

## The effect of seamustard on blood lipid profiles and glucose level of rats fed diet with different energy composition\*

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### Abstract

Recently, Korean people are consuming seaweeds almost 3.5 times more now than three decades ago. It is well known that seaweeds contain lots of soluble dietary fiber in addition to micronutrients such as  $\beta$ -carotene, iodine and some bioactive components. Seaweeds are considered to be effective for preventing chronic diseases including obesity, diabetes mellitus, atherosclerosis, cancer or constipation. This study was conducted to investigate the effect of seamustard intake on body weight gain, blood glucose level and lipid profiles in rats fed diets with different energy nutrient composition. Male Sprague-Dawley rats (average initial weight 103.7 g) were divided into groups for two experiments as follows; Control, M2.5 & M5 groups (Exp. I) and M5, M10, HCM5, HCM10, HFM5 & HFM10 groups (Exp. II). The rats were fed diet and water ad libitum for 4 weeks. In general, there was no significant difference in blood glucose and triglyceride concentration among groups. In Exp. I, serum LDL-cholesterol level of rats fed diet with 5% seamustard powder (M5) was significantly lower than that of control group, while HDL-cholesterol level, TC/LDL ratio and weight of adrenal gland were higher. In Exp. II, food intake, body weight gain and EER of high fat diet with 10% seamustard group (HFM10) were the lowest among groups. Except gastrocnemius muscle, all organ weights of HFM10 group were the lowest. Fecal cholesterol excretion and serum LDL-cholesterol concentration of HFM10 group were the highest, while serum HDL-cholesterol level was the lowest among groups. Interestingly, HDL-cholesterol concentration was the highest in HCM5 group among groups. From these results, it was suggested that seamustard intake might be more effective for body weight control, but not for improving blood lipid profiles in high fat diet than in high carbohydrate diet.

**Key Words:** Seamustard, dietary energy composition, blood lipid profile, weight loss, Sprague-Dawley rats

### Introduction

According to the third National Health and Nutrition Survey (Ministry of Health and Welfare, 2006), Korean consumed more animal foods and less plant foods during the past 30 years. However, the consumption of seaweeds such as sea tangle, seamustard, and sea lettuce has continued to increase 3.5 times from 2.4 g/d in 1970 to 8.5 g/d in 2005. Traditionally, many postpartum women in Korea have used a seamustard soup in order to return to the pre-pregnant state physiologically as fast as possible. According to Donguibogam written by Heo Jun who was the famous doctor in the Joseon Dynasty, seamustard was used to stimulate the excretion or removal of watery components after delivery (Choi, 2003). Nowadays marine natural products are considered to be effective for preventing chronic diseases such as obesity, diabetes mellitus, atherosclerosis, cancer as well as constipation (BFN, 2005). Various seaweeds contain lots of alginic acid, in addition to beta carotene, iodine or bioactive components such as fucoidan. Among them, alginic acid, a kind of soluble viscous fiber, is one of the important healthy components. One of its well-known physiological functions is a LDL cholesterol-lowering effect

(Anderson & Hanna, 1999; Fernandez, 2001).

Hypertriglyceridemia and cholesterolemia seem to confer a higher risk of atherosclerotic cardiovascular disease in diabetic person than non-diabetics (Hu *et al.*, 2001; Rubins *et al.*, 1999). Including animal fat and trans-fatty acid, dietary modification of protein, carbohydrate, fiber or phytosterol could affect cholesterol metabolism. Recently, a low fat and high carbohydrate diet is considered to protect against heart disease. However, the fasting triglyceride level would be increased if there were no dietary fiber in high carbohydrate diet (Robins *et al.*, 2003).

This study was conducted to investigate the effects of seamustard powder on body weight change, blood glucose concentration and lipid profiles in rats fed diet with different energy nutrient composition.

### Materials and Methods

#### *Animals and diets*

Male Sprague-Dawley rats (average initial weight 103.7 g, n=48) were divided into eight groups as follows: Control diet,

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**Table 1.** Dietary composition of groups in Exp. I (g/kg diet)

Components	Experiment Groups <sup>1)</sup>		
	Control	M2.5	M5
Starch		400.684	
Casein		250.0	
Fat+oil*		150.0	
Sucrose		100.0	
Mineral mixture <sup>2)</sup>		35.0	
Vitamin mixture <sup>3)</sup>		10.0	
L-cystine		1.8	
t-Butylhydroquinone		0.016	
Choline bitartrate		2.5	
Cellulose	50.0	25.0	-
Seamustard	-	25.0	50.0
Total	1000	1000	1000

<sup>1)</sup> All diets were based on AIN -93G diet.

