), indicating an accelerated difference with storage time.

Whereas supplemental vitamin E increased vitamin E retention during frozen storage in the l. dorsi and omental (group 1) tissues, retention in the omental (group 3) and perinephric (groups 1 and 3) tissues was diminished. Feeding of saturated coconut oil in comparison to unsaturated corn oil improved vitamin E retention in l. dorsi and omental tissues but not in perinephric tissues. With few exceptions, the percent of vitamin E retained based on total tissue was similar to that based on the lipid content (Table VIII).

Thus, overall evidence indicates that the coconut oil diet in association with vitamin E supplementation enhanced vitamin E stability of the l. dorsi and omental tissues but not of the perinephric tissues. Furthermore, maintenance of high levels of vitamin E in tissues, as distinct from extracted lipids, does not necessarily protect against autoxidation during frozen storage. Despite the possible lack of relevance of vitamin E levels to lipid autoxidation in tissues, it is nevertheless of interest to predict changes in vitamin E content with time in frozen storage. The present work indicated that the level of vitamin E and length of time in frozen storage for a given treatment follows the simple linear equation: $\hat{y} = a + bx$, in which $\hat{y}$ = the dependent variable, a = the intercept, b = the slope, and x = the independent variable. It is, therefore,

Table VIII. Changes in Means and Standard Deviations of Vitamin E Levels in Veal Calf Tissues during Frozen Storage at −18 °C[a]

tissue treatment			vitamin E levels[b] at time in freezer storage of							
			0 month		1 month		3 months		6 months	
muscle	diet[c]	lipid, %	μg/g	% initial value	μg/g	% initial value	μg/g	% initial value	μg/g	% initial value
l. dorsi	coconut oil + vitamine E (1)	1.66 ± 0.39	3.3 ± 2.6	100	2.6 ± 2.3	78.4	2.6 ± 2.2	77.5		
			(189 ± 124)	100	(147 ± 119)	(77.7)	(137 ± 111)	(72.2)		
	coconut oil − vitamin E (2)	1.43 ± 0.20	5.1 ± 0.9	100	4.0 ± 1.1	77.7	2.8 ± 1.7	53.7		
			(363 ± 82)	100	(286 ± 100)	(78.7)	(202 ± 134)	(55.6)		
	corn oil + vitamin E (3)	1.76 ± 0.35	5.2 ± 2.2	100	3.6 ± 1.7	69.2	2.2 ± 0.6	41.8		
			(342 ± 169)	100	(236 ± 125)	(69.0)	(153 ± 83)	(44.6)		
	corn oil − vitamin E (4)	1.44 ± 0.25	3.4 ± 0.8	100	2.4 ± 0.5	70.6	0.9 ± 0.7	25.0		
			(244 ± 64)	100	(174 ± 55)	(71.5)	(62 ± 55)	(25.3)		
omental	coconut oil + vitamin E (1)	66.0 ± 4.4	41.1 ± 10.0	100	35.8 ± 3.6	86.0	30.6 ± 4.5	74.3	24.8 ± 8.2	58.5
			(63 ± 17)	100	(55 ± 8)	(87.1)	(46 ± 5)	(73.6)	(36 ± 11)	(59.3)
	coconut oil − vitamin E (2)	56.9 ± 8.0	15.0 ± 2.6	100	10.4 ± 2.1	69.7	10.0 ± 1.5	66.9	7.6 ± 3.0	50.6
			(27 ± 7)	100	(19 ± 6)	(69.9)	(18 ± 5)	(66.9)	(14 ± 6)	(50.6)
	corn oil + vitamin E (3)	26.0 ± 30.8	23.0 ± 16.4	100	18.8 ± 16.0	82.0	14.3 ± 16.4	62.1	10.7 ± 16.0	46.8
			(188 ± 154)	100	(141 ± 109)	(74.8)	(75 ± 73)	(40.1)	(39 ± 40)	(20.8)
	corn oil − vitamin E (4)	42.6 ± 26.8	23.5 ± 6.7	100	19.2 ± 7.0	81.9	15.8 ± 15.2	67.5	11.2 ± 6.9	47.5
			(101 ± 101)	100	(73 ± 62)	(72.8)	(65 ± 104)	(65.1)	(25 ± 9)	(24.8)
perinephric	coconut oil + vitamin E (1)	73.4 ± 17.4	43.7 ± 16.9	100	37.1 ± 13.6	84.8	26.2 ± 10.7	60.1	14.0 ± 5.1	31.9
			(65 ± 40)	100	(55 ± 32)	(84.3)	(39 ± 20)	(60.1)	(19 ± 6)	(29.3)
	coconut oil − vitamin E (2)	60.5 ± 14.7	19.1 ± 3.6	100	14.3 ± 2.0	74.8	12.0 ± 2.9	62.8	7.4 ± 3.3	38.8
			(33 ± 11)	100	(24 ± 3)	(73.0)	(20 ± 4)	(61.3)	(13 ± 5)	(37.8)
	corn oil + vitamin E (3)	21.4 ± 27.1	16.4 ± 12.0	100	14.0 ± 13.0	84.9	11.0 ± 15.7	67.2	4.9 ± 8.8	29.6
			(170 ± 161)	100	(117 ± 94)	(72.0)	(35 ± 29)	(20.6)	(10.0 ± 11.6)	(6.3)
	corn oil − vitamin E (4)	68.6 ± 18.7	18.3 ± 9.3	100	14.1 ± 6.2	77.0	10.6 ± 4.7	57.9	7.7 ± 5.7	42.0
			(29 ± 14)	100	(23 ± 7)	(79.0)	(17 ± 11)	(61.5)	(13 ± 10)	(46.5)

