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RESEARCH

Resting Energy Expenditure and Metabolic Adaptation Following Sleeve Gastrectomy in Hispanic Adults with Obesity

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Abstract

Background Prospective studies examining the changes in resting energy expenditure, metabolic adaptation (MA), and body composition following sleeve gastrectomy (SG) are limited, especially among minority populations.

Methods Data were included from a secondary analysis of participants ($n=24$; mean ± SD: 42 ± 11 y, 62.5% Female, 58.3% Hispanic, 20.8% non-Hispanic White, 37.5% with type 2 diabetes not on insulin, BMI, 41.9 ± 4.7 kg/m^2) undergoing SG from a 24-month prospective study. REE was measured using indirect calorimetry at baseline, 1.5, and 6 months. Body composition (fat mass [FM] and fat-free mass [FFM]) was measured via bioelectrical impedance analysis at baseline, 1.5, 6, 12, and 24 months. Metabolic Adaptation (MA) was calculated by subtracting measured REE from predicted REE using a linear model that included FFM.

Results REE significantly decreased from baseline to 1.5 months (-371.8 kcal/day, 95%CI: -577.5 to -166.1, $p=0.0005$) and 6 months (-374.2 kcal/day, 95%CI: -647.3 to -101.0, $p=0.006$), but was not significantly different from 1.5 to 6 months ($p=0.99$). MA was −328.8 kcal/day (95%CI: -480.6 to -128.9, $p=0.008$) and −306.7 kcal/day (95%CI: -553.9 to -59.4, $p=0.02$) at 1.5 and 6 months, but was not significantly different from 1.5 to 6 months ($p=0.97$). REE and MA at 1.5 and 6 months were not associated with FM or FFM at 12- and 24-months following SG.

Conclusion The acute changes in REE and MA did not blunt weight loss and were not associated with body composition at 12 and 24 months; however, our findings should be interpreted with caution, given the small sample size and potential inaccuracies in body composition measurements.

Key Points

- REE decreases rapidly following SG in the first 1.5 months, subsequently plateauing from 1.5 to 6 months.
- MA is present at 1.5- and 6-months following SG, although the magnitudes may be overestimated due to the methodological use of BIA.
- Despite lower REE and MA at 1.5 and 6 months, body weight and fat mass continued to decline through 12 months.
- REE and MA at 1.5 and 6 months did not predict FM or FFM at 12 or 24 months, suggesting early MA may not blunt weight loss or drive later body composition changes; although these findings could be reflected by limited power, measurement inaccuracies using BIA to measure FFM, and not true physiologic irrelevance.

Keywords Adaptive thermogenesis · Weight loss · Weight regain

Introduction

Obesity, a chronic disease characterized by excessive adiposity, is a significant health issue globally, but particularly in the United States. In the United States, 39.8% of adults have obesity, with projections suggesting that this percentage will reach 50% by 2030 [1, 2]. Even more concerning, obesity increases the risk of co-morbid conditions, such as hypertension, cardiovascular disease, cancer, and type 2 diabetes (T2D) [3]. The economic burden of overweight and

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obesity was estimated to be US$705.72 billion in the United States in 2019 [4].

The first line of treatment for excess adiposity includes lifestyle interventions targeting caloric restriction and increased physical activity to induce negative energy balance. However, lifestyle interventions result in minor total weight loss (TWL) of 5–10%, which is often accompanied subsequently by weight regain [5]. Therefore, metabolic and bariatric surgery (MBS) is a highly effective obesity treatment. Weight loss-induced MBS results in significant improvements in clinical markers associated with co-morbid conditions [6]. MBS reduces cardiovascular disease risk by nearly 50% [7]. The most common surgery presently performed is sleeve gastrectomy (SG) [8]. Average TWL from SG is approximately 28% within 1 year, with subsequent weight regain, reaching approximately 22% by 5 years [9].

