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Long- and Medium-Chain Triacylglycerols in Nutritional Support of Liver Regeneration of Partially Hepatectomized Rats

V. BLÁHA, Z. ČERVINKOVÁ[1], J. ŠIMEK[1], H. LOTKOVÁ[1], Z. ZADÁK

Department of Metabolic Care and Gerontology, Faculty Hospital and [1]Department of Physiology, Faculty of Medicine, Charles University, Hradec Králové, Czech Republic

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Summary

An appropriate choice for a suitable diet during liver regeneration still remains an enigma. To investigate the effect of isocaloric enteral feeding with medium-chain triacylglycerols (MCT) and long-chain triacylglycerols (LCT) supplement (MCT+LCT, 40 %: 60 % w:w) (178 kJ/kg b.w./24 h), rat liver regeneration was studied 24 and 72 h after partial hepatectomy. The liver DNA synthesis 24 h after partial hepatectomy was significantly higher in the MCT+LCT-supplemented rats (30.2±8 x 2.10^3 dpm/mg liver DNA) compared to MCT-treated animals (18.1±5.7 x 10^3 dpm/mg liver DNA). Liver protein synthesis was non-significantly elevated both 24 and 72 h after surgery in MCT+LCT-supplemented rats (13.7±1.1 and 10.9±3.1 x 10^3 dpm/mg liver protein). Seventy-two hours after partial hepatectomy, the hepatocyte mitotic activity was significantly increased in MCT+LCT- supplemented group vs. LCT- or MCT-fed rats (3.3±0.7 vs. 1.9±0.7 or 1.0±0.6 mitoses per 1000 hepatocytes), thus exhibiting an increased proliferative potential. The results showed a qualitative difference according to the proportion of MCT to LCT in the enteral supplements. Overfeeding with MCT decreased body weight, increased liver weight by its fatty infiltration, increased rat mortality rate and reduced spontaneous caloric intake. We conclude that the balanced supplement of MCT+LCT (40 % : 60 % w:w) preserves liver regeneration, whereas overfeeding with MCT seems to be deleterious.

Key words

Medium-chain triacylglycerols • Long-chain triacylglycerols • Liver regeneration • Partial hepatectomy • Nutrition

Introduction

Dietary factors substantially affect hepatocyte proliferation. The rise in nutrient concentration initiates many biochemical processes in a sequence of events that are thought to be required for eventual DNA synthesis and cell division (Bettger and McKeehan 1986). It has been shown that the remnant liver after major hepatectomy is mainly dependent upon the oxidation of fatty acids in the early postoperative period, whereas glucose oxidation prevails thereafter (Šimek and Sedláček 1965, Ngala Kenda *et al.* 1984). During liver regeneration, the energy charge level was decreased significantly by portal infusion of (+)-octanoylcarnitine, a potent inhibitor of fatty acid oxidation. This was even true during intravenous glucose infusion. In contrast, the intraportal infusion of an inhibitor of glycolysis, sodium fluoride, did not affect the energy load 12-24 h after

Fax +420 2 293 894
E-mail physres@biomed.cas.cz
http://www.lf2.cuni.cz/physiolres/physiolres.htm

hepatectomy (Nakatani *et al.* 1981a,b). The elevated $NADH/NAD^+$ reflects β-oxidation of fatty acids and inhibits the pyruvate dehydrogenase complex (Schofield 1986). A rise of total ketone body concentration and a rise in non-esterified fatty acid levels in the plasma suggests that oxidation of fatty acids is enhanced (Bode *et al.* 1990).

Recently, fat emulsions enriched with medium-chain length triacylglycerols (MCT) have been recommended in liver failure (Blackburn and Stein 1991) and in liver transplant patients (Kuse *et al.* 1990). We had shown that MCT-enriched enteral nutrition could be beneficial in nutritional support of the prereplicative period of liver regeneration after major hepatectomy (Bláha *et al.* 1991) or after application of hepatotoxic agent (Červinková and Drahota 1998). Liver DNA synthesis and protein synthesis were enhanced. Hence, the aim of the present study was to evaluate the effect of medium-chain and long-chain triacylglycerols (MCT+LCT, 40/60 w:w) at later stages of liver regeneration. The nutritional regimen was investigated in relation to posthepatectomy liver DNA synthesis, liver protein synthesis and hepatocyte mitotic activity.

