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A Convergence Study on the Effects of Improving Buckwheat Dietary Fiber in Mice with Hyperlipidemia and Oxidative Stress

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고지혈증과 산화적 스트레스가 유도된 생쥐에서 메밀 식이섬유의 개선 효과에 대한 융합 연구

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Abstract The effect of buckwheat dietary fiber (BDF) as hypolipidemic and antioxidant agent were determined in C57BL/6 mice fed a high fat diet (HFD) with different doses of 500 (low, BDF-L) or 1,000 (high, BDF-H) mg/kg of body weight, compared with the HFD-diet control group (HFD). The negative control group (ND) was fed the basal diet. Body weights in the BDF-L and BDF-H groups were significantly decreased as compared to those in the HFD group ($p<0.05$). BDF also improved the lipid profile in a dose-dependent manner; serum lipid profiles and levels of insulin, glucose, and free fatty acid were significantly decreased in the BDF-L and BDF-H groups, whereas HDL-C and adiponectin significantly increased as compared to the HFD group ($p<0.05$). Meanwhile, BDF lowered serum malondialdehyde (MDA) in comparison with the HFD group ($p<0.05$). The results demonstrate that the intake of BDF might prevent obesity and its related metabolic disorders by inducing dyslipidemia and oxidative stress.

Key Words : Tartary buckwheat, Dietary fiber, High-fat diet, Hypolipidemic effect, Antioxidant effect

요 약 고지방 식이로 고지혈증과 산화적 스트레스를 유도한 생쥐에서 메밀식이섬유의 개선 효과를 확인하고자 하였다. 고지방 식이와 함께 메밀식이섬유(저농도, 500 mg/kg; 고농도, 1,000 mg/kg)를 제공한 그룹의 체중은 고지방 식이만 제공한 그룹과 비교하여 유의적으로 감소하였다($p<0.05$). 또한 메밀식이섬유를 제공한 그룹의 지질 프로파일 개선 효과는 저농도에서 고농도로 용량 의존적으로 관찰되었다. 혈청 지질 프로파일과 인슐린, 포도당 및 유리 지방산의 수준은 메밀식이섬유 제공으로 유의하게 감소한 반면, HDL-C와 디포넥틴은 유의하게 증가했다($p<0.05$). 한편, 고지방 식이만 제공한 그룹과 비교하여 메밀식이섬유 제공으로 혈청 말론다이알데히드(MDA) 수준이 농도 의존적으로 감소하였다($p<0.05$). 결과적으로 메밀식이섬유의 섭취는 이상지질혈증과 산화 스트레스를 개선함으로써 비만과 관련 대사 장애를 예방할 수 있을 것으로 기대할 수 있었다.

주제어 : 메밀, 식이섬유, 고지방식이, 혈청 지질저하효과, 항산화효과

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1. Introduction

A high-fat diet (HFD) is associated with adversely effects, such as insulin resistance, type 2 diabetes mellitus, hyperlipidemia, and cardiovascular disease [1]. It has been also reported that HFD increase fat-mediated oxidative damage and decrease antioxidant enzyme activity [2]. The increased intakes of dietary fiber (DF)-rich diet would be expected to manage the chronic health problem and other high-fat-associated disease [3]. However, as the over the past 200 years, the DF intake has gradually decreased and thus, it is of great interest to explore DF with the most favorable effects, and to understand the related mechanisms [4].

Tartary buckwheat (*Fagopyrum tartaricum*) is a traditional crop and is known as anti-diabetic hypoglycemia agent by managing diabetes mellitus and attenuating hyperglycemia [5]. Tartary buckwheat is rich sources of a high content of DF and multiple flavonoids, including quercetin and rutin [6], which have been reported to present positive effects in exerting anti-diabetic activity by reducing energy intake and the expression off at metabolism related-gene [7]. However, the hyperlipidemic effects of the dietary fiber from Tartary buckwheat on glucose and lipid metabolism has not been explored. Therefore, the present study was designed to investigate the effects of Tartary buckwheat dietary fiber (BDF) on the biochemical parameters and activities of some antioxidant enzymes in serum and hepatic of a high-fat-diet-obese mouse model.

2. Materials and Methods

2.1 Materials

The dietary fiber (DF, 95% purity) from Tartary buckwheat was used in this study (Hunan huacheng Biotech, Inc, China).

