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Effects of a Single Oral Ingestion of Wheat Bran on Postprandial Energy Metabolism in Healthy Participants: A Randomized, Double-blind, controlled Trial

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Abstract

This study investigated the effects of a single oral ingestion of wheat bran (WB) on energy metabolism in healthy participants. A randomized, double-blind, controlled trial with a washout period of at least 5 d was performed to compare the effects of a single oral ingestion of 8 g WB contained dietary bar (2 g arabinoxylan) with the effects of a no WB contained control dietary bar on energy metabolism. On the trial day, energy metabolism was measured in the participants using an indirect calorimeter to estimate postprandial energy metabolism. The postprandial fat oxidation was significantly enhanced by a single oral ingestion of WB compared with the control, whereas neither postprandial energy expenditure or carbohydrate oxidation differed significantly between treatments. A single oral ingestion of WB enhanced fat oxidation with no effect on total energy expenditure or carbohydrate oxidation in the postprandial state.

Keywords: Carbohydrate oxidation; Energy metabolism; Fat oxidation; Human; Wheat bran.

Introduction

Approximately 10% to 40% of the world population is affected by metabolic syndrome [1], which increases the risk for type 2 diabetes and cardiovascular disease [2]. Dietary therapy is generally believed to be effective against the development of metabolic syndrome.

Postprandial blood glucose and lipid responses are delayed by the consumption of dietary fiber [3,4]. Ingestion of dietary fiber is thought to improve and prevent such diseases as type 2 diabetes and metabolic syndrome [5-7]. The effects of dietary fiber on physiological function are reported to differ according to the physical properties of the fiber, such as its viscosity and water-holding capacity [8]. It is suggested that the positive effects induced by viscous fibers are due to delaying the absorption of nutrients. Ingestion of dietary fiber is also thought to have anti-obesity effects as a result of increased satiety and decreased energy intake [9]. The specific mechanisms underlying the anti-obesity effects of dietary fiber, especially insoluble fiber, however, are not clear. On the basis of a previous finding that postprandial fat oxidation negatively correlates with the body fat [10], one potential mechanism of the anti-obesity effect is enhanced postprandial fat oxidation.

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Wheat bran (WB) is a naturally occurring cereal crop with a long history of human consumption and is abundantly harvested worldwide. WB contains 50% dietary fiber, 60% of which is insoluble [11]. Arabinoxylan is the major fiber component in WB [12]. As a physiological function after continued ingestion of WB, increased stool weight and a shortened intestinal transit time in human are well established [13-16]. A previous study also reported that WB reduces food intake [17], but it is unclear whether continuous intake of WB prevents obesity [18].

Several human studies report that a single oral ingestion of WB or arabinoxylan has positive effects on postprandial glucose and insulin responses [3,19]. Fat metabolism is well known to be regulated by postprandial glucose metabolism, but a recent rodent study reported increased fat oxidation after a single ingestion of WB without a significant change in the glucose and insulin responses [20]. To our knowledge, no human studies have evaluated the fat oxidation rate after a single ingestion of WB. Therefore, the present study in humans investigated the effect of a single ingestion of WB on postprandial fat oxidation.

Materials and Methods

Ethics and registration

This study was performed in accordance with the Declaration of Helsinki (2013), and with the approval of the Clinical Trials Ethics Committee of the Kao Corporation (Tokyo, Japan). The study protocol was registered with the University Hospital Medical Information Network Center (UMIN-CTR,http://www.umin.ac.jp/ctr/index-j.htm) prior to inclusion of the first participant (No. UMIN000024510). The study was performed under a physician's supervision. All participants provided written informed consent after receiving a full explanation of the study.

Participants

The inclusion criterion was healthy adult male volunteers. The exclusion criteria included hepatic, renal, or cardiac disease; respiratory impairment; endocrine disorders; metabolic impairment; neuropathy; impaired mental status; diabetes; other disorders that would make participation in a study difficult; treatment with medication to improve glucose metabolism, fat metabolism, or blood pressure; regular consumption of Food with Health Claims (Food for Specified Health Uses, Foods with Functional Claims, and nutritional food supplements) and supplements; surgery for disease or injury within 2 months before the study; donation of 200 ml or more of blood within 1 month before the study; potential to exhibit allergic symptoms to the ingredients in the test diets; excessive alcohol consumption (≥30 g of alcohol for several days in a week); unable to undergo breath analysis; intention to live or travel overseas for an extended period (10 or more consecutive days) during the study period (including wash-out periods); lack of consent to view previous medical records; current participation in other clinical studies or intention to participate in one during the study period; and those judged to be unsuitable by the physician in charge of the study from the perspective of ensuring participant safety on the basis of medical exams or other factors.

