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ORIGINAL ARTICLE

Effect of resistance training on resting metabolic rate and its estimation by a dual-energy X-ray absorptiometry metabolic map

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BACKGROUND/OBJECTIVES: Fat-free mass (FFM) is the major predictor of resting metabolic rate (RMR). As protein supplementation during resistance training may augment gains in FFM, we investigated the effects of resistance training combined with protein supplementation on RMR and whether RMR responses could be estimated by a dual-energy X-ray absorptiometry (DXA) metabolic map.

SUBJECTS/METHODS: Healthy adults completed a whole-body periodized resistance training program consisting of 96 workouts (~9 months). Participants were randomly assigned to supplement with whey protein (whey; $n = 18$), soy protein (soy; $n = 21$) or carbohydrate (carb; $n = 22$). RMR was measured using indirect calorimetry (RMR_{IC}) and estimated by DXA metabolic mapping (RMR_{MM}) pretraining and posttraining.

RESULTS: RMR_{IC} increased from pretraining to posttraining in the whole cohort (1653 ± 302 to 1726 ± 291 kcal/day, $P = 0.001$) without differences between the groups. Delta RMR_{IC} and RMR_{MM} (73 ± 158 vs 52 ± 41 kcal/day were not significantly different by *t*-test ($P = 0.303$), although they were not significantly correlated ($r = 0.081$; $P = 0.535$). Stepwise regression identified 43% of the shared variance in delta RMR_{IC} using total serum thyroxine, RMR_{IC} and FFM at baseline ($P = 0.009$).

CONCLUSIONS: These results indicate that 9 months of resistance training significantly increased RMR ~ 5% on average, but there was wide variability between individuals, which can be partially accounted for by changes in FFM and thyroid hormones.

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INTRODUCTION

Health agencies recommend resistance training as part of an adult fitness program to maintain overall good health and physical independence.[1,2] However, the effects of resistance training on resting metabolic rate (RMR) are less clear, with studies showing increases,[3,4] decreases[5] and no changes.[6,7] Resistance training has the potential to increase RMR and daily energy expenditure having a positive impact on weight management.[3,8] The principal mechanism by which resistance training seems to increase RMR is by augmenting the fat-free mass (FFM).[9,10] As protein supplementation during resistance training stimulates additional gains in FFM,[11,12] we investigated the effects of resistance training with protein supplementation on RMR and whether RMR responses could be predicted by the major metabolic components of the FFM as determined by dual-energy X-ray absorptiometry (DXA) metabolic mapping.

FFM and thyroid hormones have a profound effect on RMR.[13,14] Resistance training increases FFM, which is the major determinant (50–75%) of the resting metabolism.[15,16] Although it is clear that thyroid hormones increase RMR, the exact mechanism remains unclear. Uncoupling of ATP synthesis and changes in the efficiency of futile cycles are among the mechanisms proposed.[17,18] Particularly, at the skeletal muscle, thyroid hormones may increase RMR by uncoupling the ATP hydrolysis from the sarcoplasmic calcium cycling during contraction and rest; increasing the expression of the Na-K-ATPase and various myosin heavy-chain isoforms; and altering the glycogen–glycogenolysis cycle.[17,18]

FFM is a heterogeneous compartment that represents the sum of high and low metabolically active tissues and organs.[19,20] Skeletal muscle has a low-metabolic rate (13 kcal/kg/day) compared with the brain and visceral organs (200–440 kcal/kg/day).[21] Thus when FFM increases due to elevations in the skeletal muscle, a reduction in the metabolic rate per kilogram of FFM may occur,[19,20] which makes interpretation of RMR responses to resistance exercise problematic.[22,23] The use of metabolic mapping may overcome this difficulty.[24,25] In contrast to indirect calorimetry, which provides an estimation of the energy consumed by the body as a whole, a metabolic map estimates the masses and energy consumed by the more active tissues and organs of the body to predict the RMR.[24,25] Thus metabolic mapping may be useful to compare the changes in masses and metabolic activities of the organs and tissues in response to exercise interventions.[24,25] Metabolic mapping has been shown to predict RMR in cross-sectional studies ($R^2 = 0.67$–0.82)[26–28] but, to our knowledge, has not been tested to estimate RMR changes in response to a resistance training program.

