Plant Foods Hum Nutr (2013) 68:18–23
DOI 10.1007/s11130-013-0336-2

ORIGINAL PAPER

Oat Prevents Obesity and Abdominal Fat Distribution, and Improves Liver Function in Humans

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Published online: 31 January 2013

Abstract Obesity is associated with a great diversity of diseases including non-alcoholic fatty liver disease. Our recent report suggested that oat, rich in beta-glucan, had a metabolic-regulating and liver-protecting effect in an animal model. In this study, we performed a clinical trial to further confirm the effect of oat. Subjects with BMI ≧27 and aged 18–65, were randomly divided into a control (n=18) and an oat-treated (n=16) group, taking a placebo or beta glucan-containing oat cereal, respectively, for 12 weeks. Our data showed that consumption of oat reduced body weight, BMI, body fat and the waist-to-hip ratio. Profiles of hepatic function, including AST, but especially ALT, were useful resources to help in the evaluation of the liver, since both showed decrements in patients with oat consumption. Nevertheless, anatomic changes were still not observed by ultrasonic image analysis. Ingestion of oat was well tolerated and there was no adverse effect during the trial. In conclusion, consumption of oat reduced obesity, abdominal fat, and improved lipid profiles and liver functions. Taken as a daily supplement, oat could act as an adjuvant therapy for metabolic disorders.

Keywords Oat · Obesity · Abdominal body fat · Fatty liver

Abbreviation

ALT	Alanine transaminase
AST	Aspartate transaminase
BMI	Body mass index
BUN	Blood urea nitrogen
FFA	Free fatty acids
FS	Fatty liver scores
γ-GT	gamma-glutamyl transpeptidase
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
TG	Triacylglycerol

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Introduction

Obesity, generally measured by body mass index (BMI), is associated with a great diversity of diseases involving the cardiovascular and metabolic systems. In recent years, there has been increased recognition that body fat deposition and abdominal obesity play a critical role in the pathogenesis of related disorders [1].

There is a metabolic link among abdominal fat, high triacylglycerol (TG), high low-density lipoprotein cholesterol (LDL-C), low high-density lipoprotein cholesterol (HDL-C) and flux of free fatty acids (FFA) [2]. Since the liver regulates the plasma lipid level through low density lipoprotein (LDL) clearance and high density lipoprotein (HDL) recruitment, non-alcoholic fatty liver is generally considered to be the liver component of metabolic syndrome, which is defined by over waist circumference, dyslipidaemia, hyperglycaemia, and hypertension [3, 4]. It showed that obesity induced steatosis, lymphocyte infiltration, and the subsequent development of hepatic illness [5]. As commonly found in ultrasound screening, liver disorders are often accompanied with an elevation of AST (aspartate transaminase) and ALT (alanine transaminase) [4]. In addition, gamma-glutamyl transpeptidase (γ-GT) was often accompanied with hepato-cholecystic disorders and may be involved in the promotion of carcinogenesis [6].

Previous reports have suggested that oat has metabolic-regulating effects. Oat bran decreased the total cholesterol level in serum, and decreased cholesterol and TG contents in the liver [7]. Whole-grain oat cereal reduced LDL-C in overweight and obese adults [8]. Oat contains vitamins, minerals, antioxidants, and phenolic compounds, and is rich in beta-glucan [9]. Oat-derived beta-glucan increased HDL-C, while diminished LDL-C and non-HDL cholesterol in overweight individuals [10]. It has been suggested that daily intake of at least 3 g of oat beta-glucan reduces total cholesterol and LDL-C in normo- or hyper-cholesterolemic subjects [11]. Using the high-fat-diet (HFD)-fed rat model, we recently demonstrated that oat reduced body weight and fat, and improved the serum lipid profile via increasing liver LDL clearance, inhibiting hepatic lipogenesis, and stimulating lipolysis. [12].

