Functional Foods in Health and Disease 2013; 3(11):428-440

Research Article Open Access

Krill protein hydrolysate reduces plasma triacylglycerol level with concurrent increase in plasma bile acid level and hepatic fatty acid catabolism in high-fat fed mice

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Submission date: September 10, 2013; Acceptance date: November 12, 2013; Publication date: November 18, 2013

ABSTRACT

Background: Krill powder, consisting of both lipids and proteins, has been reported to modulate hepatic lipid catabolism in animals. Fish protein hydrolysate diets have also been reported to affect lipid metabolism and to elevate bile acid (BA) level in plasma. BA interacts with a number of nuclear receptors and thus affects a variety of signaling pathways, including very low density lipoprotein (VLDL) secretion. The aim of the present study was to investigate whether a krill protein hydrolysate (KPH) could affect lipid and BA metabolism in mice.

Method: C57BL/6 mice were fed a high-fat (21%, w/w) diet containing 20% crude protein (w/w) as casein (control group) or KPH for 6 weeks. Lipids and fatty acid composition were measured from plasma, enzyme activity and gene expression were analyzed from liver samples, and BA was measured from plasma.

Results: The effect of dietary treatment with KPH resulted in reduced levels of plasma triacylglycerols (TAG) and non-esterified fatty acids (NEFAs). The KPH treated mice had also a marked increased plasma BA concentration. The increased plasma BA level was associated with induction of genes related to membrane canalicular exporter proteins (*Abcc2*, *Abcb4*) and to BA exporters to blood (*Abcc3* and *Abcc4*). Of note, we observed a 2-fold increased nuclear farnesoid X receptor (*Fxr*) mRNA levels in the liver of mice fed KPH. We also observed increased activity of the nuclear peroxiosme proliferator-activated receptor alpha (PPARα) target gene carnitine plamitoyltransferase 2 (CPT-2).

Conclusion: The KPH diet showed to influence lipid and BA metabolism in high-fat fed mice. Moreover, increased mitochondrial fatty acid oxidation and elevation of BA concentration may regulate the plasma level of TAGs and NEFAs.

Key words: Krill protein hydrolysate, triacylglycerol, fatty acids, TNFα