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Nutritional effect of oral supplement enriched in beta-hydroxy-beta-methylbutyrate, glutamine and arginine on resting metabolic rate after laparoscopic gastric bypass

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Abstract

Background—Weight regain that begins 12–18 months after laparoscopic gastric bypass has been attributed to changes in resting metabolic rate (RMR), which is largely determined by lean body mass (LBM). An oral supplement containing beta-hydroxy-beta-methylbutyrate, glutamine, and arginine (HMB/Glu/Arg) has helped to restore LBM in cachexia due to cancer and in critically ill trauma patients. The objective of this study was to evaluate the effect of oral HMB/Glu/Arg on LBM and RMR following laparoscopic gastric bypass (LGB).

Methods—Patients who underwent LGB were randomized to receive 24 g of HMB/Glu/Arg dissolved in water twice daily for 8 weeks or to receive no supplement. Weight loss, LBM, and RMR were assessed preoperatively, 2 and 8 weeks postoperatively. LBM was determined by dual emission x-ray absorptiometry (DXA) and RMR was measured by indirect calorimetry.

Results—Thirty patients were enrolled: 80% white; 20% African American; 96.7% women; mean age 46.9 ± 8.4 years; mean weight 113.4 ± 11.6 kg; and mean body mass index (BMI) 43.3 ± 4.1 kg/m². The experimental and control groups included 14 and 16 patients, respectively, and there was no difference in baseline demographics and characteristics between the two groups. At 8

weeks, weight, BMI, LBM, and RMR significantly decreased by 15.7 ± 2.5 kg, 6.0 ± 1.0 kg/m², 7.8 ± 4.0 kg, and 290.6 ± 234.9 kcal/day, respectively ($P < 0.0001$ for each variable). However, when comparing these changes between the two groups, no statistical significance was observed.

Conclusions—There is a significant decrease in weight, BMI, LBM, and RMR in all subjects after LGB, and these changes were not affected by the use of HMB/Glu/Arg. Potential preservation of LBM as a result of HMB/Glu/Arg requires further investigation. However, its consumption (78 calories per serving) did not adversely affect weight loss in the experimental group.

Keywords

Resting metabolic rate; Laparoscopic gastric bypass; Lean body mass; Beta-hydroxy-beta-methylbutyrate; Glutamine; Arginine

It is well recognized that obesity is associated with increased morbidity and mortality [1]. During the past 20 years, there has been a dramatic increase in the prevalence of obesity in the United States [2]. According to the 2005–2006 National Health and Nutrition Examination Survey (NHANES), it is estimated that 32.7% of adults in the United States are overweight (BMI ≥ 25 kg/m²), 34.3% are obese (BMI ≥ 30 kg/m²), and 5.9% are extremely or morbidly obese (BMI ≥ 40 kg/m²) [3]. In addition, a survey conducted by The Behavioral Risk Factor Surveillance System indicates that between 2000 and 2005, the prevalence of severe obesity increased at a much faster rate than that of moderate obesity [4]. It is often noticed that severely obese individuals have a long history of obesity and repeated failures with conventional methods of weight loss, such as diet and lifestyle modifications [5]. Due to the trend of increasing obesity, lack of success with traditional approaches and severity of comorbidities associated with severe obesity, bariatric surgery has become a popular and effective treatment option for achieving sustained weight loss and resolution of comorbidities, such as type 2 diabetes, hypertension, hyperlipidemia, osteoarthritis, and sleep apnea [6–9].

Laparoscopic gastric bypass (LGB) is an effective method of inducing significant weight loss and improving metabolic parameters in severely obese subjects [10]. A recent meta-analysis indicated that patients who undergo LGB lose 66–75% excess weight and 35–38% of their initial weight in the first year after surgery [11]. This rapid weight loss diminishes during the next several months and eventually stops. The exact mechanism by which weight loss ceases is not clearly understood. It has been established that in addition to fat mass, lean body mass (LBM) also decreases after gastric bypass. This loss in muscle mass contributes to a reduction in resting metabolic rate (RMR), which slows the body's use of calories and results in weight regain in bariatric patients [12–16]. This gradual weight regain may be associated with reappearance of comorbidities [17]. If the loss of LBM and resultant effect on RMR can be modulated, better weight maintenance and long-term improvement of health outcomes may be possible.