Although many previous reports have shown that oat improved lipid profiles and reduced body weight, few of them emphasized the effects of oat on liver function, hepatic steatosis, and body fat distribution. In this study, we performed a clinical trial to further confirm the effect of oat on metabolic regulation. We examined whether oat could attenuate obesity, body fat deposition, waist circumstance, and improve serum parameters and liver function to prevent hepatic steatosis in obese subjects.

Materials and Methods

Subjects The study was approved by the Institute Review Board of Chung Shan Medical University Hospital (CSMUH No: CS09072). All participants gave their informed consent in writing. Subjects with BMI ≧27 and aged 18–65 were recruited for the study. Those who had one of the following were excluded: a drinking habit (≧20 g alcohol daily), ALT 3-fold higher or bilirulin above 2 mg/dl, kidney dysfunction, cardiovascular disease, endocrine or severe systemic disturbance, mental disorder, or taking any OTC or prescribed medication and nutraceutics. Forty subjects fulfilled the above criteria were recruited for the study (Table 1).

Study Design The study was conducted from July 2009 to June 2010. Before and after the experiment, the basal serum parameters (glucose, TG, cholesterol, LDL-C, HDL-C, FFA, AST and ALT), BMI, waist-to-hip ratio, body fat and the fatty liver score (FS, described below) were measured as curative indexes. The subjects were double-blinded, randomized and divided into two groups (20 subjects in each), one taking beta-glucan-containing oat cereal and the other a placebo (with a similar external but without beta-glucan), respectively. One cereal pack (37.5 g) was prescribed to be mixed with 250 mL

Table 1 Baseline demographic data of the subjects

	Control (n=18)	Oat-treated (n=16)	p value
Biometrics			
Age (y/o)	37.67±10.59	39.44±11.69	0.65
Height (m)	1.65±0.12	1.64±0.08	0.61
Weight (kg)	81.63±17.39	78.36±11.74	0.53
BMI (kg/m^2)	29.54±2.54	29.18±2.34	0.66
Body fat (%)	37.46±5.55	36.64±6.72	0.70
Waist-to-hip ratio	0.93±0.04	0.94±0.02	0.43
Systolic pressure (mmHg)	122.78±13.05	124.00±12.24	0.78
Diastolic pressure (mmHg)	76.44±8.75	78.88±7.97	0.41
Diabetes indicators			
TCHO (mg/dl)	191.17±29.51	189.31±22.46	0.84
LDL-C (mg/dl)	133.11±23.13	126.25±24.69	0.41
HDL-C (mg/dl)	42.72±6.70	45.75±10.06	0.30
TG (mg/dl)	135.83±57.86	132.81±69.63	0.89
FFA (U/min/mg protein)	0.79±0.31	0.82±0.47	0.85
Glucose (mg/dl)	100.67±34.31	94.62±8.12	0.50
Uric acid (mg/dl)	6.17±1.46	5.84±1.34	0.51
Hepatic function			
AST (U/L)	20.17±10.51	27.06±19.74	0.21
ALT (U/L)	28.72±26.80	42.44±34.14	0.20
FS	4.78±2.41	3.75±2.89	0.27

Data are presented as mean ± SD and analyzed by the Student's t-test. $p<0.05$ was considered statistically significant

hot water and replaced some staple food of meals twice daily. The calorific capacity in each pack was 144.8 kcal, with carbohydrate 25.3 g; protein 4.0 g; lipid 2.5 g; fiber 3.7 g and beta-glucan 1.5 g. Body weight, waist-to-hip ratio and body fat were measured at weeks 0, 2, 6, 8 and 12. Serum parameters and safety evaluations, including heart rate, respiration, blood pressure, γ-GT, creatinine, blood urea nitrogen (BUN), albumin, uric acid, blood and urine routine were also measured at weeks 0, 6 and 12. The recruited subjects were asked to take 3-day records of daily meals and physical activity at each of the time points before the trial, first six weeks and last six weeks, respectively. At the end of the study, 34 subjects had completed all the experiments: 18 controls (6 males and 12 females) and 16 (6 males and 10 females) in the beta-glucan group. Six subjects withdrew from the trial: one with hyperglycemia, one with higher TG, and four did not adhere to the following appointments.

