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# Use of Olive Oil Based Emulsions as an Alternative to Soybean Oil Based Emulsions in Total Parenteral Nutrition and Their Effects on Liver Regeneration following Hepatic Resection in Rats

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**Key Words**
Olive oil based emulsions · Liver regeneration · Total parenteral nutrition · Carnitine

**Abstract**

***Background/Aim:*** The main objective of this study was to compare the effects of olive oil to those of soybean oil on liver tissue regeneration following hepatic resection in rats. ***Methods:*** Seventy albino Wistar rats were randomly assigned to seven groups which contained 10 rats each. Group 1 was the sham-treated group; groups 2 and 3 received total parenteral nutrition (TPN) containing soybean oil lipid emulsions (20% Lipofundin MCT/LCT) for 48 or 72 h; groups 4 and 5 received TPN containing olive oil (80%)/soybean oil (20%) lipid emulsions (Clin-Oleic 20%) for 48 or 72 h; group 6 was the control group for 48 h, and group 7 was the control group for 72 h. TPN was given via internal jugular vein, and 70% hepatic resection was performed in the study groups. In addition, hepatic resections with no TPN were performed in the control groups, except the sham group. Relative liver weight, mitotic index, proliferating cell nuclear antigen labeling index, and carnitine levels in liver tissue samples were used to assess hepatic regeneration. Thiobarbituric acid reactive substances were measured as an index of lipid peroxidation and oxidative tissue damage. Alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase were parameters for the liver function. ***Results:*** The relative liver weight increased significantly with minimal hepatosteatosis in the olive oil treated groups. Lipid peroxidation significantly decreased with near-normal serum levels of the liver function parameters in the olive oil/soybean oil treated groups, and mitotic index and proliferating cell nuclear antigen labeling index reached their maximum level in rats receiving TPN containing any kind of lipid emulsions for 48 h following resection. ***Conclusions:*** The use of olive oil/soybean oil lipid emulsions in TPN has important beneficial effects on the liver regeneration, and the antioxidant properties of olive oil originating from its natural components indirectly contribute to the liver regeneration in rats.



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## Introduction

Major hepatic resection carries considerable operative and perioperative risks, often secondary to hepatic failure. Nutritional depletion may increase the risk of resection. Nutritional therapy aimed at providing a more rapid and efficient return of hepatic functions would be expected to decrease morbidity and mortality of hepatic resections. It has been shown that the decreases in energy status after major hepatic resections may partly be due to the metabolic overload to the remnant liver and energy demand for tissue regeneration [1, 2]. Glucose and fat are important energy substrates during the regeneration period after partial hepatectomy [1]. It has been proved that the change in energy substrate from glucose to free fatty acids (FFA) takes place only during the early posthepatectomy stage [1, 3, 4].

Lipids are commonly used enterally and parenterally because of their high caloric density and isotonicity. Pure long-chain triglyceride emulsions may be less appropriate than medium-chain triglyceride/long-chain triglyceride (MCT/LCT) containing fat emulsions for parenteral nutrition. It has been shown in animal experiments that MCTs produce less strain on the liver reticuloendothelial system than LCT [5–7]. It has also been published [8] that MCTs are stored in the liver to a lesser extent than LCTs and that manifestation of cholestasis is less common as compared with MCT/LCT fats. Rapid metabolism of MCTs is relatively independent of the carnitine transport system [9]. All these data favored the use of lipid emulsions containing MCT/LCT following hepatic resections.

Olive oil containing lipid emulsions (80% olive oil, 20% soybean oil) seem to have decisive advantages. Their oleic acid content allows to modulate the soybean oil composition and to elaborate lipid blends with fatty acid families (monounsaturated: oleic acid; polyunsaturated ω-6: linoleic acid; polyunsaturated ω-3: α-linolenic acid). A comparable energy source with a limited risk of in vitro and in vivo peroxidation due to the high levels of monounsaturates [10, 11] and an increased content of natural antioxidants are among the main advantages of olive oil over soybean oil emulsions [12–14].