[a] Mean of four animals. [b] Expressed as micrograms of vitamin E per gram with vitamin E in micrograms per gram of lipid shown in parentheses. [c] The number in parentheses following the diet refers to the group number.

Table IX. Comparison between Observed and Predicted Levels of Vitamin E Using the Regression Equation[a]

frozen storage period, months	mean vitamin E level, μg/g of tissue		difference, μg/g
	predicted	observed	
1	38.8	37.1	1.7
3	29.0	26.2	2.8
6	14.3	14.0	0.3

[a] $\hat{y} = a + bx$, where $\hat{y}$ = the dependent variable, a = the intercept, b = the slope, and x = the independent variable.

possible after determination of changes in vitamin E levels over several points in time to calculate b and predict vitamin E levels during a limited period of storage. Values for different groups and different tissues showed good agreement between determined and predicted values as demonstrated by results for the perinephric tissues of group 1 (Table IX).

Lipid Oxidation in the Tissues during Frozen Storage at −18 °C. TBA values ranged from nondetectable initial levels to as high as 1.83 after 6 months of storage. In general, the threshold of rancidity set at a TBA value of 1–2 by Watts (1962) was not reached. As the number of observations did not permit a valid analysis of variance covering all tissues, results for the l. dorsi, omental, and perinephric tissues were considered separately.

L. Dorsi Tissues. The mean TBA values for l. dorsi increased from 0.04 at slaughter to 0.16 after storage for 6 months. Analysis of variance showed highly significant increases in TBA values with time and a highly significant retardation with supplemental vitamin E (Table X). The interaction between dietary oils and vitamin E supplementation was nonsignificant as was the effect of the dietary oils on development of TBA values. Results appear to conflict with those of Ellis et al. (1974), who found that susceptibility of veal perinephric fats to oxidation increased with linoleic acid content. L. dorsi muscle in contrast to perinephric tissues, however, contained only small amounts of lipid (2%), comprising 43–57% phospholipid, which contained most of the polyunsaturated fatty acids. Moreover, phospholipids included substantial amounts of polyunsaturated C_{20} and C_{22} acids with four to six double bonds (Table IV), which are much more susceptible to autoxidation than linoleic acid (Holman and Elmer, 1947). As the levels of C_{20} and C_{22} polyunsaturated fatty acids were largely unaffected by the dietary oils, changes in the levels of linoleic acid (Tables III and IV) would be expected to have only a minor effect on the susceptibility of l. dorsi tissues to autoxidation. Thus, protection of veal (l. dorsi) muscle lipids against autoxidation by dietary vitamin E, but not by dietary saturated coconut oil compared to dietary unsaturated corn oil, was largely anticipated. On the other hand, omental and perinephric tissues, in which the polyunsaturated fatty acids consisted largely of linoleic acid (Table III), showed a 5-fold increase in this acid in comparing dietary corn oil with dietary coconut oil and would be expected to be markedly more susceptible to autoxidation. This is confirmed by data in Table X.

Omental and Perinephric Tissues. As shown in Table X, TBA values of the fatty tissues after storage for 6 months, especially perinephric tissues, were much greater than those of the l. dorsi. If TBA values were based on lipid content and not on total tissues, the difference between l. dorsi and perinephric tissues was less extreme. In contrast to l. dorsi, TBA values of omental tissues were increased significantly by dietary corn oil compared to coconut oil, but supplemental vitamin E had no significant effect. The interaction between dietary oils and vitamin E was also nonsignificant. Thus, the effect of dietary oils on stability of the omental tissues was associated with a 5-fold increase in linoleic acid content from dietary intake of corn oil compared to coconut oil, which contains negligible amounts of this acid.

In perinephric tissues, corn oil in comparison to coconut oil increased TBA levels during storage ($P < 0.01$), whereas supplemental vitamin E resulted in a decrease ($P < 0.05$). There was also an interaction between supplemental vitamin E and dietary oils ($P < 0.10$). Results indicated that incorporation of substantial levels of dietary linoleic acid into omental and perinephric tissues had a detrimental effect on the stability of lipids, which is in agreement with the results of Ellis et al. (1974). Thus, for omental and perinephric tissues, inclusion of saturated fat (coconut oil) compared with polyunsaturated fat (corn oil) was advantageous in the diet.