SG results in significant weight loss as a result of negative energy balance. A significant determinant of energy balance is the contribution of resting energy expenditure (REE), the largest component of total daily energy expenditure (TDEE), accounting for 60–70% of the variance [10]. Following significant weight loss, REE decreases as a result of losses in fat-free mass (FFM), which consists of organs (e.g., liver, brain), skeletal muscle mass, bone, and total body water (intracellular and extracellular water) [11]. Prior studies examining REE suggest a rapid, post-weight loss decrease and subsequent plateauing, likely a combined effect of severe energy imbalance and long-term maintenance of FFM [12–14]. Furthermore, metabolic adaptation (MA), the change in energy expenditure that is independent of changes in both FFM and fat mass (FM), has been well-documented following MBS and may contribute to weight regain. MA has been postulated to persist as a homeostatic response to weight loss maintenance in individuals maintaining > 10% weight loss for more than 1 year [15]. MA may contribute to the blunting and plateauing of body weight following significant weight loss; however, there is considerable speculation around its contribution. A systematic review of intervention weight loss studies (e.g., non-bariatric surgery) examining the various components of energy expenditure suggests that, while weight loss results in MA in some energy expenditure components, the values may be small or non-statistically significant. In contrast, the seminal paper of "The Biggest Loser" competition, weight loss was induced by caloric restriction, and high-volume exercise resulted in MA that persisted 6 years following the competition despite significant weight regain [16]. Despite the intriguing findings regarding energy balance in both low-calorie diet and MBS interventions, the implications and timing of REE and MA after MBS warrant further exploration, as some weight regain appears to be common for most adults as long-term weight loss maintenance is a lifelong challenge [17]. Understanding the mechanisms associated with energy balance provides further insights into weight loss maintenance strategies (e.g., physical activity). Therefore, the current study aims to describe changes in body weight, FM, FFM, REE, and MA following SG in a 24-month prospective study, and to determine the associations between acute changes in energy expenditure (REE and MA) at 1.5 and 6 months with FM and FFM at 12 and 24 months post-surgery.

Methods

Design

Data for these secondary analyses were included from a prospective longitudinal cohort study that enrolled participants with obesity between 2017 and 2020, who were eligible for SG, at NYU Langone Health and Bellevue Hospital [18]. All procedures involving human participants were in accordance with the ethical standards of the NYU Langone Health's Institutional Review Board (IRB) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from each participant before enrollment.

Inclusion criteria included individuals between the ages of 18–75 years and candidates for SG. Participants were English or Spanish-speaking. Exclusion criteria included a history of CVD and stroke. Other exclusion criteria are listed in *Supplementary Table 1*. Measurements were obtained at baseline, 1.5, and 6 months at the Clinical Research Center of the New York University Clinical and Translational Science Institute. For this secondary analysis, the primary outcomes include FM, REE, and MA. Additionally, we define two periods of body weight change as the weight loss phase and the weight loss maintenance phase, whereby the weight loss phase is 0 to 12 months, and the weight loss maintenance phase is 12 to 24 months.

Anthropometrics

Height was measured to the nearest 1 cm using a portable stadiometer (SECA 213, Seca GmBH & Co. KG, Hamburg, Germany), and body weight was measured in light clothing without shoes to the nearest 0.1 kg using a Stow-A-Weigh scale (Scale Tronix, Welch Allyn, Skaneateles, NY, USA). All measurements were performed in a fasted state. Weight loss was defined as the total percentage of weight loss (%TWL) from baseline.

Body Composition, Resting Energy Expenditure and Metabolic Adaptation

Body composition was defined using the two-compartment model, where body weight was divided into FM and FFM. FM and FFM were assessed using bioelectrical impedance analysis (BIA; InBody 270, InBody, Inc. Cerritos, CA, USA). Prior to REE measurement, participants were asked to confirm the following: (1) no vigorous physical activity within the previous 48 h, (2) a minimum of 8 h fast, and (3) no use of caffeine or nicotine products on the day of testing. Participants were directed to lie supine for 10 min, during which the flow meter and gas analyzer were calibrated per the manufacturer's instructions. Open-circuit indirect calorimetry was performed using a COSMED Quark RMR® metabolic cart with flow-dilution canopy hood (COSMED, Rome, Italy) at NYU Langone Health's Clinical and Translation Science Institute, where environmental conditions (e.g., room temperature) were controlled as best as possible (temperature range: 68–74 °F). All indirect calorimetry measurements were done in the morning. VO_2 consumption and VCO_2 production were measured for 20 min following a 5-min run-in stabilization period. Participants were instructed to abstain from sleeping and talking during the test. Gas exchange variables (VO_2 and VCO_2) were measured every 10 s. REE was calculated from the Weir equation and expressed in kilocalories per day (kcal/d) [19]. Respiratory quotient (RQ) was determined by dividing VCO_2 by VO_2. REE was determined using the first steady-state period in which both VO_2 and VCO_2 were ≤ 10% of the coefficient of variation [20]. For those who did not meet these criteria, we used the VO_2 and VCO_2 from the 5-minute period following the initial 10-minute measurement period. To assess the validity of the test, the range of RQ was determined and compared to the physiological range (0.67–1.3). Indirect calorimetry was collected at baseline (0), 1.5 and 6 months only. MA was used to detect differences in REE that are independent of changes in body composition. MA was calculated as the difference between measured REE and predicted REE (REEp). REEp was estimated using a linear regression model fitted at baseline with FFM as the predictor, and FFM measured at 1.5 and 6 months was then entered into this model to obtain REEp at the corresponding time points MA (kcal/d) = $\{[REE_{(1.5,6)mo} - REEp_{(1.5,6)mo}] - [REE_{baseline} - REEp_{baseline}]\}$, REEp ~ 921.1 + 18.2 * FFM. The rationale for the use of this MA equation was described by Nunes et al. [21].