Liver regeneration is one of the most rapid forms of tissue growth known in mammals. Many factors such as hormones, growth factors, nutritional factors, and pharmacological agents have been proved to affect liver regeneration directly or indirectly [15, 16]. Carnitine, an important carrier of long-chain FFA into the mitochondria for beta oxidation, is believed to play one of the key roles in the regulation of this regeneration [17]. In rats, the liver is the sole site of carnitine biosynthesis [18]. The remnant liver promotes the generation of carnitine, whereas kidney and skeletal muscle release their stored carnitine at an early stage after partial hepatectomy. The influx of carnitine into hepatocytes increases at the regenerative stage; therefore, the carnitine content of the remnant liver may remain sufficiently high during the early posthepatectomy stage [17, 19].

The main objective of this study is to compare the effects of olive oil to those of soybean oil administered via total parenteral nutrition (TPN) on liver tissue regeneration following 70% hepatic resection in rats.

## Materials and Methods

*Experimental Protocol*

Seventy albino Wistar rats, bred in the Hakan Çetinsaya Experimental and Clinical Research Center, were used. All rats weighed between 200 and 310 g at the age of 24–32 weeks, and they were randomly assigned to seven groups, each consisting of 10 animals:

Group 1: sham treated; group 2: TPN containing soybean oil lipid emulsions (20% Lipofundin MCT/LCT; Braun Melsungen) for 48 h (SO48); group 3: TPN containing soybean oil lipid emulsions (20% Lipofundin MCT/LCT) for 72 h (SO72); group 4: TPN containing olive oil (80%)/soybean oil (20%) lipid emulsions (ClinOleic 20%; Clintec Parentéral) for 48 h (OO/SO48); group 5: TPN containing olive oil (80%)/soybean oil (20%) lipid emulsions (ClinOleic 20%; Clintec Parentéral) for 72 h (OO/SO72); group 6: control group for 48 h (C48), and group 7: control group for 72 h (C72).

After overnight fasting, all procedures were performed between 09.00 and 11.00 h. The rats were anesthetized using xylazine 3 mg/kg i.p. and ketamine 90 mg/kg i.p. for surgical procedures. The right jugular vein was cannulated with a Silastic catheter under sterile conditions. The catheter was tunneled subcutaneously to the back of the neck and stabilized with a precision single-channel rodent swivel drug infusion system and rodent jacket with tether. Partial hepatectomy (70%) was performed via midline abdominal incision using the technique described by Emond et al. [20]. All operations were performed by the same surgeon to reduce the effect of any surgical technique variations. All rats but the sham group received cefazolin 25 mg/kg/day to prevent bacterial translocation which occurs during the first 48 h after hepatectomy [21]. In the sham group, after midline incision, blood samples were taken, liver tissues were totally extirpated, and the total liver wet weight was measured and found to be approximately 4% of the total body weight. After decapitation at 48 and 72 h, the remnant liver weight was measured. The ratio of the remnant liver weight to the initial body weight was calculated. The liver regeneration rate was calculated according to following formula [22]:

$$\text{Regeneration rate (\%)} = 100 \times [C - (A - B)]/A$$

where A represents the estimated whole liver weight at operation, B the excised liver weight, and C the remnant liver weight at a given time. All results are expressed as percentages. The rats in groups 2, 4, and 6 were scarified by decapitation 48 h after the operation, and those in groups 3, 5, and 7 were sacrificed after 72 h by the same

method. Blood samples and remnant liver tissues were kept at −80°C until analyses. The rats which died during any stage of the study were replaced.

*Preparation of TPN Solutions*

TPN was not given to either the control groups 6 and 7 or the sham group (group 1). All other groups received TPN which contained 42.5% dextrose, 20% protein (8% amino acids), and 37.5% fat through the catheter. By this formulation 56 ml/24 h of TPN was infused to each rat which provided 78 kcal and 240 mg nitrogen also containing vitamins, minerals, and trace elements. Groups 2 and 3 received 20% Lipofundin MCT/LCT, a mixture of MCT and LCT, at a 1:1 ratio. Groups 4 and 5 received 80% olive oil and 20% soybean oil combined (ClinOleic 20%; table 1). The TPN solutions were infused for the entire day at room temperature using infusion pumps, and each rat was kept alone in a separate cage. Normal saline, vitamin, mineral, trace element, and dextrose infusions were applied for 48 and 72 h under the same conditions to control groups 6 and 7. In the control groups total calculated calories were provided by dextrose solutions only.