An oral supplement containing beta-hydroxy-beta-methylbutyrate, glutamine, and arginine (HMB/Glu/Arg) has been shown to restore muscle mass in cachexia due to cancer, rheumatoid arthritis, AIDS, and in critically ill trauma patients [18–22]. Although each component of the supplement has been shown to slow muscle proteolysis, beta-hydroxy-beta-methylbutyrate, a leucine metabolite, is the most active ingredient in the mixture and has been shown to improve nitrogen balance in critically ill subjects [22]. Arginine also promotes wound healing [23], and glutamine is a regulator of muscle turnover [24, 25]. However, their synergistic impact on preserving LBM or reversing loss of LBM after LGB has not been investigated. The objective of this study was to evaluate the impact of HMB/Glu/Arg on LBM and RMR in the short-term after LGB.

Materials and methods

Study design

This was an unblinded, randomized control pilot study in which 30 morbidly obese patients (as defined by NIH criteria) who underwent LGB were consented and enrolled in this institutional review board-approved protocol. Exclusion criteria included age younger than 18 years, pregnancy, weight greater than the limit of the dual emission x-ray absorptiometry (DXA) table (300 lb), or known allergy to any component of the supplement. Postoperatively, all patients were instructed to take an adult strength, chewable multivitamin with iron and 1,200 mg of elemental calcium citrate daily for postoperative supplementation. Each patient received a monetary incentive in a graduated manner to participate and complete the protocol.

Patients in the experimental group ($n = 14$) were provided with a supply of a commercially available oral supplement containing HMB/Glu/Arg at no cost. The patients were instructed to consume 24 g (1 packet) of the mixture dissolved in 8–10 ounces of water twice daily and record their consumption on a log sheet. Each packet of the supplement contains 1.5 g of calcium beta-hydroxy-beta-methylbutyrate (which provides 1.2 g of HMB), 7 g of glutamine, 7 g of arginine, 2 g of sugar, 7.8 g of carbohydrates, and 78 calories. Primary outcome measures of interest included body weight, LBM, total fat mass, and RMR. These were assessed preoperatively, 2 weeks postoperatively, and 8 weeks postoperatively.

Dual-emission x-ray absorptiometry

Regional and whole body lean and fat tissue was determined by dual-energy x-ray absorptiometry (LUNAR Prodigy, GE Medical Systems LUNAR, Madison, WI). The scans were analyzed using the enCORE 2002 software, Version 6.10.029 (GE Medical Systems LUNAR, Madison, WI). Subjects were scanned in the supine position wearing light clothing free of metal and other dense objects.

Resting metabolic rate

Resting metabolic rate was measured by indirect calorimetry using a Deltatrac II metabolic monitor (SensorMedica, Yorba Linda, CA) fitted with a clear plastic ventilated canopy. The test was performed between 5:00 and 6:00 A.M. after an overnight stay in the hospital and a 12-h fasting period but before getting out of bed. Patients were required to stay awake and at rest in the supine position in a quiet room at thermoneutrality. RMR was calculated from registrations of the CO₂ and O₂ concentrations in the inhaled and exhaled air during a 30-min period. Recordings were averaged over the test period.

Statistics

The primary endpoints were the differences in LBM and RMR at 2 and 8 weeks postoperatively between the two cohorts. The mean difference was defined by the change between baseline LBM and the 2-week and 8-week measurements. The change in RMR was calculated using the same definition. Secondary endpoints included changes in body weight, BMI, and fat mass (FM) between the two groups. All results are expressed as means $\pm$ standard deviation unless otherwise specified. Patient characteristics at baseline were analyzed using Fisher's exact test and two independent samples *t* test as appropriate. After checking for normality assumptions, the paired *t* test was used to compare preoperative values to 2-week and 8-week postoperative results in respect to weight, BMI, FM, LBM, and RMR in both groups. To detect the effect of HMB/Glu/Arg on the experimental group, independent *t* test was used to determine whether the mean changes in postoperative parameters were significant between the experimental and control group. All *P* values are

two-sided with statistical significance evaluated at the 0.05 alpha levels. All analyses were performed using Statistical Analysis Systems, version 9.2 (SAS, Institute, Cary, NC).

