Surgical Weight Loss: Impact on Energy Expenditure

David Thivel, Katrina Brakonieki, Pascale Duché, Béatrice Morio, Yves Y. Boirie, Blandine Laferrère

Dear Author

Here are the proofs of your article.

- You can submit your corrections **online,** via **e-mail** or by **fax**.
- For **online** submission please insert your corrections in the online correction form. Always indicate the line number to which the correction refers.
- You can also insert your corrections in the proof PDF and **email** the annotated PDF.
- For **fax** submission, please ensure that your corrections are clearly legible. Use a fine black pen and write the correction in the margin, not too close to the edge of the page.
- Remember to note the **journal title**, **article number**, and **your name** when sending your response via e-mail or fax.
- **Check** the metadata sheet to make sure that the header information, especially author names and the corresponding affiliations are correctly shown.
- **Check** the questions that may have arisen during copy editing and insert your answers/corrections.
- **Check** that the text is complete and that all figures, tables and their legends are included. Also check the accuracy of special characters, equations, and electronic supplementary material if applicable. If necessary refer to the *Edited manuscript*.
- The publication of inaccurate data such as dosages and units can have serious consequences. Please take particular care that all such details are correct.
- Please **do not** make changes that involve only matters of style. We have generally introduced forms that follow the journal's style.
- Substantial changes in content, e.g., new results, corrected values, title and authorship are not allowed without the approval of the responsible editor. In such a case, please contact the Editorial Office and return his/her consent together with the proof.
- If we do not receive your corrections **within 48 hours**, we will send you a reminder.
- Your article will be published **Online First** approximately one week after receipt of your corrected proofs. This is the **official first publication** citable with the DOI. **Further changes are, therefore, not possible.**
- The **printed version** will follow in a forthcoming issue.

Please note

After online publication, subscribers (personal/institutional) to this journal will have access to the complete article via the DOI using the URL:

`http://dx.doi.org/10.1007/s11695-012-0839-1`

If you would like to know when your article has been published online, take advantage of our free alert service. For registration and further information, go to: http://www.springerlink.com.

Due to the electronic nature of the procedure, the manuscript and the original figures will only be returned to you on special request. When you return your corrections, please inform us, if you would like to have these documents returned.

Metadata of the article that will be visualized in OnlineFirst

Please note: Images will appear in color online but will be printed in black and white.

1	Article Title	**Surgical Weight Loss: Impact on Energy Expenditure**	
2	Article Sub- Title		
3	Article Copyright - Year	**Springer Science+Business Media New York 2012 (This will be the copyright line in the final PDF)**	
4	Journal Name	Obesity Surgery	
5	Corresponding Author	Family Name	**Thivel**
6		Particle	
7		Given Name	**David**
8		Suffix	
9		Organization	Clermont University, Blaise Pascal University
10		Division	Laboratory of the Metabolic Adaptations to Exercise under Physiological and Pathological Conditions (AME2P), EA 3533
11		Address	BP 80026, Aubière cedex 63171, France
12		Organization	St. Luke's Roosevelt Hospital Center
13		Division	New York Obesity Nutrition Research Center, Department of Medicine
14		Address	New York 10025, NY, USA
15		e-mail	thiveldavid@hotmail.com
16	Author	Family Name	**Brakonieki**
17		Particle	
18		Given Name	**Katrina**
19		Suffix	
20		Organization	St. Luke's Roosevelt Hospital Center
21		Division	New York Obesity Nutrition Research Center, Department of Medicine
22		Address	New York 10025, NY, USA
23		Organization	St. Luke's Roosevelt Hospital Center
24		Division	Division of Endocrinology and Diabetes, Department of Medicine
25		Address	New York 10025, NY, USA
26		e-mail	
27	Author	Family Name	**Duche**

