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Newer Concepts of the Role of Essential Fatty Acids[1]

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THE RECENT INTEREST in the possible role of unsaturated fatty acids in the regulation of serum cholesterol levels has stimulated a great deal of research to determine whether a relationship between essential fatty acids and cholesterol metabolism exists and, if so, to understand what this relationship is.

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The connection between essential fatty acids and cholesterol is not a new idea but was suggested as early as 1923, when Bloor (6) in his studies of unsaturated fatty acids in the plasma of various species of animals found that these unsaturated fatty acids existed mainly in combination with cholesterol. Bloor (7) and other workers (10, 20) extended these observations and found that the unsaturated fatty acid ester—cholesterol linoleate—was the chief ester of cholesterol in the plasma. Linoleic acid cannot be synthesized by the animal body, but it is necessary for growth and maintenance of normal body processes and is therefore called "essential." Three fatty acids are classified in this way: linoleic acid, linolenic acid, and arachidonic acid.

The functions of essential fatty acids are not as yet completely known. Essential fatty acids are necessary for growth (15), reproduction, and lactation (16, 17, 18, 25) in the rat. The lack of essential fatty acids in the diets of rats causes them to be more susceptible to X-irradiation injury (11, 14). The absence of essential fatty acids from the diet also produces a deficiency syndrome characterized by capillary fragility (21), increased skin permeability (26), a typical eye condition, scaliness of the paws and tail (9), alopecia, and a plateau in weight. Leveling off in weight and growth is caused by reduction in the number of bone proliferating cells (5). As an example, normally in the proximal head of the tibia (Figure 1) there is a wide section composed of columnar cells in which cell division occurs. This cell division is responsible for the growth of the bone. In the fat-free animals this area is markedly reduced. In addition, in the fat-free animal at the diaphyseal border there is a thin layer of bone sealing off the epiphyseal plate, and in the diaphysis there is a loss of bone cells, which is replaced by fat globules.

In 1953 a further result of EFA deficiency was reported from this laboratory (1). Male rats had been placed on a diet of 16–20 weeks, adequate in all respects but deficient in fat and therefore deficient in essential fatty acids.[2] The rationale was that the rats were able to synthesize any fat they require, with the exception of essential fatty acids, from the two-carbon fragment which is formed as a result of carbohydrate and protein metabolism. On autopsy these rats were found to have abnormal deposits of cholesterol in certain tissues of the body (Table I), increased amounts of cholesterol in liver and adrenal, and slightly decreased amounts in the plasma. The liver was fatty in appearance. Histological sections of the liver confirmed the analytical results and showed abnormal deposits of fat and a depletion of glycogen. Sections of the adrenal also showed increased deposition of fat, but a decrease in the area of the cortex (5).

The most striking effect of EFA deficiency was noticed in the gonadal tissue. Degeneration of spermatic development was a common alteration observed in EFA-deficient animals. In the epididymis the lumens of the ducts are filled with mature sperms in the control animals, but there is almost a complete loss of sperms in the lumen of the ducts of the EFA-deficient animals. In the testes themselves a depletion of EFA produces a degeneration of the tubules and a loss of maturation of the primary spermatogonial cells (5).

[1] Contribution No. 438 from the Harry J. Deuel Jr. laboratory.

[2] The fat-free diet consisted of 20.0% casein, 70.7% sucrose, 4.0% salt mix, 4.0% cellulose and fat-soluble and water-soluble vitamins in adequate amounts. When fat was added to the diet, it was done at the expense of carbohydrate.

TABLE I

The Effect of a Diet Deficient in Fat on Cholesterol Levels in Various Organs of the Rat

Diet	Exp. No.	No. of rats	Mg. cholesterol/g.		Mg. cholesterol per 100 ml.
			Liver	Adrenal	Plasma
12.5% fat	1	10	2.04	35.4	65.6
	2	9	2.08	35.3	63.2
Fat-free (Vitamin-test casein)	3	8	3.15	48.9	38.4
	4	7	4.06	50.4	50.4
Fat-free (Commercial casein)	5	10	4.72	49.3	41.1
	6	9	4.24	46.2	44.9