Statistical Analysis

Data are reported as means (± standard deviation) or N (percent) for continuous and categorical variables, respectively. Statistical comparisons of body weight, FM, FFM, REE, and MA between baseline and other time points were conducted using paired t-tests when the data satisfied the Shapiro–Wilk normality test; otherwise, Wilcoxon signed-rank tests were applied. For analyses of relative changes in body weight, FM, and FFM (i.e., percent change from baseline), one-sample t-tests were used when normality was met, and Wilcoxon signed-rank tests were used otherwise. P values from related tests presented within each figure were adjusted using the Benjamini–Hochberg procedure to control the false discovery rate (FDR) at 0.05. To evaluate the potential impact of progressive sample attrition on the analyses, we assessed whether the longitudinal measurements were missing completely at random (MCAR) using Little's MCAR test. For REE, the test did not indicate a significant departure from the MCAR assumption ($p=0.708$). Similarly, for FM and FFM, the results were not statistically significant (FM: $p=0.578$; FFM: $p=0.373$), suggesting that the missing data patterns for these longitudinal outcomes were consistent with the MCAR assumption. Analyses were performed in R 4.3.0, and figures were generated with GraphPad Prim10.

Results

Table 1 shows the baseline characteristics of study participants. At baseline, participants were 42 ± 11 years, 62.5% Female, 58.3% Hispanic, 20.8% non-Hispanic White, and BMI, 41.9 ± 4.7 kg/m^2. The percentage of subjects with diabetes not on insulin was 37.5%. Missing data in Table 1 were due to surgery being cancelled without clinical testing being rescheduled and the unavailability of equipment (e.g., FM/FFM and BIA scale). There was a statistically significant decline in body weight across each time point (Fig. 1). Body weight loss reached a nadir from baseline (0 months) of -25.3 ± 7.9 kg and a %TWL of -22.0 ± 5.9% at 12 months ($p<0.0001$). Weight regain from 12 to 24 months was 4.3 ± 3.8 kg (range: -6.2 to 10.6 kg) and 4.8 ± 4.3% (range: -6.4 to 11.1%; $p<0.05$). Figure 2A-D shows the change in absolute and relative FM and FFM following SG. FM decreased significantly across all timepoints until reaching a nadir of -22.6 ± 6.0 kg and −44.9 ± 13.3% at 12 months. FM accounted for 65.3 ± 14.0%, 61.9 ± 28.6% and 78.6 ± 11.9% %TWL at 1.5, 6 and 12 months, respectively (*Supplemental Table 2*). There was a significant change in FM regain from 12 to 24 months, 4.8 ± 3.1 kg or 15.2 ± 8.6% (Fig. 2A and B). FFM decreased significantly from baseline to 1.5 months (-6.2 ± 3.7 kg; -11.9 ± 7.5%, Fig. 2C and D), reaching a nadir at 1.5 months and remained stable from 6 to 12 and 24 months. FFM was not regained from 12 to 24 months (+1.1 ± 1.9 kg, $p=0.11$, 1.7 ± 3.6%, $p=0.21$), suggesting that the majority of weight regain was from FM.