28		Particle	
29		Given Name	**Pascale**
30		Suffix	
31		Organization	Clermont University, Blaise Pascal University
32		Division	Laboratory of the Metabolic Adaptations to Exercise under Physiological and Pathological Conditions (AME2P), EA 3533
33		Address	BP 80026, Aubière cedex 63171, France
34		e-mail	
35	Author	Family Name	**Béatrice**
36		Particle	
37		Given Name	**Morio**
38		Suffix	
39		Organization	Clermont Université, Université d'Auvergne
40		Division	Unité de Nutrition Humaine
41		Address	BP 10448, Clermont-Ferrand 63000, France
42		Organization	CRNH Auvergne
43		Division	INRA, UMR 1019, UNH
44		Address	Clermont-Ferrand 63000, France
45		e-mail	
46	Author	Family Name	**Yves**
47		Particle	
48		Given Name	**Boirie**
49		Suffix	
50		Organization	Clermont Université, Université d'Auvergne
51		Division	Unité de Nutrition Humaine
52		Address	BP 10448, Clermont-Ferrand 63000, France
53		Organization	CRNH Auvergne
54		Division	INRA, UMR 1019, UNH
55		Address	Clermont-Ferrand 63000, France
56		Organization	CHU Clermont-Ferrand
57		Division	Clinical Nutrition Department
58		Address	Clermont-Ferrand 63003, France
59		e-mail	
60	Author	Family Name	**Laferrère**
61		Particle	
62		Given Name	**Blandine**
63		Suffix	

64		Organization	St. Luke's Roosevelt Hospital Center
65		Division	New York Obesity Nutrition Research Center, Department of Medicine
66		Address	New York 10025, NY, USA
67		Organization	St. Luke's Roosevelt Hospital Center
68		Division	Division of Endocrinology and Diabetes, Department of Medicine
69		Address	New York 10025, NY, USA
70		Organization	Columbia University College of Physicians & Surgeons
71		Division	
72		Address	New York, NY, USA
73		e-mail	

74		Received	
75	Schedule	Revised	
76		Accepted	

77	Abstract	Diet-induced weight loss is often limited in its magnitude and often of short duration, followed by weight regain. On the contrary, bariatric surgery now commonly used in the treatment of severe obesity favors large and sustained weight loss, with resolution or improvement of most obesity-associated comorbidities. The mechanisms of sustained weight loss are not well understood. Whether changes in the various components of energy expenditure favor weight maintenance after bariatric surgery is unclear. While the impact of diet-induced weight loss on energy expenditure has been widely studied and reviewed, the impact of bariatric surgery on total energy expenditure, resting energy expenditure, and diet-induced thermogenesis remains unclear. Here, we review data on energy expenditure after bariatric surgery from animal and human studies. Bariatric surgery results in decreased total energy expenditure, mainly due to reduced resting energy expenditure and explained by a decreased in both fat-free mass and fat mass. Limited data suggest increased diet-induced thermogenesis after gastric bypass, a surgery that results in gut anatomical changes and modified the digestion processes. Physical activity and sustained intakes of dietary protein may be the best strategies available to increase non-resting and then total energy expenditure, as well as to prevent the decline in lean mass and resting energy expenditure.

78	Keywords separated by ' - '	Bariatric surgery - Severe obesity - Energy expenditure - Weight loss

79	Foot note information	

OBES SURG
DOI 10.1007/s11695-012-0839-1

REVIEW ARTICLE

Surgical Weight Loss: Impact on Energy Expenditure

David Thivel · Katrina Brakonieki · Pascale Duche · Morio Béatrice · Boirie Yves · Blandine Laferrère

