Changes in fatty acid composition of rat liver and serum induced by distal small bowel resection

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The serum lipid composition and the fatty-acid profiles of the major lipid fraction (triglycerides, esterified cholesterol, and phospholipid) of liver and serum were examined 6 weeks after both 50% and 75% distal small bowel resection (DSBR). Total serum lipid content did not modify after DSBR. Esterified cholesterol and phospholipid levels of the serum did not significantly change after the operation. However, a significant increase in both free cholesterol and triglyceride levels was observed after DSBR. Different fatty acid changes in the liver and serum lipid fractions were found after DSBR, with the greatest differences in the hepatic esterified cholesterol fraction. These results suggest that DSBR affects both the lipid composition and the fatty acid composition of major lipid fraction of liver and serum.

Keywords: intestinal resection; lipid composition; fatty acids; liver; serum

Introduction

The interruption of the entero-hepatic circulation produces a decrease in the input of bile acids into the liver, leading to an increase in hepatic synthesis of both bile acids and cholesterol.[1]

The effect of the interruption of the entero-hepatic circulation on the metabolism of lipids (i.e., cholesterol) has been extensively studied.[2] Thus, we have reported recently that many of the enzymes of cholesterol and fatty acid metabolism, such as hepatic 3-hydroxy-3-methylglutaryl Coenzyme A (HMG-CoA) reductase and acyl-CoA:cholesterol acyl transferase (ACAT) (the major rate-controlling enzymes in cholesterol biosynthesis and its conversion to cholesterol ester, respectively) are modified after distal small bowel resection (DSBR).[3,4] In addition, a significant decrease in hepatic esterified cholesterol levels was observed after DSBR, with a significant increase in the esterified cholesterol content of the livers of 75% DSBR compared with the 50% DSBR. However, hepatic total triglycerides, free cholesterol, and phospholipid levels were not modified after the surgical operation.[4]

On the other hand, changes in both lipid composition and fatty acid profile of hepatic microsomal phospholipid fraction were also observed after intestinal resection.[4] Thus, it is quite likely that these and other changes observed on the metabolism of lipids after DSBR are reflected in the liver and/or serum lipids of these animals.

The present study was designed to determine the effect of DSBR upon serum and liver fatty-acid profiles in rats 6 weeks after the surgical operation. Also, serum lipid composition as affected by DSBR has been studied.

Materials and methods

Animals

Male Wistar-strain rats purchased from Iffa-Credo (Lyon, France), weighing about 300 g, were used. The animals were given food (Panreac A-04) and water ad libitum and housed in a room maintained at 21 ± 2°C with lights on from 8 a.m. to 8 p.m. The composition of the diet was as follows: lipids, 3.5%; protein, 19.0%; starch, 66.0%; non-nutritive cellulose, 5.0%; mineral mix, 5.5%, and vitamin mix, 1.0%. The fatty acid com-

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Table 1 Fatty acid composition of the diet (%, W/W)

Fatty acid	Composition (% W/W)
16:0	18.0
16:1(n-7)	2.3
18:0	4.0
18:1(n-9)	20.7
18:2(n-6)	49.4
18:3(n-3)	3.4
20:0	0.1
20:1(n-9)	1.6
22:0	0.3
ΣSaturated	22.1
ΣMonounsaturated	24.6
Σn-6	49.4
Σn-3	3.4

position of the diet is shown in *Table 1*. The rats were randomly assigned to one of three groups: sham operated, 50% DSBR, and 75% DSBR. Operative details have been described previously.[5] Briefly, the rats were anesthetized with intraperitoneal sodium pentobarbital (4.5 mg/100 g body weight) after a 24 hour fast period, laparotomy was performed, and rats assigned for SBR underwent either 50% or 75% SBR by excision of the distal small intestine beginning 1 cm proximal of the ileocecal junction. Prior to SBR, the blood vessels of the resected intestinal segment were tied and sectioned, and the blood supply and the innervation of the remaining intestine were carefully maintained. Intestinal continuity was re-established by an end-to-end anastomosis. Finally, both muscle and cutaneous layers were sutured separately with appropriate thread. Rats from the sham-operated group underwent simple mid-small intestinal transection, without removal of any tissue, followed by reanastomosis. After the surgical operation, the rats were housed in a temperature-controlled laboratory with a strict 4 a.m.-to-4 p.m./4 p.m.-to-4 a.m. dark/light cycle.

