autoxidation. Previous studies (10) on distilled oxidative dimers (not chromatographically fractionated) indicate diene conjugation as high as 23%, and with double bonds randomly distributed from the C-6 to C-10 carbon atom of the fatty-acid chains.

Chromatographic fractionation offers a method of characterization and analysis based on polarity of the various components in oxidative polymers. Although confirming our results with the chromatographic method, Bernard and Rost (4) question the nature of the dimeric material and maintain that in normally processed soybean oil, thermal polymers constitute less than 0.1%. Since Rost's method (21,22) determines thermal polymers only, it offers a means of checking the type of dimer found in the chromatographic peak I areas obtained from the oxidative dimers.

Distilled oxidative dimers do not give highly resolved chromatographic fractions, but show a large peak of ca. 30% of the same polarity as the thermal dimer, and have a major peak approx 50% in an area of much higher polarity. Saponification may not be complete, and reesterification of these two chromatographically isolated fractions shows that they are not composed of homogenous material because fractions of the various polarities are recovered. Internal ester linkages in oxidative dimer offer a partial explanation for polarity changes where hydrolysis would release hydroxyl groups to give a polar monomeric unit within the dimer structure. Polarity of a hydroxylated dimer would be different from the original dimer.

Many parameters which influence the conditions of oxidation and hydroperoxide decomposition must be investigated, and the various interactions evaluated before any chromatographic method of dimer analysis can be fully evaluated. So far results indicate that considerable chemical and physical information regarding the composition of oxidative dimers is available through a detailed analysis of the chromatographic fractions. Temp of oxidation and the environment of peroxide breakdown are extremely important, and these two factors probably contribute most to the diversity of results recorded in the literature. Many of the usual analytical techniques need critical evaluation in dimer analysis since basic distinction of dimer types (thermal, dehydro and oxidative) are made on unsaturation, type of unsaturation, mol wt, saponification value, functional group analysis and the presence of various cyclic and heterocyclic groups. No definition of oxidative polymers is possible until these materials are fractionated and the components chemically and physically characterized. Currently it might be advantageous to define, or at least partially describe, oxidative dimers in terms of polarity as determined by some chromatographic procedure.

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A Long-Term Nutritional Study with Fresh and Mildly Oxidized Vegetable and Animal Fats[1]

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Abstract

Fresh and oxidized cotton seed oil (CO) olive oil (OO), chicken fat (CF) and beef fat (BF) were fed to male weanling rats for 33 to 108 weeks. Groups fed oxidized fats except OO showed a higher death rate than those fed the corresponding fresh fats. Groups fed oxidized CO and BF had the highest death rate. Histological studies of animals dying from natural causes showed more pronounced cardiac lesions in the animals fed oxidized CO. Serum, liver and brain cholesterol levels were not influenced by oxidized fats. Fatty acid composition of depot fats and of heart and liver lipids did not show significant differences between groups fed fresh and the corresponding oxidized fats.

[1] Presented at the Spring Meeting of the AOCS 1964.

IN SPITE OF THE GREAT interest in the biological effects of natural fats which have been exposed to heat and/or air, there are comparatively few reports as to any pathological findings after long-term feeding of such fats. In acute experiments, McKay (1) induced the generalized Shwartzman reaction in pregnant rats by feeding them fractions of oxidized cod liver oil during the gestation period. These episodes of disseminated intravascular clotting, which are especially noticeable in lungs and kidneys, can be prevented by the feeding of tocopherol. Raulin (2) found that the degenerative heart lesions are more frequent in rats on a low tocopherol diet. Earlier short-term studies carried out in the authors' laboratory with highly polymerized fractions of autoxidized fats revealed no recognizable histological lesions except for some edema of the gut despite the fact that the animals had enlarged livers, kidneys, and adrenals before they died (3).