Abstract Diet-induced weight loss is often limited in its magnitude and often of short duration, followed by weight regain. On the contrary, bariatric surgery now commonly used in the treatment of severe obesity favors large and sustained weight loss, with resolution or improvement of most obesity-associated comorbidities. The mechanisms of sustained weight loss are not well understood. Whether changes in the various components of energy expenditure favor weight maintenance after bariatric surgery is unclear. While the impact of diet-induced weight loss on energy expenditure has been widely studied and reviewed, the impact of bariatric surgery on total energy expenditure, resting energy expenditure, and diet-induced thermogenesis remains unclear. Here, we review data on energy expenditure after bariatric surgery from animal and human studies. Bariatric surgery results in decreased total energy expenditure, mainly due to reduced resting energy expenditure and explained by a decreased in both fat-free mass and fat mass. Limited data suggest increased diet-induced thermogenesis after gastric bypass, a surgery that results in gut anatomical changes and modified the digestion processes. Physical activity and sustained intakes of dietary protein may be the best strategies available to increase non-resting and then total energy expenditure, as well as to prevent the decline in lean mass and resting energy expenditure.

Keywords Bariatric surgery · Severe obesity · Energy expenditure · Weight loss

D. Thivel · K. Brakonieki · B. Laferrère
New York Obesity Nutrition Research Center,
Department of Medicine, St. Luke's Roosevelt Hospital Center,
New York, NY 10025, USA

D. Thivel (✉) · P. Duche
Laboratory of the Metabolic Adaptations to Exercise under Physiological and Pathological Conditions (AME2P),
EA 3533, Clermont University, Blaise Pascal University,
BP 80026, 63171 Aubière cedex, France
e-mail: thiveldavid@hotmail.com

K. Brakonieki · B. Laferrère
Division of Endocrinology and Diabetes, Department of Medicine,
St. Luke's Roosevelt Hospital Center, New York, NY 10025, USA

B. Laferrère
Columbia University College of Physicians & Surgeons,
New York, NY, USA

M. Béatrice · B. Yves
Unité de Nutrition Humaine, Clermont Université,
Université d'Auvergne, BP 10448,
63000 Clermont-Ferrand, France

M. Béatrice · B. Yves
INRA, UMR 1019, UNH, CRNH Auvergne,
63000 Clermont-Ferrand, France

B. Yves
Clinical Nutrition Department, CHU Clermont-Ferrand,
Clermont-Ferrand 63003, France

Introduction

The worldwide alarming progression of obesity and severe obesity has led to an array of diverse efforts aimed at developing effective weight loss strategies. Dietary restriction combined or not with physical activity programs are mainly used to induce a negative energy balance and subsequent weight loss. However, the weight loss is often of small magnitude and not sustained over time. Obesity surgery is currently the most effective treatment for severe obesity, resulting in significant and long-term weight loss, decreasing comorbidities, improving quality of life, and decreasing mortality [1–4]. The number of surgical procedures performed annually is increasing [5]. Restrictive surgical procedures such as laparoscopic adjustable gastric banding or vertical banded gastroplasty, malabsorptive procedures such as biliopancreatic diversion or duodenal

switch, or mixed intervention such as gastric bypass (GBP) are currently the most used surgical techniques for the treatment of severe obesity [6]. Although it was believed that GBP induced weight loss only via calorie restriction and nutrient malabsorption [7], it is now thought that this surgery may also increase satiety [8] via enhanced gut peptide release, alter palatability toward high-fat and sweetened food [9], modify taste [10–12], and alter the metabolism of bile acids [13], all processes that may favor weight loss and maintenance of reduced weight.

Diet-induced weight loss results in adaptative decrease in energy expenditure (EE), which may explain the difficulty to sustained weight loss overtime. On the contrary, patients undergoing bariatric surgery often experience sustained weight loss years after the surgery [14]. The mechanisms of sustained weight loss after the surgery are not well understood. Some have suggested that changes in postoperative energy expenditure could explain the sustained weight loss. Better understanding of the changes of various component of energy expenditure after surgery, and their relation to weight loss, may provide insight into the mechanism for weight loss after bariatric surgery. The aim of this review is to highlight existing literature on the impact of bariatric surgery on total energy expenditure (TEE), resting energy expenditure (REE), and diet-induced thermogenesis (DIT) in obese patients and to review the evidence, or absence, of a differential effect between diet- and bariatric surgery-induced weight loss on EE. The implication of the physiological mechanisms affected by massive weight loss such as body composition, gastric regulations, or nutrient partitioning will be discussed and considered in a clinical perspective.