Results

Thirty morbidly obese subjects (96.7% women; 80% whites, and 20% African Americans; age 46.9 ± 8.4 years; weight 113.4 ± 11.6 kg; and mean BMI 43.3 ± 4.1 kg/m²) were included in the study. Fourteen patients were randomized to the experimental group and 16 to the control group. Baseline demographics and characteristics of experimental and control group are summarized in Table 1, and no significant differences were observed in any of the baseline variables. All patients ($N = 30$) completed both the 2-week and 8-week follow-up evaluations as outlined by the protocol.

Changes at 2 weeks

The entire study population ($N = 30$) demonstrated a significant decrease in weight, BMI, FM, LBM, and RMR as illustrated in Table 2. A highly significant reduction in RMR (226.2 ± 208.9 kcal/day, $P < 0.0001$) was observed in the total population. Patients lost a mean of 6.8 ± 2.3 kg ($P < 0.0001$) of their initial body weight in the first 2 weeks. Mean decrease in BMI (2.6 ± 0.9 kg/m², $P < 0.0001$), fat (1.8 ± 2.8 kg, $P = 0.0013$), and LBM (5.7 ± 3.2 kg, $P < 0.0001$) were highly significant at 2 weeks (Table 3).

The changes at 2 weeks in both the groups are outlined in Table 2. Although total body weight significantly decreased in the experimental (114 ± 11.1 vs. 107 ± 11.3 kg, $P < 0.0001$) and in the control group (112.8 ± 12.3 vs. 106.1 ± 12.6 kg, $P < 0.0001$), there was no significant difference when comparing the changes (Table 4) between the two groups (6.9 ± 1.4 vs. 6.7 ± 2.8 kg, $P = 0.8$). Similarly, BMI decreased in the experimental (42.9 ± 4.1 vs. 40.3 ± 4.4 kg/m², $P < 0.0001$) and control groups (43.6 ± 4.2 vs. 41.0 ± 4.3 kg/m², $P < 0.0001$), but the mean changes between the groups was insignificant (2.6 ± 0.5 vs. 2.6 ± 1.1 kg/m², $P = 0.99$). Fat mass (58.7 ± 7.9 vs. 56.4 ± 8.9 kg, $P = 0.003$), LBM (52.4 ± 6.9 vs. 46.8 ± 5.6 kg, $P < 0.0001$), and RMR (1798.7 ± 280.5 vs. 1610 ± 212.3 kcal/day, $P < 0.0001$) also significantly decreased at the end of 2 weeks in the experimental group. Similarly in the controls, LBM (54 ± 8.1 vs. 48.2 ± 7.5 , $P < 0.0001$) and RMR (1847.9 ± 299.4 vs. 1588.8 ± 236.2 , $P < 0.0001$) changed significantly at the end of 2 weeks. However, FM did not decrease significantly in the control group (55.4 ± 8.3 vs. 53.9 ± 8.9 kg, $P = \text{NS}$). When comparing the mean changes between the experimental versus control group in terms of fat mass (2.2 ± 2.3 vs. 1.5 ± 3.3 kg, $P = 0.51$), LBM (5.6 ± 2.7 vs. 5.8 ± 3.7 kg, $P = 0.9$), and RMR (188.7 ± 264.3 vs. 259.1 ± 146.3 kcal/day, $P = 0.39$), no statistical significance was observed (Table 4).

Changes at 8 weeks

Changes between baseline and 8-week parameters (Table 3) in all patients revealed that weight (15.7 ± 2.5 kg, $P < 0.0001$), BMI (6 ± 1 kg/m², $P < 0.0001$), FM (8.5 ± 3.4 kg, $P < 0.0001$), and LBM (7.8 ± 4 kg, $P < 0.0001$) all significantly decreased. The RMR also significantly decreased (290.6 ± 234.9 kcal/day, $P < 0.0001$) at the end of 8 weeks in all patients compared with baseline.