Table 1 Baseline demographic and anthropometric characteristics

Variable	Baseline ($n=24$)
Age, years	42 ± 11
Sex, % (n)	
Male	37.5 (9)
Female	62.5 (15)
Race and Ethnicity % (n)	
Hispanic	12.5 (3)
Hispanic White	8.3 (2)
Hispanic Black or African American	8.3 (2)
Non-Hispanic Black or African American	20.8 (5)
Non-Hispanic White	20.8 (5)
Hispanic more than one race	29.2 (7)
Ethnicity, % (n)	
Non-Hispanic	41.7 (10)
Hispanic	58.3 (14)
HbA1c, % (n)[a]	6.7 ± 2.2
NIDDM, % (n)	37.5 (9)
Body weight, kg	114.9 ± 14.5
Height, cm	165.6 ± 8.1
BMI, kg/m^2	41.9 ± 4.7
WC, cm	120.1 ± 11.4
FM, kg[b]	53.0 ± 11.2
FFM, kg[b]	62.7 ± 9.9
Body Fat, %[b]	45.7 ± 6.2
REE, kcals/day	2002.7 ± 344.2
RQ	0.86 ± 0.1

Baseline data are reported as mean and standard deviation

BMI body mass index, *FM* fat mass, *FFM* fat-free mass, *NIDDM* non-insulin dependent diabetes mellitus, *REE* resting energy expenditure, *RQ* Respiratory Quotient, *WC* waist circumference

[a]HbA1c: $n=23$

[b]FM, FFM and %Body fat: $n=21$

There was a statistically significant decline from baseline to 1.5 months and subsequent plateauing of REE from 1.5 to 6 months (Fig. 3A). The difference in REE from baseline to 1.5 months and baseline to 6 months was −371.8 kcal/day (95%CI: -577.5 to -166.1, $p=0.0005$) and −374.2 kcal/day (95%CI: -647.3 to -101.0, $p=0.006$), respectively. This suggests an acute and rapid decline in REE with a subsequent plateau as the difference from 1.5 to 6 months was non-significant (1.56 kcal/day, 95%CI: -131.9 to 135.0, $p=0.99$). The change in REE is reflected in the magnitude of MA, as shown in Fig. 3B. MA at 1.5 months was −328.8 kcal/day (95%CI: -480.6 to -128.9, $p=0.008$) and −306.7 kcal/day (95%CI: 95%CI: -553.9 to -59.4, $p=0.02$) at 6 months, but there was no significant difference between 1.5 and 6 months ($p=0.97$). Despite acute changes in REE and MA following SG, BW and FM continued to decrease from baseline to 12 months. The Pearson correlations indicate no association between REE and MA at 1.5 and 6 months with FM and 12 and 24 months, and FFM at 12 and 24 months (Table 2).

Discussion

Weight loss from MBS, regardless of surgery type, induces adaptations in energy expenditure that are independent of changes in FM and FFM [12, 22]. Our main findings indicate that REE rapidly decreases following SG, induced by severe energy imbalance (e.g., semi-starvation state), resulting in MA at 1.5 and 6 months post-surgery. Despite the rapid and significant change in energy expenditure, weight loss and FM loss continued. Furthermore, we found that the acute changes in REE and presence of MA at 1.5- and 6-month post-surgery were not associated with FFM or FM at 12 and 24 months. These findings suggest that acute changes in energy expenditure following SG, and those independent of changes in FFM (i.e., MA), may not blunt weight loss nor do they result in weight regain; however, these findings could be reflected by limited power due to the sample size, measurement noise with FFM using BIA, or attrition and not true physiologic irrelevance.