Method

Animals

Male albino Wistar rats (190-210 g initial body weight) were kept individually in a temperature-controlled room with free access to water and stock rat pellets throughout the experiment (DOS 2b pellets, Velaz, Prague, Czech Republic; energy content 15.8 MJ/kg of diet; energy content covered 25 % by protein, 22 % by fat and 53 % by carbohydrates).

Surgery

The animals were submitted to partial hepatectomy (65-70 % of the liver tissue was removed comprising the left and median lobules of the liver) under mild ether narcosis (Higgins and Anderson 1931) between 09:00 and 11:00 h. The rats were decapitated 24 and 72 h after partial hepatectomy. After exsanguination, the liver was removed for analysis.

Experimental protocol

The spontaneous consumption of the standard laboratory diet was measured between 08:00-09:00 h every morning throughout the experiment. The effect of MCT+LCT (40 % : 60 % w:w) supplemented enteral nutrition in partially hepatectomized rats was studied in the following groups of rats:

1. Control rats (a = 21, partial hepatectomy; water and stock rat pellets *ad libitum*).

After partial hepatectomy, the animals of following groups were fed the lipid supplement besides the water and stock rat pellets *ad libitum*:

2. MCT rats (n = 18, partial hepatectomy, MCT supplement);
3. LCT rats (n = 19, partial hepatectomy, LCT supplement);
4. MCT+LCT rats (n = 19, partial hepatectomy, MCT+LCT supplement).

MCT, LCT or MCT+LCT supplements were given by intragastric tube in two doses daily, at 11:00 and 18 :00 h, in total 178 kJ/kg b.w./24 h. Miglyol 812 (Dynamit Nobel, Germany) was the source of MCT (C6 3 % max., C8 60-65 % , C10 30-45 %, C12 5% max.). For long-chain triacylglycerols Intralipid 20% was used (Kabi Vitrum, Sweden).

Liver DNA synthesis

For the estimation of liver DNA synthesis 24 h after partial hepatectomy, the rats were labeled by an intraperitoneal injection of ^{3}H-thymidine (200 μCi/kg b.w. one hour before sampling). The peak of liver DNA synthesis might be recorded 21-24 h after 65-70 % hepatectomy (Bucher 1963). The radioactivity of livers was measured according to Short *et al.* (1969) using a Delta 300 scintillation counter (Nuclear Chicago). The liver DNA concentrations were determined by the diphenylamine reaction (Burton 1956) from which the specific DNA activity was calculated.

Liver protein synthesis

Liver protein synthesis was measured 24 h and 72 h after partial hepatectomy in the rats labeled by an intraperitoneal injection of ^{14}C-leucine (25 μCi/kg b.w. 30 min before sampling) (Bláha *et al.* 1991). The radioactivity of the liver was measured by means of a Delta 300 scintillation counter. The liver protein content was measured according to Lowry *et al.* (1951).

Hepatocyte mitotic activity

The mitotic activity of hepatocytes was evaluated 72 h after partial hepatectomy from 7 μm thick paraffin liver sections stained with haematoxylin and eosin. The mitotic index was expressed as the number of mitoses per 1000 hepatocyte nuclei (3000 nuclei in each rat were

evaluated). The shape of the mitosis-versus-time curve shows an increase 30 h after partial hepatectomy and cyclical activity later (Bucher 1963).

Statistical methods

The results are expressed as mean ± S.E.M. The statistical analyses were based on Student's t-test and ANOVA. Differences were considered as significant if the P value was less than 0.05.