TABLE I

Death Rate at Different Ages of Male Rats Fed Various Fats

Age (wk)	Cottonseed oil		Olive oil	
	Fresh	Autoxidized	Fresh	Autoxidized
4–33	1/40	0/40	0/40	0/40
33–73	2/33	4/34	4/34	0/34
73–99	7/25	9/24	8/24	6/28
99–108	4/12	5/9	4/10	3/16
	Chicken fat		Beef fat	
4–99	2/20	5/20	2/20	4/20
99–108	6/12	5/9	2/12	4/12

TABLE II

Average Body Weights and Food Intake of Rats on Different Fats at 96 Weeks

	Fresh oil		Autoxidized oil	
	Food intake, g	Body weight, g	Food intake, g	Body weight, g
Cottonseed oil	108.5 ± 5.10[a]	613 ± 25	108.8 ± 3.88	633 ± 23.9
Olive oi	94.1 ± 4.67[b]	588 ± 19.8	97.3 ± 3.32	565 ± 20.0
Chicken fat	86.5 ± 3.73	591 ± 16.3	85.0 ± 5.49	583 ± 21.5
Beef fat	123.6 ± 3.94	599 ± 19.4	113.8 ± 4.76	582 ± 23.3

[a] Standard error of the mean.
[b] P = <05 *vs.* fresh cottonseed oil.

In cooperation with the Human Nutrition Research Division of the U. S. Department of Agriculture, a long-term study of several food fats commonly used in the United States was undertaken. This report will give some of our findings with regard to cottonseed and olive oils and chicken and beef fats.

The fats were aerated for 40 hr at 60C at an airflow of 1–2 liters/min. Peroxide values for successive batches of rancid cottonseed oil were 122.6, 43.8 and 97.0 and for rancid olive oil, 12.7, 324.0 and 20.8. Although these values varied considerably, no attempt was made to keep the peroxide values at the same levels because conditions of oxidation had been predetermined and were kept as constant as possible. Variations in peroxide number may have been due to different amounts of antioxidants present in different batches of the oils. The fresh and oxidized oils were included at a level of 20% in a diet composed of 30% alcohol-washed casein, 44% dextrose, 3.5% USP XIII salt mixture, 0.5% calcium carbonate, 2% cellulose, and the following vitamin supplements (in mg/kg): (choline dihydrogen citrate 1000, inositol 1000, nicotinamide 100, p-aminobenzoic acid 300, thiamine · HCl 2, pyridoxine · HCl 4, riboflavin 4, Ca pantothenate 10, folic acid 2.5, biotin 0.025, ascorbic acid 25, vitamin K 10, vitamin B_{12} (0.1% trituration in mannose) 5, crystalline beta-carotene 5, alpha-tocopherol acetate 50, free alpha-tocopherol 10 and crystalline vitamin D_2 0.5). To insure an adequate vitamin intake despite the oxidized fat in the diet, each rat was given a weekly oral supplement of 3 drops of Vi Penta Multivitamin suspensions from Hoffman-LaRoche.

The studies were carried out on groups of weanling male rats of the Columbia-Sherman strain. The groups fed cottonseed and olive oils contained 40 rats each and those fed chicken and beef fats contained 20 rats each. All rats were observed for weight gain, food intake, life span and pathology at autopsy. From the groups fed the cottonseed and olive oils, six rats each were sacrificed at 33, 73, 99 and 108 weeks of age for histological examinations and for lipid analyses of serum and tissues. Groups of six rats each were sacrificed at 99 and 112 weeks of age from those fed the chicken and beef fats.

The rats were killed by drawing blood from the heart under chloroform anesthesia. Their organs were removed and immediately weighed; sections were fixed in 10% formalin for histological examination and the rest, as well as the serum, were quickly frozen and stored at −20C. Serum cholesterol was determined according to Bloor et al. (4), and tissue cholesterol, by the method of Sperry and Webb (5). Lipid was extracted from serum and tissues with chloroform:methanol according to Folch et al. (6). Fatty acid compositions were determined by gas-liquid chromatography of their methyl esters prepared by transesterification with 5% methanolic HCL in benzene (2:1). GLC was carried out on a Perkin-Elmer Model 154C vapor fractometer with a 2 meter column packed with diethylene glycol succinate on chromosorb W and with a hydrogen flame ionization detector. The carrier gas was helium at 20 psi; the operating temperature was 225C.

Table I gives the number of rats in each group dying between successive withdrawals of rats. The data shows that the rats fed oxidized cottonseed oil and oxidized chicken fat had a higher death rate than all other groups; the groups with the lowest rates were those fed fresh beef fat and oxidized olive oil. With the exception of the latter, the groups fed oxidized oils had higher death rates than did the corresponding groups fed the fresh oils.

Table II gives the body weight and food intake of all groups at two years of age. At one year, the group fed fresh cottonseed oil had the highest average weight. After two years, the average weights of the animals fed the cottonseed oils were still higher than those of the other groups but not significantly so. Monthly records of body weights and food intake for all groups were maintained through the study.