Changes in body composition and energy at the end of 8 weeks in both the groups are depicted in Table 5. Total body weight decreased in the experimental group (114 ± 11.1 vs. 98.2 ± 11.4 kg, $P < 0.0001$) and in the control group (112.8 ± 12.3 vs. 97.2 ± 12.4 kg, $P < 0.0001$). Comparing the mean changes in weight (Table 6) between the experimental and control groups, no significance difference was observed (15.8 ± 2.6 vs. 15.7 ± 2.5 kg, $P = 0.93$). Decrease in BMI in the experimental (42.9 ± 4.1 vs. 37 ± 4.3 kg/m², $P < 0.0001$) and

control (43.6 ± 4.2 vs. 37.5 ± 4.2 kg/m², $P < 0.0001$) groups were found to be significant. However, comparing the change in BMI between the groups (5.9 ± 0.9 vs. 6.1 ± 1.1 kg/m², $P = 0.66$) revealed no significant difference. Fat mass (58.7 ± 7.9 vs. 49.5 ± 8.8 kg, $P < 0.0001$), LBM (52.4 ± 6.9 vs. 44.7 ± 5.9 kg, $P < 0.0001$) and RMR (1798.7 ± 280.5 vs. 1512.1 ± 188.4 kcal/day, $P < 0.0001$) significantly decreased in the experimental group. In the control group, fat mass (55.4 ± 8.3 vs. 47.5 ± 8.8 kg, $P < 0.0001$), LBM (54 ± 8.1 vs. 46.1 ± 6 , $P < 0.0001$), and RMR (1847.9 ± 299.4 vs. 1553.8 ± 146 , $P < 0.0001$) also significantly decreased at the 8-week time point. When comparing between experimental and control groups at 8 weeks, the mean changes (Table 6) in fat mass (9.2 ± 3.2 vs. 8 ± 3.5 kg, $P = 0.35$), LBM (7.7 ± 3.5 vs. 7.9 ± 4.5 kg, $P = 0.92$), and RMR (286.6 ± 271.1 vs. 294.1 ± 207.2 kcal/day, $P = 0.93$) were insignificant.

Discussion

The results of this study show that at the end of 8 weeks, all participants lost an average of 13.9% of their initial body weight and 26.4% of their excess weight (actual body weight – ideal body weight). A progressive decline in BMI, FM, LBM, and RMR was observed at 2 weeks and 8 weeks. The only difference between the two groups occurred at 2 weeks when the FM loss was insignificant in the control groups, whereas it was significant in the experimental group. This may be real or a function of the small number of subjects in each group. The postoperative changes in body weight, BMI, FM, LBM, and RMR reported in this study agree with previous studies that examined the effects of bariatric surgery on body composition and resting energy expenditure [10–15]. However, these changes were not significantly different when comparing the experimental group that received HMB/Glu/Arg and the control group with LBM and RMR decreasing comparably at 2 weeks and 8 weeks postoperatively (Tables 4, 6). Therefore, this study was unable to demonstrate an effect of HMB/Glu/Arg on LBM and RMR in postoperative LGB patients. A number of factors may explain these results.

Although HMB/Glu/Arg has been shown to significantly reverse the loss of muscle mass in cancer and critically ill trauma patients within 8 weeks [18–22], extended follow-up duration might be needed in bariatric patients because weight loss usually continues for 12–18 months after LGB. Additionally, it may be unreasonable to expect HMB/Glu/Arg to have the same effect in LGB patients as it does in cancer and critically ill or injured patients. These patients usually recover to nearly normal function after medical and surgical therapy or they succumb to the illness or injury. The dramatic effect of LGB on the digestion and absorption of nutrients in morbidly obese individuals is expected to endure for the remainder of the increased life expectancy after bariatric surgery. Another reason for lack of effect of HMB/Glu/Arg is that the decrease in RMR is not only determined by LBM but also by fat tissue that is metabolically active [26–28]. It also suggests that after gastric bypass, RMR decreases progressively for the first 6 months with stabilization in the following months, and weight regain can begin 12–18 months after the surgery [29]. It might be beneficial for future studies to treat and follow patients who receive HMB/Glu/Arg until after the period of rapid weight loss to assess its true effect on RMR. It should be mentioned that the changes in the hormonal milieu of the gastrointestinal tract after LGB are probably not comparable to those of the ill and injured patient, making the assumption that HMB/Glu/Arg should have the same effect fallacious.

