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EFFECTS OF SATURATED AND UNSATURATED LONG CHAIN FATTY ACIDS FED WITH AND WITHOUT ESSENTIAL FATTY ACID ON VARIOUS COMPONENTS OF LIPID AND CARBOHYDRATE METABOLISM

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The degree of saturation of long chain fatty acids and the availability of essential fatty acids in diets fed to rats have been shown to affect metabolic pathways in the liver of the rat. Variations in the type or amount of dietary fat seem to alter serum and liver cholesterol patterns. Essential fatty acid deficiency has resulted in increased basal metabolic rate and increased oxidation of several acids of the Kreb's cycle.

The purpose of this experiment was to investigate what influence, if any, the degree of saturation of long chain fatty acids, when fed with and without essential fatty acid, would have on various components of lipid and carbohydrate metabolism.

The experiment (pilot and follow-up) was designed to permit comparisons between effects of the following factors on various metabolic functions: (1) presence of essential fatty acids in naturally occurring fats containing long chain polyunsaturated fatty acids (corn oil versus tuna oil), (2) supplementation of fats containing long chain saturated fatty acids with an essential fatty acid (hydrogenated peanut oil versus hydrogenated peanut oil plus linoleic acid), (3) combination of saturated and unsaturated fats with or without essential fatty acid (hydrogenated peanut oil plus linoleic acid versus hydrogenated peanut oil plus tuna oil), and (4) degree of saturation of dietary fatty acids, with and without essential fatty acids (corn oil versus hydrogenated peanut oil plus linoleic acid, tuna oil versus hydrogenated peanut oil, and hydrogenated peanut oil plus tuna oil versus either tuna oil or hydrogenated peanut oil).

All rations contained 20% by weight of protein, approximately 380 calories per 100 grams ration and adequate amounts of minerals and vitamins. The rats fed corn oil served as the control.

The pilot study groups were fed 15% fat diets and the follow-up study groups were fed 5% fat. The group given tuna oil ration in the pilot study did not accept the oil, and after one week was given a 5% tuna oil (TO) ration. The vitamin E content of TO rations was doubled to prevent oxidation of this highly unsaturated oil.

Analysis was made at two and four weeks in the pilot study, and at one day, and two and three weeks in the follow-up study.

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Livers were assayed for activities of the glucose-6-phosphatase and fructose diphosphatase enzyme systems, for glycogen, total lipid, cholesterol and phospholipid. Serum was assayed for cholesterol.

Results will deal only with the pilot study except when noted, because all data for follow-up have not been collected.

As expected from previous reports, rats fed an essential fatty acid deficient diet did not gain as much weight as did those fed corn oil (CO) or hydrogenated peanut oil plus linoleic acid (HPO+L). After four weeks, the CO fed group had gained only slightly more weight than the group fed HPO+L, thus degree of saturation did affect weight gain, but to a lesser extent than did essential fatty acid deficiency (Fig. 1).

Food efficiency, grams weight gained per gram of food eaten, was greater for CO fed group than for the other three groups; and the next to highest food efficiency was shown by the rats fed TO. Supplementing HPO with L resulted in greater food efficiency than feeding HPO with no supplement. These data suggest that food efficiency is more dependent on degree of saturation than on presence of essential fatty acid (EFA). Food efficiency ratios for each group at 4 weeks were as follows: CO, 0.46 ± 0.01 ; HPO + L, 0.35 ± 0.01 ; HPO, 0.31 ± 0.01 ; and TO, 0.41 ± 0.01 .

The activity of glucose-6-phosphatase (G-6-Pase), the enzyme which removes phosphorus from glucose-6-phosphate to give glucose, was slightly altered by the type of fat in the diet. There were only slight differences in G-6-Pase activity among groups at two weeks. By four weeks feeding TO in place of CO resulted in greater G-6-Pase activity. This may have been due to EFA deficiency in the TO group, or to differences in the utilization of the two oils, or to the lower level (5%) of TO compared to the 15% CO diet. If this rise in G-6-Pase activity in the TO group had been due only to EFA deficiency, it seems that feeding HPO would have resulted in greater activity than would feeding HPO+L, but this was not the case. Thus, EFA deficiency was not solely responsible for increased G-6-Pase activity (Fig. 2).

Feeding TO or HPO+L in place of CO caused higher fructose diphosphatase activity at four weeks when compared to the control. The enzyme, fructose diphosphatase (FDPase), removes phosphorus from fructose 1,6 phosphate to give fructose-6-phosphate. Because of lack of a 5% fat control for the 5% TO diet, I cannot conclude that either EFA or degree of saturation had an influence on FDPase activity. However, you will notice that by four weeks EFA deficient groups showed slightly increased FDPase activity over the activity in these groups at two weeks (Fig. 3).