Serum Parameters Serum glucose, TG, total cholesterol, LDL-C, HDL-C, AST, ALT, BUN, creatinine, γ-GT, albumin and uric acid were analyzed on a Beckman Synchron CX9 clinical system. FFA was analyzed using a Free Fatty Acid Quantification Kit (ab65341, abcam).

Body Fat and Waist-to-Hip Ratio Body fat was measured with a Tanita TBF-300GS analyzer. Waist-to-hip ratio was calculated by the waist circumference (just above the upper hip bone) divided by the hip circumference at its widest part.

Ultrasonic Image and Fatty Liver Scores (FS) The liver ultrasonic image examination was performed with the Aloka system (Prosound SSD-4000, with 5.0-MHz convex transducer). Five items, including hepatic clearance, far gain attenuation, and opaque of the bladder wall, portal area and hepatic vein, were evaluated. Each item was classified as 0, 1, or 2 to indicate normal, mild to moderate, or severe, respectively. FS was calculated as the sum of the scores of the five items [13]. In general, the recruited subjects showed mild liver steatosis (Table 1).

Statistical Analysis Using an unpaired Student *t*-test for the control and oat-treated groups, and a paired Student *t*-test for the pre- and post-trial, a *p* value of less than 0.05 was considered statistically significant. All the analyses were performed with SigmaPlot 11.0.

Results

Oat Reduced Body Weight and BMI Weight and BMI were slightly increased in the control group, but significantly lowered by 2.08±2.05 kg and 0.81±0.80 kg/m^2, respectively, in the oat-treated group (Table 2). Almost 90 % of the

Table 2 Treatment effects

	Control (n=18)				Oat-treated (n=16)				
	6 wk – 0 wk	p value*	12 wk – 0 wk	p value*	6 wk – 0 wk	p value*	12 wk – 0 wk	p value*	p value**
Biometrics									
Weight (kg)	−0.21±1.09	0.422	0.52±1.74	0.225	−1.54±1.64	0.002*	−2.08±2.05	0.001*	0.000**
BMI (kg/m^2)	−0.18±0.37	0.050*	0.15±0.62	0.317	−0.59±0.65	0.002*	−0.81±0.80	0.001*	0.000**
Body fat (%)	0.07±1.32	0.820	0.39±1.94	0.406	−0.34±1.97	0.495	−0.93±1.73	0.048*	0.045**
Waist-to-hip tatio	0.01±0.01	0.029*	0.01±0.03	0.013*	−0.01±0.02	0.094	−0.01±0.02	0.115	0.003**
Diabetes indicators									
TCHO (mg/dl)	−11.22±39.43	0.244	−3.94±11.36	0.159	−11.75±19.69	0.031*	−19.69±21.71	0.002*	0.011**
LDL-C (mg/dl)	−4.28±13.76	0.205	−0.83±14.56	0.811	−9.44±18.10	0.055	−13.50±14.81	0.002*	0.017**
HDL-C (mg/dl)	0.94±4.28	0.362	1.61±5.75	0.251	2.94±4.78	0.027*	0.94±6.29	0.560	0.746
TG (mg/dl)	−2.61±45.52	0.811	8.50±51.99	0.497	−26.38±48.18	0.045*	−15.81±49.91	0.224	0.175
FFA(U/min/mg protein)	−0.02±0.42	0.856	−0.15±0.37	0.109	−0.08±0.50	0.548	−0.14±0.38	0.156	0.946
Glucose (mg/dl)	7.89±8.60	0.001*	11.44±20.52	0.030*	1.50±7.71	0.449	5.38±6.57	0.005*	0.266
Uric acid (mg/dl)	−0.02±0.61	0.878	−0.32±0.86	0.136	0.06±0.60	0.713	−0.22±0.64	0.190	0.711
Hepatic function									
AST (U/L)	0.78±4.85	0.505	0.17±5.50	0.351	−4.25±15.18	0.280	−6.69±15.17	0.027*	0.083
ALT (U/L)	2.11±8.53	0.309	2.67±11.80	0.899	−7.81±18.41	0.110	−14.25±22.42	0.098	0.009**
FS			−1.00±1.75	0.027*			−0.81±1.60	0.060	0.748

Data are presented as mean ± SD and analyzed by paired *t*-test

*p<0.05 indicates the significance of each difference at 6 or 12 weeks compared with the baseline

**p<0.05 indicates the significance of each 12 wk – 0 wk difference between the control and oat-treated groups

oat-treated subjects had reduced body weight and BMI (data not shown).