M2.5 : control diet with 2.5% seamustard powder as fiber source (4.67 kcal/g)

M5 : control diet with 5% seamustard powder as fiber source (4.70 kcal/g)

<sup>2)</sup> Mineral mixture : calcium carbonate (35.7%), potassium phosphate monobasic (25.0%), potassium citrate monohydrate (2.8%), sodium chloride (7.4%), potassium sulfate (4.66%), magnesium oxide (2.43%), ferric citrate (0.606%), zinc carbonate (0.165%), manganese carbonate (0.063%), cupric carbonate (0.031%), potassium iodate (0.001%), sodium selenate (0.001%), ammonium paramolybdate (0.001%), sodium metasilicate (0.145%), chromium potassium sulfate (0.028%), lithium chloride (0.002%), boric acid (0.008%), sodium fluoride (0.006%), nickel carbonate hydroxide tetrahydrate (0.003%), ammonium vanadate (0.001%) and sucrose (20.95%)

<sup>3)</sup> Vitamin mixture : nicotinic acid (0.3%), calcium pantothenate (0.16%), pyridoxine-HCl (0.07%), thiamin HCl (0.06%), riboflavin (0.06%), folic acid (0.02%), D-biotin (0.002%), Vit B12 in 0.1% mannitol (0.25%), DL- $\alpha$ -tocopherol acetate, 500 IU/g (1.50%), retinol palmitate, 500,000 IU/g (0.08%), Vit D3 (50,000 IU/g) (0.02%), Vit K (0.007%) and sucrose (97.47%)

\* lard : soybean oil = 1:1 (w/w)

**Table 2.** Dietary composition of groups in Exp. II (g/kg diet)

Components	Experiment Groups <sup>1)</sup>					
	M5	M10	HCM5	HCM10	HFM5	HFM10
Starch	400.684	350.684	625.684	575.684	300.684	250.684
Casein	250.0	250.0	120.0	120.0	120.0	120.0
Fat+oil*	150.0	150.0	75.0	75.0	280.0	280.0
Sucrose	100.0	100.0	80.0	80.0	200.0	200.0
Mineral mixture <sup>2)</sup>			35.0			
Vitamin mixture <sup>3)</sup>			10.0			
L-cystine			1.8			
t-Butylhydroquinone			0.016			
Choline bitartrate			2.5			
Seamustard	50.0	100.0	50.0	100.0	50.0	100.0
Total	1000	1000	1000	1000	1000	1000

<sup>1)</sup> All diets were based on AIN -93G diet.

M5 : control diet with 5% seamustard powder as fiber source (4.70 kcal/g)

M10 : control diet with 10% seamustard powder as fiber source (4.67 kcal/g)

HCM5 : high carbohydrate diet with 5% seamustard powder as fiber source (4.34 kcal/g)

HCM10: high carbohydrate diet with 10% seamustard powder as fiber source (4.26 kcal/g)

HFM5: high fat diet with 5% seamustard powder as fiber source (5.67 kcal/g)

HFM10: high fat diet with 10% seamustard powder as fiber source (5.53 kcal/g)

<sup>2-3)</sup> See Table 1.

\* lard : soybean oil = 1:1 (w/w)

M2.5 (2.5% seamustard diet), M5 (5% seamustard diet), M10 (10% seamustard diet), HCM5 (high carbohydrate+5% seamustard diet), HCM10 (high carbohydrate+10% seamustard diet), HFM5 (high fat+5% seamustard diet) and HFM10 (high fat+10% seamustard diet). In fact, two experiments were designed concomitantly. Experiment I was consisted of three groups (control, M2.5 & M5) according to the level of seamustard powder in control diet. Experiment II was consisted of six groups (M5, M10, HCM5, HCM10, HFM5 & HFM10) according to seamustard level and dietary energy composition. All diets were based on AIN-93G diet (Reeves, 1997) as shown Table 1. All ingredients of diets were purchased from Labanimal Co. (Dyets, Bethlehem, PA). Total experiment period was 4 weeks. The animals were fed diet and tap water ad libitum with alternating 12-h light/12-h dark cycle in temperature controlled (20-22 °C) room at a relative humidity (50-60%). From the food intake and body weight gain of rats, food efficiency ratio (FER) and energy efficiency ratio (EER) were calculated. For three days a week, feces were collected and weighed immediately and stored at -20 °C in the refrigerator. All animal procedures conformed to "Guide for the Care and Use of Laboratory Animals" (National Research Council, 1996).