A small sample size of 30 patients and errors in patient-reported intake of HMB/Glu/Arg supplement are additional limitations of this study. Because this is a pilot study, the sample size was limited to 30 participants, which might have affected the results of the trial. Earlier studies that have successfully proven the restoration of LBM as a result of nutritional supplementation with HMB/Arg/Glu have used larger sample sizes and different techniques

to monitor supplement consumption [19–22]. One of these studies involved feeding of HMB/Glu/Arg to patients through a feeding tube while they were admitted in the trauma unit [22]. This not only ensured the consumption of the supplement as prescribed by the study but also allowed the clinician to control nutritional intake more accurately. Whereas each patient was counseled regarding high protein intake while limiting refined sugars, carbohydrates, and fat, we were not able to control substrate intake that may impact the effect of HMB/Glu/Arg. Because patients were taking the supplement after discharge, lack of compliance due to inconvenience or taste and errors in self-reporting may have affected the results of this study. Compliance issues have been reported in earlier studies due to gastrointestinal side effects and also may have been a factor in this study [18].

It is important to note that the mechanism of LBM loss, change in RMR, and long-term weight regain is multifactorial. It may be due to behavioral issues, inadequate or inappropriate eating habits, adaptation of the small bowel to improve absorption of nutrients, normal energetic adaptation to decreased energy intake resulting in a decline in RMR, or gastrointestinal hormonal changes [30–32]. This study was not designed, and was underpowered, to examine the effect of these factors in addition to HMB/Glu/Arg on changes in RMR and weight recidivism in bariatric patients.

Conclusions

Laparoscopic gastric bypass produces a significant decrease in body weight, excess weight loss, BMI, total fat mass, LBM, and RMR at 2 weeks and 8 weeks postoperatively. The study failed to demonstrate any modulation of these changes by the use of HMB/Glu/Arg. Potential preservation of LBM and RMR as a result of nutritional supplements in early postoperative laparoscopic gastric bypass patients needs further investigation.

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Table 1

Baseline characteristics

Variable	Total (N = 30)	Experimental (n = 14)	Control (n = 16)	P value
Age (year)	46.9 ± 8.4	47.9 ± 9.6	46 ± 7.5	0.56 [†]
Gender				
Female	29 (96.7)	14 (100)	15 (93.8)	1.00*
Male	1 (3.3)	0	1 (6.2)	
Race				
White	24 (80)	11 (78.6)	13 (81.2)	1.00*
African American	6 (20)	3 (21.4)	3 (18.8)	
Total body weight (kg)	113.4 ± 11.6	114 ± 11.1	112.8 ± 12.3	0.80 [†]
BMI (kg/m ²)	43.2 ± 4.1	42.9 ± 4.1	43.6 ± 4.2	0.65 [†]
FM (kg)	56.9 ± 8.1	58.7 ± 7.9	55.4 ± 8.3	0.29 [†]
LBM (kg)	53.2 ± 7.5	52.4 ± 6.9	54 ± 8.1	0.58 [†]
RMR (kcal/day)	1824.9 ± 286.8	1798.7 ± 280.5	1847.9 ± 299.4	0.65 [†]

Data are means ± standard deviations or numbers with percentages in parentheses unless otherwise indicated

BMI body mass index; *FM* fat mass; *LBM* lean body mass; *RMR* resting metabolic rate

[†] Based on two independent sample *t* test

* Based on Fisher's exact test

Table 2

Changes in body composition and resting metabolic rate at 2 weeks

Variable	Total (N = 30)		Experimental (n = 14)		Control (n = 16)	
	Baseline	2 week	Baseline	2 week	Baseline	2 week
Total body weight (kg)*	113.4 ± 11.6	106.8 ± 11.8	114 ± 11.1	107 ± 11.3	112.8 ± 12.3	106.1 ± 12.6
BMI (kg/m ²)*	43.3 ± 4.1	40.7 ± 4.3	42.9 ± 4.1	40.3 ± 4.3	43.6 ± 4.2	41 ± 4.3
FM (kg)	56.9 ± 8.1	55.1 ± 8.8 ^a	58.7 ± 7.9	56.4 ± 8.9 ^b	55.4 ± 8.3	53.9 ± 8.9 ^c
LBM (kg)*	53.2 ± 7.5	47.5 ± 6.6	52.4 ± 6.9	46.8 ± 5.6	54 ± 8.1	48.2 ± 7.5
RMR (kcal/day)*	1824.9 ± 286.8	1598.7 ± 221.8	1798.7 ± 280.5	1610 ± 212.3	1847.9 ± 299.4	1588.8 ± 236.2

Data are expressed as means ± standard deviations

BMI/ body mass index; FM fat mass; LBM lean body mass; RMR resting metabolic rate