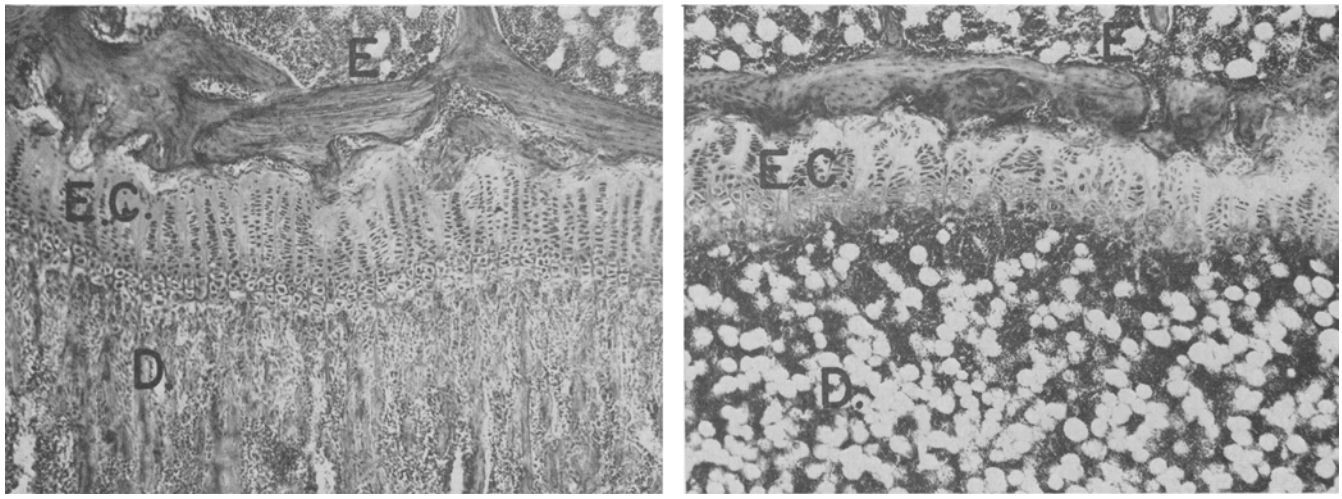

FIG. 1. Proximal epiphyseal region of the tibia.

1. Rat fed Purina Chow, 20 weeks; width of epiphyseal cartilage, 182 μ. Note the four zones of the cartilage, especially the proliferating region (E.C.), which is characterized by the parallel columns of cells. The trabeculae of the diaphysis are long and slender.

2. Rat fed fat-free diet, 20 weeks; 150 μ. Note the decrease in the number of cells in all regions, especially the proliferating region with a relative increase in the amount of intercellular matrix. There is a loss of trabeculation projecting into the diaphysis and sealing off by a thin layer of bone preventing further growth. In addition, there is a replacement of the myeloid elements by adipose tissue.

EXPERIMENTS performed in this laboratory have resulted in the observation that the liver reflects changes in cholesterol metabolism much more sensitively than does plasma. Plasma cholesterol levels in the rat are resistant to change; a more detailed examination of the liver lipide values is shown in Table II. In spite of the fact that the diet of the rats was deficient in fat and essential fatty acids, producing an insufficiency of these acids for esterification with endogenous cholesterol, the increase in cholesterol content in the liver was caused by an accumulation of cholesterol esters. An increase in total liver lipide was also observed, which confirmed the histological results. Little change was observed in the free cholesterol fraction.

TABLE II
The Effect of a Diet Deficient in Fat on Cholesterol and Lipide Levels in Rat Liver

Diet	Exp. No.	Liver			F/T ×100	Mg. lipid/g.
		% of body weight	Mg. chol./g.			
			Total	Free		
12.5% fat	1	2.8	2.0 (1.6–2.5)	1.7 (1.5–2.1)	85.0	28.8 (11.1–53.5)
	2	2.7	2.1 (1.8–2.5)	1.8 (1.6–2.1)	85.7	42.5 (21.1–55.8)
Fat-free (Vitamin-test casein)	3	4.0	3.2 (2.7–4.6)	1.9 (1.6–2.1)	59.4	57.9 (26.4–131.3)
	4	3.4	4.1 (3.1–4.8)	2.1 (1.7–2.4)	51.2	57.8 (43.2–79.1)
Fat-free (Commercial casein)	5	3.5	4.7 (3.2–6.2)	2.4 (1.5–3.4)	51.1	74.8 (50.2–118.2)
	6	3.5	4.2 (3.1–5.9)	2.5 (2.0–2.8)	59.5	63.7 (42.3–87.9)