Oat Reduced Body Fat and Waist-to-Hip Ratio During the trial, the body fat and waist-to-hip ratio of more than 60 % of the oat-treated subjects was reduced (data not shown). The mean difference significantly decreased by 0.93± 1.73 % and 0.01±0.02, respectively, in the oat-treated group (Table 2).

Oat Decreased Serum Cholesterol and Improved the Lipoprotein Profiles Serum cholesterol was significantly decreased in the oat-treated subjects (Table 2). Although HDL-C was not altered, oat decreased LDL-C. The serum TG and FFA were not significantly reduced after oat treatment, while serum glucose was slightly altered in both groups.

Oat Reduced ALT and AST, and Improved FS In the oat-treated subjects, AST significantly decreased from 27.06± 19.74 to 20.38±7.94 U/L, while no significance was found compared with the control. ALT decreased 36 %, but no significance was found between the pre- and post-trial. However, compared with the control, ALT was significantly decreased with oat treatment (Table 2). As seen in the ultrasonic images, FS of the control and oat-treated groups decreased 21 and 22 %, respectively (Table 2), without a significant difference.

Oat did not Change the Safety Evaluation Markers With the exception of the body temperature of the control group, the safety evaluation markers were not altered after the trial. However, γ-GT decreased 25 % in the oat-treated group, with a marginal p value of 0.052, close to the criteria of significance (Table 3). Hence, the oat treatment showed no harm in terms of vital signs and blood safety markers.

Discussion

In the present study, we demonstrated the anti-obesity and liver-protection benefit of oat for the human body. Oat reduced weight and body fat, especially inhibiting abdominal fat distribution. Oat decreased serum cholesterol and LDL-C, thereby improving the lipoprotein profiles. The liver function indexes were ameliorated by oat treatment, and thus the occurrence or aggravation of fatty liver was avoided. While exerting a benefit on metabolic regulation, oat did not burden any of the systemic life signs.

Although BMI is widely used in body weight classification and was adopted in this study [14], it may have limitations and lead to a mis-estimation of the prevalence of overweight and obesity. For some special populations who have shorter lower limbs, using standing height alone to calculate BMI may overestimate the number of individuals that are overweight and obese, and at risk for type 2 diabetes mellitus and cardiovascular disease [15].

Table 3 Safe evaluation markers

	Control (n=18)			Oat-treated (n=16)		
	pre-trial	post-trial	p value	pre-trial	post-trial	p value
Heart rate (BPM)	72.67±8.26	76.44±8.85	0.059	72.25±9.19	75.50±6.13	0.214
Respiration rate (BPM)	17.44±1.15	17.50±1.65	0.912	16.88±1.59	16.75±1.77	0.847
Systolic pressure (mmHg)	122.78±13.05	118.44±8.61	0.170	124.00±12.24	119.12±7.23	0.150
Diastolic pressure (mmHg)	76.44±8.75	74.89±5.10	0.444	78.88±7.97	78.62±8.79	0.922
Body temperature (°C)	36.79±0.27	36.60±0.19	0.033*	36.69±0.34	36.53±0.22	0.129
WBC (*10^3/μl)	6.55±1.34	6.26±1.08	0.353	6.25±1.50	5.99±1.66	0.217
RBC (*10^6/μl)	4.83±0.49	4.94±0.56	0.110	5.06±0.85	5.08±0.89	0.718
Hb (mg/dl)	14.30±1.70	14.60±1.93	0.152	14.91±1.60	14.98±1.94	0.749
BUN (mg/dl)	11.87±1.95	11.46±2.33	0.493	11.49±3.09	12.28±2.81	0.309
Creatinine (mg/dl)	0.86±0.18	0.84±0.15	0.331	0.86±0.16	0.86±0.15	0.855
Albumin (g/dl)	4.39±0.31	4.33±0.18	0.305	4.49±0.31	4.41±0.27	0.227
γ-GT (U/L)	31.78±18.03	31.33±17.54	0.806	35.62±30.65	26.75±19.35	0.052
TSH (μIU/ml)	1.33±0.69	1.66±0.82	0.063	1.40±0.72	1.33±0.74	0.574
T4 (μg/dl)	7.10±1.19	6.78±1.13	0.212	6.68±0.98	6.66±1.04	0.926