Lipid analysis

Six weeks after DSBR, rats were starved overnight (with access to water only) and then killed by decapitation between 9 a.m. and 10 a.m. Both the control and the experimental group were treated in the same manner to prevent effects which could mask differences between groups. Blood was collected and the serum was separated (1500 × *g* for 10 min.). Livers were excised quickly, rinsed in ice-cold physiological saline, blotted, and weighed. Lipids from serum and liver were extracted by the method of Folch et al.[6] The lipids were separated by thin layer chromatography on silica Gel H plates using a solvent system composed of hexane-ether-acetic acid (80:20:1), respectively. After the development on the plate, the solvent was allowed to evaporate. Spots were made visible in iodine vapor, and the rows of spots were delineated. Each row of spots (phospholipids, free cholesterol, esterified cholesterol, and triglycerides) were removed from the plate and eluted from the silica gel with 14 ml of either diethyl ether or chloroform-methanol (2:1 v/v) for free cholesterol or esterified cholesterol and phospholipids or triglycerides, respectively. Free cholesterol and esterified cholesterol were assayed individually by the method of Huang et al.[7] Phospholipids and triglycerides were estimated by the Vioque and Holman method.[8] Recovery of the different lipid fractions ranged between 90 to 100% by these methods.

Fatty acid methyl esters of the major lipid fraction (triglycerides, esterified cholesterol, and phospholipid) were determined by gas-liquid chromatography. Silica gel spots containing the lipid fractions were scraped off and saponified by heating for 5 minutes with 5 ml of sodium methylate 0.2 N and obtained samples heated again at 80°C for 5 minutes with 6% H_2SO_4 in anhydrous methanol. The fatty acid methyl esters thus formed were eluted with hexane and analyzed with a gas chromatograph (Hewlett Packard, model 5710A) equipped with a flame-ionization detector (FID). SP-2310 (3%) and SP-2300 (2%) on 100/120 chromosorb W AW were used in a 200 cm glass column, and N_2 (20 ml/min.) served as the carrier gas. The temperature was programmed to rise from 190°C to 220°C at a rate of 2°C per minute. The fatty acid methyl esters were identified by injecting authentic standard mixtures of fatty acid methyl esters and comparing their retention times.

Statistical analysis

All data are presented as the mean ± SD. The effect of DSBR was examined using analysis-of-variance (ANOVA) procedures. The unpaired student's t-test was used to test the significance of the difference between the means of sham-operated and resected rats.

Results

Serum lipid concentrations

Total serum lipid content did not significantly change after DSBR (355 ± 15 and 304 ± 15 mg/dl for 50% and 75% resected rats, respectively, versus 332 ± 9 mg/dl for sham rats). Analysis of the serum lipid composition in these studies demonstrated that esterified cholesterol and phospholipid levels of the serum did not significantly change after the surgical operation. However, a significant increase in both free cholesterol and triglyceride levels was observed after DSBR (*Table 2*).

Table 2 Effect of intestinal resection on serum lipid content

Serum lipid (mg/dl)	Sham	50%	75%
Free cholesterol	12.1 ± 1.2	19.0 ± 1.3**	15.9 ± 1.4*
Esterified cholesterol	70 ± 5	72 ± 2	61 ± 4
Phospholipid	167 ± 12	187 ± 13	160 ± 12
Triglycerides	49 ± 4	76 ± 6***	65 ± 3**

Results are given as means ±SD of six animals in each group. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$ 50% or 75% resected rats compared with sham animals.