Bromer and Day (8) have also reported a connection between essential fatty acids and cholesterol metabolism. They found that feeding cholesterol to rats on an essential fatty acid-deficient diet hastened the appearance of the deficiency syndrome and increased the severity of the deficiency symptoms. They confirmed our observation that the total liver lipide was increased to an extent over and above that attributable to the accumulation of cholesterol. Somewhat later Peifer and Holman (23) found that EFA deficiency in the diabetic animal and EFA deficiency intensified by dietary cholesterol in the non-diabetic animal were similar in that the deficiency syndrome was obtained in both conditions within a month. They proposed the idea that both syndromes are caused by an accelerated transport of EFA caused either by endogenous hypercholesterolemia in the diabetic rat or by exogenous hypercholesterolemia in the normal rat; both conditions resulted in a rapid depletion of body stores of essential fatty acids.

One of the interesting facts connected with our investigation on essential fatty acids was our discovery that female rats, when placed on the essential fatty acid-deficient diet for the same length of time as the male rats in our previous experiment, failed to exhibit the fatty liver or accumulation of cholesterol in the liver observed in the male animals. Evidently a sex difference exists in the need for essential fatty acids. That this requirement was sex-linked was shown in experiments in which both male and female rats were gonadectomized before being placed on essential fatty acid-deficient diets (Table III). Gonadectomized females started to show the accumulations of cholesterol esters in the liver; gonadectomized males had a much lower cholesterol concentration in the liver than intact males on the same diet (12).

An investigation was undertaken to relate the rate

TABLE III
The Effect of Gonadectomy at Weaning on the Cholesterol and Total Lipide Concentrations in the Liver of Male and Female Rats Fed a Fat-Free Diet for 20 Weeks

Sex	Diet	Cholesterol in liver			Total lipides in liver mg./g.
		Total mg./g.	Free mg./g.	% Free	
Intact male	C[a]	2.48	2.15	86.6	38.9
Intact male	FF[b]	5.73	1.89	35.0	85.3
Gonadectomized male	FF	3.35	1.93	57.6	52.0
Intact female	C	2.37	2.07	87.3	46.0
Intact female	FF	2.29	1.70	74.2	51.5
Gonadectomized female	FF	2.95	1.75	59.3	57.6

Ten animals/group.
[a] 15% cotton oil diet.
[b] Fat-free diet.

TABLE IV

The Effect of Linoleate, Oleate, and B Vitamins on Cholesterol and Total Lipide Levels of Plasma and Liver in Essential Fatty Acid-Deficient Rats

Category	Cholesterol in plasma			Cholesterol in liver			Total lipides in liver (mg./g.)
	Free (mg. %)	Total (mg. %)	Free (%)	Total (mg./g.)	Free (mg./g.)	Free (%)	
I EFA-deficient, 16 weeks	12.9	60.1	21.5	3.46	1.99	57.5	65.2
II I + 100 mg. linoleate, 4 weeks	13.1	69.1	19.0	2.76	1.95	70.7	61.5
III I + 100 mg. oleate, 4 weeks	12.6	63.9	19.8	3.95	2.03	51.4	67.2
IV I + added B vitamins, 4 weeks	17.1	63.1	27.1	3.39	2.08	61.4	51.2
V EFA-deficient, 20 weeks	8.4	56.1	15.0	4.32	2.09	48.4	60.7
VI Purina control, 20 weeks	17.0	71.5	23.8	2.46	2.04	84.5	45.4

Ten animals/group.

of appearance of elevated liver cholesterol values and depressed plasma cholesterol values in male rats placed on essential fatty acid-deficient diets with the appearance of the skin symptoms. Rats were sacrificed after 1, 4, 7, 10, and 13 weeks on diet, and cholesterol was determined in liver and plasma (13) (Figure 2). It can be seen that as early as one week after being on the EFA-deficient diet and much before dermal symptoms were evident, there was a decrease in plasma cholesterol which continued over the 13-week period. Concomitantly there developed a marked increase in liver cholesterol (Figure 3).

The animals fed hydrogenated coconut oil, a saturated fat containing no essential fatty acids, reacted atypically. It was found in our laboratory that the presence of hydrogenated coconut oil as the sole source of fat in the diet of rats hastened the onset of the dermal symptoms. It was concluded at that time that hydrogenated coconut oil either increased the metabolism of stored essential fatty acids or interfered in some way with their metabolism, rendering them ineffective. It was expected that effects paralleling those obtained with the fat-free diet would result. The fact that an initial rise in cholesterol concentration in the liver (Figure 3), obtained on the hydrogenated coconut oil diet, was followed by a slow drop toward normal led us to the conclusion that perhaps the animal could adapt and possibly use the short chain fatty acids contained in hydrogenated coconut oil for cholesterol esterification in the absence of the essential fatty acids. Obviously though the same mechanisms were not responsible for both cholesterol accumulation and skin symptoms.