#### Sample collection and biochemical analysis

The blood samples were collected from the carotid of rats anesthetized with ethyl ether. Samples were then centrifuged at 3,000 rpm for 20 minutes and the serum was separated. The organs such as liver, spleen, kidneys, adrenal glands, epididymal fat pads and gastrocnemius muscle were removed and weighed immediately. All samples were stored at -70 °C until analysis. The concentrations of total- and HDL-cholesterol, triglyceride and glucose in serum were determined enzymatically by using commercial kit (Asan Pharmaceutical, Seoul, Korea). Serum LDL-cholesterol level was calculated from the equation of Friedewald *et al.* (1972). The total lipid contents in liver and feces were extracted with chloroform: methanol mixture (v/v, 2:1) and measured using the method of Bligh and Dyer (1959). Hepatic and fecal cholesterol concentrations in the lipid extracts were measured enzymatically by using commercial kit (Asan Pharmaceutical, Seoul, Korea).

#### Statistical analysis

All data were expressed as mean  $\pm$  SD. The significance of difference among groups was determined by one way analysis of variance (ANOVA) using the SPSS program v.14 (SPSS, Chicago, IL, USA). The result was considered to be significantly different if the p value was <.05, and then Duncan's multiple range test was performed if differences were identified among groups.

## Results

### Exp. I

The food and energy intake, body weight gain, food efficiency ratio (FER) and energy efficiency ratio (EER) of rats are shown in Table 3. Energy intake of M5 group was the lowest among groups. However, food intakes, body weight and body weight gain were not significantly different among groups. Consequently, there were no significant differences in FER and EER among groups according to seamustard level.

As shown in Table 4, for weights of liver, spleen, kidney and gastrocnemius muscle there were no significant differences among three groups. However, seamustard intake resulted to affect the weights of epididymal fat pads and adrenal glands

( $p < .05$ ). Adrenal glands weight of M5 group was the highest, but the weight of epididymal fat pads was significantly lower than that of M2.5 group ( $p < .05$ ).

As shown in Table 5, all of daily fecal excretion, fecal lipid and cholesterol contents, hepatic lipid and cholesterol contents did not show any significant differences among groups according to seamustard level.

The serum concentrations of glucose, triglyceride and total cholesterol (TC) did not show any significant differences among three groups. Interestingly, serum LDL-cholesterol (LDL) concentration of M5 group was the lowest among groups. However, serum HDL-cholesterol concentrations of M2.5 and M5 groups were higher than that of control group (Table 6). Collectively shown in Table 7, the ratios of TC / LDL of M2.5 and M5 groups were significantly higher than that of control

**Table 3.** Food intakes, body weight and body weight gain of rats in Exp. I

Experiment Groups	Food Intakes (g/4 wks)	Energy Intakes (kcal/4 wks)	Final BW (g)	BW Gain (g/4 wks)	FER	EER
Control	64.5 ± 4.4 <sup>1)N.S.</sup>	299.9 ± 20.5 <sup>ab2)</sup>	256.8 ± 17.7 <sup>N.S.</sup>	152.8 ± 11.7 <sup>N.S.</sup>	2.38 ± 0.28 <sup>N.S.</sup>	0.51 ± 0.06 <sup>N.S.</sup>
M2.5	68.8 ± 5.5	321.7 ± 25.5 <sup>a</sup>	281.6 ± 30.8	177.7 ± 31.0	2.59 ± 0.45	0.55 ± 1.21
M5	59.1 ± 7.6	277.9 ± 20.5 <sup>b</sup>	254.0 ± 35.0	150.0 ± 33.4	2.51 ± 0.34	0.54 ± 0.09

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

N.S, no significant difference

BW body weight

FER (food efficiency ratio) = body weight gain/food intakes

EER (energy efficiency ratio) = body weight gain/energy intakes

**Table 4.** Organ weights of rats in Exp. I

(g)