Protein supplementation during a resistance training program produces additional increases in FFM,[29–31] but some controversy exists regarding the most effective source of dietary protein to stimulate FFM gains. We recently showed that supplementing daily with whey protein supplementation was more effective than

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soy or carbohydrate in augmenting gains in FFM in response to resistance training.[32] Here we examine the effects of resistance training and supplementation on RMR in this same cohort and examine, for the first time, whether RMR responses can be predicted by the major metabolic components of the FFM using DXA metabolic mapping.

MATERIALS AND METHODS

Experimental approach

Details of the experimental approach and body composition results have previously been reported.[32] A prospective parallel three-group study design was used to evaluate the effects of resistance training combined with protein supplementation on RMR. Healthy adults were randomly assigned in a double-blind manner to supplement daily with whey protein (whey), soy protein (soy) or carbohydrate (carb). All participants performed a resistance training program of 96 workouts (~9 months).

Participants

Recreationally active men and women aged 18–35 years who had not resistance trained for a minimum of 1 year prior to the study were recruited. Exclusion criteria were health conditions that restricted the practice of resistance training, hypertension, diabetes, a change in body weight >3 kg within the past 3 months, use of cholesterol-lowering and blood pressure medications, use of anti-inflammatory medications (aspirin, non-steroidal anti-inflammatory drugs), use of tobacco products, alcohol consumption >3 drinks/day or >21/week, pregnancy or the intent to become pregnant during the study and allergy to whey or soy protein. This study was approved by the University of Connecticut-Storrs Institutional Review Board, and all participants signed a written informed consent.

Resistance training program

All participants performed a whole-body, progressive, non-linear, periodized resistance training program 3 days/week until subjects accrued 96 workouts (~9 months). The program was divided into three 12-week mesocycles and included heavy (3–6 repetitions, long rest periods of 2–3 min, high intensity); medium (8–10 repetitions, rest periods of 1–2 min, moderate intensity), light (12–15 repetitions, short rest period of 60–90 s, light intensity) and power (whole-body exercises, 30–45% of the estimated 1 repetition maximum, 3-min rest period) days. Exercises consisted of squats, hang cleans, bench press, biceps curls, calf exercises, abdominal exercises, lat pull downs, lunges, upright rows, push presses and weight plate lifts. Multiple sets (3–5) of each exercise were performed.

Supplements

The supplements were isocaloric and isonitrogenous (whey and soy). Nutrient composition was for whey: 21.6 g of whey protein concentrate plus 22.5 g of maltodextrin; for soy: 20.0 g of soy isolate (isoflavone free) plus 24.5 g of maltodextrin, and for carb: 45.2 g of maltodextrin. Participants consumed the supplement every day, with breakfast on non-training days and immediately after exercise on training days. For supplement compliance, 200 mg of para-aminobenzoic acid was added to the supplements and subsequently measured from urine samples collected once per month on random days. Para-aminobenzoic acid was measured using the DACA (P-dimethylamino cinnamaldehyde) method as described by Yamato *et al.*[33] The supplement compliance was 82%.

Diet counseling

Participants followed a diet meeting their individual energy requirements with a protein intake of 1.0–1.2 g/kg body mass (not including supplementation). Energy requirements were estimated by multiplying RMR times an activity factor according to the subjects' physical activity level. Participants received diet counseling every 6 weeks or more frequently when needed. Prior to the diet counseling, the participants filled the 5-day diet records that were reviewed for monitoring energy and protein intake. To improve the quality of the diet information, volunteers were provided with a Personal Digital Assistant, a food scale and a booklet with portion sizes. The Personal Digital Assistant that was loaded with a dietary program Weightmania (Edward A. Greenwood, Inc., Brookline, MA, USA) facilitated tracking participants' energy and protein intake.

Anthropometrics, body composition and DXA metabolic mapping

Body weight was measured to the nearest 0.1 kg on a calibrated digital scale (Defender 5000, Ohaus, Florham Park, NJ, USA). Height was measured to the nearest 0.1 cm at baseline using a portable stadiometer (Seca, Hamburg, Germany). Body composition was measured using DXA (Lunar Prodigy, Madison, WI, USA) at baseline and 9 months. FFM and fat mass were calculated using the commercial software (enCore version 10.1 (GE Healthcare, Madison, WI, USA)). A DXA metabolic map was built by estimating the masses of brain, bone, trunk organs (that is, heart, liver kidneys and spleen), skeletal muscle, fat and residual mass from DXA measurements, as described by Bosy-Westphal *et al.*[25] The masses of organs and tissues were multiplied by their specific metabolic rates described by Wang *et al.*[21] and added to obtain the whole-body RMR.[24,25]