IT WAS OF INTEREST to determine whether the "fatty liver" and increased cholesterol concentration in

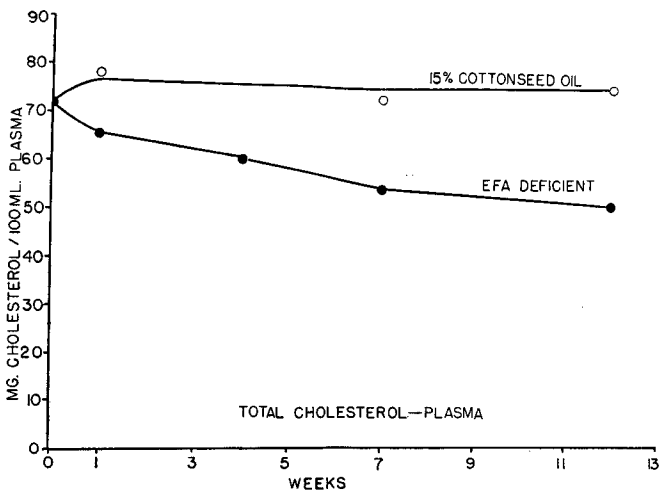

FIG. 2.

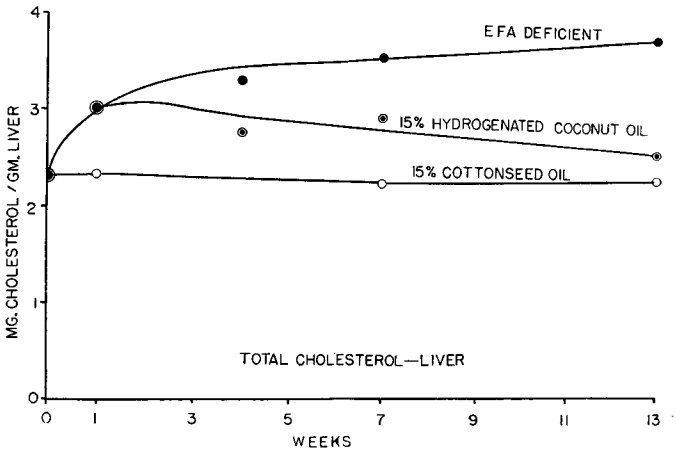

FIG. 3.

the liver of essential fatty acid-deficient rats could be reversed similar to the alleviation of the dermal symptoms when essential fatty acids were restored to the diet. Therefore rats which had been on essential fatty acid-deficient diets for 16 weeks were treated for four weeks thereafter with linoleate, oleate, and vitamins (choline, inositol, B_6, and B_{12}) known to be lipotropic. The results on plasma were not startling (Table IV); there was a slight increase in plasma cholesterol in the group supplemented with linoleate. In the liver there was a definite decrease in cholesterol concentration after linoleate supplementation. Oleate and B vitamins were ineffective; in fact, oleate caused a slight increase in liver cholesterol content. The B vitamins however were able to decrease the total liver lipide. EFA supplements were effective then not only against the external deficiency symptoms but, in time, could decrease the elevated liver cholesterol concentrations as well (2).

The problem of feeding cholesterol to animals on an essential fatty acid-deficient diet had been approached by Bromer and Day (8), also by Peifer and Holman (24). Both groups of investigators found that the addition of cholesterol to the diet of animals on essential fatty acid-deficient diets aggravated the deficiency symptoms. The latter investigators were able to alleviate the dermal symptoms of rabbits by removing cholesterol from the diet and supplementing with corn oil. If our theories were valid, then feeding cholesterol to animals on diets deficient in essential fatty acids should result in a greater deposition of cholesterol in the liver than would occur if essential fatty acids were present. Therefore EFA-deficient animals were placed on diets supplemented with cholesterol alone or with cholesterol and linoleate. Plasma and liver cholesterol values were measured after two weeks (Table V). Again

TABLE V

The Effect of Feeding Cholesterol for Two Weeks to Rats Previously Fed Diets Deficient in Essential Fatty Acids