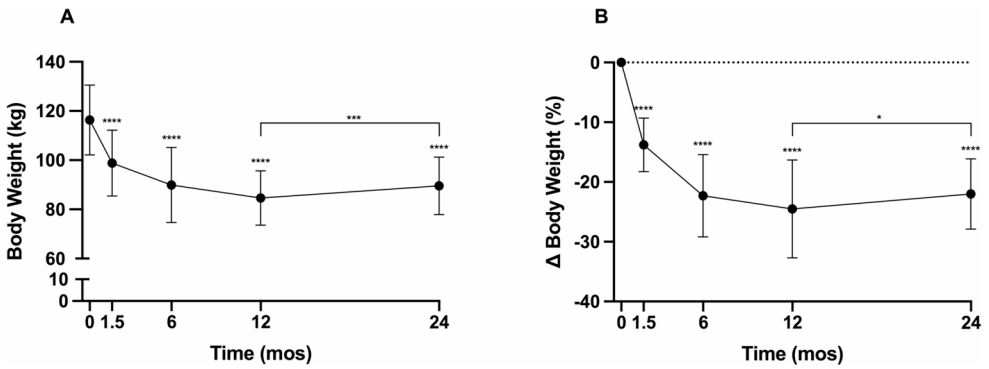

Fig. 1 **A**-**B** Body weight change following sleeve gastrectomy. Mean change in body weight (**A**) and the percent total weight loss (**B**) from baseline (0 months) to post-surgery at 1.5, 6, 12, and 24 months. Data are reported as mean ± SD. Start denote statistical significance: *, $p<0.05$, **, $p<0.01$, ***, $p<0.001$, ****, $p<0.0001$; Sample sizes compared to baseline (0 months): 1.5 mos: $n=23$; 6 mos: $n=20$; 12 mos: $n=21$; 24 mos: $n=17$

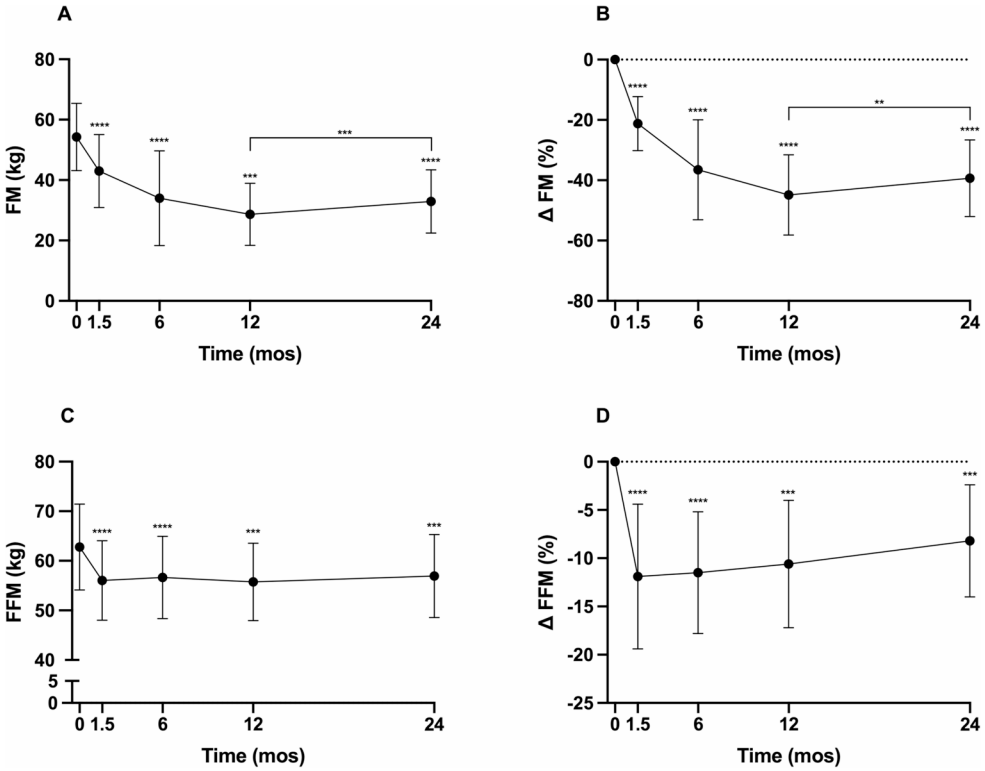

Fig. 2 **A-D** Fat mass and fat-free mass change following sleeve gastrectomy. Mean change in FM (**A**), percent FM loss (**B**), change in FFM (**C**), and percent FFM loss (**D**) from baseline (pre-surgery) to 1.5, 6, 12, and 24 months. Data are reported as mean ± SD. Stars denote statistical significance: *, $p<0.05$, **, $p<0.01$, ***, $p<0.001$, ****, $p<0.0001$; Panel (**A**) and (**C**) sample sizes compared to baseline (0 months): 1.5 mos: $n=19$; 6 mos: $n=15$; 12 mos: $n=12$; 24 mos: $n=13$; Panel (**B**) and (**D**) sample sizes compared to baseline (0 months): 1.5 mos: $n=18$; 6 mos: $n=13$; 12 mos: $n=12$; 24 mos: $n=13$

