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Inhibitory effect of perilla oil on hepatic lipid accumulation in the apoE knock-out mice fed high cholesterol diet

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Perilla oil is dietary source for the omega-3 fatty acid in which alpha-linolenic acid (ALA) is consisted approximately 50% of total fatty acids. Dietary ALA suppress hepatic cholesterol concentrationr[1] but the mechanism is yet to understand. The aim of this study is to investigate the preventive effect of perilla oil on hepatic lipid accumulation in apo E knock-out mice fed with high-cholesterol diet. High-cholesterol diet containing different oils were fed to apo E knock-out mice and its wild type, C57BL/6 mice for 10 weeks. The experimental diet contained 1.25% cholesterol, 4% cocoa oil, 2% coconut butter and 10% different oil-lard (LD), perilla (PE) or sunflower oil (SN). The animal protocol used in this study has been reviewed by the Pusan National University-Institutional Animal Care and Use Committee (PNU-IACUC) on their ethical procedures and scientific care, and it has been approved (Approval Number PNU-2010-00031).

Liver steatosis is studied with histological examination followed by oil red o (ORO) staining. The degree of steatosis induced by high-cholesterol diet was determined quantitatively using the histogram function in Photoshop 7.01 $^{TM(2)}$. Lipid peroxidation was determined as thiobarbituric acid reactive substances (TBARS) concentration[3]. Hepatic lipid concentration was determined after total lipid extraction by Folch method[4]. Western blotting was performed to detect the transcription factor protein responsible for the regulation of cholesterol and lipid metabolism.

Table 1. Hepatic lipid concentration, TBARS, and degree of steatosis

		TG (mg/g liver)		TC (mg/g liver)		TBARS (nmol/g liver)		Steatosis (%)	
	Group	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Wild-type	LD	51.93†	6.43	25.59†‡	2.85	6.88*	2.47	41.37†	12.46
	PE	28.41*	8.47	17.76*	3.78	16.96*	7.10	29.40*	3.98
apo E knock-out type	LD	61.67†	5.14	37.23§	9.13	17.74*	6.81	57.27†	16.14
	PE	56.78†‡	7.97	23.93*†	3.85	35.71†	13.02	43.84†	8.24
	SN	58.78†‡	3.63	30.96‡§	4.04	41.97†	16.81	45.23†	8.56

Values are means four independent experiment. *†‡§Mean values were significantly different with $P<0.05$(ANOVA, followed by Duncan's multiple test).

As shown in Table 1, lipid accumulation for the WPE group was lower than that for the WLD group. This phenomenon was also observed in Apo E knock-out mice. Degree of steatosis for APE was the lowest. Hepatic TG and TC concentration for the PE group in wild type mice were lower than that for the LD groups ($P<0.05$). Among the apo E knock-out type mice, there was no significant difference in hepatic TG concentration, while the hepatic TC concentration for the PE group was significantly lower than that for the LD group ($P<0.05$). But hepatic lipid peroxidation was enhanced in the both type of mice fed PE or SN diet than LD diet. It might be due to the degree of the polyunsaturation in these oils. According to these results, PE seems to decrease TC accumulation in the liver of mice regardless of strain, when high cholesterol diet was fed. But hepatic TG concentration reduction by PE diet was observed only in wild type mice. According to Kubo *et al*, hepatic lipid peroxidation depends on the relative peroxidisability index[4] sterol-regulatory-element-binding protein -1 (SREBP-1, the transcription factor protein responsible for the regulation of cholesterol and lipid metabolism was significantly reduced in WPE ($P<0.05$), as well as in APE mice. In conclusion, PE oil seems to have beneficial effects on hepatic lipid accumulation via suppressing the transcription factor protein that regulate cholesterol and lipid metabolism in the liver.

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