Table 1. Body weight loss (g) and liver weight (50.1±2.3 g/kg b.w. in the intact rats) 24 and 72 h after partial hepatectomy

Weight loss (g)	24 h	72 h
Control	20±4	1±5
LCT	22±4	3±6
MCT	13±6^{+}	28±1*$^{+o}$
MCT+LCT	18±4	8±8
Liver weight (g/kg b.w.)	24 h	72 h
Controls	17.2±8.3	35.6±2.6
LCT	18.1±2.9	36.7±1.8
MCT	19.9±1.8	39.9±0.7*$^{+}$
MCT+LCT	17.9±1.2	37.8±3.4

*Results are means ± S.E.M., *p<0.05 vs controls, $^{+}$p<0.05 vs LCT, ^{o}p<0.05 vs MCT+LCT.*

Results

The MCT-supplemented rats showed a decreased pellet intake and did not gain body weight. The mortality in this group was 28 %, i.e. 5 animals did not survive 48 h of the experiment.

Body weight loss and liver weight (Table 1). The surgical stress induced by partial hepatectomy reduced the body weight in all experimental groups after 24 h, with recovery 72 h after surgery. The body weight loss 24 h after partial hepatectomy was the lowest in MCT animals, which was statistically significant in comparison to the LCT rats. However, 72 h after surgery the weight loss was most marked in the MCT group, but the relative liver weight was significantly higher and the livers exhibited steatosis.

Table 2. Total liver DNA content in the partially hepatectomized rats (2.62±0.32 mg/g wet weight in the intact rats)

Liver DNA content (mg/g wet weight)	24 h	72 h
Controls	3.38±0.11	2.70±0.08
LCT	3.05±0.23	2.58±0.15
MCT	3.32±0.47	3.08±0.11*
MCT+LCT	3.02±0.21*	3.65±0.46*

*Results are means ± S.E.M, *p<0.05 vs controls.*

Liver DNA content (Table 2). Three days after partial hepatectomy, the liver DNA content was significantly higher in MCT and MCT+LCT group compared to the controls. In contrast, 24 h after surgery it was still reduced in MCT+LCT-supplemented animals as compared to controls.

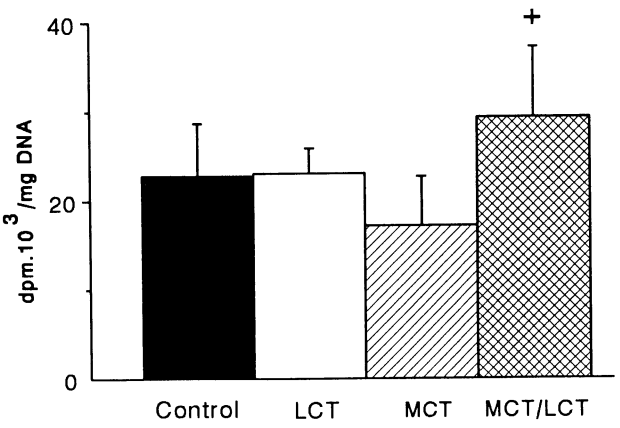

Fig. 1. *Liver DNA synthesis 24 h after partial hepatectomy. Results are means ± S.E.M, $^{+}$p<0.05 vs MCT.*

Liver DNA synthesis (Fig. 1). The specific DNA activity 24 h after partial hepatectomy was significantly higher in MCT+LCT-supplemented rats as compared to MCT-treated animals (30.2±8.2 vs 18.1±5.7 x 10^{3} dpm/mg liver DNA).

Table 3. Liver protein content 24 and 72 h after partial hepatectomy (161.2±40.4 mg/g wet weight in intact rats)

Liver protein content (mg/g wet weight)	24 h	72 h
Controls	115.0±9.7	162.1±38.1
LCT	114.3±4.5	180.1±40.1
MCT	136.1±21.9	160.1±50.3
MCT+LCT	102.1±9.2	147.2±18.1

Results are means ± S.E.M.

