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Effect of adding exercise to a diet containing glucomannan

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Abstract

The aim of this study was to examine the effect of adding a total-body exercise program to an 8-week diet supplemented with glucomannan on weight loss, body composition, blood parameters, and physical performance in overweight men and women. Sedentary men and women who were overweight (body mass index >25 kg m^{-2}) (men: 18-57 years, 27.0 ± 11.2 years, 177.5 ± 7.6 cm, 102.4 ± 14.9 kg; women: 18-52 years, 33.4 ± 12.1 years, 160.6 ± 4.6 cm, 79.9 ± 9.3 kg) completed an 8-week diet with 3000 mg glucomannan combined with either no exercise (No-Ex) (10 men, 10 women) or a resistance and endurance exercise training program (Ex) (12 men, 10 women). The diet emphasized healthy food choices and portion size control. The exercise training consisted of 3 weekly sessions of approximately 1 hour of a nonlinear periodized total-body resistance exercise program followed by 30 minutes of endurance exercise. After the intervention, there were reductions ($P < .05$) in body mass (men, −2.7 ± 1.4 and −3.0 ± 4.0 kg; women, −2.2 ± 1.5 and −3.3 ± 1.5 kg; No-Ex and Ex, respectively), fat mass (men, −2.3 ± 1.6 and −3.9 ± 2.5 kg; women, −2.6 ± 1.4 and −3.6 ± 1.1 kg; No-Ex and Ex, respectively), total cholesterol (TC) (men, −17.9 ± 21.5 and −18.8 ± 19.4 mg dL^{-1}; women, −9.3 ± 20.0 and −10.1 ± 19.5 mg dL^{-1}; No-Ex and Ex, respectively), and low-density lipoprotein cholesterol. Exercise significantly improved high-density lipoprotein cholesterol (HDL-C) (No-Ex, −2.0 ± 4.7 and −2.3 ± 4.5 mg dL^{-1} vs Ex, 4.4 ± 10.8 and 1.6 ± 3.6 mg dL^{-1}; men and women, respectively), TC/HDL-C ratio, squat and bench press 1-repetition maximum, and distance covered during a shuttle-run test. In addition, exercise appeared to augment the reduction in fat mass (by 63% and 50%; men and women, respectively) and waist circumference, but did not affect total weight loss. Addition of a resistance and endurance exercise training program to a glucomannan diet regimen significantly improved measures of body composition, HDL-C, and TC/HDL-C ratio.

1. Introduction

A growing body of research supports beneficial health effects of dietary fiber, particularly soluble fibers, on weight loss [1,2], diabetes mellitus [3,4], and risk factors for cardiovascular disease [3,5-9]. The soluble fiber shown to have the greatest gel volume and viscosity properties is glucomannan [10], a viscous, water-soluble polysaccharide that is a natural constituent of *Amorphophallus konjac* (konjac root). A number of placebo-controlled studies have shown that relatively small amounts of glucomannan (1 to 4 g d^{-1}) enhance weight loss when part of a calorically restricted diet [1,11-14], even when no dietary guidance to restrict calories is provided [14-16]. Glucomannan appears to reduce appetite because of its powerful bulk-forming properties in the stomach. When taken with meals, glucomannan decreases the rate by which food exits the stomach and enters into the intestines for absorption. This reduces the rate of absorption of nutrients, decreases the glucose and insulin response to meals by as much as one half [17,18], and promotes satiety [13].

Glucomannan also has cholesterol-lowering effects [3,5,7-9,14,15,19-21]. The hypocholesterolemic effects of glucomannan are evident with small doses (<4 g d^{-1}) and have been observed in a variety of healthy and clinical populations. In a recent randomized, placebo-controlled, double-blind, crossover clinical trial in diabetic subjects, glucomannan (3.6 g d^{-1}) decreased low-density lipoprotein cholesterol (LDL-C) by 21% and improved the total cholesterol (TC) to high-density lipoprotein cholesterol

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(HDL-C) ratio by 16% [3]. The mechanism for this potent lipid-lowering effect of glucomannan appears to be via interference with intestinal cholesterol absorption [7] as well as enhanced cholesterol loss caused by increased fecal bile and neutral sterol excretion [3].

Exercise induces favorable alterations in body composition [22], blood lipids [23], and physical performance [22,24]. Exercise also amplifies the positive effects of a dietary weight loss program [25,26]; thus, it is important to consider the impact of adding a total-body exercise program to a glucomannan-supplemented hypocaloric diet. The purpose of this study was to examine the effect of adding a total-body exercise program to an 8-week diet program with glucomannan supplementation on weight loss, body composition, blood lipid parameters, and physical performance in overweight men and women. A secondary purpose was to examine potential sex differences to the 8-week intervention.

2. Methods

2.1. Experimental design

Forty-two men (n = 22) and women (n = 20) who were sedentary and overweight (body mass index [BMI] >25 kg m^{-2}) successfully completed one of two 8-week experimental conditions: (1) a diet with glucomannan with no exercise (No-Ex) or (2) a diet with glucomannan combined with a resistance and endurance exercise training program (Ex). Each participant ingested 1500 mg of glucomannan before each of the 2 largest meals every day for 8 weeks. The diet promoted an emphasis on healthy food choices and portion size control. The exercise training involved 3 weekly sessions consisting of about 1 hour of a nonlinear periodized total-body resistance exercise program followed by 30 minutes of cardiovascular endurance training. The exercise program was a typical total-body conditioning program. Body mass, body composition, maximal strength, cardiovascular endurance, blood lipids, and metabolic markers were measured before and after the 8-week intervention.