In the groups fed cottonseed oil, neither age nor the oxidation of the fat influenced the amount of food eaten, which was approximately 15 g per day. The rats fed the olive oils tended to eat somewhat less—particularly those fed the oxidized oil after about one year of age, when the second batch of oxidized olive oil with the higher peroxide number was being fed. However, they evidently adapted to the diet because their intake increased significantly although never to the levels of the rats fed cottonseed oil. The best survival rate was attained by this group fed an oxidized oil with a peroxide number of about 300 for over one year. This suggests that at least these peroxides taken orally were not toxic. This confirms previous studies (7). The food intakes of the groups fed chicken and beef fats for almost the first one and a half years did not differ from those of the rats fed vegetable oils. Later, however, the intakes of those fed chicken fat declined to about 12 g per day, whereas those fed beef fat ate approximately 17 g per day. Fecal fat analyses showed that the latter group excreted 200 mg fat per day whereas those fed chicken fat and the vegetable oils excreted 20

TABLE III

Serum, Liver and Brain Cholesterol Levels in Male Rats Fed Various Fats, for 108 Weeks

	Cholesterol, mg		
	Serum[a]	Liver	Brain
Fresh cottonseed oil	118 ± 9.9	209	3920
Oxidized cottonseed oil	118 ± 9.6	264	4320
Fresh olive oil	99 ± 9.9	562	3930
Oxidized olive oil	95 ± 9.0	607	4190
Fresh chicken fat	123 ± 11.3	527	3870
Oxidized chicken fat	138 ± 13.1	320	3780
Fresh beef fat	155 ± 16.9	336	3670
Oxidized beef fat	119 ± 3.8	384	4110

[a] The serum values are averages of six samples; the liver and brain values were derived from pooled samples. ± values are standard errors.

TABLE IV

Fatty Acid Compositions (%) of Tissue Lipid Extracts from Male Rats Fed Fresh and Oxidized Fats. Each Analysis was Carried Out on the Pooled Tissue from Six Rats.