2.2 Animal and experimental design

The female C57BL/6mice, aged 6 weeks (16~19 g), were purchased from Samtako BIO KOREA (Seoul, Korea). All mice were housed in individual clean cages at 22 °C with a 12 h dark/light cycle in a humidity control (60~70% humidity) and were allowed free access to water and food. After acclimation of one week, the mice were divided into four groups (8 mice/group): control group (ND) fed standard diet (AIN-76A purified rodent diet 65% corn starch #111753 (Dyets Inc., Bethlehem, PA, USA), high-fat diet group (HFD, Rodent diet with 60% kcal% fat), HDF with low-dose BDF group (BDF-L, BDF 500 mg/kg), and a high-dose BDF group (BDF-H, BDF 1,000 mg/kg). The weight of the mice in each group was recorded weekly during the twelve-week intervention. Serum was collected by centrifugation (3000 g, 4°C, 20 min) from blood sample and was frozen at -70°C until further use. All procedures complied with the Institutional Animal Care and Use Committee (17143 AON) according to the guidelines of the Korea Center for Disease Control and Prevention.

2.3 Oral glucose tolerance test (OGTT)

One day before the termination of the experiment, animals were subjected to OGTT. After overnight fasting, animals orally received a glucose load (2 mg/kg). At that time, the serum glucose levels were measured with the use of a One Touch Select Plus (LifeScan, Johnson and Johnson, New Brunswick, NJ, USA) at 0, 15, 30, 60, and 120 min after glucose load.

The area under the blood glucose response curve was calculated using the following Eq. (1) according to Wolever and Jenkins [8]. GI = (Area

under the curve for sample) / (Area under the curve for glucose) (1)

2.4 Serum biochemical parameters

Concentrations of fasting serum triglycerides (TG) and cholesterol (Total-C, LDL-C, and HDL-C) were determined by enzymatic colorimetric methods using commercial test kits, and all the results were expressed in mg/dL. Serum glucose and free fattyacid (FFA) was measured using a commercially available diagnostic kit (Wako Pure Chemical Industries Ltd, Japan). Serum insulin and adiponectin concentrations were determined by a competitive inhibition method of ELISA using ELISA kit (Insulin cat no. AKRIN-011RU and Adiponectin cat no. AKMAN-011, respectively). Activities of serum function enzyme, alanine transaminase and aspartate transaminase (ALT & AST) were measured using diagnostic kits (Hitachi 2030, Hitachi Ltd, Japan) and were presented as units per liter (U/L).

2.5 Lipid peroxidation Assay

Determination of the lipid peroxidation end product in liver, malondialdehyde (MDA) by thiobarbituric acid (TBA) was analyzed using lipid peroxidation (MDA) assay kit (Abcam Plc., USA). The concentrations of protein were measured by commercial kit (Bio-Rad Laboratories, Hercules, CA) and the results were expressed as MDA formation/mg protein.

2.6 Statistical analysis

All the *in vivo* experimental data are presented as the mean ± SD. Statistical analysis were performed using one-way analysis of variance (one-way ANOVA) and the comparisons of means were determined by Duncan's multiple range test at $p<0.05$. The SPSS software version 21 (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses.

3. Results and Discussion

3.1 Influence of BDF diet on body weight

During first six weeks, normal diet (ND) group had less initial weight than other groups fed the with high-fat diet, whereas body weight of all HFD groups had no significant difference ($p>0.05$). However, the final body weight of BDF-L and BDF-H groups significantly decreased compared to the HFD group ($p<0.05$) since supplement of six weeks. Weight gain was 19.90 ± 2.30%, 11.92 ± 9.90% and 10.41 ± 1.96% for mice fed with HFD, BDF-L, and BDF-H, respectively. In addition, at the end of the trial, BDF-L and BDF-H groups had decrease in the weight gain by a 59.90% and 52.31%, respectively, compared with the HFD group Table 1. Since hyperlipidemia was related to increases in body weight, liver weight, or obesity index, supplement with BDF led to significant loss of body weight and weight gain, indicating protective effects against obesity or type 2 diabetes.