Total Energy Expenditure

Decreased TEE has been observed after diet-induced weight loss in relation to decreased lean body mass (LBM) in obese adults and adolescents [15–17] and persist well beyond the period of dynamic weight loss [18]. In animal models, it has been shown that postoperative weight loss is not restrictively due to decreased energy intake, with operated rats losing more weight than pair-fed ones, which raises the hypothesis of other surgery-induced modifications likely affecting energy expenditure [19]. A higher total energy expenditure (assessed by open circuit indirect calorimetry in diet-induced obesity male Wistar rats) has effectively been found in rats after gastric bypass compared with fed- and body weight-matched controls [20]. Stylopoulos et al. also underlined an increase in both total (19 %) and resting energy expenditure (31 %) after gastric bypass in rats (Sprague–Dawley, Levin Sprague–Dawley, and Osborne Mendel) [21]. In this study, energy expenditure was also assessed in rats that underwent other surgical methods such as sleeve gastrectomy or gastric banding, but no energy expenditure modification was found postoperatively [21], suggesting that the type of surgical procedure may modulate subsequent changes in energy metabolism. Such an increased TEE in rats is however contradictory with the available literature in humans. Few studies have investigated the impact of bariatric surgery on TEE in humans (Table 1). Recently, Tamboli et al. assessed 24-h energy expenditure, using metabolic chambers, in 29 obese patients (body mass index (BMI) 43.6±5.5 kg/m^2) before, 6 and 12 months after Roux-en-Y gastric bypass (RYGBP) [22]. Their results show that the reduced fat mass and fat-free mass, assessed by DXA, was accompanied by a significantly decrease in TEE (−25 %) 6 months after surgery, with no further changes at 12 months. Previous studies have shown a 25 % decrease in TEE 14 months after RYGBP (using doubly labeled water) [23], and 3 and 12 months after vertical banded gastroplasty (VBG) (by indirect calorimetry) [24] (accompanied by a decreased of both fat mass (FM) and fat-free mass (FFM), as detailed in Table 2). The literature shows then the discrepancies between animal and human studies which are mainly explained by the fact that animal studies express EE relatively to body size while in human exploration EE is expressed relative to time. Comparisons between animal and human studies are then not possible.

Non-resting Energy Expenditure

While REE is the main component of TEE, some authors have been interested in non-resting energy expenditure (NREE). NREE accounts for approximately 30 to 35 % of TEE and is mainly determined by spontaneous physical activity [25]. It has been suggested that the decline in weight loss-induced TEE may be partly explained by a decreased in NREE in people decreasing their habitual physical activity level while dieting [15, 16, 26]. Even with unchanged physical activity behaviors, the lower energy needs during activity after weight loss, for the same activity, can also explain such a decreased NREE [26, 27]. After bariatric surgery, NREE has been found to decrease independent of the level of physical activity [23, 24]. Although Das et al. did not show any difference in physical activity level after surgery compared with preoperative values, a recent review suggest that the level of physical activity tends to increase after bariatric surgery [28]. Physical activity behaviors and the NREE can be modified by lifestyle interventions, contrary to REE, the main parameter of TEE, and/or DIT.