*Mitotic Index (MI), Proliferated Cell Nuclear Antigen (PCNA) Labeing Index, and Steatosis*

Sections (5 μm thick) were cut from the paraffin-embedded liver tissue specimens, routinely processed, and stained with hematoxylin and eosin. Mitotic figures were counted in routine hematoxylin/eosin-stained sections as outlined by van Diest et al. [23]. The MI was calculated by dividing the mitotic count determined in 30 consecutive high-power fields by the total cell number in this field and expressed as the number of mitotic figures per 1,000 cells [24].

After fixation with formalin and paraffin embedding, immunohistochemical assessment was performed in 5 μm thick specimens. The proliferation index of PCNA-stained tissue was determined in 30 high-power fields. Data were expressed as PCNA-stained cell number per 1,000 cells [25].

Macrovesicular steatosis was graded as absent or minimal (<1% of the hepatocytes; score 0), mild (<30% of the hepatocytes; score 1), moderate (between 30 and 60% of the hepatocytes; score 2), or severe (>60% of the hepatocytes; score 3), as described previously [26].

*Biochemical Measurements*

An isocratic reverse-phase HPLC method was used to quantify the carnitine contents in the liver tissue samples using the method previously described by Arakawa et al. [27]. Liver tissue samples (wet weight approximately 0.5 g) were homogenized in 3 vol of 6% cold perchloric acid using a Teflon homogenizer (Bilser). The homogenates were then centrifuged at 5,000 *g* for 10 min. The pellets were again washed with 6% cold perchloric acid, and the supernatants as well as the tissue washings were kept at −80°C until analysis. The HPLC system consisted of an intelligent high-pressure pump (Jasco JC 980), an UV-VIS detector (Jasco UV-975), a sample injector (Rheodyne 7725), and a computer unit with a chromatography program (Dizge Analitik). A stainless steel analytical HPLC column (Higgins Analytical) packed with ODS C8 (5 μm; 25 cm × 4.6 mm inner diameter) was used, and a solvent mixture of 190 m*M* $KH_2PO_4$ and methanol (87:13, v/v) at a flow rate of 0.7 ml/min was used for the mobile phase. A 10-μl aliquot of each sample and standard was injected, and the peak heights were automatically used in the calculation of concentrations in samples according to those obtained from external standards.

**Table 1.** Main fatty acid and α-tocopherol contents of oils for clinical nutrition

| Fatty acids | Soybean | Lipofundin MCT/LCT | Olive/soybean 80%/20% |
|---|---|---|---|
| Saturated[a] | 18.1 | 57.6 | 17.5 ± 0.2 |
| Monounsaturated[a] | 22.3 | 10 | 59.5 ± 0.5 |
| Polyunsaturated ω-6[a] | 54.5 | 26.4 | 18.5 ± 0.5 |
| Polyunsaturated ω-3[a] | 8.3 | 5.8 | 2.12 ± 0.2 |
| Ratio ω-6/ω-3 | 6.7 | 4.6 | 8.8 |
| α-Tocopherol, mg% | 8.0–10.0 | 6.9 | 15.5 ± 2.0 |
| α-Tocopherol/PUFA ratio mg/g | 0.1–0.2 | 0.2 | 0.8−0.9 |
| β-Carotene, mg% | n.d. | n.d. | 0.085−0.496 |

[a] As a % of total fatty acids.

PUFA = Polyunsaturated fatty acids: 18:2 (linoleic acid) and 18:3 (α-linolenic acid); n.d. = not determined.

For free carnitine analysis, tissue supernatants were neutralized with KOH and allowed to stand for 30 min on ice. After centrifugation, the supernatant was processed through a Millipore filter (0.45 μm) and used for the enzyme reaction assay. The reaction mixture, in a final volume of 1 ml, contained 0.5 μmol EDTA, 10 μmol phosphate buffer (pH 7.5), 40 nmol acetyl-coenzyme A, and the tissue supernatant (0.1–5.0 nmol carnitine). The reaction was initiated by the addition of 1 U carnitine acetyltransferase. After incubation at 25°C for 30 min, the mixture was adjusted to pH 2 with $H_3PO_4$ and then analyzed by HPLC. Coenzyme A, of the products of the reaction mentioned above, was measured by the HPLC method and stoichiometrically corresponded to the carnitine concentrations in all samples.