Liver protein content (Table 3). Liver protein content did not change significantly. The liver protein synthesis 24 h after hepatectomy (controls 10.8±2.4 x 10^3 dpm/mg liver protein) was significantly lower in MCT-fed rats (9.7±3.5) compared to LCT and MCT+LCT animals (15.8±3.8 and 13.7±1.1 x 10^3 dpm/mg liver protein, respectively). No significant differences were found in liver protein synthesis 72 h after hepatectomy.

Hepatocyte mitotic activity (Fig. 2). The mitotic activity of hepatocytes was significantly decreased in LCT and MCT groups vs. MCT+LCT-supplemented and control rats.

Total energy load and spontaneous stock pellet consumption (Fig. 3). The spontaneous stock pellet consumption was measured, and a lipid supplement (178kJ/kg b.w.) was added in the LCT, MCT and/or MCT+LCT groups. The energy load-versus-time curve shows that 24 h after partial hepatectomy, a significant decrease in energy consumption in MCT and MCT+LCT group occurred, whereas an increase was found in LCT-fed rats. We found in the MCT group 48 h after surgery that the energy intake was lower as compared to the LCT rats, but there were no significant differences among control, LCT and MCT+LCT animals. Finally, the energy intake 72 h after surgery was the lowest in MCT-fed rats and remained significantly low in MCT+LCT animals. A further increase was recorded in control and LCT animals.

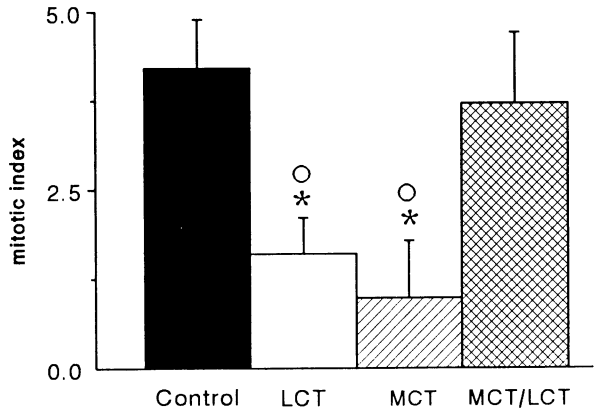

Fig. 2. *Hepatocyte mitotic activity 24 and 72 h after partial hepatectomy. Results are means ± S.E.M, *p<0.05 vs controls, °p<0.05 vs MCT+LCT (40 % : 60 % w:w).*

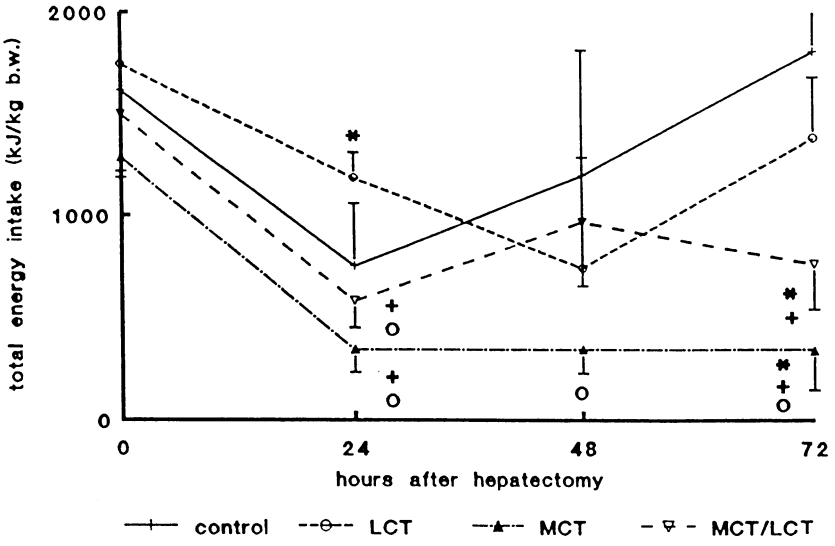

Fig. 3. *Total energy load (spontaneous stock pellet consumption plus lipid supplement) 24 and 72 h after partial hepatectomy. The lipid supplement was 178kJ/kg b.w. in LCT, MCT and MCT+LCT (40 % : 60 % w:w) group. Results are means ± S.E.M, *p<0.05 vs controls, +p<0.05 vs LCT, °p<0.05 vs MCT+LCT (40 % : 60 % w:w).*