Resting Energy Expenditure

REE corresponds to the minimum energy needed to maintain an individual integrated system and homeothermic

Q4 **Table 1** Data referring to the impact of bariatric surgery on total energy expenditure

Authors	Population (n/BMI)	Surgery	Assessment periods	Energy expenditure measure	TEE
Das et al. [23]	30/50±9.3 kg/m^2	GBP	Preoperative After weight stabilization (WS) (14±2 months)	Doubly labeled water (15 days)	↓ by ≈25 % 14.8±2.6 to 11.2±3.1 MJ/day
van Gemert et al. [24]	8/45.87± 5.1 kg/m^2	Vertical banded gastroplasty	Preoperative 3 months post 12 months post	Doubly labeled water (14 days)	↓ Preoperative: 9,400± 1,300 J/min 3 months post: 6,700± 1,000 J/min 12 months post: 6,900± 1,200 J/min
Tamboli et al. [22]	29/43.6± 5.5 kg/m^2	RYGBP	Preoperative 6 months post 12 months post	Metabolic chamber	↓ at 6 months ↓ at 12 months Preoperative: 2,768± 474 kcal/day 6 months post: 2,010± 260 kcal/day 12 months post: 1,987± 228 kcal/day

Data are presented as mean ± standard deviations

↓, decrease, *post* postoperative, *n* sample size, *BMI* body mass index, *TEE* total energy expenditure, *RYGP* Roux-en-Y gastric bypass, *GPB* gastric bypass

temperature at rest. Diet-induced weight loss induces an important reduction in the REE (6 to 10 %), in association with decreased LBM, measured by dual-energy X-ray absorptiometry [29] or densitometry [30].

Bariatric surgery results in 30–40 % weight loss, both of FM and FFM, which may then highly impact REE. Although postoperative REE reduction has been mainly explained by the decreased FFM that accompanies weight loss [31–36], Das et al. suggests that both FFM and FM losses are responsible for the REE reduction (please see Table 2 for body composition assessment methods) [37]. However, body composition studies, particularly the measure of LBM, are difficult in the severely obese individuals since the physical size limitations imposed by severe obesity pose challenges to the measurement of body composition [37]. As illustrated in Table 2, various methods have been used to assess body composition in bariatric patients, which limits comparisons between studies. Further studies are needed to clearly establish the implication of body composition on the REE modifications during large weight loss, particularly after bariatric surgery. Interestingly, recent studies have determined the specific resting metabolic rates of major organs and tissues in the body in order to better adjust for the REE changes in relation to changes in specific regions of the body [38]. Current data available on the impact of bariatric surgery on REE are presented in Table 2. Patients who undergo surgical intervention experience decreased REE within few day postoperatively and some data underlined significant decreases at 6 weeks postoperatively [39], regardless of the surgical method used (RYGBP, open or laparoscopic RYGBP, vertical gastroplasty (VBG), or adjustable gastric banding) or the limb-length of the bypass [40]. Two different surgical methods and their impact on postoperative REE were compared in 36 obese patients undergoing RYGBP and 39 having VBG [9]. The two Q6 groups were matched in terms of preoperative REE, and both showed decreased REE 12 months after the operation (−498±273 and −481±234 kcal, respectively). REE at 12 months was not significantly different between groups [9]. According to these data and others [41] (Table 2), it is not the nature of the bariatric surgery but rather factors such as energy balance status (active weight loss, weight stability, or weight regain) or body composition that impact the postoperative change in REE.

Many factors can be implicated in the weight regain experienced by some patients after surgery such as unhealthy eating habits [42], progressive increase in food intake [43], or anatomical and physiological adaptations occurring over time [44]. In 2009, Faria et al. measured REE in patients that underwent RYGBP 2 years before their investigations [45]. Among the 36 patients enrolled, 15 were classified as healthy weight (no weight regain observed) whereas 21 experienced weight regain. According to the results of this cross-sectional study, individuals who experienced weight regain 2 years after RYGBP had lower REE, compared to the healthy weight group. Such results could

Table 2 Publications related to the effects of bariatric surgery on resting energy expenditure