Fatty acid composition of the major lipid fractions of the liver and serum

The fatty acid composition of hepatic triglycerides is shown in *Table 3*. Total saturated fatty acids were not significantly different among the groups, though an increase in 18:0 was observed after DSBR. An enhancement in monounsaturated fatty acids (oleic acid, 18:1, n-9) was obtained in resected rats. Regarding the (n-6) fatty acids, a decrease in the levels of 18:2 (n-6) accompanied by no changes in 20:4 (n-6) was observed after intestinal resection. 22:4 (n-6) and 22:5 (n-6) levels were not detected in resected rats. Therefore, taken together these levels are responsible for the lower value in total (n-6) fatty acids found in resected animals in comparison with those in sham rats. On the other hand, DSBR reduced the levels of total (n-3) fatty acids in this fraction.

Total saturated fatty acids (stearic acid, 18:0) were reduced in the hepatic phospholipid fraction by DSBR (*Table 4*). However, a significant increase in oleic acid (18:1, n-9) was observed in this fraction only after 75% resection. Contrary to what occurred in the triglyceride fraction, the levels of total (n-3) fatty acids were elevated in resected animals.

Analysis of the fatty acid composition of hepatic esterified cholesterol fraction is illustrated in *Table 5*. A decrease in both saturated and monounsaturated fatty acids was observed in 75% resected animals. The level of linoleic acid (18:2, n-6) was elevated after massive resection. However, a reduction in arachidonic acid (20:4, n-6) was obtained in resected animals, and it was not found in detectable values after 75% resection.

Table 3 Effect of intestinal resection on fatty acid composition of hepatic triglycerides (%, W/W)

Fatty acid	Sham	50%	75%
16:0	23.58 ± 2.50	23.34 ± 0.97	25.91 ± 0.44
16:1(n-7)	5.99 ± 0.70	5.01 ± 0.08	6.84 ± 1.17
17:0	0.36 ± 0.04	1.04 ± 0.31*	1.16 ± 0.18*
18:0	1.99 ± 0.20	3.26 ± 0.13***	3.78 ± 0.28***
18:1(n-9)	28.47 ± 2.05	37.36 ± 3.01**	34.53 ± 0.38*
18:2(n-6)	28.71 ± 3.15	17.27 ± 0.51*	16.85 ± 1.76*
18:3(n-3)	1.09 ± 0.12	0.45 ± 0.10	—
20:0	0.22 ± 0.03	—	—
20:1(n-9)	0.39 ± 0.05	0.63 ± 0.13	0.24 ± 0.03*
20:2(n-6)	0.21 ± 0.02	—	—
20:4(n-6)	3.98 ± 0.42	3.72 ± 0.39	4.21 ± 0.64
20:3(n-9)	0.53 ± 0.04	0.45 ± 0.09	—
22:4(n-6)	0.34 ± 0.04	—	—
22:5(n-6)	0.31 ± 0.04	—	—
22:5(n-3)	0.36 ± 0.05	—	—
22:6(n-3)	1.51 ± 0.17	1.24 ± 0.05	1.30 ± 0.66
ΣSat.	26.15 ± 2.8	27.64 ± 1.41	30.86 ± 0.9
ΣMUFA[a]	34.85 ± 2.8	43.00 ± 3.22**	41.61 ± 1.58**
ΣPoly	37.04 ± 4.05	23.13 ± 1.14**	22.36 ± 3.06*
Σn-6	33.55 ± 3.67	20.99 ± 0.95**	21.06 ± 2.4*
Σn-3	2.96 ± 0.34	1.69 ± 0.15*	1.30 ± 0.66**

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

Table 4 Effect of intestinal resection on fatty acid composition of hepatic phospholipid (%,W/W)

