Whey Protein Supplementation Does Not Affect Exercise Training-Induced Changes in Body Composition and Indices of Metabolic Syndrome in Middle-Aged Overweight and Obese Adults

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Abstract

Little is known about the effects of different quantities of whey protein on exercise training–induced changes in body composition and indices of metabolic syndrome in middle-aged overweight and obese adults. Therefore, we examined the effects of consuming 0.8-MJ supplements with 0 (n = 126), 10 (n = 112), 20 (n = 44), or 30 (n = 45) g whey protein twice daily in conjunction with resistance (2 d/wk) and aerobic (1 d/wk) exercise training in a double-blind, randomized, placebo-controlled, community-based 9-mo study in men (n = 117) and women (n = 210); (age: 48 ± 7.9 y; BMI: 30.0 ± 2.8 kg/m²). Whey protein supplementation did not influence any of the following outcomes, some of which were affected by training. Among all participants, strength increased by 15% (P < 0.001) and maximal oxygen uptake capacity (VO2max) increased by 9% ± 15% (P < 0.001). Body weight was unchanged (0.1 ± 3.7 kg, P = 0.80), lean body mass increased by 1.9 ± 2.8% (0.95 ± 1.3 kg, P < 0.001), and fat mass decreased by 2.6 ± 9.4% (–0.86 ± 3.1 kg, P = 0.001). Oral-glucose-tolerance testing showed that plasma glucose AUC was unchanged (–18.0 ± 170 mmol/L·3 h, P = 0.16), insulin AUC decreased by 2.6 ± 32% (–7.5 ± 29 mmol/L·3 h, P = 0.01), and HOMA-IR (0.2 ± 2.0, P = 0.81) and the insulin sensitivity index (0.3 ± 3.0, P = 0.63) were unchanged. Plasma concentrations of TG; total, LDL, and HDL cholesterol; C-reactive protein; plasminogen activator inhibitor-1; blood pressure; and waist circumference were unchanged. Whey protein supplementation did not affect exercise training–induced responses in body composition and indices of metabolic syndrome in middle-aged overweight and obese adults who maintained body weight. J. Nutr. 142: 1532–1539, 2012.

Introduction

Metabolic syndrome is characterized by a combination of metabolic abnormalities that increase the risk of the development of diabetes and cardiovascular disease and include dyslipidemia (elevated TG and LDL cholesterol, and low HDL cholesterol), elevated blood pressure, insulin resistance or glucose intolerance, a prothrombotic state, and a proinflammatory state (1). Overweight, obesity, and physical inactivity are well-established risk factors for type 2 diabetes and cardiovascular comorbidities (2), which may be countered by improvements in body composition (decreased fat mass and increased lean body mass) (3,4) and physical fitness (5–7). Exercise training is an effective way to increase lean body mass and reduce fat mass (3,8), as well as to improve lipid-lipoprotein profile (particularly to increase HDL cholesterol) (5), blood pressure (6,9), and insulin sensitivity (7) in weight-stable adults. Limited research suggests that the consumption of higher protein diets may enhance the exercise-induced improvements in body composition (10–12) and indices of metabolic syndrome (13).