Experiment Groups	Liver	Spleen	Kidney	Adrenal gland	Epididymal Fat pad	Gastrocnemius
Control	8.48 ± 1.22 <sup>1)N.S.</sup>	0.46 ± 0.10 <sup>N.S.</sup>	2.05 ± 0.28 <sup>N.S.</sup>	0.024 ± 0.007 <sup>b2)</sup>	2.10 ± 0.46 <sup>b</sup>	2.78 ± 0.94 <sup>N.S.</sup>
M2.5	9.58 ± 1.20	0.59 ± 0.14	2.12 ± 0.19	0.030 ± 0.015 <sup>ab</sup>	2.81 ± 0.80 <sup>a</sup>	2.04 ± 0.94
M5	8.39 ± 1.87	0.55 ± 0.13	2.19 ± 0.26	0.046 ± 0.024 <sup>a</sup>	2.01 ± 0.32 <sup>b</sup>	2.58 ± 0.86

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

N.S, no significant difference

**Table 5.** Fecal lipid and hepatic lipid contents of rats Exp. I

Experiment Groups	Fecal Weight* (g)	Fecal Lipid (g/g feces)	Fecal Cholesterol (mg/g feces)	Hepatic Lipid (g/g tissue)	Hepatic Cholesterol (mg/g tissue)
Control	18.58 ± 2.26 <sup>1)N.S.</sup>	0.081 ± 0.019 <sup>N.S.</sup>	6.05 ± 0.85 <sup>N.S.</sup>	0.110 ± 0.018 <sup>N.S.</sup>	62.19 ± 5.93 <sup>N.S.</sup>
M2.5	17.48 ± 4.96	0.086 ± 0.016	7.80 ± 1.32	0.111 ± 0.031	80.02 ± 28.34
M5	12.75 ± 3.37	0.116 ± 0.023	6.32 ± 1.87	0.126 ± 0.039	59.27 ± 7.63

<sup>1)</sup> Mean ± SD

N.S, no significant difference

\*The feces were collected from 3 week to 4 week during experimental periods.

**Table 6.** Serum glucose concentration and lipid profile of rats in Exp. I

Experiment Groups	Serum Glucose Concentration (mg/dl)	Serum Lipid Concentration			
		TG (mg/dl)	TC (mg/dl)	LDL (mg/dl)	HDL (mg/dl)
Control	119.31 ± 23.26 <sup>1)N.S.</sup>	19.45 ± 9.10 <sup>N.S.</sup>	70.32 ± 13.04 <sup>N.S.</sup>	51.44 ± 14.14 <sup>a2)</sup>	14.98 ± 7.82 <sup>b</sup>
M2.5	119.34 ± 10.98	25.90 ± 3.67	77.02 ± 18.18	45.45 ± 18.42 <sup>a</sup>	26.38 ± 7.03 <sup>a</sup>
M5	118.63 ± 24.12	25.84 ± 24.01	64.31 ± 11.34	36.16 ± 8.95 <sup>b</sup>	22.98 ± 3.43 <sup>ab</sup>

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

N.S, no significant difference

**Table 7.** The risk for cardiovascular disease of rats in Exp. I

Experiment Groups	Ratio of Lipid Profiles		
	TC/LDL	TC/HDL	LDL/HDL
Control	1.41 ± 0.20 <sup>1)bc2)</sup>	3.94 ± 0.91 <sup>N.S.</sup>	2.74 ± 0.88 <sup>N.S.</sup>
M2.5	1.80 ± 0.38 <sup>a</sup>	3.08 ± 1.10	1.87 ± 1.09
M5	1.82 ± 0.28 <sup>a</sup>	2.85 ± 0.61	1.62 ± 0.52

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

N.S., no significant difference

group. However, in the ratio of TC to HDL and the ratio of LDL to HDL, there were no significant differences among groups, even though the ratios tended to decrease according to seamustard level.

### Exp. II

The food intake, energy intake, body weight gain, food efficiency ratio (FER) and energy efficiency ratio (EER) of rats are shown in Table 8. Food intakes of high fat diet groups (HFM5 & HFM10) were significantly lower ( $p<0.05$ ) than those of high carbohydrate diet groups (HCM5 & HCM10). Energy intake was

the highest in HCM5 group and the lowest in M5 group; final body weight and body weight gain were the highest in M10 group and the lowest in HFM10 group ( $p<0.05$ ). FER was the lowest in HCM5 group, and EER was the lowest in HFM10 group, while both of FER and EER were the highest in M10 group. Conclusively, seamustard affected only body weight gain, while dietary energy composition affected both food intake and body weight gain of rats.

