

fed with fish oil (TAMA Biochemical Co. Tokyo, Japan) (FO, $n = 17$). Another group of rats was fed soybean oil (Taiwan Sugar Co., Taipei, R.O.C.) (SO, $n = 19$). After 4 weeks, each experimental group was further divided into septic and control subgroups. Four groups were classified as follows: fish oil control group (FO-C, $n = 7$), fish oil sepsis group (FO-S, $n = 10$), soybean oil control group (SO-C, $n = 7$), and soybean oil sepsis group (SO-S, $n = 12$). Sepsis in rats was induced by cecal ligation and puncture (CLP) according to the method of Wichterman *et al.*¹⁶ Briefly, rats were lightly anesthetized with ether. The abdomen was opened through a midline incision, and the cecum was punctured twice with an 18-gauge needle and then replaced into the abdomen. The abdominal wound was closed in layers. For confirmation of sepsis, blood culture was performed in a half of the septic rats 22-24 h after surgery, and positive cultures were obtained in all of them. The clinical symptoms of the septic rats were similar to those described by Wichterman *et al.*¹⁶ Sham operation with manipulation of the cecum but without ligation or puncture of cecum was performed on control rats.

Twenty-four hours after surgery, all rats were anesthetized and sacrificed by drawing arterial blood from the aorta. The blood samples were collected in tubes containing heparin and centrifuged immediately. Plasma TG,¹⁷ total cholesterol,¹⁸ nonesterified fatty acids (NEFAs),¹⁹ and high-density lipoprotein-cholesterol (HDL-C) were determined by colorimetric methods after enzymatic reaction with peroxidase (Randox Co., Antrim, Ireland). HDL was precipitated from plasma by a modification of the dextran-sulphate method, as described by Warnick *et al.*²⁰ The precipitation reagent for apoprotein B-containing lipoproteins contained phosphotungstic acid and magnesium chloride. Liver lipid was extracted with a chloroform:methanol 2:1 mixture according to Folch *et al.*²¹ Total lipid was gravimetrically measured after drying in an evaporator to constant weight.²² TG was determined by the method of Soloni.²³ The packed erythrocytes were washed twice with isotonic saline, and then stored at -70 °C until analysis of superoxide dismutase (SOD) and glutathione peroxidase (GSHPx) activities (Randox Co.). GSHPx was measured by the

consumption of NADPH at 37 °C on the basis of an absorption coefficient for NADPH at 340 nm of 6.3×10^2 ($\text{mol}^{-1} \text{min}^{-1}$).²⁴ SOD was measured by the competitive inhibition of the superoxide radical produced by xanthin reacting with xanthin oxidase.²⁵ The enzyme activities were expressed in U/mg of hemoglobin (Hb). Hb was determined by cyanomethemoglobin assay (Sysmex F-500, San Tung Instrument Co., Taipei, Taiwan).

Statistics

Data are expressed as means $\pm$ SD. Statistic analysis was performed by two-way ANOVA using a SAS program (SAS Version 6.12, SAS Institute, Cary, NC). Specific comparison of group means was made when F values were significant for diet or treatment. Differences among groups were analyzed by analysis of variance using Duncan's test. A p value < 0.05 was considered statistically significant.

RESULTS

There were no differences in initial body weights and weights after feeding the experimental diet for 4 weeks (data not shown). Plasma concentrations of TG and total cholesterol were significantly higher in septic groups than in corresponding groups without sepsis. The FO-S group had significantly higher plasma TG levels than did the SO-S group, whereas no difference was observed in plasma concentration of cholesterol between the 2 sepsis groups (Table 2). There were no differences in concentrations of NEFA and HDL-C among all groups. The HDL-C/total cholesterol ratio in the FO-S group was significantly lower than that in the other groups (Table 2).

The hepatic total lipid and TG contents of sepsis groups were significantly lower than those in groups without sepsis, irregardless of whether the rats were prefed soybean oil or fish oil. However, the hepatic total lipids and TG contents in fish oil groups were significantly lower than those in soybean oil groups despite the rats being septic or not (Table 3). There were no effects of oil or treatment on the activities of erythrocyte SOD or GSHPx (Table 4).