3.2 Changes in serum lipid parameters

The results of serum parameters after six weeks of BDS supplement was presented in Table 1 The serum triglyceride (TG) and total cholesterol (TC) in HFD-fed groups were higher than ND group, suggesting hyperlipidemia caused by high-fat diet. TG increased significantly from 23.13 ± 3.24 mg/dL in the ND group to 63.98 ± 7.93 mg/dL in the HFD group, while a significant decrease was observed in the BDF-L (37.80 ± 5.40 mg/dL) and BDF-H (32.50 ± 6.50 mg/dL) groups, respectively ($p<0.05$). Total cholesterol (TC) levels decreased by 5.02% and 25.99% in the

Table 1. Effects of BDF on weight and serum parameters of C57BL/6J mice fed with a high-fat diet

Sample	ND	HDF	BDF-L	BDF-H
Weight changes				
Initial weight (g)	28.13 ± 1.41b	39.15 ± 2.17a	36.91 ± 1.74a	36.46 ± 2.29a
Final weight (g)	29.98 ± 1.26c	47.03 ± 2.65a	41.34 ± 2.44b	40.27 ± 2.11b
Weight gain (g)	6.55 ± 0.91c	19.90 ± 1.30a	11.85 ± 1.48b	10.41 ± 0.67b
Serum parameters				
TG (mg/dL)	23.16 ± 3.24d	63.50 ± 7.93a	37.72 ± 5.40b	32.62 ± 6.50c
TC (mg/dL)	116.42 ± 4.74d	197.28 ± 3.83a	173.428 ± 3.26b	147.36 ± 3.11c
HDL-C (mg/dL)	67.26 ± 1.49c	72.24 ± 1.83b	74.70 ± 2.30b	93.76 ± 2.80a
LDL-C (mg/dL)	20.579 ± 1.94 d	52.48 ± 1.13a	47.08 ± 1.37b	40.64 ± 1.13c
Glucose (mg/dL)	142.00 ± 3.08c	188.60 ± 3.71a	173.46 ± 4.80b	146.24 ± 4.48c
Insulin (μg/mL)	4.53 ± 0.81d	10.74 ± 0.99a	8.29 ± 0.58b	6.14 ± 0.40c
FFA (equiv/L)	637.66 ± 24.33d	1250.92 ± 35.39a	1048.46 ± 31.67b	781.94 ± 20.29c
Adiponectin (μg/mL)	32.18 ± 2.25a	21.43 ± 1.65b	29.28 ± 1.05a	30.30 ± 0.84a

Initial weight: mean body weight at the beginning of the experimental diets.
Weight gain (%) = [(Final weight (g) − initial weight (g))/initial weight (g)] X 100.
ND (n=8), normal control group, HDF (n=8), high-fat diet group; BDF-L (n=8), high-fat group fed with 500 mg/kg of BDF; BDF-H (n=8), high-fat group fed with 1,000 mg/kg of BDF.
Different letters indicate statistically significant differences between the variables ($p<0.05$) in the same row.

mice fed with BDF-L(187.38 ± 30.85 mg/dL) and BDF-H (146.00 ± 23.69 mg/dL), respectively as compared to the HFD group (197.28 ± 19.33 mg/dL). Also, LDL-C in BDF-L (49.60 ± 18.00 mg/dL) and BDF-H groups (40.20 ± 5.70 mg/dL) were significantly decreased as compared to in HFD group (52.30 ± 10.44 mg/dL) ($p<0.05$). The increased level of serum TG, TC, and LDL-C indicate of lipid metabolic disorders (e.g. including of insulin resistance, dyslipidemia, and atherosclerosis) and incidence of cardiac dysfunction in the HFD group. These results reveals that supplement of BDF facilitates lipid metabolism in HFD-fed mice. The level of HDL-C in the BDF groups was significantly increased as compared to the HFD group (72.80 ± 5.84 mg/dL) ($p<0.05$). Particularly, BDF-H group (93.40 ± 26.90 mg/dL) showed notable performance, that increased by 22.06% for the HDL-C level. Hyperlipidemia induced the changes of lipid profile, increasing the cardiovascular disease (e.g. atherosclerosis) [9]. In previous studies, the association between low level of HDL-C and increased of cardiovascular disease has been reported [10]. Therefore, the elevated level of HDL-C by BDF indicated the protective roles of BDF-supplement against cardiovascular disease [11]. The HFD led to a significant increase in the glucose and insulin level compared to the ND group,s uggesting insulin resistance of HFD group ($p<0.05$) [12,13]. However, serum glucose and insulin level of the BDF-L group (173.75 ± 20.95 mg/dL, 8.29 ± 0.58 μg/mL, respectively) and the BDF-H group (145.83 ± 21.21 mg/dL, 6.14 ± 0.40 μg/mL, respectively) were significantly lower than that of mice from the HDF group (188.80 ± 38.10 mg/dL, 10.74 ± 0.99 μg/mL, respectively) ($p<0.05$). These results suggested that BDF reduced resistance to insulin. Also, the mice from the BDF-L and BDF-H groups significantly decreased free fatty acids (FFA) level in a dose-dependent manner ($p<0.05$), compared to HFD group. During the development of insulin resistance, the accumulated body fat and larger fat cells increase release of FFA into the bloodstream or tissue, mainly liver. As a result of, these released FFA not only affects fat storage capacity but also leads to formation of fatty liver, which induces tissue damage,