Fatty acid	Sham	50%	75%
16:0	21.35 ± 2.65	19.39 ± 0.59	19.46 ± 0.62
16:1(n-7)	0.66 ± 0.07	1.64 ± 0.12***	2.70 ± 0.51**
17:0	0.30 ± 0.02	1.59 ± 0.30**	2.06 ± 0.17****
18:0	30.45 ± 3.05	22.07 ± 0.48*	21.72 ± 0.72*
18:1(n-9)	9.46 ± 0.80	9.12 ± 0.16	12.36 ± 0.72*
18:2(n-6)	11.64 ± 1.18	12.03 ± 0.23	11.85 ± 0.14
20:1(n-9)	0.18 ± 0.02	0.14 ± 0.00	0.42 ± 0.06
20:3(n-9)	—	0.79 ± 0.02	—
20:4(n-6)	21.29 ± 1.98	21.92 ± 2.39	19.42 ± 0.24
20:3(n-6)	—	0.14 ± 0.02	0.35 ± 0.04
22:4(n-6)	—	0.59 ± 0.15	0.58 ± 0.11
22:5(n-6)	—	0.90 ± 0.07	0.62 ± 0.08
22:5(n-3)	—	1.09 ± 0.01	1.19 ± 0.15
22:6(n-3)	3.45 ± 0.21	5.43 ± 0.23*	4.33 ± 0.12
ΣSat.	52.1 ± 5.72	43.05 ± 1.37*	43.24 ± 1.51*
ΣMUFA[a]	10.30 ± 0.89	10.90 ± 0.28	15.48 ± 1.29*
ΣPoly	36.38 ± 3.37	42.89 ± 3.12	38.34 ± 0.88
Σn-6	32.93 ± 0.21	35.58 ± 2.86	32.82 ± 0.61
Σn-3	3.45 ± 0.21	6.52 ± 0.24**	5.52 ± 0.27**

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$; ****$P < 0.001$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

Table 5 Effect of intestinal resection on fatty acid composition of hepatic esterified cholesterol (%, W/W)

Fatty acid	Sham	50%	75%
16:0	17.17 ± 2.09	18.99 ± 0.11	3.75 ± 1.07**
16:1(n-7)	15.15 ± 1.10	16.72 ± 0.01	6.64 ± 0.70***
18:0	4.50 ± 1.17	2.45 ± 0.05*	2.92 ± 0.75*
18:1(n-9)	9.32 ± 0.05	7.51 ± 0.58***	14.01 ± 0.83***
18:2(n-6)	45.06 ± 2.23	47.59 ± 0.04	72.68 ± 4.97***
20:4(n-6)	7.43 ± 1.00	3.82 ± 0.01*	—
ΣSat.	21.67 ± 3.26	21.44 ± 0.16	6.67 ± 1.82***
ΣMUFA[a]	24.47 ± 1.15	24.23 ± 0.59	20.05 ± 1.53*
ΣPoly.	52.49 ± 3.23	51.41 ± 0.05	72.68 ± 5.8***
Σn-6	52.49 ± 3.23	51.41 ± 0.05	72.68 ± 5.8***
Σn-3			

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$; ****$P < 0.001$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

The changes observed in the fatty acid profile of esterified cholesterol, triglycerides, and phospholipid of serum are shown in *Tables 6, 7,* and *8,* respectively. In the case of essential fatty acids (18:2, n-6 and 20:4, n-6), an increase in the arachidonic acid is observed in both esterified cholesterol (*Table 6*) and triglyceride (*Table 7*) fraction of resected rats, accompanied by no changes in the levels of linoleic acid. However, the levels of arachidonic acid were lowered in serum phospholipid fraction after DSBR (*Table 8*). Total monounsaturated fatty acids (i.e., palmitoleic and oleic acid) were enhanced for esterified cholesterol fraction in 75% resected rats, but in triglyceride fraction a reduction in monounsaturated fatty acids was observed after both 50% and 75% resection.