* Postoperative changes at 2 weeks were significant ($P < 0.0001$) in total population, experimental, and control groups^a $P = 0.0013$ ^b $P = 0.003$ ^c $P =$ not significant

Table 3

Changes in total population ($N = 30$) compared with baseline

Variable	Mean change 2 weeks	P value	Mean change 8 weeks	P value
Total body weight (kg)	6.8 ± 2.3	<0.0001	15.7 ± 2.5	<0.0001
BMI (kg/m^2)	2.6 ± 0.9	<0.0001	6 ± 1	<0.0001
FM (kg)	1.8 ± 2.8	0.0013	8.5 ± 3.4	<0.0001
LBM (kg)	5.7 ± 3.2	<0.0001	7.8 ± 4	<0.0001
RMR (kcal/day)	226.2 ± 208.9	<0.0001	290.6 ± 234.9	<0.0001

Data are expressed as means $\pm$ standard deviations unless otherwise indicated

Mean change at 2 weeks = mean baseline – mean 2 week

Mean change at 8 weeks = mean baseline – mean 8 week

BMI body mass index; FM fat mass; LBM lean body mass; RMR resting metabolic rate

Table 4

Comparing the mean changes at 2 weeks between the groups

Variable	Experimental (<i>n</i> = 14) Mean change	Control group (<i>n</i> = 16) Mean change	<i>P</i> value
Total body weight (kg)	6.9 ± 1.4	6.7 ± 2.8	0.8
BMI (kg/m ²)	2.6 ± 0.5	2.6 ± 1.1	0.99
FM (kg)	2.2 ± 2.3	1.5 ± 3.3	0.51
LBM (kg)	5.6 ± 2.7	5.8 ± 3.7	0.9
RMR (kcal/day)	188.7 ± 264.3	259.1 ± 146.3	0.39

Data are expressed as means ± standard deviations unless otherwise indicated

Mean change = mean baseline – mean 2 week

BMI body mass index; *FM* fat mass; *LBM* lean body mass; *RMR* resting metabolic rate

Table 5

Changes in body composition and resting metabolic rate at 8 weeks

Variable	Total (N = 30)		Experimental (n = 14)		Control (n = 16)	
	Baseline	8 week	Baseline	8 week	Baseline	8 week
Total body weight (kg)*	113.4 ± 11.6	97.6 ± 11.8	114 ± 11.1	98.2 ± 11.4	112.8 ± 12.3	97.2 ± 12.4
BMI (kg/m ²)*	43.3 ± 4.1	37.2 ± 4.2	42.9 ± 4.1	37 ± 4.3	43.6 ± 4.2	37.5 ± 4.2
FM (kg)*	56.9 ± 8.1	48.4 ± 8.7	58.7 ± 7.9	49.5 ± 8.8	55.4 ± 8.3	47.5 ± 8.8
LBM (kg)*	53.2 ± 7.5	45.5 ± 5.9	52.4 ± 6.9	44.7 ± 5.9	54 ± 8.1	46.1 ± 6
RMR (kcal/day)*	1824.9 ± 286.8	1534.3 ± 165.4	1798.7 ± 280.5	1512.1 ± 188.4	1847.9 ± 299.4	1553.8 ± 146

Data are expressed as means ± standard deviations

BMI body mass index; FM fat mass; LBM lean body mass; RMR resting metabolic rate

* Postoperative changes at 8 were significant ($P < 0.0001$) in total population, experimental, and control groups

Table 6 Comparing the mean changes at 8 weeks between the groups

Variable	Experimental (<i>n</i> = 14) Mean change	Control group (<i>n</i> = 16) Mean change	<i>P</i> value
Total body weight (kg)*	15.8 ± 2.6	15.7 ± 2.5	0.93
BMI (kg/m ²)	5.9 ± 0.9	6.1 ± 1.1	0.66
FM (kg)	9.2 ± 3.2	8 ± 3.5	0.35
LBM (kg)	7.7 ± 3.5	7.9 ± 4.5	0.92
RMR (kcal/day)	286.6 ± 271.1	294.1 ± 207.2	0.93

Data are expressed as means ± standard deviations unless otherwise indicated

Mean change = mean baseline – mean 8 week

BMI body mass index; *FM* fat mass; *LBM* lean body mass; *RMR* resting metabolic rate