lipogenesis and aggravation of insulin resistance [14,15]. It is well known that abnormal cholesterol metabolism is closely associated with changes in levels of HDL-C and LDL-C. Herein, we also confirmed that BDF can reduce FFA and LDL-C levels and increase HDL-C in the serum of high-fat diet induced obese mice. Meanwhile, adiponectin is the most highly expressed and secreted adipokine. Due to its health benefits on metabolism, inflammation, and vascular function, which contribute to regulate insulinsen sitivity, LDL oxidation, inflammation suppression, and fatty acid catabolism [16,17]. Thus, many studies showed that serum adiponectin level was significantly lowered in obese/diabetic mice and human or in patient with various metabolic disorders [18]. In agreement with that, groups supplemented with BDF had significantly increased serum adiponectin as compared with HDFgroup ($p<0.05$) (Table 1).

3.3 Changes in ALT and AST activities

As represented in Fig. 1(a) and (b), high fat-diet induced changes in the severity of hepatic injury as manifested by a significant increase in serum AST (209.9 U/L) and ALT (119.8 U/L) activities compared with the effects induced by ND (104.7 and 27.9 U/L; $p<0.05$). However, the elevation of serum ALT and AST activities was dose-dependently reduced in BDF-L (160.9 and 99.1 U/L) and BDF-H (112.5 and 62.6 U/L) groups, respectively ($p<0.05$). The results suggested that BDF exhibits notable protective effects on high fat-induced liver injury [19]. Similarly, there have been reports that buckwheat extracts could increase hepatic antioxidant enzyme activities to attenuate insulin resistance [20]. It is also well known that ALT and AST are reliable biomarkers of liver function; increased ALT activity represent damage of cell membrane, while increase of AST activity serves as an indicator mitochondrial damage [21]. In this study, it was apparent that the application of high-fat diet markedly increased serum ALT and AST activities, an indication of liver injury in mice. Interestingly, BDF supplement effectively improved liver dysfunction by attenuating the AST and ALT activities in a dose-dependent manner, representing the preventive action of BDF against liver damage in high fat diet-fed mice.

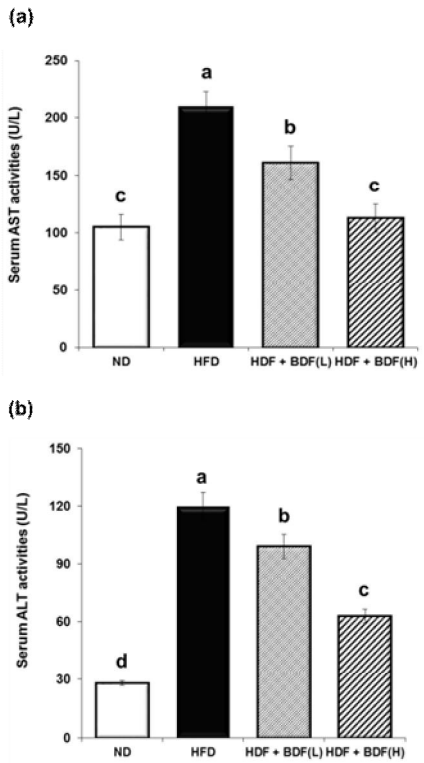

Fig. 1. Effects of BDF on serum AST (a) and ALT (b) levels in the mice fed with a high-fat diet. ND (n=8), normal control group, HDF (n=8), high-fat diet group; HDF + BDF (L) (n=8), high-fat group fed with 500 mg/kg of BDF; HDF + BDF (H) (n=8), high-fat group fed with 1,000 mg/kg of BDF. Mean values with different superscripts are significantly different ($p<0.05$).