Authors	Population	Surgery	Assessment periods	Body composition	Energy expenditure measure	REE
Flancbaum et al. [39]	70/52±10 kg/m^2	RYGBP	Preoperative	Not assessed	Indirect calorimetry (hood)	↓
			6 weeks post			Preoperative, 2,017± 700 kcal/day
			3 month post			6 weeks post, 1,983± 409 kcal/day
			6 months post			3 month post, 1,930± 352 kcal/day
			12 months post			6 months post, 1,868± 400 kcal/day
			18 months post			12 months post, 1,862± 326 kcal/day
			24 months post			18 months post, 1,831± 414 kcal/day
						24 months post, 1,873± 224 kcal/day
Das et al. [23], 2003	30/50±9.3 kg/m^2	GBP	Preoperative After weight stabilization (WS) (14±2 months)	Total body water ↓ FM (51.3±4.6 to 33.9±8.6 %) and FFM (72.2±23.0 to 30.1±13.1 kg)	Indirect calorimetry/ 30 min (hood)	↓ by ≈25 % 9.3±1.8 to 6.9±1.1 MJ/day
De Castro et al. [46]	21/47.31±5.81 kg/m^2	Banded RYGBP	Preoperative	Not assessed	Indirect calorimetry (hood)	↓
			3 months post			Preoperative, 2,006.7± 376.4 kcal/day
						3 months post, 1,763.3± 310.5 kcal/day
Tamboli et al. [22]	29/43.6±5.5 kg/m^2	RYGBP	Preoperative	DXA	Metabolic chamber	↓ at 6 months
			6 months post	↓ FM/FFM	From Sleep EE	↓ at 12 months
			12 months post	0–6 months, −38±9/−18±6 %		Preoperative, 2,092± 342 kcal/day
				6–12 months, −21±13/−1±5 %		6 months post, 1,495± 190 kcal/day
				0–12 months, −50±13/−19±7 %		12 months post, 1,513± 192 kcal/day
Faria et al. [45]	Total, 36	RYGBP	2 years postoperative	Bioelectrical multifrequency bioimpedance	Indirect calorimetry (hood)	WR REE (1,369.33 kcal/day) < HW REE (1,582.73 kcal/day)
	15 with healthy weight (HW)/27.90±3.76 kg/m^2			Fat mass WR, 34.51 % Fat mass HW, 30.59 %		

Table 2 (continued)

Authors	Population	Surgery	Assessment periods	Body composition	Energy expenditure measure	REE
	21 with weight regain (WR)/32.49 kg/m^{2a}			FFM data not provided		
Carey et al. [41]	19/48.5±2.5	16/laparoscopic RYGBP	Preoperative	Under water weighing	Indirect calorimetry (hood)	↓ significantly at 1 month
		2/open RYGBP	1 month post	↓ (FM/FFM)		Preoperative, 2,091.0±588.0 kcal/day
		1/laparoscopic adjustable banding	3 months post	Preoperative, 67.0±12.1/73.8±15.8 kg		1 month post, 1,758.4±412.1 kcal/day
			6 months post	1 month, 58.3±11.2/70.2±14.0 kg		3 months post, 1,647.1±306.0 kcal/day
				3 months, 50.5±11.6/64.4±12.5 kg		6 months post, 1,651.0±460.0 kcal/day
				6 months, 40.6±12.0/60.6±11.2 kg		
Benedetti et al. [54]	14 obese/132.66±18.90 kg 15 lean/62.96±7.46 kg	Biliopancreatic diversion (BPD)	Preoperative 30 months post	Total body water 30 months post (obese), ↓ FM (60.13 ±13.01 to 19.02±8.61 kg) and FFM (72.50±12.42 to 53.22±9.07 kg)	Indirect calorimetry	Preoperative, obese > lean 30 months post, ↓ REE in obese (2,293±284 to 1,640±254 kcal/24 h)
Carrasco et al. 2007	38/44.0±4.5 kg/m^2	RYGBP	Preoperative	Total body water	Indirect calorimetry in a ventilated chamber system	↓
			6 months post	↓ FM and FFM FM, 51.6±5.4 to 41.2±6.2 %		Preoperative, 1,845±302 kcal/day
				FFM, 56.1±10.2 to 48.0±7.3 kg		6 months post, 1,449±215 kcal/day
Bobbioni-Harsch et al.[34]	20/43.9±1.3 kg/m^2	RYGBP	Preoperative	Bioelectrical impedance	Indirect calorimetry (hood)	↓
			3 months post	↓ FFM (graphical reading)		Preoperative, 1,823±45 kcal/day
			6 months post	Preoperative, 60 kg		3 months post, 1,585±39 kcal/day
			12 months post	3 months post, 55 kg		6 months post, 1,529±34 kcal/day
				6 months post, 51 kg		12 months post, 1,475±34 kcal/day
				12 months post, 49 kg		
				FM results not provided		
Olbers et al.[9]	75	G1: RYGBP	Preoperative	DXA	Indirect calorimetry (hood)	↓
	G1, 36/42.3±4.5 kg/m^2	G2: vertical banded gastroplasty (LVBG)	12 months post	Greater FM reduction after LGBP		Preoperative,
	G2, 39/42.6±4.2 kg/m^2			1 year FM LGBP, 26.9±9.4 kg		G1, 2,156±618 kcal