Discussion

Several previous studies have suggested a favorable effect of MCT-containing parenteral or enteral nutrition over LCT-based diet in liver regeneration. The protein synthesis in partially hepatectomized rats fed for 7 days after hepatectomy with 50 % MCT/50 % LCT during total parenteral nutrition was significantly enhanced compared with that of LCT-fed animals (Farriol *et al.* 1990). In ultrasound density studies of the liver during parenteral nutrition with MCT+LCT versus LCT fat emulsions, Baldermann *et al.* (1988) found no increase in size or density of the liver. They therefore concluded that during the administration of MCT+LCT fats a "fatty liver" was less likely to develop than during the administration of pure LCT fat emulsion. Hamada (1993) suggested that the combination of LCT with MCT may be more beneficial over LCT in the recovery phase of liver regeneration. Although the liver transplant patients are more vulnerable to drug-induced and toxic influences and are therefore very sensitive to the side effects of total parenteral nutrition, the use of MCT+LCT fats instead of pure LCT fats showed no evidence of any fatty changes in the liver biopsy caused by the diet (Kuse *et al.* 1990). The influence of MCT+LCT in enteral nutrition on visceral protein synthesis in hepatectomized rats was studied, and 60 % MCT/40 % LCT enteral modular diet *ad libitum* for 8 days after partial hepatectomy was recommended Pérez-Bartolí *et al.* (1988).

The animals in our study were kept under the same conditions, as far as the temperature, noise, light and surgery are concerned. The enteral supplements only differed in the proportion of MCT to LCT. The results obtained should be attributed to this qualitative difference. Several significant differences were found in the MCT group. The body weight showed a decreasing tendency, and the final body weight gain was significantly lower (Table 1). In all other groups, the body weight increased after an initial loss. The MCT+LCT (40 % : 60 % w:w) and MCT-supplemented rats consumed significantly less energy and/or standard laboratory chow. When the mortality in the MCT-group was evaluated, we found that out of 18 animals, five (28 %) did not survive 48 h of the experiment. The medium-chain triacylglycerols are known to be highly ketogenic. After partial hepatectomy, the MCT-induced hyperketonemia might have altered the metabolic pathways. Nevertheless, we did not measure the serum levels of ketone bodies in our experiments. The intensive β-oxidation of medium-chain fatty acids increases intramitochondrial acetyl-CoA, which is known to be a potent inhibitor of some crucial metabolic enzymes. This would also result in a reduction of mitochondrial free-CoA, which is necessary for catabolizing energy substrates *via* β-oxidation of fatty acids, pyruvate decarboxylation and/or oxidative transformation of α-ketoglutarate *via* Krebs' cycle. (Pérez-Bartolí *et al.* 1988). Another mechanism involved in the free-CoA decrease could result from changes in carnitine metabolic pathways after partial hepatectomy. The catabolism of MCT is reported to be carnitine-independent (Bremer 1983). Nevertheless, the carnitine acyltranslocase and transferase system of the inner mitochondrial membrane are important for the liberation of free-CoA (Rössle *et al.* 1990). After partial hepatectomy, the total liver carnitine pool is reduced and the hepatic ratio of free-to-esterified carnitine decreases on day 1, predominantly due to increased mitochondrial acylcarnitine (French *et al.* 1985). The resulting reduction in free-CoA might accelerate the metabolic alterations mentioned above. There is a theoretical disadvantage of MCT-containing emulsions for the support of liver regeneration. The easier passage of medium-chain fatty acids through the blood-brain barrier could also have a negative effect in the survival rate of MCT-rats, since substances such as octanoate have been held partly responsible for the appearance of hepatic encephalopathy (Staeffen *et al.* 1979). However, no apparent changes in the behavior of the MCT group have been observed in comparison to other animals.