As shown in Table 9, the weights of liver and kidney of HFM10 group were significantly lower than those of the other groups ( $p<0.05$ ). Spleen weight of high fat diet groups (HFM5 & HFM10) were significantly lower than those of M5 and M10 groups ( $p<0.05$ ), and adrenal glands weight of HFM10 group was the lowest among groups. The weight of epididymal fat pads was the lowest in HFM10 group, but there were no significant differences in weight of gastrocnemius muscle among all groups. Consequently, dietary energy composition affected the weights of liver and kidney, but seamustard affected the weight of gastrocnemius.

As shown in Table 10, daily fecal weight and fecal lipid content did not show any significant difference among groups. Fecal cholesterol excretion of HFM5, HFM10 and HCM10 groups were

**Table 8.** Food intakes, body weight and body weight gain of rats in Exp. II

Experiment Groups	Food Intakes (g/4 wks)	Energy Intakes (kcal/4 wks)	Final BW (g)	BW Gain (g/4 wks)	FER	EER
M5	59.1 ± 7.6 <sup>1)bc2)</sup>	277.9 ± 20.5 <sup>c</sup>	254.0 ± 35.0 <sup>a</sup>	150.0 ± 33.4 <sup>ab</sup>	2.51 ± 0.34 <sup>ab</sup>	0.54 ± 0.09 <sup>ab</sup>
M10	61.1 ± 10.8 <sup>bc</sup>	285.2 ± 36.2 <sup>bc</sup>	272.3 ± 39.2 <sup>a</sup>	168.5 ± 40.7 <sup>a</sup>	2.74 ± 0.25 <sup>a</sup>	0.59 ± 0.11 <sup>a</sup>
HCM5	71.7 ± 4.8 <sup>a</sup>	311.2 ± 20.8 <sup>a</sup>	257.0 ± 20.9 <sup>a</sup>	153.3 ± 21.0 <sup>a</sup>	2.15 ± 0.35 <sup>b</sup>	0.49 ± 0.10 <sup>ab</sup>
HCM10	67.0 ± 5.9 <sup>ab</sup>	285.4 ± 25.1 <sup>bc</sup>	250.3 ± 21.6 <sup>ab</sup>	146.8 ± 24.1 <sup>ab</sup>	2.19 ± 0.30 <sup>b</sup>	0.51 ± 0.10 <sup>ab</sup>
HFM5	54.1 ± 6.6 <sup>c</sup>	306.6 ± 32.3 <sup>ab</sup>	237.6 ± 22.8 <sup>ab</sup>	134.1 ± 22.6 <sup>ab</sup>	2.47 ± 0.21 <sup>ab</sup>	0.44 ± 0.07 <sup>ab</sup>
HFM10	53.9 ± 4.7 <sup>c</sup>	297.9 ± 26.0 <sup>ab</sup>	219.9 ± 17.5 <sup>b</sup>	116.5 ± 19.4 <sup>b</sup>	2.17 ± 0.39 <sup>b</sup>	0.39 ± 0.07 <sup>b</sup>
Significant Factor <sup>3)</sup>	B		B	A, B		