2.2. Participants

Participants (men: 18-57 years, 27.0 ± 11.2 years, 177.5 ± 7.6 cm, 102.4 ± 14.9 kg; women: 18-52 years, 33.4 ± 12.1 years, 160.6 ± 4.6 cm, 79.9 ± 9.3 kg) were matched on sex, age, body mass, percent (%) body fat, BMI, and maximal strength and subsequently randomly assigned to either the No-Ex (10 men, 10 women) or Ex (12 men, 10 women) group. To be considered eligible for the study, participants had to be (1) overweight (BMI >25 kg m^{-2}), (2) sedentary, (3) nonsmokers, and (4) not taking any medication or supplements known to influence blood lipid or cholesterol profiles during the 6 months before the study. In addition, participants were medically screened to ensure that they were free from cardiovascular, gastrointestinal, metabolic, endocrine, or orthopedic limitations. Subjects were considered sedentary when they reported via a questionnaire to not engage in any exercise or moderate- to high-intensity physical activity more than 1 hour a week. All participants were asked to maintain their previous physical activity level throughout the duration of the study. The study was approved by the University of Connecticut Institutional Review Board; all participants were informed of potential risks and gave written informed consent to participate before the start of the study.

2.3. Testing

Participants reported to the laboratory in the morning after a 12-hour overnight fast and 36 hours of abstaining from physical activity, alcohol, and caffeine. The posttraining test day was separated from the last training day by at least 72 hours to avoid transient short-term effects from the exercise bout. Upon arrival, anthropometric measurements and a blood sample were collected. The participants then had a small breakfast that was standardized for each participant for the testing day before and after the 8-week intervention. The participants then performed the strength tests followed by the cardiovascular endurance test.

2.4. Anthropometric measurements

Height was measured to the nearest 0.1 cm, waist circumference on the skin at the height of the navel to the nearest 0.5 cm, and total body mass to the nearest 0.1 kg on a digital scale (OHAUS, Florham Park, NJ). Participants were nude or wearing only underwear for measurements of body mass. Whole and abdominal body composition (% body fat, fat mass, lean body mass, and bone mass) were assessed using fan-beam dual-energy x-ray absorptiometry (Prodigy, Lunar, Madison, WI). Total body and regional abdominal tissue were assessed using anatomic landmarks as regional borders (lumbar vertebrae 2 and 4 for abdominal tissue) using computer algorithms (enCORE version 6.00.270). Coefficients of variation for lean body mass, fat mass, and bone mineral content on repeat scans with repositioning on a group of men and women by the technician in our laboratory were 0.4%, 1.4%, and 0.6%, respectively. Each anthropometric measurement was performed by the same technician for all participants before and after the 8-week intervention.

2.5. Blood collection

Participants fasted for 12 hours and abstained from exercise for 36 hours before the blood collection, which was performed in the morning from 6:00 AM to 9:00 AM at the same time of day before and after the 8-week intervention for each participant. Upon arrival to the laboratory, participants rested quietly for 15 minutes in the supine position after which a blood sample (10 mL each for serum and EDTA plasma) was obtained by venipuncture of an antecubital vein. Blood was then centrifuged at 1500*g* for 15 minutes at 4°C and resultant serum or EDTA plasma was divided into several aliquots and stored at −80°C until analysis.

2.6. Exercise testing

Participants were familiarized with the exercises involved in the testing approximately 1 week before the test day. After a standardized dynamic warm-up (lunges, heel kicks, no-load squats, high-knee raises, and high kicks) maximal upper and lower body strength was determined as the 1-repetition maximum (1-RM) for the back squat and bench press using the methods described by Kraemer and Fry [27]. Cardiovascular endurance was assessed using the Multistage Fitness Test (CD version, Sports Coach, Headingly, UK). Briefly, participants ran back and forth between 2 sets of cones spaced 20 m apart at a speed dictated by an audio recording. Approximately every minute the speed increased and the participants proceeded until they could no longer keep up with the dictated speed. The test was stopped when participants failed to maintain the dictated speed for 2 consecutive shuttles or voluntary exhausting occurred, and the number of shuttles successfully performed at the dictated speed was recorded. The distance completed was used as an estimate of cardiovascular endurance.

2.7. Diet and glucomannan supplementation

Each subject received general instructions for healthy eating and weight loss by registered dietitians before the start of the diet intervention. In addition, diet-counseling sessions were provided every 2 weeks throughout the study. Calorie counting was not emphasized, as the diet intervention focused on portion control and improving food choices. Subjects recorded their food and beverage intake for 3 days before starting the intervention (before the initial diet counseling and supplementation) and during the last week of the intervention (week 8). All diet records were analyzed for energy and macro/micronutrient content using the NUTRITIONIST PRO software package (version 3.0.1 First DataBank, The Hearst Corporation, San Bruno, CA).

Each participant ingested 1500 mg of glucomannan with approximately 237 mL of water about 5 minutes before each of the 2 largest meals of the day for a total of 3000 mg of glucomannan per day. The glucomannan was supplied in 2-piece hard gelatin capsules (Natural Alternatives, San Diego, CA). Participants recorded the time and date for each glucomannan supplementation to allow for determination of supplementation protocol compliance.