For total carnitine analysis, 1 ml of tissue supernatant was added to 1 ml of 1 *N* KOH and incubated at 25°C for 30 min to hydrolyze acylcarnitines. The solution was neutralized with 6% $HClO_4$ and then assayed for free carnitine as described above. The short-chain acylcarnitine content was determined by subtracting the value of free carnitine from that of acid-soluble carnitine.

Thiobarbituric acid reactive substances (TBARS) in liver tissue samples were measured by the spectrophotometric method described by Ohkawa et al. [28], and the results were given as nanomoles malondialdehyde per gram wet weight. The serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (AP) were measured using commercial kits by an auotoanalyzer (Konelab 60i).

*Statistics*

Differences between the study and control groups were compared using the Mann-Whitney U test. All experimental data are expressed as the median (minimum – maximum). $p < 0.05$ was considered statistically significant.

**Table 2.** Median (minimum–maximum) and range (in parentheses) of all parameters at both time periods

| | Parameter | Sham | Control | SO | OO/SO |
|---|---|---|---|---|---|
| 48 h | Regeneration ratio, % | | 25.6 (25.5–25.8) | 31.6 (6.7–43) | 52.8 (39.5–57.0) |
| | MI, ‰ | 0 | 6.3 (5.1–7.3) | 11.6 (2.4–26) | 11.6 (4.4–24.8) |
| | PCNA, ‰ | 0 | 7.6 (6.0–9.0) | 13.9 (2.88–31.2) | 13.9 (5.28–29.7) |
| | Steatosis | 0 | 0 | 2.0 (0.0–3.0) | 1.0 (0.0–3.0) |
| | TBARS, nmol/g | 189 (179–192) | 189.3 (187.7–189.3) | 187.5 (184.5–188.8) | 177.7 (175.5–178.7) |
| | Total carnitine, nmol/g | 507 (290–730) | 464.6 (451.6–475.1) | 450.0 (342.0–563.0) | 410.5 (400.6–425.4) |
| | Acylcarnitine, nmol/g | 123 (90–309) | 114.0 (110.9–117.1) | 89.0 (30.0–144.0) | 14.0 (9.7–40.2) |
| | AP, U/l | 127 (88–279) | 444.5 (397.0–492.1) | 550 (448.0–1294.0) | 571.0 (371.0–756.0) |
| | AST, U/l | 99 (81–138) | 508.4 (412.9–604.0) | 473 (37.0–2218.0) | 334.0 (165.0–1040.0) |
| | ALT, U/l | 33 (21–42) | 233.2 (187.5–279.0) | 316.0 (7.0–550.0) | 225.0 (78.0–853.0) |
| 72 h | Regeneration ratio, % | | 25.2 (25.1–25.5) | 29.7 (22.5–43.2) | 49.1 (42.5–53.5) |
| | MI, ‰ | 0 | 4.3 (3.6–5.1) | 2.4 (0.4–16.0) | 2.8 (0.8–8.8) |
| | PCNA, ‰ | 0 | 5.5 (4.0–7.0) | 2.8 (0.5–19.2) | 3.3 (0.9–10.5) |
| | Steatosis | 0 | 0 | 1.0 (0.0–3.0) | 0.0 (0.0–3.0) |
| | TBARS, nmol/g | 189 (179–192) | 187.7 (186.2–189.3) | 185.5 (179.5–191.5) | 185.5 (178.6–187.9) |
| | Total carnitine, nmol/g | 507 (290–730) | 475.1 (464.6–475.1) | 473.0 (410.0–545.0) | 415.6 (402.6–425.6) |
| | Acylcarnitine, nmol/g | 123 (90–309) | 122.1 (117.1–127.4) | 125.0 (45.0–170.0) | 24.7 (13.0–37.0) |
| | AP, U/l | 127 (88–279) | 539.6 (492.1–587.2) | 718.0 (452.0–1290) | 510.0 (288.0–799.0) |
| | AST, U/l | 99 (81–138) | 317.3 (221.8–412.9) | 379.0 (176.0–787.0) | 205.0 (108.0–636.0) |
| | ALT, U/l | 33 (21–42) | 141.7 (96.0–187.5) | 134.0 (63.0–456.0) | 65.0 (44.0–312.0) |

## Results

The relative liver weights were significantly higher in groups which received OO/SO lipid emulsions as compared with the control groups and SO groups during both of the two time periods ($p < 0.05$). There were no significant differences between control groups and SO groups for relative liver weights at 48 and 72 h ($p > 0.05$; table 2).