Protein supplementation can be used to increase total protein intake. Studies evaluating the effects of higher protein diets and exercise training on body composition often use whey protein (13–15), which is 1 of the 2 primary proteins in milk and accounts for 20% of total milk protein (16). Evidence suggests that whey protein and its bioactive components exhibit
hypo- lipidemic (17), insulinotrophic (18,19), antihypertensive (20,21), and antiinflammatory (22) properties. Furthermore, whey is considered a high-quality protein that contains all essential amino acids, making it a potent anabolic stimulus when coupled with resistance exercise (23). Findings from studies evaluating the effects of higher protein diets and resistance exercise training on body composition in overweight and obese middle-aged adults are conflicting (3,10,12,15,24,25). Individual studies have reported no additional benefits of higher protein diets on exercise training–induced changes in body composition (3,26), whereas a retrospective reassessment in 106 men and women showed gains in fat-free mass when \( >1.0 \) g protein per kg \( \cdot d^{-1} \) was consumed (10). In terms of whey protein supplementation, men who performed resistance training and who consumed \( >100 \) g whey protein/d (\( \geq 2.0 \) g protein \( \cdot kg^{-1} \cdot d^{-1} \)) experienced greater gains in lean body mass compared with those who received a casein supplement (12) or a placebo (25). On the other hand, men supplemented with 25–35 g/d (\( \geq 1.0 \) g protein \( \cdot kg^{-1} \cdot d^{-1} \)) did not enhance resistance training–induced increases in lean body mass and decreases in fat mass compared with a placebo supplement (13,15). Collectively, these studies suggest that the quantity of supplemental whey protein may be an important factor influencing these body-composition and metabolic health responses, with intakes of whey protein \( \geq 35 \) g/d, leading to total protein intakes \( >1.0 \) g protein \( \cdot kg^{-1} \cdot d^{-1} \) possibly being more effective. Limited studies have evaluated the effects of whey protein supplements in conjunction with exercise training on indices of metabolic syndrome (13), and studies that titrate different quantities of whey protein are needed.

The primary aims of this study were to examine the effectiveness of increased total dietary protein intake, through whey protein supplementation, under free-feeding conditions to enhance changes in body composition and positively change indices of metabolic syndrome with resistance and aerobic exercise training in overweight and obese middle-aged adults. We hypothesized that increased whey protein consumption would result in 1) enhanced training-induced gain of lean body mass and loss of fat mass and 2) enhanced reductions in waist circumference, blood lipids, blood pressure, and inflammation and increased indices of insulin sensitivity.

**Participants and Methods**

**Participants.** Overweight and obese middle-aged men and women (aged 35–65 y) were recruited from the greater West Lafayette, IN, area. Inclusion criteria were as follows: body weight \( <300 \) lb (136 kg), BMI between 26 and 35 \( kg/m^2 \), blood pressure \( <160/100 \) mm Hg, fasting plasma glucose \( <6.1 \) mmol/L, total cholesterol \( <6.7 \) mmol/L, LDL cholesterol \( <4.1 \) mmol/L, TG \( <4.5 \) mmol/L, no preexisting kidney or liver conditions, not currently or previously (past 6 mo) consuming a weight-loss diet or other special/nonbalanced diets, no weight loss/gain \( \geq 4.5 \) kg) within the past 6 mo, and \( <2 \) h/wk of habitual resistive or aerobic exercise training in the past 6 mo. Participants taking medications for elevated TG, reduced HDL cholesterol, or elevated blood pressure were included. The Purdue University Institutional Review Board approved the study protocol, which complied with the Helsinki Declaration as revised in 1983. All participants provided written informed consent and received monetary compensation for participation. A flow diagram of the recruitment and retention of participants is presented in Supplemental Figure 1.

**Experimental design.** This intent-to-treat study was double-blind, placebo-controlled, community-based, and 36 wk in duration. After completing a 1-wk baseline period, participants were randomly assigned to 1 of 4 groups and were instructed to consume the assigned dietary supplements. All participants performed resistance (2 d/wk) and aerobic (1 d/wk) exercise for 36 wk at 1 of 5 local fitness facilities in the Lafayette/West Lafayette, IN, area. Details on the exercise training and testing can be found in the Online Supporting Materials. All measurements were taken pre- (baseline), mid- (wk 18), and post- (wk 36) intervention.