RMR and macronutrient oxidation

Participants were asked to abstain from food and beverages for a minimum of 12 h, alcohol and caffeine for 24 h and physical activity for 48 h before the test. They collected 24-h urine prior to the testing day and slept between 6 and 8 h the previous evening. On the testing day, participants were transported from their home to the laboratory. The test was conducted using a metabolic cart (TrueOne 2400, Parvomedics Inc., Sandy, UT, USA). After the 30-min rest period, participants were asked not to fidget, talk or sleep during the test. Continuous measures of CO_2 and oxygen (O_2) gasses were averaged and recorded every 30 s during 30 min of testing. CO_2 and O_2 values were used to calculate the 24-h RMR using the Weir equation.[34] Macronutrient oxidation was calculated using the 24-h urinary urea excretion and O_2 and CO_2 values with the Jequier equation.[35]

Blood collection

Blood was drawn at baseline, 3, 6 and 9 months. A 12-h fasting blood sample was performed at the same time (0430–0830 hours) and was obtained from an antecubital vein into serum tubes. After coagulation, blood was centrifuged at 1500 *g* for 15 min at 4 °C. Serum was aliquoted, snap frozen in liquid nitrogen and stored at − 80 °C for further analysis.

Biochemistry

Total triiodothyronine and total thyroxine were measured using an enzyme-linked immunosorbent assay (Calbiotech, Spring Valley, CA, USA). Urinary urea concentrations, in the 24-h urine collection, were measured by enzymatic methods using commercial reagents and standards (Pointe Scientific, Canton, MI, USA). Analytes were analyzed using a Versamax tunable microplate reader (Molecular Devices, Sunnyvale, CA, USA) at its appropriate wavelength.

Statistical analysis

One-way analysis of variance was used to compare baseline variables among groups and changes over specific time intervals. Two-way repeated measures analysis of variance was conducted to compare the effects of supplement (whey, soy, carb) and time (baseline, 3, 6 and 9 months). Bonferroni corrections were used for multiple comparisons. Paired *t*-test and Pearson's correlation coefficient was used to compare measured RMR and predicted RMR by metabolic mapping. Stepwise linear regression was used to identify the best predictors of the RMR change. Differences with a $P \leq 0.05$ were considered significant. Data are presented as means ± s.d. if not otherwise indicated.

RESULTS

Sixty-one participants completed the study: whey; $n = 18$ (6F/12M), soy; $n = 21$ (11F/10M) and carb; $n = 22$ (9F/13M). There were no significant differences between groups in physical characteristics at baseline (Table 1). The dietary energy intake remained constant during the study and was similar between groups, on average 28.2, 29.0 and 28.1 kcal/kg/day for whey, soy and carb, respectively. During the study, protein intake was higher in whey and soy than carb (1.3–1.4 vs 1.0–1.1 g/kg body mass, $P < 0.01$) due to protein supplementation. There were no significant differences between groups in fat or carbohydrate intake during the study. The percentage of macronutrient energy distribution in the

Table 1. Participant characteristics

Physical characteristics	*All* (n = *61*)	*Whey* (n = *18*)	*Soy* (n = *21*)	*Carb* (n = *22*)	*ANOVA P-value*
Age (years)	23.0 ± 3.3	22.8 ± 3.7	24.0 ± 2.9	22.3 ± 3.1	0.459
Height (cm)	171.2 ± 8.9	171.8 ± 10.3	170.5 ± 2.9	172.0 ± 8.7	0.829
Body weight (kg)	72.8 ± 15.7	74.1 ± 15.7	72.0 ± 8.4	72.4 ± 14.9	0.880
BMI (kg/m^2)	24.6 ± 4.0	25.2 ± 3.8	24.5 ± 4.2	24.3 ± 3.9	0.777
FFM (kg)	49.5 ± 9.9	51.7 ± 10.7	48.5 ± 10.0	49.8 ± 9.8	0.608
FM (kg)	20.2 ± 10.3	19.4 ± 11.3	20.5 ± 11.3	19.5 ± 9.0	0.893

Abbreviations: ANOVA, one-way analysis of variance; BMI, body mass index; Carb, carbohydrate; FFM, fat-free mass; FM, fat mass. Values are mean ± s.d.

groups oscillated between 14% and 19% for protein, 52–59% for carbohydrates and 25–30% for fat.