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

<sup>3)</sup> A: The effect of seamustard

B: The effect of dietary energy composition

AB: The effect of seamustard and dietary energy composition

N.S., no significant difference

BW body weight

FER (food efficiency ratio) = body weight gain/food intakes

EER (energy efficiency ratio) = body weight gain/energy intakes

**Table 9.** Organ weights of rats in Exp. II

Experiment Groups	Liver	Spleen	Kidney	Adrenal gland	Epididymal Fat pad	Gastrocnemius
M5	8.39 ± 1.87 <sup>1)ab2)</sup>	0.55 ± 0.13 <sup>ab</sup>	2.19 ± 0.26 <sup>a</sup>	0.046 ± 0.024 <sup>a</sup>	2.01 ± 0.32 <sup>ab</sup>	2.58 ± 0.86 <sup>N.S.</sup>
M10	8.28 ± 2.22 <sup>a</sup>	0.62 ± 0.15 <sup>a</sup>	2.02 ± 0.80 <sup>a</sup>	0.027 ± 0.006 <sup>b</sup>	1.92 ± 0.23 <sup>ab</sup>	2.34 ± 0.72
HCM5	8.07 ± 0.55 <sup>a</sup>	0.53 ± 0.15 <sup>ab</sup>	1.89 ± 0.31 <sup>abc</sup>	0.026 ± 0.009 <sup>b</sup>	2.17 ± 0.39 <sup>a</sup>	2.39 ± 0.93
HCM10	7.52 ± 1.81 <sup>ab</sup>	0.55 ± 0.08 <sup>ab</sup>	1.66 ± 0.29 <sup>bc</sup>	0.033 ± 0.018 <sup>ab</sup>	1.77 ± 0.36 <sup>ab</sup>	1.93 ± 1.45
HFM5	6.95 ± 1.46 <sup>ab</sup>	0.46 ± 0.10 <sup>b</sup>	1.67 ± 0.17 <sup>bc</sup>	0.028 ± 0.006 <sup>b</sup>	1.69 ± 0.30 <sup>ab</sup>	2.51 ± 1.07
HFM10	6.16 ± 1.07 <sup>b</sup>	0.41 ± 0.05 <sup>b</sup>	1.50 ± 0.21 <sup>c</sup>	0.024 ± 0.006 <sup>b</sup>	1.51 ± 0.59 <sup>b</sup>	2.06 ± 0.58
Significant Factor <sup>3)</sup>	B		B			A

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

<sup>3)</sup> A: The effect of seamustard

B: The effect of dietary energy composition

AB: The effect of seamustard and dietary energy composition

N.S., no significant difference

(g)

**Table 10.** Fecal and hepatic lipid and cholesterol contents of rats in Exp. II

Experiment Groups	Fecal Weight* (g)	Fecal Lipid (g/g feces)	Fecal Cholesterol (mg/g feces)	Hepatic Lipid (g/g tissue)	Hepatic Cholesterol (mg/g tissue)
M5	12.75 ± 3.37 <sup>1)n.s.</sup>	0.116 ± 0.023 <sup>n.s.</sup>	6.32 ± 1.87 <sup>2)</sup>	0.126 ± 0.039 <sup>n.s.</sup>	59.27 ± 7.63 <sup>n.s.</sup>
M10	21.50 ± 11.90	0.095 ± 0.014	8.07 ± 2.45 <sup>bc</sup>	0.100 ± 0.016	50.88 ± 15.45
HCM5	14.01 ± 5.01	0.175 ± 0.165	6.18 ± 1.89 <sup>c</sup>	0.118 ± 0.048	54.46 ± 32.03
HCM10	19.75 ± 5.44	0.116 ± 0.054	9.68 ± 0.51 <sup>ab</sup>	0.098 ± 0.020	37.23 ± 6.25
HFM5	16.16 ± 4.39	0.139 ± 0.084	11.56 ± 1.10 <sup>a</sup>	0.126 ± 0.030	55.94 ± 30.76
HFM10	18.50 ± 5.21	0.177 ± 0.062	11.56 ± 3.17 <sup>a</sup>	0.136 ± 0.046	41.51 ± 32.22
Significant Factor <sup>3)</sup>	A		B		A

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

<sup>3)</sup> A: The effect of seamustard

B: The effect of dietary energy composition

AB: The effect of seamustard and dietary energy composition

N.S. no significant difference

\*The feces were collected from 3 week to 4 week during experimental periods.

**Table 11.** Serum glucose concentration and lipid profile of rats in Exp. II

Experiment Groups	Serum Glucose Concentration (mg/dl)	Serum Lipid Concentration			
		TG (mg/dl)	TC (mg/dl)	LDL (mg/dl)	HDL (mg/dl)
M5	118.63 ± 24.12 <sup>1)n.s.</sup>	25.84 ± 24.01 <sup>n.s.</sup>	64.31 ± 11.43 <sup>n.s.</sup>	36.16 ± 8.95 <sup>b2)</sup>	22.98 ± 3.43 <sup>ab</sup>
M10	116.33 ± 24.89	19.15 ± 9.41	60.66 ± 7.74	37.97 ± 4.89 <sup>b</sup>	18.87 ± 6.80 <sup>abc</sup>
HCM5	123.88 ± 21.35	17.72 ± 4.80	67.87 ± 8.62	39.93 ± 7.71 <sup>ab</sup>	24.39 ± 6.23 <sup>a</sup>
HCM10	114.46 ± 20.70	19.05 ± 10.80	63.62 ± 7.92	43.83 ± 5.82 <sup>a</sup>	15.98 ± 3.32 <sup>bc</sup>
HFM5	107.80 ± 13.09	13.74 ± 3.57	61.55 ± 4.50	42.74 ± 5.06 <sup>ab</sup>	16.06 ± 2.36 <sup>bc</sup>
HFM10	109.52 ± 28.75	15.27 ± 3.39	66.83 ± 3.65	50.13 ± 4.13 <sup>a</sup>	13.65 ± 6.42 <sup>c</sup>
Significant Factor <sup>3)</sup>	B				AB