**Whey protein supplementation and nutritional intakes.** During the 36-wk intervention period, each participant was instructed to consume one supplement sachet containing 0.8 MJ (200 kcal) and 0, 10, 20, or 30 g whey protein twice daily with breakfast and lunch mixed with the food or beverage of their preference. Daily supplemental whey protein intakes were 0, 20, 40, and 60 g/d, respectively. Supplements were in powder form and manufactured by Innovative Food Processors, Inc. (Supplemental Table 1). Participants were told that the supplements provided 1.7 MJ/d (400 kcal/d) of energy but were not counseled to purposely alter their usual eating behaviors. Four-day food records (3 weekdays and 1 weekend day) were completed at wk 0, 18, and 36 to estimate daily energy intake and macronutrient composition (Nutritionist Pro, First DataBank version 1.3.36). The age- and sex-specific Schofield equations (27) were used to estimate basal metabolic rate, and food records that included the 1.7 MJ from the supplements at mid- and postintervention time points were considered valid if they fell within the previously established lower and upper 95% confidence limits (28). Data on 176 of the 220 participants (0 g/d: \( n = 72 \); 20 g/d: \( n = 65 \); 40 g/d: \( n = 16 \); and 60 g/d: \( n = 23 \)) met this criterion and were used in the food record analyses. To document group-specific differences in total protein intake, 24-h urinary urea nitrogen (UUN) was analyzed. Twenty-four-hour urine volumes (weight divided by specific gravity; Digital Probe Refractometer; Misco Products Division) were obtained from two 24-h urine collections, and aliquots were stored at \(-20^\circ C \) for subsequent analyses of urea nitrogen (COBAS Integra 400; Roche Diagnostic Systems).

**Assessment of dietary compensation.** Dietary compensation was determined by subtracting nonsupplement energy and macronutrient intakes at mid- and postintervention time points from baseline intakes. The dietary data were obtained from the 4-d food records completed at baseline, wk 18, and wk 36.

**Compliance.** Participants completed daily supplement logs to document when the supplements were consumed and a weekly exercise training log for each exercise session, which included information pertaining to the amount of weight lifted, repetitions, and specific exercises completed for resistance exercise sessions and heart rate for the aerobic exercise sessions. On the basis of the self-reported daily supplement logs and weekly exercise training logs, participants were placed into 3 compliance categories for both supplementation (80%, 50–79%, and <50% of supplements consumed) and exercise (90%, 70–89%, and <90% of sessions completed).

**Body composition and anthropometric measurements.** Body weight, lean body mass, and fat mass were determined by using dual-energy X-ray absorptiometry (LUNAR iDXA and Lunar enCORE software, version 11.2; GE Medical Systems). Waist circumference was measured in the standing position at the narrowest area between the lateral lower rib and the iliac crest. Hip measurement was taken at the largest circumference of the lower abdomen. The measurements were performed in triplicate, and mean values were reported.

**Blood collection, lipoprotein profile, and renal function.** Following a 12-h overnight fast, blood samples were collected in tubes containing a clot activator to obtain serum or sodium heparin to obtain plasma (BD Vacutainer Brand; Becton, Dickinson and Co). Serum tubes were sent to Mid America Clinical Laboratories for lipoprotein profile (total and HDL cholesterol and TG) and renal function determination (albumin

10 Abbreviations used: AU, arbitrary unit; CRP, C-reactive protein; PAI-1, plasminogen activator inhibitor-1; UUN, urinary urea nitrogen; VCO₂, carbon dioxide production; VO₂, oxygen consumption.

1533

Whey protein, exercise, lean body mass, and health
and creatinine). LDL cholesterol was calculated on the basis of the Friedewald equation (29). Glomerular filtration rate was estimated by using the Modification of Diet in Renal Disease equation (30). Plasma tubes were immediately placed on ice for 30 min, and centrifuged at 4°C for 10 min at 3000 × g. The plasma was then separated and stored in microcentrifuge tubes at −80°C for subsequent glucose, insulin, C-reactive protein (CRP), and plasminogen activator inhibitor-1 (PAI-1) analyses.

**Blood pressure.** Sitting blood pressure was measured with an automated sphygmomanometer (Advantage 6014 Advanced Blood Pressure Monitor; American Diagnostic Corporation) after the participant rested in a sitting position for ≥10 min. The measurements were taken in duplicate on the same arm and averaged.