All groups showed a significant increase in MI and PCNA-labeling index when compared with the sham group ($p < 0.001$). The rats which received OO/SO (group 4) or SO (group 2) for 48 h had significantly higher MI and PCNA values as compared with the control group 6 ($p < 0.01$), but showed no difference when compared with the two TPN groups (groups 2 and 4, $p > 0.05$; table 2), and the MI after 72 h of TPN (groups 3 and 5) was also not significantly different ($p > 0.05$). Group 7 had significantly higher MI and PCNA values than the rats receiving OO/SO (group 5, $p < 0.05$; table 2).

Enhanced steatosis was observed in both lipid groups as compared with the sham and control groups at 48 h (table 2). The most significant steatosis was observed in group 2 when compared with the control group ($p < 0.001$), although the differences between group 4 and control group were less significant ($p < 0.05$) at 48 h. At 72 h, there was no significant difference either in SO or in OO/SO groups as compared with the control group (table 2).

The TBARS levels decreased significantly in group 4 (OO/SO48) as compared with groups 1 (sham), 2 (SO48), and 6 (C48) ($p < 0.001$; table 2). The TBARS levels also decreased significantly in group 5 (OO72) versus group 1 (sham) and in group 2 (SO48) versus group 6 (C48) ($p < 0.05$; table 2). There were no significant changes in TBARS levels in all other groups.

The liver tissue total carnitine levels were significantly different among groups 4 and 5 and the sham group ($p < 0.05$). The total carnitine levels were significantly different between group 4 (OO48) and group 6 (C48) ($p < 0.05$). However, the acylcarnitine levels were significantly different between groups 2 (SO48) and 4 (OO48), between groups 4 (OO48) and 6 (C48), between groups 3 (SO72) and 5 (OO72), and also between groups 5 (OO72) and 7 (C72) ($p < 0.001$; table 2).

All liver function tests were significantly higher in control and study groups at both time periods as compared with the sham group. The AP concentration was significantly higher in the 48-hour study groups 2 and 4 as compared with the levels in control group 6 ($p < 0.02$). The

ALT and AST levels were also significantly increased in group 3 as compared with group 5 ($p < 0.05$). The ALT and AST concentrations were not significantly different in either study group at 48 h ($p > 0.05$; table 2).

## Discussion

The increased energy requirement during the early period after partial liver resection is mainly met by lipids instead of carbohydrates. As the FFA are more intensely utilized than glucose at this early posthepatectomy period, the structure of lipid emulsions used in TPN solutions and their effects on liver regeneration have led to discussions in previous studies.

After the report by Higgins and Anderson [29] in 1931, the concept of regeneration of the liver to its preoperative volume and weight after partial hepatectomy gained popularity. Preoperative and postoperative liver weights and volumes, or ratios, are frequently used as important parameters in studies showing liver regeneration following hepatic resection in experimental models. However, in such studies different formulas to express the regeneration ratio were used [15, 22, 30–32]. In our study, we used a formula to calculate the relative liver weight which indicates the regeneration ratio of remnant liver tissue. The relative liver weight of rats receiving SO type of lipid emulsions reached 31.6% at 48 h, although this proportion was 52.8% for rats receiving olive oil based lipid emulsions. Furthermore, the relative liver weight within 72 h of TPN reached 49.1% in the OO/SO groups, whereas this was 29.7% in the groups receiving SO. There were no significant differences between control and SO groups at both time points, although the differences were significant when the OO/SO groups were compared with the SO and control groups during both periods. These data show that OO/SO increases the relative liver weight (regeneration rate). Rats with fatty livers have a reduced liver regeneration after partial hepatectomy [25]. Minimal hepatosteatosis in rats receiving OO/SO-containing lipid emulsions has contributed to higher levels of relative liver weights. According to this, we believe that olive oil has beneficial effects on liver regeneration.

It has been previously shown that the MI reaches the maximal range in remnant livers at 48 h [17]. Our data are also in agreement with this finding and confirm this evidence. Significantly higher MI values in TPN groups as compared with control groups at 48 h could be evidence of an increased hepatocyte regeneration. In a study, in which hepatic regeneration after hepatectomy was assessed [33], it was reported that PCNA labeling index and MI were declined to low levels 96 h after hepatectomy. Relatively low levels of MI and PCNA after 72 h could be explained on the basis of this observation. Another explanation for these low levels may be complete regeneration, reaching the original size of the liver earlier in TPN groups.