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

<sup>3)</sup> A: The effect of seamustard

B: The effect of dietary energy composition

AB: The effect of seamustard and dietary energy composition

N.S. no significant difference

**Table 12.** The risk for cardiovascular disease of rats in Exp. II

Experiment Groups	Ratio of Lipid Profiles		
	TC/LDL	TC/HDL	LDL/HDL
M5	1.82 ± 0.28 <sup>1)a2)</sup>	2.85 ± 0.61 <sup>b</sup>	1.62 ± 0.52 <sup>b</sup>
M10	1.61 ± 0.20 <sup>abc</sup>	3.69 ± 1.40 <sup>b</sup>	2.37 ± 1.32 <sup>b</sup>
HCM5	1.73 ± 0.22 <sup>ab</sup>	2.88 ± 0.55 <sup>b</sup>	1.72 ± 0.51 <sup>b</sup>
HCM10	1.46 ± 0.10 <sup>bc</sup>	4.11 ± 0.89 <sup>b</sup>	2.87 ± 0.83 <sup>b</sup>
HFM5	1.45 ± 0.10 <sup>bc</sup>	3.90 ± 0.59 <sup>b</sup>	2.72 ± 0.58 <sup>b</sup>
HFM10	1.34 ± 0.15 <sup>c</sup>	6.10 ± 3.20 <sup>a</sup>	4.82 ± 3.05 <sup>a</sup>
Significant Factor <sup>3)</sup>	AB	A	A

<sup>1)</sup> Mean ± SD

<sup>2)</sup> Significantly different among groups by Duncan's multiple range test at  $\alpha=0.05$  level after one way ANOVA.

<sup>3)</sup> A: The effect of seamustard

B: The effect of dietary energy composition

AB: The effect of seamustard and dietary energy composition

N.S. no significant difference

significantly higher than that of HCM5 group. However, there were no significant differences in hepatic total lipid and cholesterol contents among groups. As a result, seamustard affected fecal weights and hepatic cholesterol contents, while dietary energy composition might affect fecal cholesterol excretion.

As shown in Table 11, the concentrations of glucose, triglyceride and total cholesterol (TC) in serum showed no significant differences among groups, even though serum glucose and triglyceride concentrations of high fat diet groups (HFM5 & HFM10) tended to be lower than other groups. However, serum LDL-cholesterol (LDL) concentrations of these groups were higher than those of control diet groups (M5 & M10). Serum HDL-cholesterol (HDL) concentration of HFM10 group was the lowest among all groups. As a result, 10% seamustard level and high fat diet affected serum cholesterol profiles of rats undesirably. As shown in Table 12, the ratios TC/LDL of M10, HCM10, and HFM10 groups tended to be lower than those of M5, HCM5, and HFM5 groups, respectively. However, the ratios TC/HDL and LDL/HDL were not significantly different among groups, except those of HFM10 group. Extraordinarily in HFM10 group, the risk for atherosclerotic cardiovascular diseases, the ratio of TC/HDL was the highest. From the results, it was observed that seamustard as a dietary fiber source might confer the risk for cardiovascular disease in high fat diets statistically.

## Discussion

Generally, it has been recognized that dietary fiber had numerous important physiological effects on the gastrointestinal tract, which might be attributed in large part to the viscosity or fermentability of the fiber sources. In upper gastrointestinal tract, an important attribute of fiber is its viscosity which may lead to delay in gastric emptying, interfere with or prolonged absorption of other nutrients, for example, cholesterol or glucose (Lupton & Trumbo, 2006).