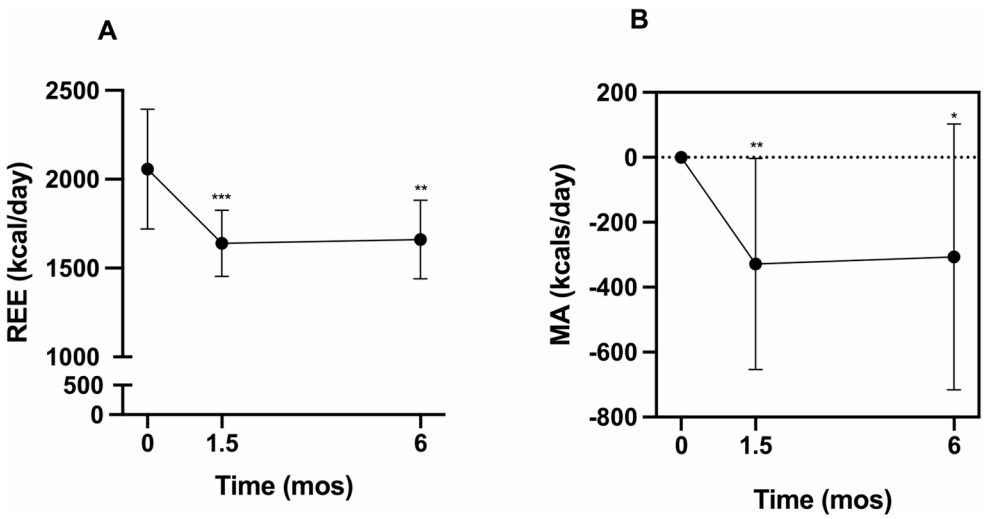

Fig. 3 **A-B** Resting energy expenditure and metabolic adaptation following sleeve gastrectomy. Changes in resting REE (**A**) and MA (**B**) before and at 1.5 and 6 months post-sleeve gastrectomy. Data are reported as mean ± SD. Stars denote statistical significance: *, $p<0.05$, **, $p<0.01$, ***, $p<0.001$, ****, $p<0.0001$; MA (kcal/d) = $\{[REE_{(1.5,6)mo} - REEp_{(1.5,6)mo}] - [REE_{baseline} - REE_{pbaseline}]\}$. REEp: A prediction equation using baseline FFM as an independent predictor. MA, metabolic adaptation; REE, resting energy expenditure. Panel (**A**) sample sizes compared to baseline (0 months): 1.5 mos: $n=19$; 6 mos: $n=16$; Panel (**B**) sample sizes compared to baseline (0 months): 1.5 mos: $n=16$; 6 mos: $n=13$

Prior literature shows the typical nadir of body weight following SG is approximately 12 months, which is supported by our data [9]. The changes in FM reflect the changes in body weight, where there was a significant decrease from baseline to 12 months, accounting for the majority of total weight loss (*Supplemental Table 2*). While the goal in any weight loss intervention, whether it be lifestyle modification, pharmacological therapy or surgery, is to optimize FM loss, FFM loss is inevitable in most cases. Interestingly, our findings show that FFM decreased from baseline to 1.5 months and remained relatively stable from 1.5 to 24 months. Our findings suggest that of the %TWL at 1.5 and 6 months,

Table 2 Associations between resting energy expenditure and metabolic adaptation, and body composition at 12 and 24 months

Variable	$REE_{1.5mos}$		REE_{6mos}		$MA_{1.5mos}$		MA_{6mos}	
	Correlation (*r*)	*P* value	Correlation (*r*)	*P* value	Correlation (*r*)	*P* value	Correlation (*r*)	*P* value
FM_{12mos}	-0.037	0.90	0.231	0.49	0.119	0.74	0.318	0.40
FM_{24mos}	0.01	0.96	0.411	0.21	0.236	0.48	0.426	0.25
FFM_{12mos}	0.526	0.06	0.455	0.16	-0.0005	0.99	-0.092	0.81
FFM_{24mos}	0.532	0.06	0.447	0.17	0.004	0.99	-0.020	0.92

Pearson correlation coefficient. MA, metabolic adaptation; FM, fat mass; FFM, fat-free mass; $n = 19$ at 1.5 mos for REE and MA; $n = 15$ at 6 mos for REE and MA