Saturated fat without EFA was the only dietary fat which significantly influenced liver glycogen when compared to the control group. Calculations for the overall period showed that group 3 had a significantly lower percentage of liver glycogen than did the group fed

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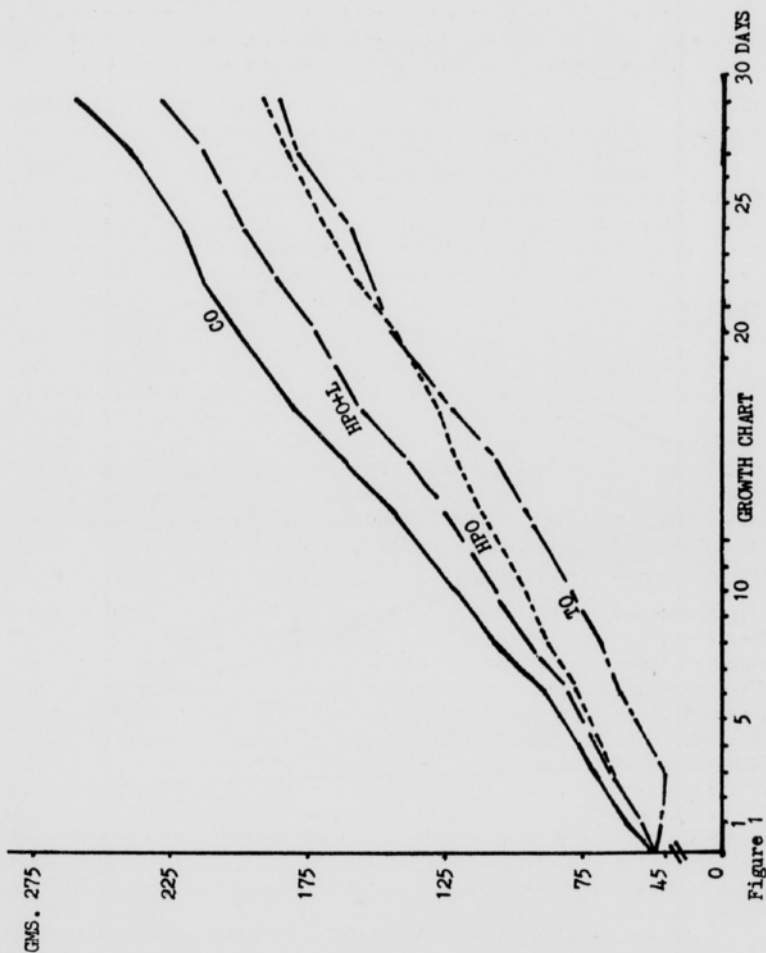


Figure 1 Growth rate of rats fed a 15% fat diet for 28 days. CO — corn oil, HPO — hydrogenated peanut oil, L — linoleic acid, and TO — tuna oil.