Category	Cholesterol in plasma			Cholesterol in liver			Total liver lipide (mg./g.)
	Free (mg. %)	Total (mg. %)	Free (%)	Free (mg./g.)	Total (mg./g.)	Free (%)	
EFA-deficient	11.3	60.2	18.8	2.14	5.58	38.4	71.4
EFA-deficient + cholesterol	13.5	77.8	17.4	2.56	9.48	27.0	79.3
EFA-defiicient + cholesterol + linoleate	19.6	88.5	22.9	2.24	6.56	34.1	76.7
EFA-deficient + linoleate	13.6	73.7	18.5	2.19	4.34	50.5	59.6
Normal control	—	71.6	—	2.01	2.23	90.1	39.5

Ten animals/group.

the plasma showed no startling changes. Slight increases were obtained where deficient animals were supplemented with linoleate, as was shown previously. The increases when cholesterol alone or when cholesterol and linoleate were added to the diet are small but probably significant. In the liver the largest increase in cholesterol concentration did occur when the cholesterol diet was given without the linoleate supplement (3).

Since the accumulation of cholesterol in the liver of the essential fatty acid-deficient animals could be caused by an increase in synthesis, experiments were performed to determine the synthesis of cholesterol in the liver *in vitro* from C-14 labelled acetate under our several dietary conditions (Table VI) (22). Rather

TABLE VI

Cholesterol Concentration and Incorporation of Acetate $-1-C^{14}$ Into Cholesterol in the Liver of Rats on Various Diets

Diet	Duration of expt. weeks	Total cholesterol mg./g.	% total counts incorporated	Counts per minute/g. tissue
I 15% CSO	1	2.39	4.01	7204±892
	4	2.47	4.40	7946±286
	16	2.51	4.14	7482±200
II Fat-free	1	3.70	0.33	587± 73
	4	3.74	0.78	1408±106
	16	4.39	0.51	914± 42
III Fat-free + 100 mg. + EFA per rat/day	1	2.79	3.45	6212±136
	4	2.81	3.78	6791±214
	16	2.69	4.17	7502±307
IV 30% HCO	1	4.02	0.29	527±105
	4	3.78	0.29	527± 56
	16	2.87	0.31	556± 40

Groups I and III contained 9 animals each; Groups II and IV contained 8 animals each.

than an increase in synthesis, there occurred a marked decrease in synthesis in the livers of rats fed the fat-free and the hydrogenated fat diet. In the animals fed the fat-free diet supplemented with linoleate, synthesis was essentially normal. Synthesis seems to be inversely proportional to the cholesterol present in the liver. Since there is an increase in cholesterol concentration in the liver of animals fed the fat-free diet, the decrease in synthesis is easily explained. However, here again the animals receiving the hydrogenated coconut oil diet reacted unpredictably since, although cholesterol concentrations in the liver dropped almost to normal levels after 16 weeks, there was no return to normal cholesterol synthesis. Fractionation studies of the livers of rats fed the hydrogenated coconut oil diet have revealed the presence of an inhibitor for cholesterol synthesis in a residue fraction consisting probably of mitochondria and microsomes.

THE REASON for the accumulation of cholesterol esters in the liver was still an unsolved problem. As a possible clue, separation, isolation, and characterization of the liver lipides of rats fed our experimental diets were then carried out, using a modification of the chromatographic separation of Fillerup and Mead (19). The column was filled with silicic acid, and pentane was used instead of the petroleum ether suggested in the original method. After each fraction was isolated, the fatty acids were liberated and characterized by iodine values and spectrophotometric analysis. The results on the fatty acid composition of cholesterol esters of the livers of rats fed various diets are shown in Table VII (4). The iodine value of fatty acids associated with the cholesterol esters in the liver of animals on the EFA-deficient diet is much lower than those obtained from the liver of animals on a diet containing fat or a fat-free diet supplemented with linoleate. This low iodine value is reflected by the absence of polyunsaturated fatty acids. In the fatty acids of the phospholipide fraction (Table VII) the same general pattern obtains. It is interesting to note that the addition to the diet of one component of cottonseed oil, the essential fatty acid, linoleic acid, is able to yield a normal fatty acid pattern in both the cholesterol ester and phospholipide fractions.

