0.99 µmol Se/100 g of diet and for vitamin E to 0.145 µmol/100 g. Sixteen diabetic rats were not supplemented (group D). All the diabetic rats were treated by insulin.

After 24 weeks the weight gain in group C was 33% and only 15% in group D ($P < 0.05$ vs group C), but when the rats were supplemented with selenium the increase was higher and not significantly different from group C. Mortality was null in group C, and from 6/14, 4/14, 6/14 and 3/16, respectively, in groups D, DSel, DSm and DSmE.

Plasma selenium levels were significantly increased in all Se-supplemented diabetic groups compared to group C. Glycemia was significantly increased in the diabetic groups compared to group C ($P < 0.0005$), but it tended to be lower in the Se-supplemented groups compared to group D. The same effect of selenomethionine was observed with glycosylated Hb. Plasma lipid levels (cholesterol, phospholipids, triglycerides) were increased in the diabetic groups compared to group C, but a large decrease in triglycerides (TG) was observed in groups DSm and DSmE compared to group D after 9 and 24 weeks of the diet ($P < 0.01$, $P < 0.05$, respectively).

The ratio vitamin E/TG did not change in the diabetic groups compared to group C, except in group DSmE, where it was significantly increased compared to all the other groups ($P < 0.01$ vs C, D, DSel, $P < 0.05$ vs DSm). In group DSmE, TBARS and conjugated diene levels were significantly decreased after 24 weeks compared to group D ($P < 0.01$). These parameters increased in all the other diabetic groups after 24 weeks but more weakly in the Se-supplemented group compared to group D. Moreover, plasma fatty acid changes were modulated in diabetic rats by selenomethionine and more efficiently by selenomethionine + vitamin E where the overcorrection of of Δ9- and Δ6-desaturases was reduced.

These results indicate that selenium and more efficiently Se + vitamin E tend to normalize the glycemic balance and to correct several plasma abnormalities observed in diabetic rats (increase of TG and peroxides), which represent risk factors for the cardiovascular complications associated with this pathology.

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Long chain polyunsaturated fatty acid (LCPUFA) assay before and after orthotopic liver transplantation (OLT) in children with extrahepatic biliary atresia (EBA). M Chambon [1], A Lapillonne [2], V Chirouze [3], V Mamoux [1], O Boillot [4], M Lievre [5], A Lachaux [1] *([1] Hépatograstroentérologie et nutrition pédiatriques; [2] Néonatologie, hôpi-*

Laboratory data (% of total fatty acids, means ± SD).

Age (months)	*Linoleic acid*	*AA*	*DHA*
1.5–4	9.45 ± 1.78	16.47 ± 0.66	4.71 ± 1.82
8–11	7.54 ± 2.44	15.35 ± 1.55	2.32 ± 0.42**
After PN	10.43 ± 3.24**	15.06 ± 0.80	3.42 ± 1.10**
6 months post-OLT	10.30 ± 1.54	19.52 ± 1.03**	3.44 ± 0.63**
1 year post-OLT	11.30 ± 1.77	19.18 ± 0.77*	4.19 ± 0.95*

* $P < 0.01$: 8–11 vs 1 year post-OLT; ** $P < 0.05$: 1.5 – 4 vs 8 – 11, 8 – 11 vs after PN, 8 – 11 vs 6 months post-OLT.

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Cholestatic children with EBA have an impaired ability to absorb dietary fat and exhibit LCPUFA deficiency. The aim of this study was to assess their LCPUFA status before and after OLT, and also the effect of an infusion of essential fatty acids (EFA) before OLT.

Eight children with EBA waiting for OLT were included in this study. All of them failed to recover from biliary obstruction 3 months after hepatic portoenterostomy and had total bilirubin levels > 250 µmol/L. LCPUFA values in their red blood cells were prospectively studied before (every 3 months) and after (every 6 months) OLT. Mean age was 9.2 ± 1.4 months at the time of OLT, and four of eight children received parenteral nutrition (PN) with EFA supplementation for 14 to 60 days before OLT.

LCPUFA deficiency was rapidly apparent in EBA; it increased when portoenterostomy failed and disappeared during the first year following OLT. Parenteral nutrition seems to partially correct LCPUFA deficiency. These results showed the important role played by fat malabsorption in the constitution of this deficiency and may indicate a positive effect of intravenous EFA supplementation during the pre-OLT period.

Antioxidant nutrients and liver transplantation. J Goudable [1], C Fleuret [1], B Delafosse [2], M Accominotti [1], O Boillot [3] *([1] Services de biochimie; [2] Réanimation; [3] Chirurgie digestive, hôpital Édouard-Herriot, Lyon, France)*

Antioxidant vitamins (A and E) and some trace elements play an important role in protection against free radical damage during biological events and disease processes. These free radical scavengers are provided by the diet.

The liver has a major role in retinoid (vitamin A) and a tocopherol (vitamin E) metabolism as well as in binding protein metabolism. This organ is critical to the regulation of the plasma levels of vitamins and in vitamin delivery to extrahepatic target tissues.

The lack of hepatic perfusion is mandatory in liver transplantation, this period being following by a reperfusion. This ischemia reperfusion is usually associated with an injury due to free radical production. In this respect, the antioxidant nutrients may be of value during this period.

We studied, therefore, the plasma levels of vitamin A, vitamin E, copper, zinc and selenium before and 1 year after a liver transplant in 110 patients following different hepatic disorders.

One hundred ten patients were studied. The main aetiologies were posthepatitic cirrhosis BD (n = 15), posthepatitic cirrhosis C (n = 25) and ethylic cirrhosis (n = 31). No patients received vitamin supplementation.

Vitamins A and E were measured in the plasma by high-performance liquid chromatography. The control population consisted of 58 subjects with normal diets (1 800–2 800 cal/day) without vitamin supplementation.

Selenium, zinc and copper levels were determined in the plasma by atomic absorption spectrometry.

All data are expressed as mean values ± SD. A one-way analysis of variance and a posthoc Scheffe's test, when indicated, was used to compare the means.

The results were as follows:

Vitamin A:

control population: 0.67 ± 0.17 mg/L;
all aetiologies: J_0: 0.20 ± 0.30 mg/L
J_{365}: 0.83 ± 0.26 mg/L