JrnlID 11695_ArtID 839_Proof# 1 - 06/12/2012

Table 2 (continued)

Authors	Population	Surgery	Assessment periods	Body composition	Energy expenditure measure	REE
				1 year FM LVBG, 20.2±8.6 kg		G2, 2,237±344 kcal
						12 months post, G1=G2
						G1, −498±273 kcal
						G2, −481±234 kcal
Busetto et al. [32]	12/46.9±6.8 kg/m^2	Adjustable silicone gastric banding	Preoperative	Bioelectrical impedance	Indirect calorimetry (hood)	↓
			6 months post	↓ (FM/FFM)		Preoperative, 7.96±1.77 MJ/day
				Preop, 68.5±10.8/52.6±4.0 kg		6 months post, 6.57±6.90 MJ/day
				6 months, 48.5±9.2/47.9±4.6 kg		
van Gemert et al. [31]	15	Vertical banded gastroplasty	G1	Deuterium oxide component of doubly labeled water	Metabolic chamber	↓
	G1, 6/48.1±7.0 kg/m^2		Preoperative	G1, ↓ (FM/FFM)		Preoperative, 11.1±1.8 MJ/day
	G2, 9/45.7±5.7 kg/m^2		3 months post	Preop, 74.0±28.6/81.5±13.6 kg		3 months post, 8.4±1.6 MJ/day
			6 months post	3 months, 49.2±22.3/73.9±13.7 kg		6 months post, 7.9±1.6 MJ/day
			12 months post	6 months, 36.6±17.0/70.8±13.6 kg		12 months post, 8.1±0.9 MJ/day
			G2	12 months, 30.0±11.4/		REE G2 (>36 months) <
			>36 months post	72.3±13.0 kg		REE G1 preoperative
van Gemert et al. [24]	8/45.87±5.1 kg/m^2	Vertical banded gastroplasty	Preoperative 3 months post 12 months post	Deuterium oxide component of doubly labeled water	Metabolic chamber	↓
				↓ (FM/FFM)		Preoperative, 5,800±800 J/min
				Preoperative, 68.3±11.7/61.8±9.2 kg		3 months post, 4,400±400 J/min
				3 months, 50.8±11.1/53.0±6.2 kg		12 months post, 4,200±300 J/min
				12 months, 31.4±12.1/52.4±4.4 kg		
Galtier et al. [55]	73/43.3±7.0 kg/m^2	Laparoscopic adjustable banding	Preoperative	Bioelectrical multifrequency bioimpedance	Indirect calorimetry (hood)	↓ for each group[b]
			6–12 months post (n=39=G1)	↓ (FM/FFM)		