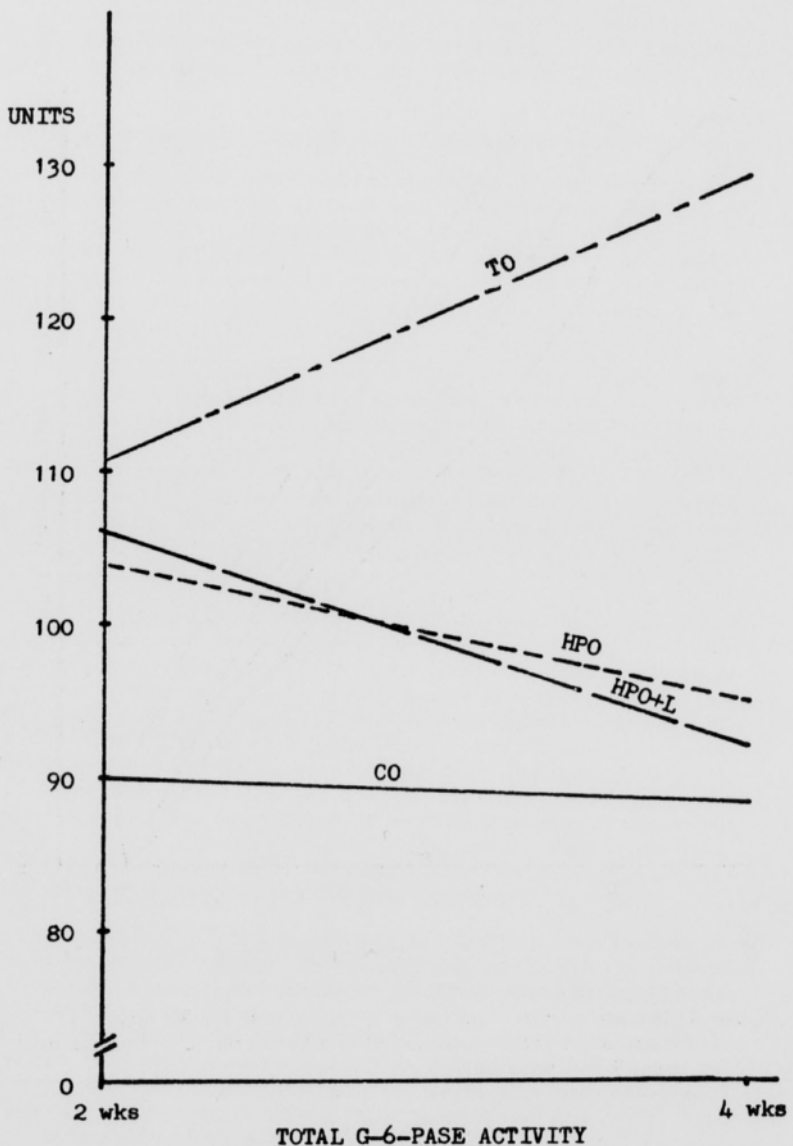


Figure 2 Total glucose-6-phosphatase activity (units/100 g body weight) of rats fed a 15% fat diet for 28 days. CO — corn oil, HPO — hydrogenated peanut oil, L — linoleic acid and TO — tung oil.

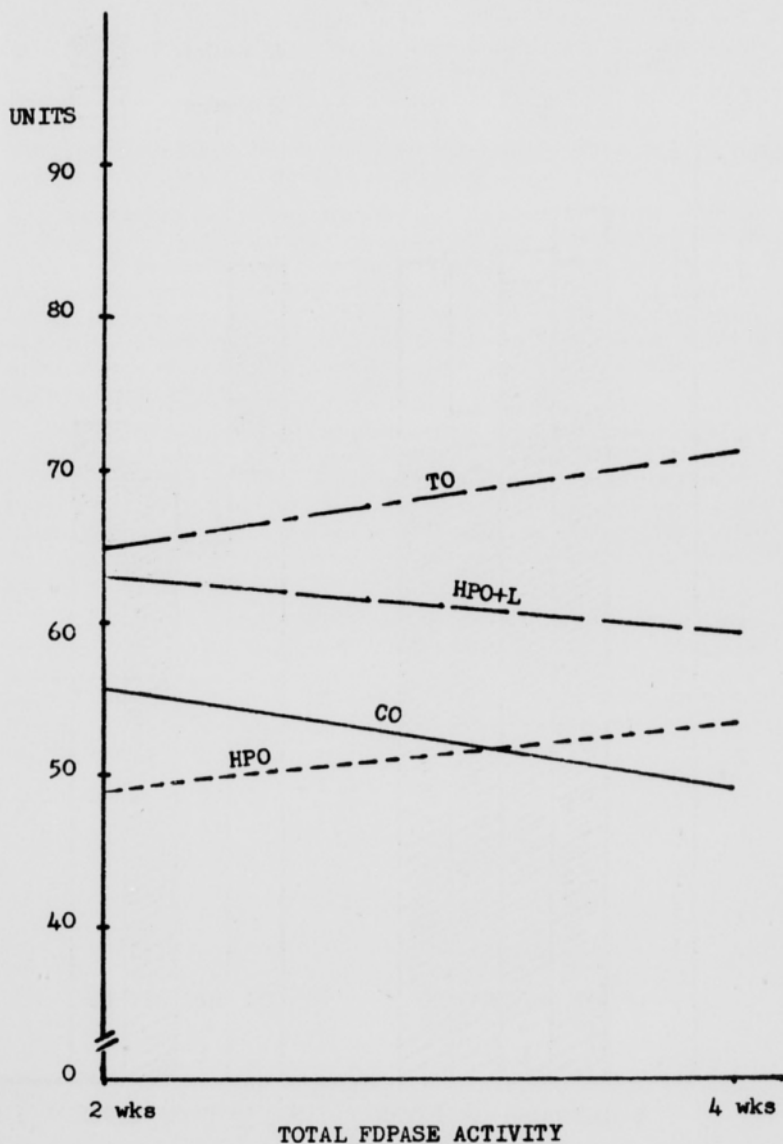
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Figure 3 Total fructose-1,6-diphosphatase activity (units/100 g body weight) of rats fed a 15% fat diet for 28 days. CO — corn oil, HPO — hydrogenated peanut oil, L — linoleic acid and TO — tuna oil.