Table 6 Effect of intestinal resection on fatty acid composition of serum esterified cholesterol (%,W/W)

Fatty acid	Sham	50%	75%
16:0	14.94 ± 0.13	14.30 ± 1.44	12.83 ± 2.00
16:1(n-7)	5.59 ± 0.02	6.73 ± 0.47*	7.03 ± 0.02***
17:0	0.38 ± 0.06	0.60 ± 0.01**	1.46 ± 0.15***
17:1	0.92 ± 0.09	0.94 ± 0.02	0.98 ± 0.01
18:0	0.29 ± 0.03	0.61 ± 0.09*	0.52 ± 0.07*
18:1(n-9)	3.69 ± 0.40	3.79 ± 0.82	6.49 ± 0.29***
18:2(n-6)	22.35 ± 0.46	20.18 ± 1.61	19.10 ± 0.72
18:3(n-6)	4.14 ± 0.12	3.14 ± 0.40	—
20:4(n-6)	38.78 ± 0.10	43.00 ± 0.46*	51.00 ± 4.55**
20:3(n-6)	1.52 ± 0.03	0.34 ± 0.01**	0.30 ± 0.05**
20:5(n-3)	0.36 ± 0.02	0.32 ± 0.05	—
22:4(n-6)	—	0.19 ± 0.01	—
22:5(n-6)	3.00 ± 0.15	3.60 ± 0.03	—
22:6(n-3)	1.39 ± 0.03	2.55 ± 0.66*	2.03 ± 0.20*
ΣSat.	15.61 ± 0.22	15.51 ± 1.54	14.81 ± 2.22*
ΣMUFA[a]	10.20 ± 0.51	11.46 ± 1.31	14.65 ± 0.32**
ΣPoly	71.54 ± 0.94	73.32 ± 3.63	72.43 ± 5.52
Σn-6	69.79 ± 0.86	70.45 ± 2.52	70.40 ± 5.32
Σn-3	1.75 ± 0.05	2.87 ± 0.71*	2.03 ± 0.2*

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$; ****$P < 0.001$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

Table 7 Effect of intestinal resection on fatty acid composition of serum triglycerides (%,W/W)

Fatty acid	Sham	50%	75%
16:0	27.79 ± 1.68	23.16 ± 0.06*	23.6 ± 0.11*
17:0	1.34 ± 0.14	—	0.75 ± 0.09*
17:1	1.24 ± 0.07	3.11 ± 0.60*	1.69 ± 0.18
18:0	2.68 ± 0.30	2.45 ± 0.08	2.62 ± 0.29
18:1(n-9)	28.54 ± 0.38	22.14 ± 1.59*	22.30 ± 1.03**
18:2(n-6)	26.4 ± 2.30	25.08 ± 1.83	25.81 ± 1.60
20:2(n-6)	—	0.63 ± 0.06	—
20:4(n-6)	3.65 ± 0.81	8.85 ± 0.75*	14.55 ± 1.42**
ΣSat.	31.81 ± 2.12	25.61 ± 0.14*	26.97 ± 0.49*
ΣMUFA[a]	29.78 ± 0.45	25.25 ± 2.19*	23.99 ± 1.21*
ΣPoly	30.05 ± 3.11	34.56 ± 2.64	40.36 ± 3.02*
Σn-6	30.05 ± 3.11	34.56 ± 2.65	40.36 ± 3.02*
Σn-3	—	—	—

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

Discussion

It is well known that intestinal resection produces lipid malabsorption.[9] This fat malabsorption is likely to derive from a combination of effective loss of absorptive surface area associated with a severely compromised entero-hepatic circulation, which could lead to decreased efficiency of the remaining functional intestine and a decrease in growth rate. It has been previously reported that animals with DSBR grew less than those without resection and that the amount of food eaten per day was similar between groups.[4] Therefore, lower body weight after DSBR could not be due to difference in energy intake, and might relate to changes in feed efficiency.

The outcome of this study can be divided into 2 parts: (1) liver and serum lipids and (2) fatty acid composition of liver and serum lipid fractions (phospholipid, triglycerides, and esterified cholesterol).