FFM loss accounted for nearly one-third of this loss. Tam et al. (2016) found that 23% of %TWL at 2 months post-surgery was from FFM, which remained relatively stable at 1 year, accounting for 22% of total weight loss despite continued weight and FM loss [22]. One concern following weight loss, regardless of type, is weight regain, specifically in the form of FM. Our findings suggest that while FM accounted for the majority of weight loss at each time point, from 12 to 24 months, there was significant weight regain, predominantly from FM. As a result, our population experienced a significant loss of FFM without subsequent recovery of FFM during weight regain. This raises concerns about sarcopenic obesity, a concurrent decline in skeletal muscle mass and muscle function, along with increased adipose tissue, in this population [23]. While our sample was middle-aged, there is growing concern for sarcopenia among aging adults with obesity, especially older adults who are at greater risk for falls and CVD [24, 25]. Mitigation strategies include weekly resistance training and physical activity paired with a high-protein diet (> 1.2 g/kg body weight) [26, 27]. We should note that FFM is not specific to skeletal muscle mass and includes tissues like organs and bone. In normal weight individuals, skeletal muscle mass accounts for 45–50% of FFM [28]. Therefore, our data are unclear as to the magnitude of skeletal muscle loss as a result of SG. Future work should consider more direct measures of skeletal muscle mass, such as magnetic resonance imaging, paired with muscle function and strength testing (e.g., sit-to-stand, handgrip).

The rapid decline in REE following SG in our study is consistent with prior studies [22, 29, 30]. Fidilo et al. found a substantial decline in REE within 1 month following SG, and no significant change from 1 month to 12 months despite continued loss of body weight and BMI [30]. Our findings reflect those of Fidilo et al. and show that despite a rapid decrease and plateauing of REE following SG, participants continue to lose weight, even up to 12 months after surgery. These trends were also documented by Falkenhain et al., which found that TDEE, measured with doubly labelled water, decreased significantly from pre-surgery to 8 weeks but did not change from 8 to 52 weeks, despite continued weight loss [31]. These findings are relevant as TDEE includes both REE and activity energy expenditure. Our findings support the notion that the acute and rapid reduction in REE immediately following MBS is period of severe energy restriction or semi-starvation, and does not blunt weight loss, likely the result of internal physiological signals to conserve energy and reduce metabolic rate. The changes in energy expenditure appear to occur acutely but seem to dissipate over time.

Similar to diet-induced weight loss studies and other MBS studies, we found significant decreases in REE independent of changes in FFM (i.e., metabolic adaptation) at 1.5 and 6 months. In our study, MA at 1.5 and 6 months was similar to prior studies. For example, Tam et al. reported that in 14 adults with obesity undergoing SG, MA calculated from REE was − 305 ± 52 kcals/day and − 318 ± 63 kcals/day at 6 weeks and 6 months, respectively [29]. In a similar study by the same lead author, Tam et al. measured MA from 24-hr energy expenditure using a whole-room calorimeter and found MA was − 254 ± 51 kcals/day at 8 weeks following SG [22]. Wolfe et al. measured REE using indirect calorimetry on three consecutive days following an overnight stay, reporting MA of -131 ± 304 kcals/day at 6 months [14]. The differences in MA from prior studies are most likely methodological, and the comparisons should be interpreted carefully. Given the lack of consensus on an agreed-upon definition of MA, previous studies report different linear regression equations to predict REE, where some use FFM only [14], FM, FFM and sex [13, 29] or FFM, age, and sex [22]. Additionally, traditional measures calculate residuals from measured and predicted values at each time point. As such, prior studies do not adjust for potential MA at baseline, as highlighted by Nunes et al. who suggest that the approach we used likely reduces the large discrepancy between data treatment regarding predicted REE from difference approaches [21]. Furthermore, the difference in MA from our studies compared to others may also be a result of our use of BIA to estimate body composition compared to others which used dual energy x-ray absorptiometry. BIA may have inflated our findings, providing inaccurate estimates of FM and FFM in adults with severe BMIs (> 35 kg/m^2) [32, 33]. Because predicted energy expenditure in our model is largely driven by FFM, systematic overestimation of FFM by BIA would tend to inflate predicted REE. When MA is calculated as the difference between REE measured and REE predicted expenditure, this bias in FFM pushes

the predicted value upward and can therefore exaggerate the apparent magnitude of MA (i.e., make the residual more negative).