The power calculation was based on an unpublished preliminary examination showing enhanced fat oxidation by a single oral ingestion of WB. As a result, the sample size required to detect a significant difference from control was 14 participants (power 0.8 and type I error 0.05). Potential participants who met the criteria were determined on the basis of a health and lifestyle habit questionnaire administered prior to the study. Assuming a 30% dropout rate, target enrollment was set at 22 individuals. Potential participants were allocated to each treatment sequence by stratified randomization counterbalanced for body mass index (BMI) and age under blinded conditions. The block size was 4.

Study Protocol

A randomized, double-blind, controlled crossover study with a washout period of at least 5 d was conducted. The primary outcome was postprandial fat oxidation. On the day before the trial, the participants were asked not to drink alcohol or perform hard exercise, and were instructed to consume a designated dinner (total energy = 735 kcal, protein: fat: carbohydrate = 10:23:65) before 10:00 pm. The participants were asked not to consume any food or drink except for water after the designated dinner and to come to the laboratory at 08:30 am the next day for measurements. After acclimatizing to the room environment (25°C), fasting breath analysis and anthropometric measurements were performed after at least 12-h fasting (~10:00 am). At 10:00 am, a WB-rich or control- dietary bar was ingested with 200 ml of milk (total energy= 373 kcal, protein: fat: carbohydrate = 20:36:44 in both meals). The WB-rich dietary bar was consisted of WB, wheat flour, soy protein, margarine, egg and sugar. The control dietary bar was designed to have equal energy content and ratios of the proteins, lipids and carbohydrates without WB use. Half of the participants ingested the WB-rich dietary bar first and then, at least 5 d later, repeated the process and consumed the control dietary bar. The other half of the participants consumed the control dietary bar first and then the WB-rich dietary bar. The WB comprised 42.1% insoluble dietary fiber, 4.2% soluble dietary fiber, 23.8% arabinoxylan, 67.9% carbohydrate, 20.6% protein, and 3.5% fat. Arabinoxylan amout was estimated by sum of amounts of arabinose and xylose. Postprandial breath analyses were performed 60, 120, 150, 180, 210, and 240 mins after ingestion of the dietary bar to estimate the amount of substrate oxidation and energy expenditure.

Measurements

Oxygen consumption (VO_2) and carbon dioxide production (VCO_2) were measured to estimate the amount of fat and carbohydrate oxidation, and energy expenditure using a mass spectrometer for respiratory gas analysis (ARCO-2000, Arco Systems Inc., Chiba, Japan) and a sampling mask (601M, Arco Systems Inc.). Urine samples were pooled

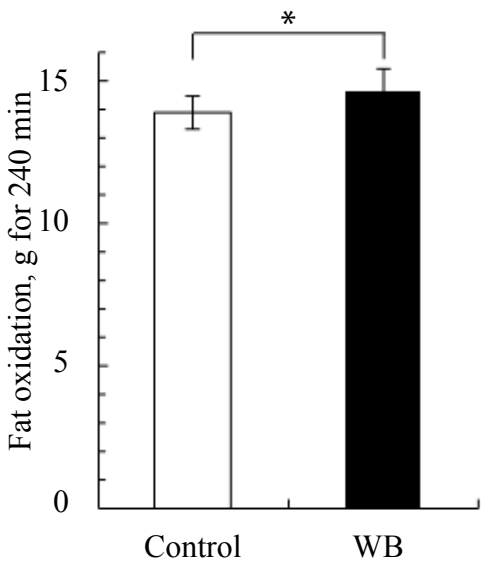

Figure 1: Area under the curve (AUC) of fat oxidation for 240 min after ingesting the control or WB dietary bar (control: 13.9 g for 240 min, test: 14.6 g for 240 min, p=0.025). Mean ± SE, n=21. *p<0.05 vs. control bar.

Table1: Clinical characteristics of the 21 participants (all men, mean ±SD).

Age (years)	40 ± 9
Weight (kg)	67.2 ± 9.1
BMI (kg/m$_2$)	22.5 ± 2.7
Fat ratio (%)	18.9 ± 3.4
Triglycerides (mg/dl)	84 ± 43
Total cholesterol (mg/dl)	197 ± 33
LDL cholesterol (mg/dl)	118 ± 27
HDL cholesterol (mg/dl)	63 ± 10
Glucose (mg/dl)	99 ± 6
Insulin (μU/ml)	3.89 ± 1.23
HOMA-R	0.92 ± 0.30

Table 2: AUC of energy expenditure and carbohydrate oxidation, and average of respiratory quotient after ingesting the control or WB dietary bar. Mean ± SE, n=21.