2.8. Resistance and endurance training

Exercise training was performed 3 times per week on nonconsecutive days during the 8-week duration of the study under the supervision of a trainer. The resistance training program consisted of 1 to 3 sets of 3 to 12 repetitions of standard resistance exercises using a nonlinear periodized (i.e., different loads on different days to provide variation in the exercise stress) and progressive overload format. Table 1 provides an overview of the sets and repetitions used during each resistance exercise session. The resistance used by each participant was determined by the trainer and was increased when participants were able to perform the prescribed repetitions using proper technique. Exercises included free weight exercises (bench press, incline bench press, back squat, calf raise, military press, sit-up, stiff-legged deadlift, upright row, and weighted sit-ups) and cable machine exercises (lateral pull down and seated row). Each resistance training session consisted of a selection of these exercises and was designed to target all major muscle groups. The endurance exercise program consisted of continuous aerobic exercise (walking and running) on a running track for 30 minutes; absolute intensity was progressively increased during the 8-week training period but was not periodized. The absolute and relative intensities were prescribed and monitored by the trainer.

2.9. Biochemical analyses

All biochemical variables were analyzed in duplicate and all samples for a particular participant were analyzed within the same assay batch to avoid interassay variability. Plasma total triglycerides (TG), TC, and HDL-C were determined by enzymatic methods using commercially available kits (TG, diagnostic kit no. 450032 and 759350; cholesterols, diagnostic kit no. 704036 and 759350; Roche Molecular Biochemicals, Indianapolis, IN) according to manufacturer's instructions and as previously described [28-30]. The Roche kit determines TG concentration adjusted for free glycerol. HDL-C was determined after precipitation of apolipoprotein B–containing lipoproteins using magnesium chloride and dextran sulfate [30]. Briefly, 300 μL of plasma was incubated with 30 μL of precipitation reagent (0.5 mol/L $MgCl_2$ and 0.4 mmol/L dextran sulfate) for 10 minutes and subsequently centrifuged for 15 minutes at 10 000 rpm at room temperature. The resultant supernatant was then assayed using the Roche kit according to the manufacturer's procedures for TC. The laboratory that determined TG, TC, and HDL-C has participated in the Centers for Disease Control/National Heart, Lung, and Blood Institute Lipid Standardization Program since 1989 for quality control and standardization for plasma TC, HDL-C, and TG assays. The intra-assay coefficient of variation (CV) assessed by the standardization program were 0.76% to 1.42% for TC,

Table 1
Number of sets and repetitions (sets × repetitions) for each exercise performed during the resistance exercise sessions

Week	Day 1	Day 2	Day 3
1	1-2 × 10-12	3 × 10-12	3 × 6-8
2	3 × 8-10	3 × 6-7	3 × 8-10
3	3 × 12	3 × 8-10	3 × 6-7
4	3 × 8-10	3 × 12	3 × 8-10
5	3 × 6-7	3 × 12	3 × 6-7
6	3 × 8-10	3 × 6-7	3 × 8-10
7	3 × 12	3 × 3-5	3 × 8-10
8	3 × 8-10	3 × 12	3 × 12

Repetition ranges equal repetition maximum in that the resistance allows only the listed number of repetitions.

1.71% to 2.72% for HDL-C, and 1.64% to 2.47% for TG. LDL-C was calculated using the method of Friedewald et al [31]. (LDL-C = TC − [HDL-C + TG × 5^{-1}]). Serum nonesterified fatty acids (NEFAs) were analyzed using an in vitro enzymatic colorimetric assay (994-75409 NEFA C, Wako Chemicals, Richmond, VA) according to manufacturer microtiter procedures. Absorbance was measured with dual wavelength measurements at 550 and 650 nm on a microplate reader (VersaMax, Molecular Devices, Sunnyvale, CA).

Serum insulin was determined using an enzyme-linked immunosorbent assay (ELISA) (DSL 10-1600, Diagnostic Systems Laboratories, Webster, TX; and EZHI-14K, Linco Research, St Charles, MO) according to manufacturers' guidelines. The sensitivity of the assays are 1.8 and 13.9 pmol L^{-1} and the intra-assay CVs were 3.9% and 4.0% for the Diagnostic Systems Laboratories and Linco assay, respectively. Plasma glucose was analyzed using an automated lactate/glucose analyzer (2300 STAT, YSI, Yellow Springs, OH). An estimation of insulin resistance was calculated using the homeostasis model analysis (homeostasis model assessment of insulin resistance [HOMA-IR]) with the formula: glucose (mmol L^{-1}) × [insulin (pmol L^{-1}) × 22.5^{-1}] [32]. Normal-weight, healthy individuals aged younger than 35 years usually have a HOMA-IR value of about 7; a value greater than 26.4 represents insulin resistance [33]. Serum homocysteine was measured using a high-pressure liquid chromatography system (Beckman, Fullerton, CA) with refrigerated autosampler and fluorescence detector (Jasco FP2020, Tokyo, Japan) [34]. Plasma leptin was analyzed using an ELISA (DSL 10-23100, Diagnostic Systems Laboratories); the sensitivity of the assay is 0.5 ng mL^{-1} and the intra-assay CV was 2.6%. Absorbance was measured with dual wavelength measurements at 450 and 620 nm on a microplate reader (VersaMax, Molecular Devices).

2.10. Statistical analyses

The data were evaluated using a 3-way analysis of variance (time × treatment × sex) with repeated measures for time. In addition, a 2-way analysis of variance (sex × treatment) was used to examine % changes from pre- to postintervention. When a significant F value was found, Fisher least significant difference post hoc test was used to determine pairwise differences and Bonferroni post hoc procedures were used to examine interactions for pre- to postintervention differences. Age and height for preintervention values only were examined using independent *t* tests within each sex. The α level used to establish significance was .05. Intraclass correlations for reliability of the dependent measures ranged from 0.88 to 0.95. Statistical power was calculated using nQuery Advisor software (Statistical Solutions, Saugus, MA). For the n size used, statistical power ranged for the variables examined in this investigation from 0.80 to 0.87. All statistical procedures were performed using the Statistica software package (StatSoft, Tulsa, OK). Data are presented as mean ± SD.