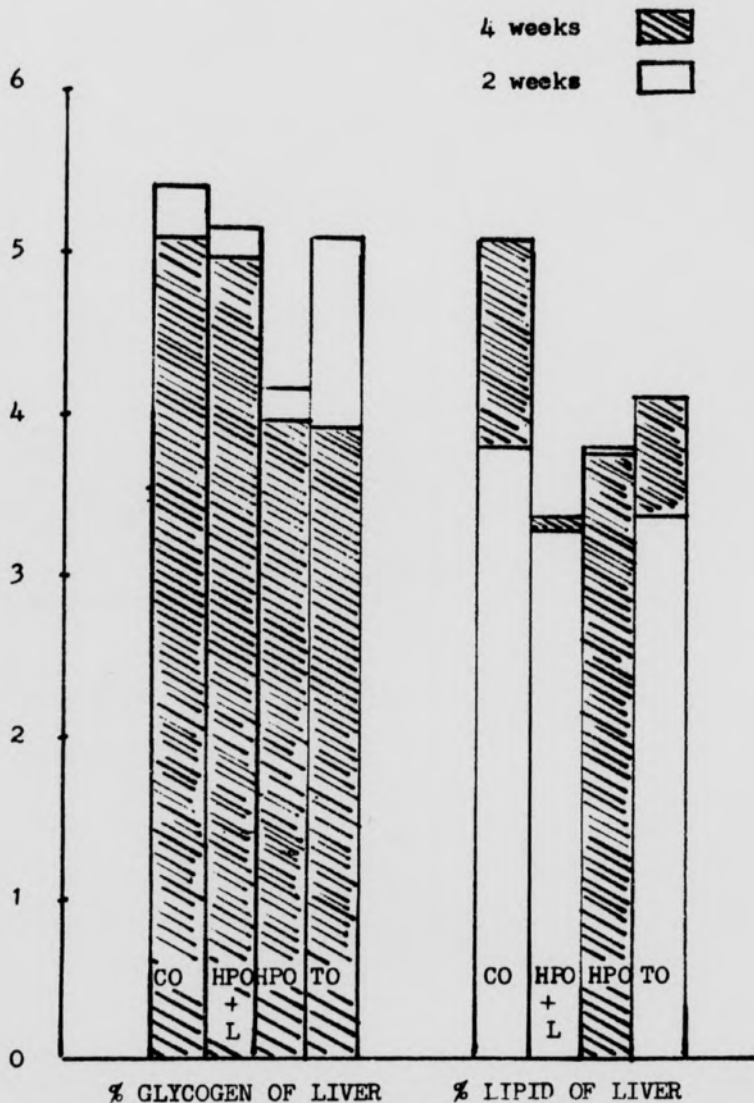


Figure 4 Total liver lipid and glycogen (percent of liver) of rats fed a 15% fat diet for 28 days. CO — corn oil, HPO — hydrogenated peanut oil, L — linoleic acid, and TO — tuna oil.

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HPO+L. If depressed glycogen levels in livers from the HPO group were due to EFA deficiency, then a comparable 15% TO diet would be expected to give similar results. Indeed, even with the 5% TO diet, the average percentage of glycogen in livers from rats given TO was somewhat lower than that of control rats (3.92 ± 0.66 versus 5.09 ± 0.37), although this difference was not significant at the 5% level due to wide variations within the TO group (Fig. 4).

The feeding of polyunsaturated fat resulted in greater changes in liver lipids within groups from two to four weeks than did the feeding of saturated fat. Feeding CO or TO for four weeks raised lipid levels significantly over those in livers from rats fed saturated fat for the same period. The higher liver lipid levels may have been due to increased lipid synthesis by the liver, mobilization of lipids from adipose tissue to liver, lipid storage in the liver, or a combination of these factors (Fig 4).

Serum cholesterol levels (mg cholesterol per 100 ml serum) for the overall experimental period were higher in the CO (209 ± 11) and TO (162 ± 13) fed groups than in the groups fed a saturated fat (HPO+L, 163 ± 7 and HPO, 146 ± 7). Serum cholesterol levels were not significantly different with or without linoleic acid supplement; thus under the conditions of this experiment, EFA did not show an effect in lowering serum cholesterol.

These results call for more work pertaining to the inter-relationships of fats in the diet with carbohydrate and lipid metabolism. Some questions may be answered in the follow-up by feeding the other fats at the level at which TO was accepted, and by doing assays at intervals from a few days to several weeks while the rats continue to eat the specified fat diet.