**Glucose-tolerance assessment.** During the 3-h oral-glucose-tolerance test, participants consumed a sugar solution containing 75 g dextrose. Blood samples were collected at 0, 30, 60, 90, 120, 150, and 180 min, and AUC for glucose and insulin were determined using the trapezoidal rule (31). HOMA-IR and whole-body (composite) insulin sensitivity were calculated as previously described (32,33). Plasma glucose concentration was measured by enzymatic colorimetry using an oxidase method on a COBAS Integra 400 analyzer (Roche Diagnostic Systems). Plasma insulin concentration was measured by an electrochemiluminescence immunoassay method on the Elecsys 2010 analyzer (Roche Diagnostic Systems).

**Proinflammatory and prothrombotic markers.** Plasma CRP was measured on a COBAS Integra 400 analyzer (Roche Diagnostic Systems). Plasma PAI-1 was analyzed through enzyme immunoassay techniques by using an ELISA and the standard manufacturer’s protocol (PAI-1 AcrEase; Technoclone GmbH). All samples from a given participant were tested in duplicate and analyzed within the same assay. The CV for CRP and plasma PAI-1 were 1.7% and 14.0%, respectively.

**Metabolic syndrome criteria.** The number of participants who met the American Heart Association and National Heart, Lung, and Blood Institute criteria for metabolic syndrome (34) were documented pre-, mid-, and postintervention. A participant was defined as having metabolic syndrome if any 3 of the 5 following criteria were met: elevated waist circumference (population- and country-specific definitions), ≥TG 1.7 mmol/L, HDL cholesterol <1.0 mmol/L, systolic blood pressure ≥130 mm Hg and/or diastolic blood pressure ≥85 mm Hg, fasting glucose ≥5.6 mmol/L, or drug treatment for elevated TG, blood pressure, and/or reduced HDL cholesterol (34).

**Metabolic measurements.** The participants reported to the laboratory after a 10-h overnight fast and reclined on a bed for 30 min to acclimate to room temperature. Indirect calorimetry was used to measure resting energy expenditure for the next 30 min. The first 10 min of data were excluded to reduce the variability in the initial adjustment period (MedGraphics Cardiopulmonary Diagnostics Systems; MedGraphics Corporation). Energy expenditure was estimated by using the Weir equation (35). Substrate oxidation (g/min) was estimated from the following equations (36): carbohydrate oxidation = 4.55 carbon dioxide production (VCO₂) (mL/min). Power calculations were performed on the dietary protein–related differential change in lean body mass. Sixty-four participants per group were needed to statistically confirm a difference in lean body mass accretion of ≥0.8 kg between the control group and the 20-g/d group. Twenty participants per group were needed to statistically confirm differences in lean body mass accretion between the control group and the 40-g/d and the 60-g/d groups (P < 0.05, >90% power). This study was not powered to evaluate differences between the 20-, 40-, and 60-g/d groups.

**Statistical analyses.** Analyses were performed by using data from participants who completed the entire 36-wk intervention (n = 188) or who dropped out after the midpoint but completed the wk 18 testing (n = 32) (Supplemental Figure 1). The appropriate transformation (i.e., log, square root, or reciprocal) was performed on data that were not normally distributed to meet the homogeneity of variance assumption needed to perform the ANOVA. A repeated-measures ANOVA was performed to determine the main effects of group and time and group-by-time interactions on all variables (n = 220 at pre-, n = 220 at mid-, and n = 188 at postintervention). Dunnett’s test of multiple comparisons was used for post hoc analyses to detect differences between treatment groups and the control group. A Tukey-Kramer test was used to examine differences between time points within groups and with the overall group means. To examine the relationship between the presence or absence of metabolic syndrome between whey protein groups and over time, chi-square and McNemar’s tests were performed, respectively. On the basis of accumulating evidence that suggests that a change in insulin sensitivity is influenced by changes in body composition (39,40), Pearson correlations were used to assess the relationship between changes in fat and lean body mass with changes in glucose and insulin AUC. Participants taking antihypertensive or antihyperlipidemic medications who either stopped taking the medication or if the dosage changed during the intervention were excluded from the blood pressure and blood lipid analyses, respectively.