3. Results

There were no groupwise differences at preintervention within sex for any of the variables examined in this study. According to 3-day diet records, dietary energy, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, and carbohydrate intake were significantly reduced during

Table 2
Average daily dietary intake preintervention (Pre) and during week 8

	No exercise		Exercise	
	Pre	Week 8	Pre	Week 8
Energy (kcal)	2029 ± 553	1653 ± 521 #	1890 ± 491	1427 ± 429 #
Protein (g)	77 ± 19	79 ± 22	75 ± 25	78 ± 25
Protein (%)	15.4 ± 3.4	19.3 ± 4.9 #	15.8 ± 3.7	22.2 ± 4.6 #
Carbohydrate (g)	248 ± 107	214 ± 102 #	230 ± 67	157 ± 62 #
Carbohydrate (%)	47.1 ± 9.2	49.7 ± 10.1	48.3 ± 9.0	43.9 ± 8.
Total fat (g)	84 ± 22	57 ± 20 #	73 ± 27	53 ± 21 #
Total fat (%)	37.2 ± 7.2	30.8 ± 7.3 *	34.1 ± 7.3	33.2 ± 7.3
Cholesterol (mg)	305 ± 155	261 ± 143	243 ± 164	257 ± 200
Saturated fat (g)	29 ± 8	20 ± 10 #	24 ± 9	17 ± 7 #
Monounsaturated fat (g)	21 ± 9	13 ± 6 #	20 ± 7	16 ± 9 #
Polyunsaturated fat (g)	11 ± 6	9 ± 4 #	12 ± 5	8 ± 4 #
Alcohol (g)	0.7 ± 2.6	0.5 ± 1.7	4.8 ± 1.5	6.8 ± 3.8
Dietary fiber [a] (g)	14 ± 7	16 ± 7	15 ± 6	14 ± 8
Soluble fiber [a] (g)	0.3 ± 0.6	0.2 ± 0.2	0.4 ± 1.0	0.2 ± 0.4
Caffeine (mg)	143 ± 143	122 ± 113	114 ± 224	95 ± 150

Values are expressed as mean ± SD.

[a] Not including 3000 mg of glucomannan.

* $P < .05$, significant different from corresponding Pre value.

\# $P < .05$, significant effect of time.

week 8 of the intervention compared with preintervention. Percent protein intake was significantly increased from preintervention to week 8. There was a trend ($P < .07$) for a lower daily carbohydrate intake during week 8 for the Ex group compared with the No-Ex group. For the women, % carbohydrate intake was significantly reduced from preintervention to week 8 for the Ex group, whereas % carbohydrate intake was significantly increased for the No-Ex group. There were no differences in % carbohydrate intake among the men. For the women No-Ex group only, % fat intake was significantly reduced from preintervention to week 8. There were no differences for daily protein, cholesterol, alcohol, total fiber, soluble fiber, or caffeine between preintervention and week 8. Results for average daily dietary intake are presented in Table 2.

Exercise training significantly ($P < .05$) increased squat 1-RM, bench press 1-RM, and distance completed during the shuttle-run test (Fig. 1). No performance changes were found for the No-Ex groups. There were no sex by exercise interactions for absolute values in any of the performance measures, indicating that absolute increases in performance were consistent across sex. There was a trend for greater increase in % change in squat 1-RM ($P = .06$) for the women (35.4% ± 43.1%) compared to the men (18.7% ± 27.8%) and in distance completed in the shuttle-run test ($P < .10$) for the women (41.7% ± 47.7%) compared to the men (25.6% ± 43.2%). For the % change in the bench press 1-RM there was a significant interaction effect for sex and exercise. The increase in % change for the women Ex group (21.1% ± 13.4%) was significantly greater than for the other groups (men Ex, 11.9% ± 9.6%; men No-Ex, 3.6% ± 9.6%; women No-Ex, −1.0% ± 7.7%).

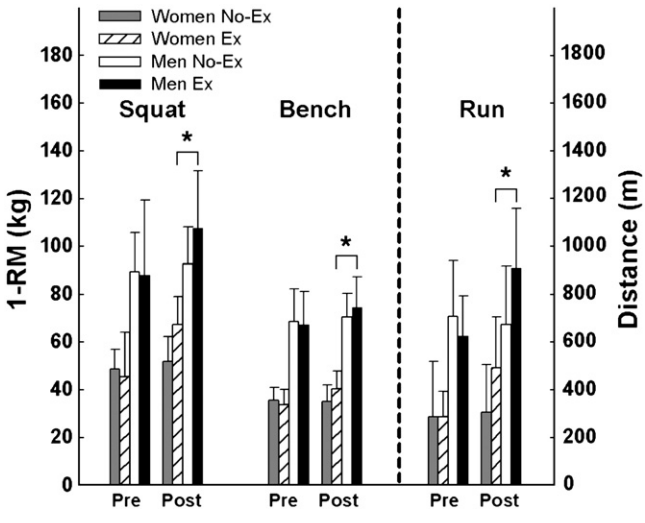


Fig. 1. One-repetition maximum (1-RM) for the back squat (Squat) and bench press (Bench) (left axis) and distance completed in the Multistage Fitness Test (Run) (right axis) pre- and postintervention. *$P < .05$, significantly different from corresponding preintervention value. Mean ± SD.