The PCNA is an important protein for DNA polymerase and plays a very important role in the initiation of cellular proliferation. Expression of this protein is a sign of the S phase of the cell cycle, and it is a proliferation marker because it shows the proliferative fraction of tissue cells [34]. Cells with PCNA antibodies are very few in the normal liver. However, they were found to increase 24 and 48 h following liver resection [35].

It is already known that PCNA, as the MI, has a close relation to the histological characteristics of the liver, and it also provides important information on hepatocyte regeneration. These MI and PCNA results showed the beneficial effects of TPN on hepatic regeneration at the cellular level.

Oxidative processes, resulting in the formation of free radicals in generation of lipid peroxides, occur in tissues and cells under various conditions. Antioxidant properties of olive oil, originating from its natural components, have been known for a long time, and synergism between these antioxidants may have an increased effect [12–14]. In this respect, synergistic interactions have been postulated and described for β-carotene and α-tocopherol, two lipid-soluble antioxidants present in olive oil which may function at different localizations in biological membranes [36]. A combination of β-carotene and α-tocopherol results in an inhibition of lipid peroxidation, significantly greater than the sum of individual inhibitions. On the other hand, components of the phenolic fraction of olive oil can inhibit platelet function and eicosanoid formation [14]. The TBARS levels were the lowest in the rats which received olive oil based emulsions when compared with the other groups after 48 h of TPN, and this may, at least partly, be due to the natural antioxidant properties of olive oil. The decrease in oxidative stress by the natural antioxidants in olive oil could also have an indirect beneficial effect on liver regeneration.

The high rate of lipolysis for energy formation, and also the cycling between fatty acid oxidation and synthesis, may occur to a higher degree in the regenerating liver [1, 37]. Under such conditions, carnitine, as an essential cofactor in fat oxidation, has an important role in posthepatectomy metabolic changes [38]. Therefore, the carnitine levels during hepatic regeneration have previously

been investigated in several reports [17–19]. In other studies, the effects of carnitine infusion on hepatic regeneration have been examined [38–40]. Sufficient amounts of carnitine have been reported to be stored in livers after partial hepatectomy [18, 19]. An increased acylcarnitine concentration has been suggested to reflect impaired liver regeneration [41].

In our study, rats receiving OO/SO showed a significant decrease in their acylcarnitine levels. The levels of acylcarnitine in livers of rats which were given OO/SO-based emulsions were significantly lower than in the controls as well as in the other groups receiving SO-containing lipid emulsions. Acylcarnitine is thought to suppress mitochondrial fatty acid transport solely through the inhibition of the carnitine-acylcarnitine translocase component [42]. Therefore, the low levels of acylcarnitine found in olive oil receiving groups may reflect an increased oxidation of FFA, providing a more efficient energy source and less steatosis. Adequate energy support during the early period postoperatively has an indirect beneficial effect on hepatic regeneration. Relatively higher levels of acylcarnitine in SO groups may decrease the transport of fatty acids into the mitochondria, resulting in accumulation of fatty acids which may explain the enhanced steatosis in the SO groups.

Near-normal serum levels of AST, ALT, and AP in the groups receiving OO/SO as compared with controls and groups receiving SO may indicate that cellular repair improved and that functional regeneration occurred earlier using OO/SO lipid emulsions. It may be concluded that regeneration in the nonparenchymal cells and their interaction with hepatocytes occurred easier and earlier in OO/SO groups as compared with control and SO groups.

In conclusion, the beneficial effects of olive oil on the regenerating liver can be summarized as follows: the relative liver weight increased significantly with minimal hepatosteatosis, the lipid peroxidation significantly decreased and near-normal serum levels of liver function parameters were observed in OO/SO groups, and MI and PCNA labeling index reached their maximum in rats receiving any kind of lipid emulsions containing TPN solutions 48 h after liver resection. We believe that the use of OO/SO lipid emulsions in TPN has important beneficial effects on liver regeneration and that the antioxidant properties of olive oil originating from its natural components contribute indirectly to liver regeneration in rats.

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