RMR and macronutrient oxidation

RMR significantly increased in the whole cohort (73 ± 158 kcal/day, $P < 0.01$) without differences between the groups (Figure 1). When RMR was normalized for FFM, no significant changes were observed (−0.10 ± 3.4 kcal/day, $P > 0.05$). There were no differences in RMR per kilogram of FFM between the groups (Table 2). Protein oxidation increased in the whole cohort (0.17 ± 57 g/kg/day, $P < 0.05$) without differences between the groups. There were no significant changes in carbohydrate and fat oxidation (Table 2).

DXA metabolic mapping

Estimated metabolic map components and their resting energy expenditure are shown in Table 3. Compared with baseline, there was an increase at 9 months in the skeletal muscle (1.73 ± 0.9 kg, $P < 0.001$), trunk organs (0.1 ± 0.1 kg, $P < 0.05$) and residual mass (0.5 ± 1.6 kg, $P < 0.05$). The estimated energy expenditure of the skeletal muscle (22.8 ± 12.2 kcal/day) and trunk organs (23.1 ± 29.8 kcal/day) also increased ($P < 0.05$) at the end of the study (Table 3). There were differences ($P < 0.001$) between measured and estimated RMR at baseline (1653 ± 302 vs 1548 ± 257 kcal/day) and 9 months (1726 ± 292 vs 1599 ± 268 kcal/day). Yet, the measured and estimated RMR changes were similar (73 ± 158 vs 52 ± 41 kcal/day, $P > 0.05$, respectively); however, they were not correlated ($r = 0.081$; $P > 0.05$).

Body mass and body composition

Compared with baseline, body mass increased at 9 months (2.3 ± 3.0 kg, $P < 0.001$) without differences between the groups (Table 4). The increase in FFM with whey (3.3 ± 1.5 kg) was higher ($P = 0.01$) than soy (1.8 ± 1.6 kg) and carb (2.3 ± 1.7 kg) (Table 4). There were no significant changes in fat mass in the whole cohort or any group.

Thyroid hormones

Total triiodothyronine concentrations decreased at 6 months (0.35 ± 0.77 nmol/l, $P < 0.001$) and returned to baseline at 9 months. There were no significant differences in triiodothyronine between the groups (Table 5). Total thyroxine concentrations decreased at 3 months (15.2 ± 18.0 nmol/l, $P < 0.001$) and returned to baseline at 6 months, with no significant changes at 9 months. There were no significant differences in thyroxine between the groups (Table 5).

The RMR correlated with FFM at baseline ($r = 0.760$, $P < 0.01$) and at 9 months ($r = 0.792$, $P < 0.01$). The RMR increase correlated with thyroxine at baseline ($r = 0.504$, $P < 0.01$), 9 months ($r = 0.271$, $P < 0.05$) and with the change in thyroxine from baseline to 9 months ($r = -0.358$, $P < 0.01$). The stepwise regression analysis included the baseline levels of thyroxine, RMR and FFM in the best model predicting RMR change (43.0%, $P < 0.01$). Thyroxine predicted 25.4% ($P < 0.001$) of the variation, RMR predicted 9.0% ($P < 0.01$) and FFM predicted 8.3% ($P < 0.01$).

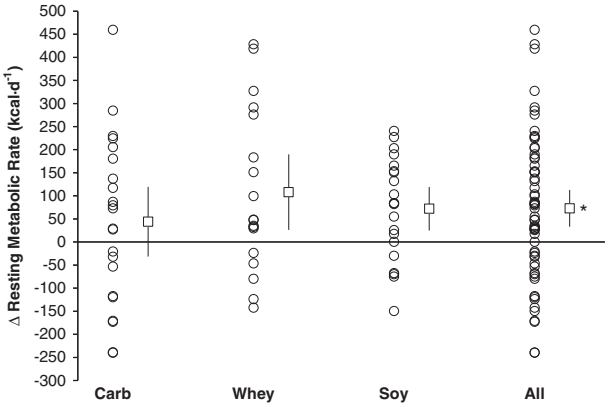

Figure 1. Individual changes in resting metabolic rate with 9 months of resistance training in subjects supplemented with carbohydrate ($n = 22$), whey protein ($n = 18$) or soy protein ($n = 21$).