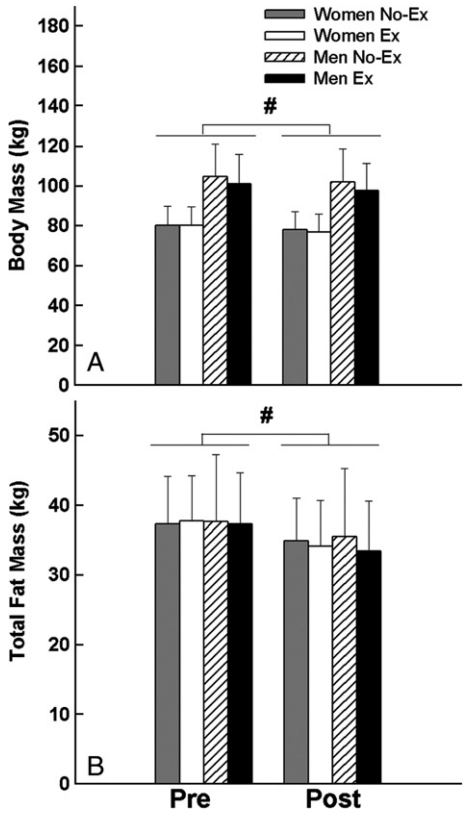


Fig. 2. A, Body mass pre- and postintervention. B, Total fat mass pre- and postintervention. #$P < .05$, significant main effect of time. Mean ± SD.

Exercise significantly augmented the absolute change in % body fat and % changes in % body fat, total fat mass, and waist circumference, but did not affect body mass or BMI. Results for body mass and total fat mass are presented in Fig. 2 and results for % changes in body mass and total fat mass are presented in Fig. 3. Remaining results for body composition are presented in Table 3. There was a significant main effect of time (pre- to postintervention) for improvements in body composition with reductions in body mass,

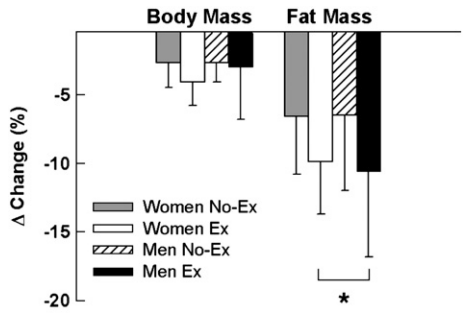
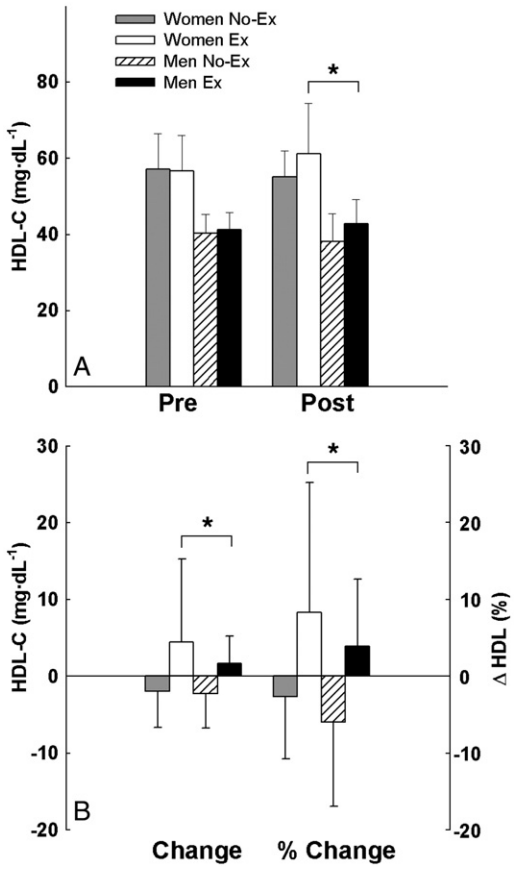


Fig. 3. Percent change in body mass and total fat mass from pre- to postintervention. *$P < .05$, significant main effect of exercise. Mean ± SD.

Table 3
BMI, % body fat, total lean mass, % abdominal fat, abdominal fat mass, and waist circumference