	C_{14}	C_{16}	C_{16}:1	C_{18}	C_{18}:1	C_{18}:2	C_{20}:4
				Depot fat			
Cottonseed oil							
Fresh							
33 weeks	0.9	20.2	2.2	1.4	22.8	52.5	.....
73 weeks	1.2	18.8	3.1	2.5	26.6	47.6	.....
99 weeks	1.3	17.2	1.6	1.6	24.2	54.1	.....
108 weeks	0.3	16.9	0.6	2.3	30.6	49.3	.....
Oxidized							
33 weeks	0.8	21.6	2.9	2.0	23.7	49.0	.....
73 weeks	0.9	19.0	1.7	2.0	28.6	47.7	.....
99 weeks	1.1	16.2	1.6	2.2	25.7	53.2	.....
108 weeks	....	14.3	0.4	0.7	28.5	56.0	.....
Olive oil							
Fresh							
33 weeks	0.5	11.2	2.6	1.4	78.5	5.9	.....
73 weeks	0.3	13.6	3.7	1.2	70.0	11.2	.....
99 weeks	....	12.4	2.7	1.5	74.6	8.8	.....
108 weeks	....	8.5	1.6	1.2	83.4	5.2	.....
Oxidized							
33 weeks	0.3	11.2	2.9	1.0	79.2	5.4	.....
73 weeks	0.4	16.4	3.7	1.7	66.8	11.0	.....
99 weeks	0.5	11.0	2.7	1.5	72.6	11.7	.....
108 weeks	....	10.2	1.8	2.1	82.8	3.1	.....
Chicken fat							
Fresh							
112 weeks	....	15.2	3.4	1.3	64.7	15.3	.....
Oxidized							
112 weeks	....	14.5	4.0	0.5	69.5	11.3	.....
Beef fat							
Fresh							
112 weeks	0.5	17.1	3.9	3.2	73.8	1.5	.....
Oxidized							
112 weeks	0.4	17.3	3.3	2.9	74.8	1.3	.....
				Liver			
Cottonseed oil							
Fresh							
33 weeks	0.8	25.3	1.2	10.4	13.2	34.1	14.3
73 weeks	0.4	21.1	1.1	11.9	12.7	32.1	17.2
99 weeks	0.2	21.1	....	12.7	10.4	33.6	22.0
108 weeks	....	21.5	0.3	20.1	15.9	19.3	22.9
Oxidized							
33 weeks	0.1	24.1	1.3	11.7	13.4	34.0	14.5
73 weeks	0.3	19.4	....	12.3	15.9	29.1	18.7
99 weeks	....	22.2	....	12.8	11.3	31.4	22.3
108 weeks	....	21.0	0.2	23.6	6.0	21.2	28.0
Olive oil							
Fresh							
33 weeks	0.6	17.9	2.9	6.0	61.6	5.5	5.1
73 weeks	0.4	19.8	1.7	7.1	47.9	13.4	9.0
99 weeks	0.3	18.2	1.1	8.3	43.7	13.1	15.3
108 weeks	....	19.7	0.6	7.9	55.1	3.8	11.4
Oxidized							
33 weeks	0.2	16.0	2.1	7.5	61.2	5.8	6.9
73 weeks	0.3	19.7	1.1	10.9	46.5	8.6	11.5
99 weeks	0.3	16.3	.	13.6	44.9	8.7	16.2
108 weeks	....	22.7	1.9	8.7	51.3	4.6	10.7
Chicken fat							
Fresh							
112 weeks	....	22.7	1.2	15.1	29.6	13.5	17.9
Oxidized							
112 weeks	....	23.7	1.5	14.9	27.1	12.6	20.2
Beef fat							
Fresh							
112 weeks	0.1	21.7	0.5	20.2	35.6	2.7	19.2
Oxidized							
112 weeks	....	19.0	1.5	22.6	40.0	2.6	14.1
				Heart			
Cottonseed oil							
Fresh							
33 weeks	1.2	17.5	2.6	23.3	10.6	30.2	12.4
73 weeks	0.6	11.9	0.7	16.9	13.2	23.4	22.6
99 weeks	....	12.4		23.1	7.9	25.9	30.7
108 weeks	....	15.3	0.1	28.8	9.2	21.9	24.7
Oxidized							
33 weeks	1.6	24.9	3.9	17.5	24.9	22.5	4.3
73 weeks	....	10.4	. .	20.5	12.7	28.7	19.2
99 weeks	....	13.4	....	22.9	10.1	23.9	29.7
108 weeks	....	13.5	0.1	30.5	6.8	23.9	24.9
Olive oil							
Fresh							
33 weeks	0.4	19.4	3.4	16.3	35.3	16.2	7.9
73 weeks	0.5	11.3	1.0	18.0	26.3	13.9	26.4
99 weeks	....	13.7	0.4	21.2	16.0	19.4	31.2
108 weeks	....	12.2	....	29.1	39.9	6.8	10.1
Oxidized							
33 weeks	0.8	14.4	1.1	24.2	35.0	9.8	14.8
73 weeks	0.1	11.1	0.9	16.8	32.8	14.3	21.8
99 weeks	....	11.7		21.2	24.8	9.0	33.3
108 weeks	....	11.5	0.2	26.7	23.8	13.1	24.1
Chicken fat							
Fresh							
112 weeks	....	12.9	0.3	25.9	15.2	17.1	28.3
Oxidized							
112 weeks	....	11.5	0.1	30.3	12.1	16.5	29.2
Beef fat							
Fresh							
112 weeks	....	13.1	0.1	30.4	18.7	6.0	31.7
Oxidized							
112 weeks		16.9	0.3	30.9	20.3	6.9	23.8

to 70 mg per day. This difference in fat excretion does not explain that the groups gained weight at similar rates despite wide variations in their food intakes. The result is in line with observations that weight maintenance can be affected by adaptation to widely varying food intakes (8).

Table III gives serum, liver and brain cholesterol values for all groups. The serum levels of the animals fed olive and cottonseed oils appeared to increase slightly with age, but were not affected by the kind of oil fed. Serum levels of the rats fed chicken and beef fats were somewhat higher but oxidation of the fat did not seem to have any effect.

The liver cholesterol values of the animals fed olive oil were higher than those of the other groups. Although strict statistical evaluation of the data is hardly possible, the consistency of the differences between the animals fed olive oils and those fed other fats suggest that the feeding of olive oils was associated with high liver cholesterol levels. When the animals were 108 weeks old, liver cholesterol values of those fed the cottonseed oils were probably lower than those of any other group.