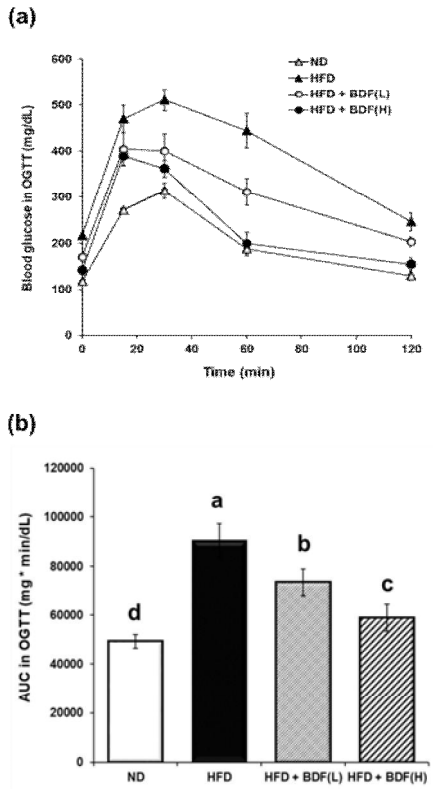

Fig. 2. Incremental blood glucose curves (a) and glycemic indices (b) of high-fat-diet-induced obese mice after BDF supplement. ND (n=8), normal control group, HDF (n=8), high-fat diet group; HDF + BDF (L) (n=8), high-fat group fed with 500 mg/kg of BDF; HDF + BDF (H) (n=8), high-fat group fed with 1,000 mg/kg of BDF. Mean values with different superscripts are significantly different ($p<0.05$).

3.4 Changes of OGTT

Fig. 2 shows the response to OGTT at the end of the six-week supplement of BDF Fig. 2(a). Blood glucose concentrations were significantly greater in HDF and maintained higher at 15, 30, 60, and 120 min after glucose loading compared with the ND group ($p<0.05$). However, a dose-dependent decrease in the glucose levels in the BDF-L and BDH-H groups was observed. Also, a notable difference was observed in incremental glucose concentrations between BDF-L and BDF-H groups, particularly after 30 min. The AUC of glucose during the OGTT of HFD increased by 1.3-folds compared with the ND group (Fig. 2(b)). This suggested that consumption of HFD leads defect in ability of insulin to stimulate glucose utilization, which plays a major role in peripheral tissues related to insulin resistance after glucose loading [19]. However, AUCs of blood glucose decreased to 18.70 and 34.8% in BDF-L and BDH-H groups, respectively, compared to the HDF group, indicating improved insulin- sensitivity ($p<0.05$).

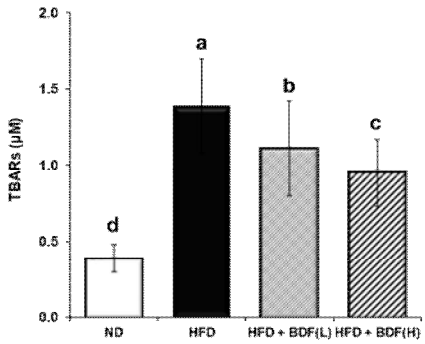

Fig. 3. Effects of BDF on hepatic MDA in the mice fed with a high-fat diet. ND (n=8), normal control group, HDF (n=8), high-fat diet group; HDF + BDF (L) (n=8), high-fat group fed with 500 mg/kg of BDF; HDF + BDF (H) (n=8), high-fat group fed with 1,000 mg/kg of BDF. Mean values with different superscripts are significantly different ($p<0.05$).

3.5 Changes in hepatic MDA (malondi-aldehyde) level

HFD are associated with oxidative stress, and diminished antioxidant enzyme. Oxidative stress not only causes lipid peroxidation, it also leads to the formation of MDA, which is a stable metabolite and biomarker of lipid-peroxidation cascade [22]. As shown in Fig. 3, mice fed with

high-fat diet exhibited a significant increase in hepatic MDA concentration ($p<0.05$), indicating that high-fat diet causes notable liver damage due to lipid peroxidation [15]. However, the high- fat-diet-induced increase was significantly attenuated in BDF-L and BDF-H groups in dose-dependent manner ($p<0.05$), which demonstrated the in vivo antioxidant properties of BDF.

4. Conclusion

Results from the experimental data clearly show that Tartary buckwheat dietary fiber (BDF) improved the effects on body weight and serum lipid profile of in HFD-fed mice. Also, BDF greatly attenuates the insulin resistance as well as peroxide formation of hepatic lipid and raised antioxidant enzymes in serum. Therefore, it is reasonable to believe that dietary fiber as major bioactive component in Tartary buckwheat may have a positive effect for treatment of HFD-related metabolic disorder such as obesity and diabetes.

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