	BMI (kg m^{-2})	Body fat (%)	Total lean mass (kg)	Abdominal fat (%)	Abdominal fat mass (kg)	Waist circumference (cm)
Men						
No exercise						
Pre	32.6 ± 3.9	35.6 ± 5.3	63.7 ± 9.1	46.6 ± 6.3	4.3 ± 1.3	111 ± 12.3
Post	31.8 ± 4.0$^{\#}$	34.2 ± 5.7$^{\#}$	63.6 ± 9.2	44.5 ± 7.8$^{\#}$	4.0 ± 1.4$^{\#}$	109 ± 12.8$^{\#}$
Exercise						
Pre	29.7 ± 10.0	36.8 ± 3.9	60.6 ± 8.7	48.7 ± 4.0	4.5 ± 1.0	112 ± 7.4
Post	28.8 ± 9.6$^{\#}$	33.9 ± 4.1$^{\#,\$}$	61.2 ± 7.2	45.0 ± 4.4$^{\#}$	3.9 ± 0.8$^{\#}$	108 ± 7.3$^{\#}$
Women						
No exercise						
Pre	30.7 ± 3.0	46.3 ± 4.7	40.3 ± 4.6	52.9 ± 5.7	3.6 ± 0.8	103 ± 7.5
Post	29.8 ± 2.7$^{\#}$	44.4 ± 4. 3$^{\#}$	40.7 ± 4.4	49.6 ± 6.9$^{\#}$	3.3 ± 0.9$^{\#}$	101 ± 8.3$^{\#}$
Exercise						
Pre	31.0 ± 3.2	47.0 ± 3.2	39.6 ± 3.6	52.5 ± 4.9	3.4 ± 0.7	102 ± 7.6
Post	29.7 ± 3.1$^{\#}$	44.2 ± 3.7$^{\#,\$}$	40.0 ± 3.2	49.0 ± 5.4$^{\#}$	3.0 ± 0.7$^{\#}$	97 ± 8.4$^{\#}$
% Change						
No exercise						
Men	−2.7 ± 1.4	−4.2 ± 4.6	−0.2 ± 1.9	−5.0 ± 7.1	−7.9 ± 8.4	−2.1 ± 1.7
Women	−2.7 ± 1.8	−4.0 ± 2.7	1.0 ± 1.5	−6.4 ± 5.4	−9.4 ± 6.1	−2.1 ± 2.4
Exercise						
Men	3.0 ± 3.8	−8.1 ± 4.1$^{\$}$	1.4 ± 4.4	−7.6 ± 5.0	−13.4 ± 8.9	−3.8 ± 2.7$^{\$}$
Women	−4.1 ± 1.7	−6.0 ± 2.9$^{\$}$	0.9 ± 2.3	−6.7 ± 4.0	−13.3 ± 6.1	−4.8 ± 3.8$^{\$}$

Values are expressed as mean ± SD; absolute pre- and postintervention values and relative changes (%) from pre- to postintervention.
$^{\#}$ $P < .05$, significant effect of time.
$^{\$}$ $P < .05$, significant effect of exercise.



total fat mass, % body fat, BMI, abdominal % fat, abdominal fat mass, and waist circumference.

The blood lipid profile improved after the 8-week intervention with significant reductions in TC, LDL-C, and LDL-C/HDL-C. There was a significant interaction (time × treatment) for HDL-C and TC/HDL-C. Post hoc follow-up revealed that exercise significantly elevated HDL ($P = .04$, effect size = 0.6 and 0.4 for men and women, respectively; % change, $P = .006$) and reduced TC/HDL-C (change, $P = .04$; effect size = 0.6 and 0.7 for men and women, respectively; % change, $P = .02$) pre- to postintervention. No changes were found in HDL-C or TC/HDL-C for No-Ex. Similarly, exercise significantly improved the % change in HDL-C and TC/HDL-C but had no effect on % changes in the remaining blood lipids. Results for HDL-C are presented in Fig. 4; results for remaining blood lipid variables are presented in Table 4.

Results for insulin, glucose, HOMA-IR, homocysteine, and leptin are presented in Table 5. There were no significant differences between pre- to postintervention in insulin, glucose, HOMA-IR, or homocysteine for any condition. Similarly, there were no significant differences between % changes in insulin, glucose, HOMA-IR, or homocysteine

Fig. 4. A, Plasma concentrations of HDL-C pre- and postintervention. B, Absolute (left axis) and relative (%) (right axis) change in plasma HDL-C concentration from pre- to postintervention. *$P < .05$, significant effect of exercise. Mean ± SD.

Table 4
Plasma concentrations of TC, LDL-C, TG, LDL/HDL, TC/HDL, and serum NEFAs

	TC (mg dL^{-1})	LDL-C (mg dL^{-1})	TG (mg dL^{-1})	LDL/HDL	TC/HDL	NEFA (mEq dL^{-1})
Men						
No exercise						
Pre	168.9 ± 43.3	108.6 ± 39.5	100.2 ± 48.5	2.7 ± 1.0	4.2 ± 1.1	0.35 ± 0.13
Post	151 ± 39.1[#]	92.5 ± 38[#]	102.2 ± 35.6	2.5 ± 0.9[#]	4.0 ± 1.0	0.26 ± 0.06
Exercise						
Pre	191.3 ± 22.0	123.3 ± 23.5	134.3 ± 53.8	3.0 ± 0.6	4.7 ± 0.7	0.27 ± 0.10
Post	172.5 ± 27.0[#]	108.1 ± 26.5[#]	108.2 ± 38.9	2.6 ± 0.8[#]	4.1 ± 0.9[$]	0.24 ± 0.12
Women						
No exercise						
Pre	180.1 ± 24.0	105.2 ± 19.5	88.9 ± 30.1	1.9 ± 0.4	3.2 ± 0.4	0.33 ± 0.12
Post	170.8 ± 28.7[#]	98.3 ± 27.9[#]	87.4 ± 37.7	1.8 ± 0.6[#]	3.1 ± 0.5	0.31 ± 0.16
Exercise						
Pre	170.1 ± 36.6	99.5 ± 29.4	69.9 ± 18.5	1.8 ± 0.5	3.0 ± 0.5	0.32 ± 0.11
Post	159.9 ± 23.7[#]	85.9 ± 21.0[#]	65.1 ± 24.6	1.5 ± 0.5[#]	2.7 ± 0.5[$]	0.35 ± 0.07
% Change						
No exercise						
Men	−9.8 ± 11.1	−14.0 ± 16.1	22.4 ± 89.9	−8.1 ± 16.7	−3.5 ± 11.7	−17.7 ± 29.1
Women	−5.0 ± 10.6	−7.2 ± 18.4	−3.8 ± 15.0	−4.8 ± 16.6	−2.3 ± 8.5	−5.2 ± 56.9
Exercise						
Men	−9.9 ± 10.0	−12.4 ± 14.6	−10.6 ± 43.5	−14.7 ± 19.0	−12.4 ± 15.1[$]	−1.2 ± 65.3
Women	−4.9 ± 8.4	−11.8 ± 14.7	−6.7 ± 24.9	−16.5 ± 19.1	−10.9 ± 10.8[$]	16.8 ± 38.8

Values are expressed as mean ± SD; absolute pre- and postintervention values and relative changes (%) from pre- to postintervention.
[#] $P < .05$, significant effect of time.
[$] $P < .05$, significant effect of exercise.

between sex or exercise condition. Leptin was significantly reduced from pre- to postintervention for both the Ex and No-Ex groups; however, exercise significantly augmented the absolute and % reductions in leptin.