The cholesterol values of the brain increased with age. The average values were 1.43% at 33 weeks, 1.83% at 73 weeks, 3.03% at 99 weeks and 3.96% at 108 weeks. Feeding of various fats was without affect. Cholesterol analyses of kidney and heart lipids did not reveal any differences.

Thus, the various fats seemed o exert only a mild influence on cholesterol levels and what differences there were did not appear to be correlated to survival rate.

Analyses of fatty acid were carried out on all lipid extracts which had been analyzed for cholesterol. For brevity, only the results obtained for depot fat, liver and heart are given in Table IV. Depot fat analyses revealed no differences other than those reflecting the composition of the dietary fats. Neither age nor feeding of oxidized fat brought about definite changes. Liver lipids, although influenced by the dietary fat, had their own characteristics. Again, no influence of age or oxidation of the dietary fat could be established. The heart lipids seemed to be somewhat less influenced by the composition of the dietary fat. Here, too, age and oxidation of the dietary fat had no detectable effect. Whether or not the characteristic lipid composition of various organs is related to special functions remains to be seen. However, it may be pointed out that all organs of the animals fed the beef fats had a low linoleate content, and that, in the case of those eating fresh beef fat the low linoleate content was associated with a low mortality rate.

In our earlier feeding studies with more highly polymerized fats, we had seen marked enlargement of liver, kidneys and adrenals. In the present studies, liver weights were not significantly influenced by the oxidized fats. The hearts of the two oldest groups fed cottonseed oil were relatively heavier than those of the other groups, especially those fed beef and chicken fats. Two of the four animals fed oxidized cottonseed oil and sacrificed at 108 weeks had particularly heavy hearts. The kidney weights were greater than those of any other group.

Table V summarizes the main histological findings in the animals dying from natural causes. In general, they died from the usual causes of death in older rats: lung infections, cardiorenal diseases and malignant tumors, and there did not appear to be definite differences between the groups. One possible exception may have been a higher incidence of malignant tumors in the groups fed olive oil and chicken fat. A chi-square analysis gave a P of about 5%, which is just enough to invite further studies. Chicken fat contained 70 μg per 100 g of stilbesterol whereas the other fats contained about one-third this amount. Among the 91 rats sacrificed at about two years of age, the difference did not exist; eight malignant tumors distributed over all the groups were observed. It is known that old rats develop unspecific cardiac and renal lesions. In the heart, muscle fibers were damaged by accumulations of round cells and the appearance of fibrous tissue. The greatest difference was found between the groups fed fresh and oxidized cottonseed oils. In the latter there were significantly more scarring and round cell infiltration, that is, muscle damage before the laying down of connective tissue. The group fed this diet and sacrificed after two years had by far the largest hearts. In other groups there seemed to be more severe lesions in the groups fed oxidized chicken and beef fat than in their controls, but not significantly so.

TABLE V

Incidence of Deaths from Various Causes Among Male Rats Fed the Different Fats. Diagnosis was Based on Histologic Evidence

	No. of deaths	Inflammatory lung disease	Cardiorenal insufficiency	Cancers[a]	Undetermined
Cottonseed oil					
Fresh	15	8	2	0	5
Autoxidized	18	6	3	3	6
Olive oil					
Fresh	16	4	3	4	5
Autoxidized	13	3	4	3	3
Chicken fat					
Fresh	6	0	2	2	2
Autoxidized	11	3	5	3	0
Beef fat					
Fresh	5	0	1	1	3
Autoxidized	8	2	1	1	4

[a] Metastasizing tumors of the pituitary and of the adrenal have been excluded.

The kidneys were graded with regard to hyaline casts and accumulation of round cells. Among those dying spontaneously, the number of lesions was higher in the animals fed oxidized fats, with the exception of oxidized olive oil. In the animals that had been sacrificed, there was not much difference between those on fresh and oxidized fats, but there was a highly significant difference between the relatively few animals with lesions among those fed the olive oils and the greater frequency in the other groups.

It is evident that even the relatively mild damage to the fats led to significant biological changes. Oxidized fats reduced the survival rate except for the curious case of oxidized olive oil. The feeding of oxidized cottonseed oil was associated with the occurrence of more marked heart and kidney lesions.

It is evident that the biological activities of the various fats could hardly be correlated with their chemical properties, which makes it more necessary to carry out long-term nutritional studies.