WBC white blood cell, *RBC* red blood cell, *Hb* hemoglobulin, *TSH* thyroid stimulating hormone, *T4* thyroxine

Data are presented as mean ± SD and analyzed by paired t test

*p<0.05 indicates the significance

On the other hand, central obesity but not generalized obesity, predicts a high prevalence of hepatic steatosis and related disorders, including impairment of glucose tolerance and type 2 diabetes. The analysis revealed that the quantitative insulin-sensitivity index, waist circumference and waist-to-height ratio had a significant association with the development of fatty liver, whereas BMI did not. Hence, in this study, we measured waist circumference and used the waist-to-hip ratio as the index, which should more adequately reflect the regulatory effect of oat on abdominal fat distribution and central obesity [16].

Our data showed that ALT was significantly lowered in the oat-treated group compared with the control, implicating that oat is beneficial for preventing fatty liver. However, the ultrasound image changes were not parallel with the functional index. As a marker of liver disorders, ALT is associated with the pathogenesis of metabolic syndrome, type 2 diabetes mellitus and subsequent cardiovascular disease [17]. In the absence of a detectable ultrasonic change, ALT and AST are associated with hyperinsulinemia and insulin resistance, indicating that a mild stage of steatosis is sufficient to mediate the association between insulin resistance and liver enzymes [18]. In this trial, the duration of oat treatment was only 12 weeks. Some liver improvement was assumed to be too mild for detection by ultrasound, thus liver enzymes might be able to reflect early changes. Considering the sub-cellular distribution and biochemical properties of hepatic enzymes, ALT is superior for monitoring early or mild liver changes [18].

We demonstrated the benefits of oat beta-glucan on metabolic regulation in this study. Oat beta-glucan may form a viscous layer at the small intestine, which attenuates the uptake of dietary cholesterol and reabsorption of bile acid. The reduced bile acid levels activate the conversion from cholesterol to bile acid, thus decreasing the hepatic cholesterol content, and stimulating LDL receptor synthesis and plasma LDL-C clearance [11]. However, controversy still exists. Consumption of beta-glucan-enriched meals did not significantly lower serum cholesterol and LDL-C in some studies. [19]. While assessing its metabolic regulating effect, the physicochemical properties of oat beta-glucan should be considered. The lowering of LDL-C by oat beta-glucan may depend on viscosity, which is dictated by molecular weight and solubility in the intestine [20]. The oat-induced LDL-C reduction significantly diminished when molecular weight was reduced [21]. Our data revealed that oat effectively reduced obesity, as well as the serum lipid and liver function indexes. The effect could be exerted by the proper molecular weight of beta-glucan. Initial moisture levels and extrusion temperatures affected the water solubility and viscosity of oat product [22]. Processing conditions also influence the amount of beta-glucan [23]. Although beta-glucan is a major polysaccharide of oat, we still cannot rule out whether other functional components participate in the metabolic regulation [9].

In conclusion, we found that oat decreased obesity, abdominal fat, serum cholesterol, LDL-C, and liver functions. Taken as a daily supplement, oat could act as an adjuvant therapy for metabolic disorders.

Acknowledgments We thank STANDARD Foods Co., Taiwan for providing all experimental diets.

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