There is considerable debate regarding the contribution of MA to weight loss, weight regain, and its long-term persistence. MA may be a transient period of severe energy restriction; however, we cannot say definitively, as we did not collect REE at 12 and 24 months. In our study, MA at 1.5 and 6 months was not associated with FM or FFM at 12 months or 24 months, suggesting that early energetic adaptations following MBS may not contribute to weight loss or weight regain. A systematic review of lifestyle interventions suggests that MA may be attenuated or non-existent after periods of weight stability [34]. This may explain why in some studies, MA is present during weight loss but not at long-term follow-up (e.g., weight stability or weight regain). Bettini et al. examined patients with obesity and prediabetes/diabetes undergoing SG, and measured REE and metabolic adaptation at 1 year and 5 years following surgery [12]. MA was present after 1 year and was inversely related to excess percent weight loss. In other words, lower MA was associated with greater weight loss. After 5 years post-surgery, MA was not statistically significantly different from baseline, nor was MA related to weight loss or weight regain. Alternatively, Tam et al. found acute (6-week) MA following SG that persisted for up to 2 years, coupled with a continued increase in weight loss until reaching a nadir at 12 months [29]. Additionally, MA estimated from TDEE was reported at 8 weeks post-surgery (-358 kcal/day, 95%CI: -651, -64), but not week 52 (-45 kcal/day, 95%CI: -339, 248) [31]. MA may be attenuated by certain behaviors, such as physical activity. MA was higher in patients who did not meet the physical activity recommendations compared to those who did practice the physical activity recommendations (-282 [-448 to -126] kcals/day vs. -119 [-272 to 22]) kcals/day) [12]. In the study by Falkenhain et al., MA was not present at 52 weeks, despite being present at 8 weeks, which the authors suggest may be explained by the increase in physical activity [31]. The authors suggest that the concomitant increase in exercise may partly compensate for the decrease in energy expenditure after surgery.

Our study had numerous strengths, which include repeated measures and longer-term follow-up at 6-, 12-, and 24-month post-surgery, use of indirect calorimetry to measure REE rather than use population-based prediction equations, the regression equation used to predict REE was developed from the measured population and not previously published equations, and the inclusion of mostly Hispanic adults [14, 22]. Prior studies examining the impact of SG on REE and MA have focused mostly on White adults. Despite these strengths, we recognize limitations in our study. The data were collected as part of a cohort, with follow-up measures at 12 and 24 months optional; therefore, our attrition rate was ~ 29%. While dietary intake and physical activity are important components of weight loss and weight loss maintenance, they were not included in this post-hoc analysis. The SG participants studied had a high incidence of diabetes (~ 40%), common in the surgical population, but our analysis (data not shown) suggests unchanged REE measurements in diabetes compared to normoglycemia. In contrast, Betteni et al. found that individuals with pre-diabetes and T2D have slightly higher REE than individuals with normal glycemia [12]. Comorbidities and changing medication regimens are common in this population; however, we did not systematically collect this information, both of which could influence our findings. Our use of paired t-tests required complete data for both time points, which resulted in listwise deletion of participants with missing values at either assessment. This approach reduced the analytic sample size and may have introduced bias if the excluded participants systematically differed from those with complete data which limits generalizability. There are known errors with BIA in individuals with severe BMIs, which can contribute to potential inaccuracies in the estimate of FM and FFM [33]. This is demonstrated by the low proportion of variance between measured REE and FFM at baseline in our findings ($r^2 = 0.31$, $p = 0.008$, *Supplement* Fig. 2), where the proportion of variance between measured REE and FFM measured using the gold standard measure, dual energy x-ray absorptiometry, is $r^2 = 0.64$ [35]. Although REE accounts for the majority of TDEE, MA is also characterized by non-REE, specifically, activity energy expenditure [36]. There may have been compensatory changes in activity energy expenditure following MBS, such that physical activity increases as a result of improved muscle efficiency.

In conclusion, our study found that REE rapidly declines in the acute period following SG, supporting prior evidence in SG of a severe imbalance in energy intake and energy expenditure (e.g., semi-starvation state). Weight and FM loss continued despite the rapid decline and subsequent plateauing of REE and presence of MA post-surgery, reaching a nadir at 12 months.

While MA at 1.5 and 6 months was similar to prior published studies, these findings should be interpreted with caution, given the small sample size and potential methodological limitations in body composition measurement (e.g., BIA). Future studies should investigate potential mechanisms associated with energetic changes following SG, including alterations in FGF21 and the gut microbiota, as well as the mechanisms underlying increased physical activity following SG.

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Data Availability The data supporting the findings of this study are available from the corresponding author upon reasonable request. Researchers interested in accessing the data should contact the corresponding author directly.

Declarations

Competing Interests The authors declare no competing interests.

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