Since TBA values are conventionally based on total tissue instead of lipid content, some variation in interpretation of the extent of autoxidation could arise if the lipid content varied greatly. In general, conclusions in the present work were not significantly affected. Because of the relatively low lipid levels in perinephric tissues of group 3 compared with group 4, however, the conclusion reached by means of conventional TBA values that vitamin E significantly reduced TBA values was not sustained if they were assessed on lipid content.

The conclusion that supplemental vitamin E had a significant effect in enhancing the stability of lipids in l. dorsi and perinephric tissues (coconut oil fed groups only) is in agreement with the findings of Ellis et al. (1974). However, present results for omental tissues did not reach

Table X. Means and Standard Deviations of Thiobarbituric Acid (TBA) Values of Calf Tissues during Frozen Storage at −18 °C[a]

storage period, months	coconut oil		corn oil	
	group 1, +vitamin E	group 2, no vitamin E	group 3, +vitamin E	group 4, no vitamin E
		L. Dorsi		
0	0.04 ± 0.04	0.04 ± 0.02	0.00 ± 0.00	0.06 ± 0.04
1	0.08 ± 0.02	0.12 ± 0.01	0.07 ± 0.02	0.12 ± 0.03
3	0.11 ± 0.02	0.16 ± 0.03	0.11 ± 0.03	0.18 ± 0.07
6	0.11 ± 0.03	0.17 ± 0.04	0.14 ± 0.04	0.21 ± 0.01
		Omental		
0	0.05 ± 0.06	0.10 ± 0.05	0.02 ± 0.04	0.08 ± 0.05
6	0.20 ± 0.10	0.29 ± 0.09	0.33 ± 0.05	0.38 ± 0.03
		Perinephric		
0	0.00 ± 0.00	0.09 ± 0.05	0.01 ± 0.05	0.04 ± 0.06
6	0.20 ± 0.02	0.31 ± 0.09	0.73 ± 0.61	1.59 ± 0.36

[a] Mean of four different animals. TBA values are expressed as milligrams of malonaldehyde per 1000 g of tissues.

the significant level. In contrast to the results of Ellis et al. (1974), which indicated that high levels of vitamin E in extracted veal lipids protect against rancidity, even in the presence of high levels of linoleic acid, the present work involving intact tissues does not entirely support this viewpoint. Thus, in the perinephric tissues of group 4 the initial values in three of the animals were respectively 28.5, 23.0, and 14.3 μg of vitamin E/g of tissue. These fell to 13.6, 7.8, and 10.0 μg of vitamin E/g of tissue, respectively, after 6 months of storage. Despite the retention of substantial amounts of vitamin E, relatively high TBA values of 1.17, 1.83, and 1.77, respectively, were found. Likewise, in omental tissue of one of the calves of group 3, a high initial value for vitamin E (47.2 μg/g of tissue) after frozen storage for 6 months was nevertheless accompanied by a relatively high TBA value of 1.16. Thus, high levels of vitamin E in tissues do not necessarily prevent the development of rancidity. This is because the system is more complex than in extracted lipids and includes inhibitors, such as glutathione peroxidase, and proxidants, such as ascorbic acid (Sato and Hegarty, 1971), that are absent in extracted fat. Thus, the results of Ellis et al. (1974) using the Swift accelerated oxidation test on extracted veal fats may not be relevant to autoxidation of lipids in intact tissues during frozen storage. Because of variations in levels of inhibiting substances and proxidants in intact tissues, it seems likely that lipids of similar composition would show variation in their susceptibility to autoxidation, depending on the nature of the tissue.

Treatments contributed differently to inhibition of autoxodation of veal tissues during frozen storage. Dietary coconut oil compared to corn oil significantly reduced autoxidation of omental and perinephric but not l. dorsi tissues. Supplemental vitamin E benefited l. dorsi tissues and perinephric tissues of the coconut oil fed calves, but omental tissues showed no measurable response. Although the separate treatments only influenced some of the tissues favorably, the combined treatments significantly improved the lipid stability of all tissues studied.

ACKNOWLEDGMENT

We are indebted to the DPI Division of Eastman Chemical Products Inc., Kingsport, TN, for providing the d-α-tocopheryl acetate. F.B.S. also acknowledges the encouragement of Norman Law, formerly Director of the Meat Industry Research Institute of New Zealand, whose discussions gave direction to the subject matter of this investigation.

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Received for review August 11, 1980. Accepted March 23, 1981. F.B.S. was supported in part by the Scientific Distribution Committee in New Zealand and by a Fulbright-Hays Fellowship. Michigan Agricultural Experiment Station Article No. 9522. Taken from a thesis presented to Michigan State University by J.O.I. in partial fulfillment of the requirements for the M.S. degree.