Viscous fibers, such as pectin, guar, oat bran and psyllium are considered to have a cholesterol-lowering effect (Fernandez, 2001), a reducing hunger or weight loss (Birketvedt, 2000) as well as a protective effect against cardiovascular disease (Truswell, 2002). The well-known physiological function of soluble viscous fiber is a LDL cholesterol-lowering effect (Anderson & Hanna, 1999; Fernandez, 2001).

In this study, there were no significant differences in fecal and hepatic cholesterol changes by seamustard consumption in control group. However, 10% seamustard level induced to increase fecal cholesterol excretion and to decrease hepatic cholesterol contents remarkably in high carbohydrate diet groups (Table 5, Table 10). From these results, it was expected that seaweeds containing soluble viscous fiber and some healthy components, might affect bile acid excretion to the intestine and affect the cholesterol metabolism at higher level in rats fed with high carbohydrate diet.

Interestingly, seamustard at below 5% level in control group induced to decrease LDL-cholesterol (LDL) and to increase HDL-cholesterol (HDL). In contrast, 10% seamustard level induced to increase LDL of rats fed with high carbohydrate diet or high fat diet significantly compared to rats fed with control diet. Assessment of risk for coronary disease is usually done by measuring TC and LDL in blood as well as the ratios TC/LDL, TC/HDL or LDL/HDL. Elevated LDL is considered to be a major risk factor, and HDL-cholesterol is a major protective factor for cardiovascular diseases (Kritchevsky, 2006). It is reported that the risk for coronary disease may be high, especially when the ratio of TC/HDL is greater than 4:1. It may be optimal when that ratio is 3.5:1 or less (Wardlaw & Smith, 2009). In this study, the TC/HDL ratios of all diet groups were similar or lower than 4:1, except HFM10 group (6:1) in which rats were fed high fat diet with 10% seamustard powder as fiber source (Table 7, Table 12). From the results, it was observed that the higher was the seamustard level, the worse serum lipid profile was in high fat diet especially.

There were no significant changes in serum triglyceride as well as glucose concentration among all groups. It has been reported that if high carbohydrate diet was also a high fiber diet, fasting triglyceride level in blood would be reduced while it is usually increased in high carbohydrate and low fiber diet (Anderson, 2000). Total fiber of seamustard powder is about 14.2 g per 100 g edible portion (National Rural Resources Development Institute,

2006). Therefore, dietary fiber levels in diets of Exp. I groups were 5% (cellulose) in control diet, 2.85% (cellulose+seamustard fiber) in M2.5 group and 0.71% (seamustard fiber) in M5 group, respectively. The seamustard fiber levels in diets in Exp. II groups were 0.71% in M5, HCM5, and HFM5 groups and 1.42% in M10, HCM10, and HFM10 groups. According to replacement of cellulose by seamustard powder partially or completely, substantial dietary fiber level was dramatically reduced. From these results, it could be speculated that seamustard effect on body weight gain or serum lipid profiles relied on its soluble fiber, alginic acid and/or other components. Seaweeds contain much  $\beta$ -carotene, iodine, or a bioactive components such as fucoidan. Nowadays, these substances are known to be effective for preventing chronic diseases such as obesity, diabetes mellitus, atherosclerosis as well as cancer. In fact, interaction of both seamustard intake and dietary energy composition resulted in significantly different effects on body weight and HDL-cholesterol between high fat diet and high carbohydrate diet.

Sometimes the reduction of LDL level implied lowering levels of circulating carotenoids and vitamin E because they are transported in LDL (Noakes *et al.*, 2002). Olistat, a chemically synthesized derivative of lipostatin, directly inhibited the lipase activity and resulted in the suppression of triglyceride digestion by around 30%, and thus increasing fecal elimination of fat, and therefore overall effects resulted in a 5 % to 10% weight loss and improved total-, LDL- and HDL-cholesterol levels without significant declines in circulating vitamin A, vitamin D, vitamin E and  $\beta$ -carotene (Hutton & Fegusson, 2004).

Conclusively, the present study suggested that seamustard might be more effective for body weight control in high fat diet than in high carbohydrate diet, but not for improving blood lipid profiles. Even though still remain the difficulties and research limitations to generalize this suggestion. In the future, more well-designed epidemiologic researches would be needed to investigate the beneficial and/or undesirable effects of seamustard consumption on modern population living under the global environments.

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