DISCUSSION

The primary aim of the study was to evaluate the effects of a long-term resistance training program combined with protein supplementation on RMR and whether the RMR response to exercise could be estimated by DXA metabolic mapping and thyroid hormones. In response to resistance training, RMR significantly increased by 5% in the whole cohort (73 ± 158 kcal/day, $P < 0.01$), but the supplement groups were not significantly different. Metabolic mapping from DXA failed to predict RMR at baseline, 9 months and the change in RMR, but inclusion of thyroid hormones improved the prediction such that 43% of the shared variance was accounted for in delta RMR.

Similar to previous studies, we found that the increase in RMR with resistance training is due, at least in part, to augmentation of FFM.[3,36,37] This hypothesis is supported by the fact that the differences in RMR disappeared after normalizing the values per kilogram of FFM and by the correlations between FFM and RMR at baseline ($r = 0.760$, $P < 0.01$) and 9 months ($r = 0.792$, $P < 0.01$). If the increase in RMR was solely due to an augment in the skeletal muscle mass, the estimated RMR increase would be 31.2 kcal/day (13 kcal/kg/day × 2.4 kg FFM), less than half of the observed change (73 kcal/day). Thus an increase in mass of other metabolically active tissues may account for the unexplained variance.

Results from DXA metabolic mapping show significant increases in the skeletal muscle, trunk organs and residual mass (Table 3). Although metabolic mapping identified changes in the FFM components, it failed to predict the RMR change and the RMR at baseline and 9 months. These results are contrary to previous studies[26–28] and could be due to an underestimation of the brain and trunk organs' masses in our subjects. This is suggested by the

Table 2. Change in resting metabolic rate and macronutrients oxidation

Variable	*Group*	*Baseline*	*9 Months*	*Change*	*ANOVA*
RMR (kcal/day)	Whey	1715 ± 318	1823 ± 312	108 ± 177	T: $P < 0.05$
	Soy	1579 ± 240	1652 ± 216	72 ± 110	G: $P > 0.05$
	Carb	1673 ± 339	1717 ± 326	44 ± 181	G × T: $P > 0.05$
	All	1653 ± 302	1726 ± 291*	73 ± 158	
RMR (kcal/kg FFM/day)	Whey	34.0 ± 4.1	33.9 ± 3.0	− 0.08 ± 3.4	T: $P > 0.05$
	Soy	33.5 ± 4.4	34.0 ± 6.0	0.55 ± 3.0	G: $P > 0.05$
	Carb	33.9 ± 5.0	33.2 ± 3.8	− 0.65 ± 3.9	G × T: $P > 0.05$
	All	33.8 ± 4.5	33.7 ± 4.5	− 0.10 ± 3.4	
Protein oxidation (g/kg/day)	Whey	1.05 ± 0.37	1.25 ± 0.40	0.203 ± 0.46	T: $P < 0.05$
	Soy	0.99 ± 0.45	1.26 ± 0.71	0.275 ± 0.57	G: $P > 0.05$
	Carb	1.02 ± 0.39	1.06 ± 0.52	0.034 ± 0.54	G × T: $P > 0.05$
	All	1.02 ± 0.40	1.19 ± 0.57*	0.168 ± 0.57	
Carbohydrate oxidation (g/kg/day)	Whey	2.16 ± 0.82	2.30 ± 1.1	0.139 ± 1.41	T: $P > 0.05$
	Soy	2.36 ± 0.87	2.36 ± 1.3	0.000 ± 1.44	G: $P > 0.05$
	Carb	2.40 ± 1.0	2.37 ± 1.0	− 0.027 ± 1.28	G × T: $P > 0.05$
	All	2.32 ± 0.93	2.35 ± 1.13	0.033 ± 1.3	
Fat oxidation (g/kg/day)	Whey	1.15 ± 0.50	1.04 ± 0.46	− 0.109 ± 0.58	T: $P > 0.05$
	Soy	0.99 ± 0.40	0.91 ± 0.72	− 0.083 ± 0.68	G: $P > 0.05$
	Carb	1.02 ± 0.43	1.02 ± 0.40	0.006 ± 0.68	G × T: $P > 0.05$
	All	1.05 ± 0.44	0.99 ± 0.54	− 0.059 ± 0.64	

Abbreviations: ANOVA, analysis of variance; Carb, carbohydrate; FFM, free-fat mass; G, main group effect; G × T, Group × Time effect; RMR, resting metabolic rate; T, main time effect. *$P < 0.05$ from the corresponding baseline value. Values are mean ± s.d.