The effect of the MCT-supplemented diet was dependent on the proportion of MCT given. We did not observe any mortality in the MCT+LCT fed animals. In addition to the reduced energy intake, the MCT+LCT rats regained their body weight after the early postoperative weight loss. The liver DNA content 72 h after hepatectomy was significantly higher in MCT+LCT-supplemented animals, and was also elevated in other groups compared to controls, mainly due to the changes of liver wet weight. The DNA content in MCT group remained comparable to the MCT+LCT group. The liver DNA synthesis 24 h after partial hepatectomy was significantly higher in the MCT+LCT-supplemented rats, as did the liver protein synthesis both 24 and 72 h after surgery. The enhancement of liver protein synthesis after partial hepatectomy was also reported by others (Farriol *et al.* 1990, Pérez-Bartolí *et al.* 1988). The hepatocyte mitotic activity in the MCT+LCT group was significantly increased 72 h after partial hepatectomy, thus showing an

increased proliferative potential. The shape of the mitosis-versus-time curve exhibited the first increase 30-32 h after partial hepatectomy (Bucher 1963) and 16-18 h cyclic activity later (Heine and Klinge 1973). The third mitotic peak was found approximately 72 h after surgery.

Replacing dietary long-chain triacylglycerols with medium-chain triacylglycerols in our study produced significant changes in caloric intake. We found that during 72 h of the experimental protocol increasing concentrations of MCT in the diet reduced spontaneous caloric intake in the MCT+LCT and MCT rats. This confirms earlier findings (Pirk and Skalla 1971, Friedman *et al.* 1983, Edens and Friedman 1984), although they did not study partially hepatectomized animals. The possible mechanisms involved are not fully understood. The MCT-containing diet are emptied from the stomach more slowly than the test meals containing LCT (Pirk and Skalla 1971), indicating that the gastric factor could be involved in the effect of MCT on food intake. Changes in the oxidation of metabolic fuels in the liver have been implicated in the control of food intake, which was reduced in MCT-fed normal and diabetic rats, and was accompanied by a greater degree of hepatic oxidation of ingested fats (Friedman *et al.* 1983, Edens and Friedman 1984).

Several mechanisms could be involved in the higher regenerative capacity of the MCT+LCT-supplemented animals in our study. The changes in ATP/ADP and Pi indicate that the liver regenerative processes depend on energy substrates that may in fact limit or modulate the rate of rat hepatic regrowth (Campbell *et al.* 1990). The metabolic changes in the posthepatectomy period showed that the energy requirements are mainly dependent upon the oxidation of fatty acids, but not of glucose, in the early postoperative period (Nakatani *et al.* 1981a). Thus the MCT as a high energy and rapidly metabolizable fat substrate could be preferably used by the regenerating liver during the period of greatly decreased ATP levels. Medium-chain triacylglycerols or ketone bodies derived from them could be oxidized in extrahepatic tissues and thus increase the total energy production of the body during the early postoperative period. Nutritional fats enriched with MCT may favorably affects mitochondrial coupling. As we have shown previously, the oxidation of shorter-chain fatty acids, namely propionylcarnitine, but not palmitoylcarnitine, was increased in the regenerating rat liver (Bláha and Šimek 1991).

The results of our study have indicated that the regenerative capacity of MCT+LCT (40 % : 60 % w:w) supplemented rats is preserved 24 and 72 h after partial hepatectomy, as measured by the liver DNA synthesis and hepatocyte mitotic activity. Overfeeding solely with MCT deteriorated the postoperative recovery and further liver regeneration.

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MUDr. V. Bláha, CSc., Department of Metabolic Care and Gerontology, Faculty Hospital, Charles University, Sokolská 408, 500 05 Hradec Králové, Czech Republic.