4. Discussion

This is the first study to examine the effect of adding a total-body exercise program to a diet containing the soluble

Table 5
Circulating concentrations of insulin, glucose, homocysteine, and leptin, and HOMA-IR

	Glucose (mmol L^{-1})	Insulin (pmol L^{-1})	HOMA-IR	Homocysteine (μmol L^{-1})	Leptin (ng mL^{-1})
Men					
No exercise					
Pre	5.13 ± 0.38	98.0 ± 70.3	17.3 ± 7.6	8.90 ± 2.13	39.8 ± 16.5
Post	5.03 ± 0.17	103.3 ± 76.6	18.0 ± 9.0	8.36 ± 1.52	34.4 ± 15.1[#]
Exercise					
Pre	5.10 ± 0.37	101.7 ± 84.0	17.3 ± 5.4	9.10 ± 1.87	41.4 ± 19.3
Post	5.12 ± 0.43	95.6 ± 85.1	16.5 ± 8.8	9.50 ± 1.44	26.4 ± 16.8[#,$]
Women					
No exercise					
Pre	4.77 ± 0.46	57.9 ± 31.2	12.5 ± 7.5	8.22 ± 1.82	63.3 ± 18.3
Post	4.79 ± 0.26	53.0 ± 30.1	11.4 ± 6.9	7.97 ± 2.44	54 ± 16.7[#]
Exercise					
Pre	4.75 ± 0.28	100.2 ± 89.7	21.0 ± 18.7	8.76 ± 2.16	85.9 ± 24.6
Post	4.82 ± 0.30	97.5 ± 76.5	21.2 ± 17.1	9.59 ± 3.00	59.7 ± 20.5[#,$]
% Change					
No exercise					
Men	−11.7 ± 27.3	10.2 ± 30.9	11.8 ± 33.0	−3.98 ± 16.76	−10.9 ± 19.1
Women	0.9 ± 8.1	−4.5 ± 37.6	−3.6 ± 45.5	−1.42 ± 22.83	−15.2 ± 21.7
Exercise					
Men	−7.3 ± 26.4	−8.4 ± 24.5	−9.1 ± 26.8	6.47 ± 17.90	−37.6 ± 25.8[$]
Women	1.6 ± 7.6	−4.0 ± 32.8	5.4 ± 31.7	11.95 ± 35.02	−30.4 ± 14.8[$]

Values are expressed as mean ± SD; absolute pre- and post intervention values and relative changes (%) from pre- to postintervention.
[#] $P < .05$, significant effect of time.
[$] $P < .05$, significant effect of exercise.

fiber glucomannan. We showed that a dietary intervention with glucomannan alone promoted weight loss, fat loss, and improvement in cholesterol levels, whereas the inclusion of exercise 3 days per week resulted in further improvements in body composition and had additional benefits on HDL-C and TC/HDL-C and measures of physical performance.

The exercise program successfully increased 1-RM in the bench press and squat exercises and distance completed in the shuttle-run test in the Ex groups. The No-Ex groups exhibited no changes in performance on any of the exercise tests from pre- to postintervention. The large increases in physical performance in the Ex groups are consistent with previous research using comparable exercise training programs [25,35] and confirms the effectiveness of the resistance and cardiovascular endurance training program used in this investigation.

As expected, the diet intervention, which included 3000 mg of glucomannan per day, successfully reduced daily caloric intake from preintervention to the end of the study. This reduction in caloric intake was caused by a reduction in total fat and carbohydrate intake. Surprisingly, the reduction in total fat intake was not accompanied by a change in cholesterol intake. Exercise had no major effects on dietary intake, although a significant reduction was found for % fat for the women in the No-Ex group and total carbohydrate intake appeared to be reduced in the exercise group.

The reduction in daily caloric intake was manifested in a significant reduction in body mass and BMI. This reduction in body mass is consistent with previous findings on the effect of a diet with glucomannan [1,15]. The loss of body mass in the present study mainly was due to a loss of total fat mass. The reduction in fat mass accounted for 85% and 130% of body mass reduction in the men and 118% and 109% of body mass reduction in the women, for No-Ex and Ex groups, respectively. Because total lean mass was not affected by the intervention this led to a reduction in % body fat. Similarly, abdominal fat mass, % abdominal fat, and waist circumference significantly declined from pre- to postintervention, with no change in abdominal lean mass. The exercise program did not increase the body mass loss compared to the diet and glucomannan intervention alone. However, exercise augmented the reduction in % body fat and appeared (significant treatment × time interaction approached significance after Bonferroni correction) to augment the reduction in total fat mass (by 63% and 50% in men and women, respectively), abdominal fat mass, and waist circumference. This apparent discrepancy between changes in body mass and total fat mass for the Ex groups is likely explained by the larger but nonsignificant increases in lean mass in the Ex groups compared with the No-Ex groups. As expected with an 8-week intervention for resistance exercise, no changes were yet observed in the lean mass, but large increases in 1-RM were observed, suggesting that neuromuscular adaptations had occurred. Exercise also significantly augmented % reduction in waist circumference. Excess waist circumference [36,37] and abdominal fat are associated with numerous cardiovascular risk factors [38,39]; the apparent added reduction in fat by exercise might have important clinical implications for overweight individuals beyond those examined in this study.