Table 3. Estimated metabolic map components and their resting metabolic rate

	Metabolic map components (kg)			*Resting metabolic rate (kcal/day)*		
	Baseline	*9 Months*	*Change*	*Baseline*	*9 Months*	*Change*
Sk. muscle	27.19 ± 6.51	28.92 ± 6.9‡	1.73 ± 0.9	357 ± 85.7	380 ± 91.0‡	22.8 ± 12.2
Adipose	23.85 ± 12.1	23.76 ± 12.7	− 0.09 ± 3.8	108.3 ± 55.2	107.8 ± 57.8	− 0.4 ± 17.4
Bone	5.54 ± 1.0	5.61 ± 1.1	0.07 ± 0.2	12.7 ± 2.3	12.9 ± 2.4	0.2 ± 0.6
Brain	1.49 ± 0.2	1.50 ± 0.2	0.01 ± 0.02	359.7 ± 39.2	361.5 ± 38.9	1.85 ± 6.7
Trk-organs	2.21 ± 0.4	2.30 ± 0.4*	0.1 ± 0.1	619.7 ± 114.0	642.9 ± 120.1‡	23.1 ± 29.8
Residual	12.45 ± 3.4	13.0 ± 3.9*	0.5 ± 1.6	89.3 ± 24.5	93.3 ± 27.7	4.0 ± 11.8
Total	72.74 ± 15.7	75.1 ± 16.1‡	2.4 ± 3.0	1548 ± 257#	1599 ± 268*,#	51.5 ± 41.0
Mes. RMR	—	—	—	1653 ± 302	1726 ± 292*	73.0 ± 158

Abbreviations: Mes. RMR, measured resting metabolic rate; Sk. muscle, skeletal muscle; Trk-organs, trunk organs (that is, heart, liver, kidneys and spleen). *$P < 0.05$ from the corresponding baseline value. ‡$P < 0.001$ from the corresponding baseline value. #$P < 0.001$ from the corresponding measured RMR value. Values are mean ± s.d.

Table 4. Body mass and body composition by group and time

Variable	*Group*	*Baseline*	*9 Months*	*Change*	*ANOVA*
Body mass (kg)	Whey	74.1 ± 15.7	77.2 ± 16.7*	3.1 ± 3.0	T: $P < 0.05$
	Soy	72.0 ± 8.4	74.2 ± 17.0*	2.2 ± 4.0	G: $P > 0.05$
	Carb	72.4 ± 14.9	74.2 ± 14.8*	1.8 ± 2.4	G × T: $P > 0.05$
	All	72.8 ± 15.6	75.1 ± 15.9*	2.3 ± 3.0	
Fat-free mass (kg)	Whey	51.7 ± 10.7	55.0 ± 11.1*	3.3 ± 1.5#	T: $P < 0.05$
	Soy	48.5 ± 10.0	50.3 ± 10.7*	1.8 ± 1.6	G: $P < 0.05$
	Carb	49.8 ± 9.8	52.1 ± 10.3*	2.3 ± 1.7	G × T: $P < 0.05$
	All	50.0 ± 10.0	52.4 ± 10.7*	2.4 ± 1.7	
Fat mass (kg)	Whey	19.4 ± 11.3	18.8 ± 11.4	− 0.6 ± 2.7	T: $P > 0.05$
	Soy	20.5 ± 11.3	20.7 ± 12.5	0.2 ± 4.1	G: $P > 0.05$
	Carb	19.5 ± 9.0	19.0 ± 8.6	− 0.5 ± 2.2	G × T: $P > 0.05$
	All	19.8 ± 10.4	19.5 ± 10.9	− 0.3 ± 3.2	

Abbreviations: ANOVA, analysis of variance; Carb, carbohydrate; G, main group effect; G × T, Group × Time effect; T, main time effect. *$P < 0.001$ from the corresponding baseline value. #$P < 0.01$ from the corresponding Carb and Soy value. Differences in changes in fat-free mass are described in the text. Values are mean ± s.d.