We have reported recently that free cholesterol, total triglycerides, and phospholipids in total liver lipid did not change after DSBR. However, a decrease in esterified cholesterol levels in liver lipid was observed after DSBR, this decrease being higher after 50% than after 75% resection.[4] In the present studies, a significant increase in both free cholesterol levels and total triglycerides was observed in the serum of resected rats, both increases being higher after 50% than after massive resection (75%). However, esterified cholesterol and phosholipid serum levels were not significantly modified by the surgical operation (*Table 2*). Therefore, no good correlation was found between serum and liver lipids after intestinal resection.

In the post-absorptive condition, serum lipid level depends mainly on the balance between release of lipid from the liver and uptake of lipid in peripheral tissue. Since no good correlation was found between serum and liver lipid after resection, an impairment of this balance might have been produced by the surgical operation. Furthermore, the effect of 50% DSBR was more pronounced than that of 75% DSBR. This was evident in esterified cholesterol content in liver,[4] free cholesterol, and total triglycerides in serum (*Table 2*).

Table 8 Effect of intestinal resection on fatty acid composition of serum phospholipid (%,W/W)

Fatty acid	Sham	50%	75%
16:0	22.70 ± 1.50	22.45 ± 2.20	23.28 ± 0.70
16:1(n-7)	2.94 ± 0.50	2.21 ± 0.80	2.27 ± 0.02
17:0	0.47 ± 0.04	0.99 ± 0.16**	1.75 ± 0.00****
17:1	1.55 ± 0.16	1.49 ± 0.18	1.37 ± 0.03
18:0	19.65 ± 1.08	21.49 ± 1.09	18.48 ± 0.56
18:1(n-9)	9.85 ± 1.05	9.05 ± 0.66	9.93 ± 1.07
18:2(n-6)	17.2 ± 1.09	17.00 ± 0.78	18.79 ± 2.28
18:3(n-6)	—	0.51 ± 0.05	0.65 ± 0.09
20:0	—	—	0.14 ± 0.01
20:1(n-9)	—	0.42 ± 0.03	0.36 ± 0.01
20:2(n-6)	—	0.37 ± 0.04	0.38 ± 0.10
20:3(n-9)	0.47 ± 0.02	0.86 ± 0.02***	0.81 ± 0.02***
20:4(n-6)	17.28 ± 0.38	15.82 ± 0.61*	15.18 ± 0.29**
20:5(n-3)	—	0.98 ± 0.27	0.17 ± 0.01
20:4(n-3)	0.27 ± 0.01	0.24 ± 0.02	0.46 ± 0.02***
22:4(n-6)	1.10 ± 0.11	1.41 ± 0.43	1.23 ± 0.05
22:5(n-6)	1.50 ± 0.06	0.93 ± 0.25	0.95 ± 0.02
22:5(n-3)	0.85 ± 0.09	0.47 ± 0.13*	0.58 ± 0.02*
22:6(n-3)	3.50 ± 0.28	2.87 ± 0.16*	2.48 ± 0.26*
ΣSat.	42.82 ± 2.62	44.93 ± 3.45	43.64 ± 1.27
ΣMUFA[a]	14.34 ± 1.71	13.17 ± 1.67	13.93 ± 1.13
ΣPoly	42.17 ± 2.04	41.46 ± 2.76	41.68 ± 3.16
Σn-6	37.08 ± 1.64	36.04 ± 2.16	37.18 ± 2.83
Σn-3	4.62 ± 0.38	4.56 ± 0.58	3.69 ± 0.31

Results are given as means ±SD of 6 separate determinations. *$P < 0.05$; **$P < 0.01$; ***$P < 0.005$; ****$P < 0.001$ 50% or 75% resected rats compared with sham animals.
[a]Monounsaturated fatty acid.

At present, there appears to be no reasonable explanation for these findings.