**Secondary analyses.** Because this was an intent-to-treat intervention, all participants who completed the entire 36-wk intervention or who dropped out after the midpoint but completed wk 18 testing were included in the original analyses; however, secondary statistical analyses were performed to assess the influence of compliance on the outcome variables. The mean compliance of each participant between wk 1–18 and 19–36 for the mid- and postintervention time points, respectively, was determined. Participants who achieved the highest compliance to both the supplementation (80% consumed) and exercise sessions (90% completed) were then included in repeated-measures ANOVA and post hoc (Tukey-Kramer and Dunnett’s) analyses.

All data are presented as means ± SD unless otherwise specified. Significance was accepted at P < 0.05. Data were analyzed by using PROC MIXED (SAS version 9.1.2; SAS Institute, Inc.).

**Results**

**Participant characteristics and compliance**

Characteristics of participants included in the analyses (n = 220; age: 48 ± 7.9 y; BMI: 30.0 ± 2.8 kg/m²) did not differ between the 4 groups at baseline (Supplemental Table 2). Of the 327 participants who started the study, 140 dropped out. Reasons included the following: too busy (n = 33), noncompliant with the supplements and/or exercise (n = 19), exercise-related injury (n = 1), medical issue (non–study related; n = 32), gaining weight/disliked supplements (n = 20), family issues/relocated (n = 25), cost of gas (n = 2), and no excuse provided (n = 8). Twenty-four-hour UUN output was greater in the 60-g/d group than in the 0-g/d group at the midpoint and end of the study, which is consistent with greater total protein intake (group-by-time, P < 0.05; Supplemental Table 3). During the first half of the study (baseline to wk 18), 89% of the participants consumed >50% of the supplements and 96% completed >80% of the exercise sessions (Supplemental Table 4). During the second half of the
study, fewer participants were in the highest compliance category (supplements: >80%; exercise: >90%), whereas the number of participants increased in the lower compliance categories (supplements: 79–50% and <50%; exercise: 89–70% and <70%).

**Whey protein supplementation**

Except for total protein intake, whey protein supplementation did not affect any of the physiologic, metabolic, nutritional intake, body composition, or appetite responses or indices of metabolic syndrome over time as described below (i.e., there were no significant group-by-time interactions).

**Physiologic and metabolic responses to training**

Among all participants, whole-body strength (1 repetition maximum) increased 15 ± 12% (66 ± 49 kg, P < 0.001) and VO₂max (maximal oxygen uptake capacity) increased by 9 ± 15% (3.0 ± 5.0 mL·kg⁻¹·min⁻¹, P < 0.001) from pre- to postintervention (Supplemental Table 3). There was a trend for an increase in resting energy expenditure over time (pre- vs. postintervention; 0.6 ± 2.0 MJ/d, P = 0.08), with 62% of the change occurring between baseline and wk 18. Carbohydrate oxidation was lower at wk 18 (0.15 ± 0.09 g/min, P < 0.01) and wk 36 (0.17 ± 0.17 g/min, P < 0.01) vs. baseline (0.18 ± 0.09 g/min). Fat oxidation was higher (P < 0.01) at wk 36 (0.06 ± 0.05 g/min) vs. baseline (0.05 ± 0.04 g/min). Protein oxidation increased (P < 0.01) from baseline (0.01 ± 0.01 g/min) to wk 18 (0.04 ± 0.03 g/min) but decreased (P < 0.01) from wk 18 to wk 36 (0.01 ± 0.01 g/min).

**Nutritional intakes**

**Non-supplement intakes.** Among all groups, non-supplement energy intake decreased from baseline to wk 18 (−1.8 ± 1.9 MJ/d, P < 0.001) and pre- to postintervention (−1.4 ± 2.2 MJ/d, P < 0.001); thus, the participants maintained body weight by compensating for the 1.7-MJ/d supplements (Fig. 1A, Supplemental Tables 5 and 6). The macronutrient distribution of non-supplement energy intakes did not largely differ from baseline macronutrient distributions (Fig. 1A). The percentage of energy contribution from protein increased (16 ± 3% vs. 17 ± 4%, P < 0.01) and from carbohydrate decreased (48 ± 8% vs. 46 ± 8%, P = 0.03) from pre- to postintervention. The percentage of energy contribution from fat did not change (35 ± 6% vs. 36 ± 6%, P = 0.47).