These results have led to the evolution of the following theory of the interrelationship between essential fatty acids and cholesterol metabolism.

a) Essential fatty acids are used for the esterification of cholesterol. Cholesterol esters of essential fatty acids have a lower melting point than esters of the more saturated fatty acids. From a purely physical point of view these unsaturated fatty acid esters are more labile.

b) In the absence of essential fatty acids, cholesterol is esterified with more saturated fatty acids, which are less labile and tend to accumulate. When a saturated fat containing short chain fatty acids is present, it may be possible in time for the animal

TABLE VII

The Effect of Diet on the Fatty Acid Composition of Liver Lipide Fractions

Fatty acids	Diet		
	15% CSO	Fat-free	Fat-free + linoleate
I. Cholesterol Esters			
S	17	27	19
M	60	73	58
D	18	—	18
P	5	—	5
I.V.	104	67	100
II. Phospholipides			
S	42	36	41
M	30	64	28
D	12	—	13
P	16	—	18
I.V.	105	58	113

S = Saturated fatty acids.
M = Monoenoic.
D = Dienoic.
P = Polyenoic.

to make certain adaptations; cholesterol can become esterified with short chain fatty acids, and a more or less normal cholesterol metabolism can be resumed.

c) Essential fatty acids are required for phospholipide synthesis. Phospholipides are probably necessary for transport of cholesterol esters. In the absence of essential fatty acids there may be reduced phospholipide synthesis and therefore interference with cholesterol ester mobilization and transport. It is possible that short chain fatty acids by combining with cholesterol are able to spare essential fatty acids for phospholipide synthesis.

On a 15% cottonseed oil diet the ratio of cholesterol esters to phospholipide in the liver was 1 to 2.6; on a diet deficient in fat and essential fatty acids the ratio was 1 to 1.3; on the essential fatty acid-deficient diet containing hydrogenated coconut oil, the ratio was 1 to 1.9.

d) It is also possible that essential fatty acids are involved in certain enzyme systems which regulate cholesterol metabolism. Tulpule and Williams (27) found that EFA deficiency affected the activity of certain enzyme systems and that one of the sites of action of EFA is the phosphate esterification system, coupled with the oxidation of reduced cytochrome C.

The absolute necessity for essential fatty acids has not been proven in man. It is known that certain human skin diseases are helped by a diet supplemented with essential fatty acids. Diets containing vegetable oils rich in essential fatty acids are now being advised by many investigators for the reduction of elevated serum cholesterol levels. Although it is improbable that a human dietary regime is entirely deficient in essential fatty acids, it is possible that the requirements for EFA are elevated in certain disease conditions. Certainly there is a need for much more investigation in this promising field.

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Nutritional Quality of Frying Fats in Commercial Use[1]

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THERE ARE two currents of thought and investigation in the problem of fats and nutrition. The first deals with the general problem of the effect of the amount and type of fat in the diet on nutrition and health. This is the major problem, and the other papers in this symposium are devoted to a discussion of this problem. There is however a second consideration, and that involves changes that may take place in fats during processing and use which might affect the nutritional properties of otherwise adequate fats. These considerations involve principally modifications resulting from hydrogenation and heat treatment.

Daniel Melnick

An earlier report (1) from this and Deuel's laboratory describes the changes which occur in the hydrogenation of fats and presents data in support of the complete biological utilization of fatty acid isomers. Alfin-Slater and associates (2) have recently reported on the nutritive value and safety of hydrogenated fats, following a most comprehensive investigation with rats involving studies of 46 consecutive generations, three longevity studies, carcass analyses, and histopathological examination of the tissues.

The present paper deals with the changes that take place in heated fats and more specifically in fats during frying operations. That commercially fried products represent a significant portion of the foods consumed by the American public is supported by the findings in one industry alone; about one-eighth of all the potatoes raised in this country are consumed in the form of potato chips.

Nutritional and toxicological studies of the fats absorbed by fried foods are unfortunately scanty in number. In Table I are listed conclusions drawn from reports from the most active laboratories in this field. In the studies from Deuel's laboratory (3) attempts were made to simulate commercial frying operations in testing both the frying fat after eight hours of continuous use and the last batch of potato chips fried in

[1] Presented in the symposium on Fats in Nutrition and Health at the 48th Annual Meeting, American Oil Chemists' Society, New Orleans, April 30, 1957. For a more extensive review of the subject the reader is referred to a paper published in J. Am. Oil Chemists' Soc., *34*, 351–356 (1957). In this paper and in another submitted to the same Journal will be found additional experimental details and findings to support conclusions drawn.