Table 5. Hormone concentrations by group and time

Variable	*Time*	*Baseline*	*3 Months*	*6 Months*	*9 Months*	*ANOVA*
TT3 (nmol/l)	Whey	2.18 ± 1.05	2.30 ± 1.09	1.76 ± 0.73	1.93 ± 0.84	T: $P < 0.05$
	Soy	2.07 ± 0.80	1.74 ± 0.56	1.69 ± 0.42	2.04 ± 0.89	G: $P > 0.05$
	Carb	1.91 ± 0.44	1.89 ± 0.50	1.66 ± 0.65	1.72 ± 0.55	G × T: $P > 0.05$
	All	2.05 ± 0.78	1.97 ± 0.76	1.70 ± 0.60*	1.90 ± 0.77	
TT4 (nmol/l)	Whey	148.5 ± 49.9	130.9 ± 42.7	153.1 ± 57.2	152.0 ± 48.7	T: $P < 0.05$
	Soy	126.8 ± 30.9	118.5 ± 35.1	132.7 ± 35.2	129.9 ± 35.5	G: $P > 0.05$
	Carb	130.2 ± 41.7	110.4 ± 40.8	146.5 ± 45.4	142.5 ± 38.9	G × T: $P > 0.05$
	All	134.5 ± 41.5	119.2 ± 39.7*	143.7 ± 46.2	141.0 ± 41.3	

Abbreviations: ANOVA, analysis of variance; Carb, carbohydrate; G, main group effect; G × T, Group × Time effect; T, main time effect. *$P < 0.001$ from the corresponding baseline value. Values are mean ± s.d.

higher RMR per kg of FFM in our group compared with previous studies (~33.7 and ~28, respectively) given the constancy of the metabolic rates of organ and tissues in healthy adults.[21,38]

The increased RMR was best predicted using a model that included thyroxine, RMR and FFM at baseline ($R^2 = 0.43$, $P < 0.01$). Higher initial values of thyroxine and FFM predict larger increases in RMR in response to resistance training, which is consistent with the known actions of thyroid hormone on the skeletal muscle. A study by Clement *et al.*[39] evaluated the effects of supplementing with 75 μg/day of triiodothyronine during 14 days in healthy volunteers. They reported an increase of 13% in RMR and upregulation in the vastus lateralis muscle of 381 genes related to energy expenditure, protein turnover, signal transduction and protein trafficking.[39]

Protein supplementation and an accretion in brown adipose tissue activity in response to exercise training could have contributed to the increase in RMR. Protein supplementation may have contributed by: (a) increasing protein oxidation, observed in the whole group, as production of 1 mol of urea uses four ATPs,[40] (b) increasing protein turnover, which might account as much as 20% of resting metabolism,[41] and (c) increasing the food thermogenic effect, as the effects of protein is around 25–40% of the energy intake compared with carbohydrate, which is about 6–8%.[40,42] Similarly, the presence of brown adipose has been confirmed in adult humans, and some evidence suggests that it can be activated by exercise.[43,44] Recently, it was demonstrated that Irisin, a hormone released from the skeletal muscle during contraction, stimulates brown adipose tissue and energy expenditure in mice, and a similar process may occur in humans.[45,46]

The increase in RMR with whey was more than double that with carb supplementation; however, there were no statistical differences between the groups. The lack of differences may be due to a high individual variability in the RMR response to exercise (see Figure 1), which has been reported.[47,48] The variability in RMR has a significant genetic component as was demonstrated by Poehlman *et al.*[48] They studied the RMR changes to an endurance program of 22 days, where twins cycled ~116 min/day at 58% of maximal oxygen uptake. They found that the variability in RMR response between phenotypes was more than twice the variability among genotypes.[48] This highlights the role that genetic background has in the RMR response and the fact that people may differ in the exercise intensity or work volume necessary to impact the RMR.[3]

The fact that the RMR increase was detected >48 h after the last exercise bout suggests that this is a chronic effect. This is encouraging given that resistance exercise is recommended by health agencies to be performed every other day.[3] The effects of RT on RMR (increased by 73 kcal/day) and body composition (2.4 kg FFM increase and 0.3 kg loss of fat mass) were moderate and probably not of sufficient magnitude to justify its sole use as a treatment strategy to manage obesity, but it may be an excellent strategy to prevent obesity in college students, a population at high risk.[49,50]

In conclusion, a 9-month resistance training program significantly increased RMR on average by 5%; the addition of protein supplementation did not produce additional increases in RMR. DXA metabolic mapping failed to predict RMR at baseline and at 9 months and was not associated with the RMR change. Just under half the variability in RMR response to resistance training was accounted for by FFM and thyroxine, suggesting that other factors contributed to the metabolic response.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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