**Total energy and protein intakes (including supplements).**

Total energy intakes were not different between time points or protein groups (Fig. 1B and Supplemental Tables 5 and 6). Total protein intake (g/d and g·kg⁻¹·d⁻¹) decreased from pre- to postintervention in the 0-g/d group (P = 0.01) and increased (P < 0.001) from pre- to postintervention in the 20-, 40-, and 60-g/d groups (Fig. 2 and Supplemental Table 5). Over time, the percentage of energy from protein decreased in the 0-g/d group and increased in the 20-, 40-, and 60-g/d groups (P < 0.05). The relative contribution of total protein was lower in the 0-g/d group compared with the 20-, 40-, and 60-g/d groups (Supplemental Fig. 2).

**Body composition**

Among all participants, body weight from pre- to postintervention was unchanged (P = 0.80), whole-body lean mass increased by 1.9 ± 2.8% (P < 0.001), and fat mass decreased by −2.6 ± 9.4% (P < 0.01) (Fig. 3 and Supplemental Table 7). Appendicular lean mass increased (0.5 ± 0.8 kg, P < 0.01) and appendicular fat mass decreased (−0.3 ± 1.0 kg, P < 0.01) over time (pre- to postintervention) (Supplemental Table 7).

**Indices of metabolic syndrome**

Among all participants, waist circumference, waist-to-hip ratio, plasma lipid lipoprotein profile (total, LDL, and HDL cholesterol and TG), and blood pressure were unchanged (Supplemental Table 8). Glucose AUC was unchanged (−18.0 ± 170 mmol/L·3 h, P = 0.16), insulin AUC decreased by 2.6 ± 32% (−7.5 ± 29 mmol/L·3 h, P = 0.01), and HOMA-IR (0.2 ± 2.0, P = 0.81) and insulin sensitivity index (0.3 ± 3.0, P = 0.63) were unchanged (Supplemental Table 8). Change in insulin AUC, but not in glucose AUC, was associated with changes in body fat mass (r = 0.26, P < 0.001). Changes in lean body mass were associated with both changes in insulin (r = 0.16, P = 0.03) and glucose (r = 0.16, P = 0.03) AUC. CRP (−0.5 ± 6.0 mg/L, P = 0.14) and PAI-1 (−4.6 ± 47.0 μg/L, P = 0.29) were unchanged (Supplemental Table 8).

The number of participants that met the American Heart Association and National Heart, Lung, and Blood Institute criteria for metabolic syndrome was not affected by whey protein.
supplementation and did not change over time (baseline = 46%, wk 18 = 45%, and wk 36 = 47%).

Renal function
Among all participants, plasma albumin and creatinine and estimated glomerular filtration rate were within ranges of clinical normalcy (albumin: 32–52 g/L; creatinine: 53–114 μmol/L; glomerular filtration rate: >60 mL/min) at baseline and at wk 18 and wk 36.

Appetite
Desire to eat increased from mid- to postintervention (0.15 ± 1.1 AU, P = 0.02), and there was a trend for an increase in hunger from pre- to postintervention (0.15 ± 1.6 AU, P = 0.05). Fullness did not change (P = 0.25).

Secondary analysis of compliant participants
Overall, comparable results were achieved when only participants who achieved the highest compliance to both the supplementation and exercise sessions were included in the analyses (wk 0, n = 155; wk 18, n = 154; wk 36, n = 91). Similar to the original analyses, there was no effect of whey protein supplementation on any outcomes, except for UUN and dietary protein intakes. Findings for both nonsupplement and total nutritional intakes as well as body weight and composition were comparable to the primary analyses. However, a main effect of time for carbohydrate and fat oxidation was no longer present.