Glucomannan has a lowering effect on circulating TC and LDL-C by mechanisms that include blocking uptake in the gut and possibly increasing excretion into the bile [3,19]. In the present study, TC, LDL-C, and LDL-C/HDL-C were significantly reduced after the diet and glucomannan intervention despite no changes in dietary cholesterol intake. Although cholesterol uptake in the gut and excretion into the bile were not measured in the present study, it appears likely that glucomannan aided in the reduction of circulating TC and LDL-C levels via these mechanisms. Exercise did not influence changes in TC or LDL-C; however, exercise favorably influenced HDL-C and TC/HDL-C. Diets with glucomannan supplementation led to either a decrease [5,7,9] or no change in HDL-C [3,6,8]. In the present study, exercise increased HDL-C (3.9% and 8.3% for men and women, respectively), whereas the No-Ex groups experienced a decline in HDL-C (−6.0% and 2.7% for men and women, respectively). This follows the findings of Varady et al [40] who showed that 8 weeks of endurance exercise 3 times a week increased HDL-C by approximately 10%. It appears that exercise, although not affecting changes in TC and LDL, has a beneficial effect on HDL-C, which also leads to an improved TC/HDL-C. Increased HDL-C as well as reduced TC, LDL-C, and TC/HDL-C are each associated with a decreased risk of cardiovascular disease [41-44]; thus, adding exercise to a glucomannan-containing diet regimen appears to be an important factor in improving the blood lipid profile of overweight individuals.

There were no changes in fasting insulin, glucose, or insulin resistance, as measured by the HOMA-IR, in the present study. This follows the previous finding that fasting glycemic control is not affected by long-term glucomannan supplementation in nondiabetic individuals [6]. Glucomannan has been found to reduce the insulin and glucose response in a glucose tolerance test during the first 2 hours of the test in nondiabetics [18]. Thus, it appears that the effect of glucomannan on glycemic control in nondiabetics is mainly during the postprandial period and likely involves reduced gastric emptying and/or interference with glucose uptake in the gut. Contrary to what was expected, 8 weeks of combined resistance and endurance training did not affect fasted insulin or HOMA-IR. This finding is in contrast to several studies that found that exercise training reduces fasted insulin [45-47]. A possible explanation for this discrepancy could be that exercise mainly has a transient short-term effect on fasted insulin concentrations, and limited long-term effect [45,48-50]. Boule et al [45] showed that after 20 weeks of endurance training, insulin was reduced but returned to pretraining levels 72 hours after the last exercise session. In contrast to the studies by Houmard et al [47] and O'Donovan et al [46], posttesting in the present study was conducted more than 72 hours after the last

training session, thereby reducing the influence of potentially transient short-term effects of exercise. It is also possible that training 3 times a week was not frequent enough to induce a reduction in resting insulin.

Leptin is produced by the adipocytes and plays an important role in the regulation of body mass via energy expenditure and intake [51]. Obesity is linked with leptin resistance and elevated levels of circulating leptin [52]. Previous studies have found that exercise and diet regimens leading to reductions in body and fat mass in obese adults also lead to reduced circulating leptin concentrations [53,54]. In the present study, the addition of exercise to an 8-week diet containing glucomannan significantly augmented the reduction in leptin (by ~245% and ~100% for the men and women, respectively). It seems unlikely that the added reductions in fat mass from exercise can cause this large reduction alone; we speculate that exercise also reduced leptin resistance and, thus, circulating leptin levels beyond that explained by the augmented reduction in fat mass alone.

In the present study, circulating homocysteine concentrations did not change from pre- to postintervention for either condition. Certain weight loss treatments (eg, gastroplasty or high fat-low carbohydrate diets without vitamin supplementation) can lead to increased circulating homocysteine concentrations [55,56]. This is a potential concern for any weight loss regimen because elevated concentrations of serum homocysteine are correlated with an increased risk for cardiovascular disease [57]. The elevation in homocysteine is likely due to inadequate intake of folic acid [58]. Because glucomannan may reduce the uptake of certain nutrients (ie, cholesterol), a concern could be that glucomannan affects the uptake of folic acid; however, no significant differences over time or between groups were observed.

There were no sex effects in absolute or % changes for any of the variables examined in this investigation with the exception of the exercise tests. Thus, it appears that the improvements in % body fat and HDL-C from adding exercise to a diet containing glucomannan are not sex specific. A limitation of this study is that there were no control groups with respect to the diet and glucomannan intervention. Previous studies, however, have already established the beneficial effects of glucomannan supplementation on body mass, body composition, and blood lipids in the context of hypocaloric diets [1,11-14], as well as free living conditions (no dietary guidance to restrict calories) [14-16]. The results from the present study complement these previous findings by showing that the addition of a resistance and endurance exercise program to a glucomannan diet regimen significantly improves body composition, HDL-C, TC/HDL-C, and leptin. Future research should further explore the potential added benefits of exercise to a glucomannan supplementation intervention. Of special interest could be the effect in diabetic individuals because glucomannan and exercise independently have been shown to improve glycemic control in diabetics and no negative effects were observed between the 2 treatments.

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