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Nutritive Value of Heated Vegetable Oils

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Abstract

Peanut, sesame and coconut oils were heated at 270C for 8 hr, in an open iron pan. These fats were fed to albino rats at 15% level in otherwise adequate diets. All rats fed heated fats showed a growth depression. Livers of rats receiving heated oil were congested and showed extensive periportal fatty infiltration. Rats on heated peanut oil showed i) reduced B-vitamin storage in the liver, ii) increased glucose and cholesterol levels in the blood and iii) a disruption in the digestion and absorption of carbohydrates.

Introduction

HEATED OILS have been shown to be poorly absorbed (13), to produce cancerous tumors (11) and to cause symptoms resembling that due to vitamin E deficiency (9). Lower nutritive value is also believed to be due to the destruction of certain vitamins, especially vitamin A (6). Further, it has been shown that the oxidation products of fats produced inhibitory effect on certain enzyme systems (4,12).

Consumption of fried foods in India is probably higher than in any other country. The conditions of heating are known to vary widely. The oils are usually heated in open air pans to about 200–300C depending on the type of preparation and the heated oils are reused.

The present investigations were carried out to evaluate the effect of heat on some edible oils commonly used in the country.

Experimental and Results

Preparation of Heated Oils

Peanut, sesame and coconut oils were heated continuously at 270C for 8 hr in an open pan made of iron. The heated oils were stored in pyrex glass bottles at 0C.

Influence of Heating the Oil on Its Nutritive Value and Fat Deposition in the Liver

Thirty-six Wistar strain albino rats, five weeks old and weighing about 40–50 g from our laboratory stock colony were allotted to six groups in a randomized block design and were housed in individual cages. They were fed *ad lib.* on purified diets (fat, 15%; casein, 12%; sugar, 10%; salt mixture, 4%; vitaminized starch, 1% and corn starch, 58%) in which the fat was supplied by raw or heated peanut, sesame or coconut oils. At the end of six weeks feeding, the animals were killed by bleeding through the abdominal aorta. Livers, spleens, stomachs and kidneys were removed and weighed. The liver lipids were estimated by the method of Hawk (7). The results are presented in Table I.

The results show clearly that in all the three cases the heated oil has adversely affected the gain in weight. The feed efficiency ratio, calculated as increase in weight per gram of fat consumed, is significantly lower in groups receiving the heated oils. The livers of rats receiving the heated oil are significantly heavier than the controls. The lipid content of the livers is nearly twice that of the control groups. The livers showed signs of congestion and extensive periportal fatty infiltration. No significant changes were found in the weights of stomach, kidney and spleen of the two groups of animals.

Influence of Heated Oils on the Levels of B-Vitamins in the Livers

Eighteen male weanling albino rats, about 4 weeks old, were distributed into three groups in a random block design and were depleted of their B-complex stores by feeding a deficient diet for a period of two weeks. One group was sacrificed at this stage for determining the basal stores of B-vitamins in the liver which were as follows: thiamine, 11.7 μg; riboflavin, 44.8 μg; niacin, 285 μg; pantothenate, 185 μg and pyridoxine, 12.5 μg. The two other groups were fed on a purified diet containing 15% of raw or heated peanut oil for a period of two weeks, after which

TABLE I

Influence of Heating the Oil on Its Nutritive Value and Fat Deposition in the Liver

Dietary fat	Body weight		Mean gain per week (g)	Mean daily food intake (g)	Feed efficiency[a]	Liver weight		Liver fat %
	Initial g	Final (g)				(g)	g/100 g body weight	
Peanut oil:								
Raw	42.5	120.7	13.0 ± 0.5[b]	11.3	1.1	4.58	3.8 ± 0.3	3.9 ± 0.3
Heated	42.0	72.1	5.0 ± 0.9	7.1	0.67	4.11	5.7 ± 0.6	7.1 ± 0.4
Sesame oil:								
Raw	42.5	102.5	10.0 ± 0.8	9.5	1.0	4.30	4.2 ± 0.7	4.8 ± 0.3
Heated	42.4	68.3	4.3 ± 0.7	5.9	0.7	4.10	6.0 ± 0.5	7.8 ± 0.3
Coconut oil:								
Raw	43.1	109.2	11.0 ± 1.1	9.5	1.1	4.70	4.3 ± 0.3	4.5 ± 0.3
Heated	42.8	69.9	4.5 ± 0.7	6.1	0.7	4.05	5.8 ± 0.4	7.0 ± 0.3

[a] Increase in weight per gram of fat intake.
[b] Standard error of the mean.