Discussion
This is the first study, to our knowledge, to examine the effect of different quantities of whey protein supplementation during 36 wk of exercise training on body composition and indices of metabolic syndrome in overweight and obese middle-aged adults. The primary findings were that increasing total protein intake through whey protein supplementation was not effective in enhancing exercise training–induced improvements in body composition and indices of metabolic syndrome. The participants compensated for the additional dietary energy from the supplements by reducing nonsupplement energy intakes, which led to weight maintenance. The increase in UUN with higher intakes of whey protein and increases in strength, aerobic capacity, and lean body mass support the success of this free-feeding, community-based intervention to increase total protein intakes and improve fitness and body composition over the long term.

The finding that exercise-induced increases in lean mass and decreases in fat mass were not altered with whey protein supplementation is consistent with other training studies in middle-aged adults (13,15), but contrasts with findings in younger adults (12,25). Training studies in young men showed an additional 2–6% increase in lean body mass with whey protein supplementation, but the participants consumed very high quantities of whey protein (>100 g/d for 6–10 wk) (12,25). The studies in middle-aged men supplemented with 26–35 g whey protein/d for 12–14 wk and found no additional benefit. Because intakes of whey protein >100 g/d can be difficult to maintain over the long term, the current study examined consumption of whey protein ranging from 0 to 60 g/d. The current study also extended the intervention period to 36 wk to evaluate changes over a longer term. The results suggest that neither whey protein amount nor extending the supplementation period to 36 wk influenced the exercise-induced improvements in body composition in overweight and obese middle-aged adults.

Previous studies in 50–80-y-old adults indicated no difference in lean body mass gains between adults who consumed 1.2–1.6 g protein · kg⁻¹ · d⁻¹ compared with those who consumed close to the recommended dietary allowance (41) for protein (0.8–0.9 g protein · kg⁻¹ · d⁻¹) during a 12-wk progressive-resistance exercise program (3,24). Total protein intakes in the current study were above the RDA (0.8 g protein · kg⁻¹ · d⁻¹), with intakes of 0.93, 1.13, 1.43, and 1.63 g · kg⁻¹ · d⁻¹ in the 0-, 20-, 40-, and 60-g/d groups, respectively. The lack of a lower protein group who consumed the RDA (0.8 g protein · kg⁻¹ · d⁻¹) for protein may account for the inability to detect a differential response.
between the 0-g/d group and the higher protein groups. Findings from some resistance-training studies indicated no change (42) or decreases in lean body mass (43) when older participants (>50 y) consumed 0.8 g protein·kg⁻¹·d⁻¹, and a regression analysis combining findings from 6 studies supported these findings (10). Collectively, our results support a growing body of literature that suggests that a higher protein intake in middle-aged and older weight-stable adults does not enhance resistance training–induced gains in lean body mass when adequate dietary protein is consumed.

To date, only one study has examined the effect of whey protein supplementation during exercise training on indices of metabolic syndrome. Denysschen et al. (13) supplemented overweight hyperlipidemic men with 26.6 g whey protein/d during 12 wk of resistance training (3 d/wk). After the intervention, total cholesterol decreased, and TG and LDL and HDL cholesterol remained unchanged independent of whey protein. In the absence of exercise training, consumption of 54 g whey protein/d for 12 wk resulted in reductions in fasting TG, total and LDL cholesterol, insulin, and HOMA scores compared with a control group in overweight and obese adults; however, body composition was not altered (44). In the current study, blood lipids and lipoproteins were unaltered, whereas blood pressure decreased. Furthermore, a decreased insulin response (45) to an oral-glucose challenge was seen independent of whey protein intake and was associated with reductions in fat mass. The current study extends the previous findings to include a titration of whey protein quantities spanning 0–60 g/d and a more comprehensive evaluation of metabolic syndrome indices in combination with resistance and aerobic exercise training. Collectively, these findings suggest that, in the longer term, whey protein supplementation does not enhance exercise training–induced improvements in indices of metabolic syndrome and that changes in insulin sensitivity are likely a result of the reductions in fat mass in overweight and obese middle-aged adults. Although speculative, the lack of lipid- and cholesterol-lowering effects of whey protein during exercise training, specifically with the higher doses (40 and 60 g/d), may be a result of the participants having clinically normal lipoprotein profiles.

