feeding inhibits hepatic VLDL secretion, and this consequently leads to lower plasma TG.[8] However, a study by Lin *et al.*[26] revealed that, without dietary cholesterol supplementation, animals on a fish oil diet had significantly lower plasma TG and VLDL-TG levels than those on a soybean oil diet. However, with dietary cholesterol supplementation, no significant difference in plasma TG between fish oil and soybean oil groups was observed; the fish oil group even had higher plasma cholesterol than did the soybean oil group. Lu *et al.*[27] also demonstrated that there is no difference in plasma TG between n-3 and n-6 fatty acid enriched groups when cholesterol is supplemented in the diet. The difference of plasma TG concentrations between groups with or without dietary cholesterol supplementation may be explained by the alteration of the lipoprotein particles in the fish oil-cholesterol group. The changes in conformation and structure of VLDL and low-density lipoprotein particles may affect the interactions of the lipoproteins and enzymes or receptors,[28,29] thus reducing the catabolic rate of the lipoprotein, and resulting in higher levels of these lipoproteins in the plasma.[26,27] Since a reduced catabolic rate is the main cause of hypertriglyceridemia in sepsis,[3,4] it is possible that the catabolic rate is further inhibited in the FO-S group when cholesterol is supplemented. This resulted in a higher plasma TG in the FO-S group than in the SO-S group. Results in this study demonstrate that plasma cholesterol is higher in septic groups than in those without sepsis; this result is consistent with a report by Lanza-Jacoby *et al.*[15] The reason for hypercholesterolemia in sepsis is not clear.

According to Lin *et al.*,[26] the HDL-C in the cholesterol-supplemented fish oil group was significantly lower than that in the soybean oil group. In this study we also observed a tendency for HDL-C in the FO-S group to be lower than that in the SO-S group, although no statistical significance was seen between the 2 groups. Because the plasma total cholesterol was elevated in sepsis, HDL-C/total cholesterol in the FO-S group was lower than that in other groups.

Otto *et al.*[30] demonstrated that hepatic lipid accumulation was not observed with a low (10% of total calorie) or medium (20%) fish oil diet; however, 30% fish oil in the diet as performed in this study is considered a high level of fat and may result in fat accumulation in the liver. Our previous reports showed that liver fat content is about 6% for normal rats fed a chow diet.[12,13] The result in this study showed that the hepatic total fat contents were 9% in the FO-C and 12% in the SO-C group, which indicates that fat accumulation is obvious in the livers of control rats fed a high fat diet for 4 weeks. The fish oil feeding group showed a lower fat accumulation than did the soybean oil group; this result is similar with that reported by Rustan *et al.*,[11] which showed liver fat was lower in rats fed a high fish oil diet than those fed a safflower oil diet. *In vivo* and *in vitro* studies have shown that n-3 fatty acid rather than n-6 fatty acid administration leads to a greater extent of peroxisomal fatty acid oxidation and suppression of liver TG synthesis,[11,31] which may contribute to the lower liver fat content in fish oil feeding groups. Compared with control groups, sepsis groups did not show higher fat accumulation in the liver; in contrast, liver fat content was lower in the septic groups than in the corresponding control groups. The mechanisms are unclear. Since liver fat accumulation in sepsis is caused by increased liver TG synthesis without a concomitant enhancement of liver TG secretion,[5] whether a preexisting condition of fatty liver prior to sepsis inhibits the enzyme activities responsible for hepatic TG synthesis requires further investigation.

SOD and GSHPx are enzymes which protect tissues from the effects of free radicals and lipid peroxides, and the activities of both SOD and GSHPx increase after free-radical-mediated injury and lipid peroxidation.[25,32] The result in this study demonstrates that the erythrocyte SOD and GSHPx activities do not differ between the sepsis and control groups. It is possible that the duration of sepsis in this study was not long enough to change the enzyme activities in erythrocytes, or that erythrocyte antioxidant enzyme activities are not a sensitive indicator for oxidative stress of these diseases. Fish oil feeding groups did not show higher antioxidant enzyme activities than those of the soybean oil group, and this might mean that lipid peroxidation products do not accumulate in erythrocytes of the fish oil groups. This might be explained by the presence of vitamin E supplemented in the fish oil by the manufacturer which prevented lipid peroxidation. However, this result is consistent with our previous reports.[12,13]

In conclusion, the elevation of plasma TG and cho-