Alteration of fatty acid composition of liver and serum as a result of DSBR has not been reported previously. The data presented in *Tables 3* to *8* reveals that DSBR causes different fatty acid changes in the liver and serum lipid fractions, and that the largest differences were observed in the hepatic esterified cholesterol fraction.

The fatty acid composition of hepatic triglycerides shows a decrease in the family of (n-6) fatty acids after resection, mainly due to both a reduction in the level of 18:2 (n-6) and to a disappearance of (n-6) metabolites (22:4 and 22:5) produced by this operation (*Table 3*).

In the phospholipid fraction, the ratio monounsaturated/saturated was elevated after resection (0.25 and 0.36 for 50% and 75% resected rats, respectively, versus 0.19 for sham rats), which might indicate that at this level there is an activation of the enzyme Δ_9 desaturase. Contrary to what occurred in the triglyceride fraction, resected animals show an enhancement in the metabolites of (n-6) fatty acids (22:4 and 22:5) for phospholipid fraction, whose value was not detected in sham animals (*Table 4*).

Hill et al.[10] have demonstrated previously that liver phospholipid fatty acid composition reflects effectively and reliably the in vivo activity of essential fatty acid desaturation. The levels of 18:2 (n-6) and 20:4 (n-6) were not modified in this fraction by resection. However, we have reported previously that the fatty acid profile of microsomal hepatic phospholipid changes after resection to resemble that of an essential fatty acid deficiency (EFAD) state.[4] Thus, a decrease in both linoleic acid and arachidonic acid together with the appearance of 20:3 (n-9), a marker for EFAD, was found in 75% resected rats. Therefore, in this case, no consistent relationship was found between liver phospholipid fatty acid composition and desaturation enzymes.

The major important changes were found in the esterified cholesterol fraction of liver (*Table 5*), in which 18:2 (n-6) was significantly increased after the massive resection, and 20:4 (n-6) strikingly reduced after 50% resection with no detectable values in 75% resected rats. If both 18:2 (n-6) and 20:4 (n-6) were decreased after resection, it might be suggested that these results could be due to an increased utilization of 20:4 (n-6) for cholesterol ester formation. However, in the present study, the decrease in 20:4 (n-6) was accompanied by either no changes or an increase of 18:2 (n-6). Therefore, 18:2 (n-6) was readily available for any increased demand of utilization. Furthermore, the ratio 20:4/18:2 was reduced by resection (0.08 and 0 for 50% and 75% resected animals, respectively, versus 0.16 for sham rats). Therefore, these results are a suggestive evidence for a possible block in the synthesis of 20:4 (n-6) from 18:2 (n-6), probably due to a decrease in the desaturation of essential fatty acids produced by an inhibition of the enzymes Δ_6 and Δ_5 desaturases.[11] These findings are consistent with previous results obtained by direct hepatic microsomal studies.[4]

The analysis of fatty acid profile of lipid fractions of serum show that arachidonic acid is the major fatty acid utilized for esterification in the esterified cholesterol fraction (*Table 6*). However, in hepatic esterified cholesterol, the major fatty acid was linoleic acid (*Table 5*). These results are in agreement with previous reports.[12,13] As a consequence of DSBR, the serum levels of arachidonic acid were increased in both esterified cholesterol (*Table 6*) and triglyceride (*Table 7*) fractions, and decreased in phospholipid fraction (*Table 8*). The enhancement of arachidonic acid found in both esterified cholesterol and triglyceride fractions of serum after DSBR might be transferred to the plaquets for the formation of TXA_2[14], and it might explain the reduction of this fatty acid in hepatic esterified cholesterol in these animals.

Taken together, these results indicate that the fatty acid composition shows large differences between liver and serum and from fraction to fraction, which might be explained both by the different mechanisms of fatty acid incorporation into different fractions, and also by different patterns of desaturation and elongation.

The desaturase enzymes bringing about the conversion of fatty acids, as affected by DSBR, are currently under investigation in our laboratory.

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