Studies that use protein supplementation as a means to increase total protein intake need to document dietary compensation because it can alter the daily energy and macronutrient intakes of participants. Dietary compensation data during long-term supplementation and exercise training are sparse, especially in overweight and obese middle-aged adults. In the current study, participants successfully compensated for the energy in the supplements, and as a result nonsupplement intakes of protein, carbohydrate, and fat decreased. Interestingly, the decrease was to a similar extent in all groups (−6%, −18%, and −12% for protein, carbohydrate, and fat, respectively) and did not largely change nonsupplement macronutrient distribution pre- and postintervention (Supplemental Table 5). This shows that participants did not preferentially compensate with a specific macronutrient. During resistance training, shorter-term supplementation studies have generally reported complete compensation for the supplemental energy and macronutrients (e.g., no increase in total protein intake), leading to no change in body weight in younger and middle-aged adults (12,15) or a small increase in body weight (1.5 kg) that can be attributed to exercise-induced increases in lean body mass (2.3 kg) in young men (25). Middle-aged men were also shown to successfully compensate for the supplemental energy, but the resulting decrease in nonsupplemental protein was less than the protein content of the supplements, leading to increased total protein intakes (13). Our findings show that higher total protein intakes are achievable with supplementation as little as 20 g protein/d, and the impact of the supplements on dietary intakes is sustainable in the longer term.

The strengths of this study include using an exercise training program that effectively improved physiologic, metabolic, and anthropometric characteristics of the participants. The changes in fitness and body composition observed in this study are comparable to those in other community-based exercise training studies and include similar increases in aerobic capacity (7–19%) (46–48) and strength (8–39%) (49–51) and gains in lean body mass that were similar (50) or greater (51) than in other studies. Evidence from some (52–54), but not all (55), shorter-term studies (12 wk) suggests that consuming protein supplements immediately before or after resistance exercise sessions may be more effective in enhancing gains in lean body mass. Because the aim of the current study was to evaluate the effects of prolonged protein supplementation (9 mo) on gains in lean body mass, participants were instructed to consume the supplements with breakfast and lunch to maintain consistency with the timing of supplement consumption and to provide flexibility as to when they chose to perform the weekly exercise sessions. Future longer-term interventions should evaluate the effect of the timing of protein supplement intake on resistance exercise–induced lean body mass gains in middle-aged and older adults.

This study is limited in that it was not powered to statistically detect changes in indices of metabolic syndrome or sex differences. The original aim of the investigation was to evaluate the effect of whey protein supplementation during exercise training on changes in lean body mass. Therefore, power calculations were performed from available data on changes in lean body mass in men and women, and participants were recruited on the basis of BMI and not sex or the presence of metabolic syndrome. The lack of change in some of the indices of metabolic syndrome may be because <50% of the participants met the criteria for diagnosis of metabolic syndrome (34). Studies powered to evaluate the effect of whey protein and exercise training on sex-specific outcomes as well as on indices of metabolic syndrome are warranted. This study is also limited by the high dropout rate (43%). However, secondary analyses on compliant participants as well as analyses comparing completers vs. noncompleters (data not shown) showed no differences in any of the outcomes, suggesting that the dropout rate had a minor impact on the results.

In conclusion, whey protein supplementation that increased total protein intakes to as high as twice the RDA during exercise training did not enhance exercise-induced responses in strength, aerobic fitness, or body composition or indices of metabolic syndrome in weight-stable overweight and obese middle-aged adults. Although results from this study must be interpreted with caution due to the high dropout rate, this study supports the success of a community-based aerobic and resistance exercise training intervention to positively influence fitness and body composition and promote metabolic health.

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editorial input to finalize the manuscript. All authors read and approved the final manuscript.

**Literature Cited**