	Control	WB	p-value
Energy expenditure (kcal for 240 min)	282.9	282.5	0.640
Carbohydrate oxidation (g for 240 min)	19.9	19.0	0.880
Respiratory Quotient ((L/L/240 min)	0.803	0.802	0.492

for 4 hours to analyze urinary nitrogen and estimate protein oxidation (POX). The urinary nitrogen concentration was measured using a nitrogen analyzer (TN-100, Mitsubishi Chemical Analytech Co., Ltd.). The amount of POX was estimated using 6.25 as the protein conversion factor [21]. All measurements were conducted under blinded conditions among the participants, care givers, and collaborators. Fat and carbohydrate oxidation, and energy expenditure were calculated according to the following equations [22,23].

$$\text{Fat oxidation (g)} = 1.689 \times VO_2 - 1.689 \times VCO_2 - 0.324 \times POX$$

$$\text{Carbohydrate oxidation (g)} = 4.113 \times VCO_2 - 0.375 \times POX$$

$$\text{Energy expenditure (kcal)} = 3.9 \times VO_2 - 1.1 \times VCO_2$$

Statistical Analysis

The general characteristics of the participants are presented as mean ± standard deviation (SD). The other values are presented as mean ± standard error (SE). The difference between the treatments was compared using the area under the curve (AUC) from the initial time-point to the 240 min time-point with a mixed model adjusted by the baseline and treatment sequence (ingestion order of WB and control). A P-value less than 0.05 was considered statistically significant. All statistics were performed using SPSS version 18.0 (IBM Inc., Tokyo, Japan).

Results and Discussion

Eligibility for participation in the study was assessed in 26 individuals and 22 individuals who were deemed eligible were allocated to one of the two treatment sequences. One participant withdrew his consent to participate in the study before the 1st visit due to a scheduling conflict. Therefore, a total of 21 participants completed the study and were included in the analyses. The characteristics of those that completed the study are shown in Table 1.

No significant effect on treatment sequence was observed. The order effect of test food was not confirmed. The AUC of fat oxidation was significantly increased after ingesting the WB compared with the control (Figure 1, $p<0.05$). The AUCs of energy expenditure, carbohydrate oxidation, and respiratory quotient did not differ significantly following the WB and control (Table 2, $p = 0.657$, $p = 0.880$, and $p = 0.492$, respectively). POX also did not significantly differ following ingestion of the WB and control.

Several mechanisms are suggested to underlie the enhanced fat oxidation by WB ingestion [3,20, 24-32]. A rodent study demonstrated that mice fed a high-WB diet had significantly higher postprandial fat oxidation rates and a significantly decreased postprandial glucose-dependent insulinotropic polypeptide (GIP) response [20]. GIP is a gut hormone whose secretion from K-cells in the intestinal mucosa is induced by dietary ingestion, and fat metabolism may be involved in GIP secretion [24-27]. A previous study reported that fat utilization is increased by GIP receptor inhibition [24]. The adipose tissue mass and triglyceride deposition in the liver and muscle are decreased by suppressing GIP signaling [25]. In addition, ingesting a diet that leads to reduced GIP secretion promotes fat utilization [26,27]. Thus, fat oxidation after a single ingestion of WB may be associated with a reduction of the blood GIP response. Further studies are needed to investigate this association in humans.

A single ingestion of WB or arabinoxylan induces increased production of breath hydrogen [3] and short chain fatty acid (SCFA) [28]. These products result from fermentation by the gut microbiome, and may have a physiological function or may be utilized as an energy source for the host [29,30]. Indeed, several reports suggest that SCFA regulates host metabolism via signaling molecules such as G protein-coupled receptor (GPR) 41 and GPR43 [31,32], and improve energy metabolism and suppress accumulation of fat to adipose tissue. Thus, the GPR-mediated behavior of SCFA induced by WB ingestion is a potential mechanism underlying the enhanced fat oxidation observed in the present study.

Limitations

This study was performed only in men, and no women were included. Although we observed that postprandial fat oxidation was significantly increased after a single ingestion of WB, no data to explain the mechanism of enhanced fat oxidation were collected in this study. Postprandial fat oxidation was enhanced by the WB while energy expenditure was not. It is not clear if enhanced postprandial fat oxidation alone contributes to control obesity. Further human trials are required to investigate the GIP response after a single ingestion of WB and the effect on energy metabolism by repeated WB ingestion.

Conclusion

A single oral ingestion of a WB-rich dietary bar can enhance postprandial fat oxidation in healthy adult men.

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