Journal 11695 Article 839 Proof# 1 - 06/12/2012

Table 2 (continued)

Authors	Population	Surgery	Assessment periods	Body composition	Energy expenditure measure	REE
			12–18 months post (n=21=G2)	Preoperative, 43.0± 2.6 %/66.5±8.9 kg		
			>18 months post (n=18=G3)	Postop, 35.5±7.2 %/ 57.5±7.6 kg		
				Postoperative results are the means for all groups		

Data are presented as mean ± standard deviations

↓ decrease, *post* postoperative, *n* sample size, *FM* fat mass, *FFM* fat-free mass, *BMI* body mass index, *REE* resting energy expenditure, *RYGP* Roux-en-Y gastric bypass, *GPB* gastric bypass

[a] SD were not provided for the WR group in Faria et al. [45]

[b] The original paper only presents results graphically

support the hypothesis that FFM loss, and the quality of FFM loss (in terms of fibers typology for instance) that accompanied body weight reduction after surgery, may be responsible for decreased REE, leading to an increased risk of weight regain. However, unfortunately, similarly to other studies of EE after bariatric surgery [39, 46], LBM was not measured in the study by Faria et al. [45].

In another study, REE was assessed in 70 morbidly obese patients (52±10 kg/m^2) up to 24 months postoperatively [39]. Preoperatively, they stratified participants based on actual measured and predicted REE values. They defined patients as "hypometabolic" when their measured REE was less than 85 % of the predicted REE, based on the Harris and Benedict equation [47], or "normometabolic" when it was within ±15 % of the predicted REE. The authors showed that the preoperative measured REE correlated with postoperative weight loss in "normo metabolic" patients. In hypometabolic patients however, REE increased toward normal range immediately after surgery. These differences between the two groups have been observed while both groups were on the same very low calorie diet [39]. Others have been interested in the impact of preoperative REE, on postoperative change in REE and weight loss [35, 36]. Data from these studies remain inconsistent, with some papers stating that preoperative REE may be predictive of weight loss 6 months after surgery [35], while others did not find any association up to 1 year after operation [36].

Since measuring REE needs an elaborated protocol realized under strictly controlled condition, some predictive equations, mainly based on gender, body weight, and age, have been developed and provide satisfactory results [48, 49]. The results obtained using such equations need however to be considered carefully, especially during longitudinal weight changes in adults or in obese adolescents [16]. Ruiz et al. have for instance recently compared measured REE by indirect calorimetry, with estimated REE before and after diet-induced weight loss in obese women [50]. According to their results, the best estimations of REE were not obtained using the same equation before and after weight loss. The equation proposed by Mifflin et al. [51] provided the best REE prediction at baseline, while after the 12-week diet, the best results were obtained using the equation proposed by Owen et al. [52]. In bariatric patient, van Gemert et al. [31] compared measured REE with predicted REE, using the equation proposed by Westerterp et al. [53], before and 3, 6, 12, and 36 months after vertical banded gastroplasty. Their results indicated that preoperatively, there was no difference between measured and calculated REE. However during the weight loss stages, at months 3, 6, and 12, REE was significantly overestimated when compared with measured values. This overestimation remained true during the weight stabilization period experienced by the patients more that 36 months after surgery. Although later studies obtained

similar results confirming an overestimation of REE when using predictive equations compared to measured values during the first months after surgery [41], others found no differences between measured and predicted REE before and 30 months after surgery [54]. In their study, Carey et al. used the equations proposed by Harris and Benedict [47] to estimate REE [41]. They found that 3 months postoperatively, using LBM instead of body weight in the equation, the equations lead to a reduction of the overestimation of REE (almost 112 kcal less). However, others did not confirm these results and showed no differences between the two methods [23, 34, 55]. The use of predictive equations to estimate REE, although offering translational applicability at population or clinical level, remains approximative and results from these equations should be used as indicators and not as the basis for any nutritional or energetic interventions.

Diet-Induced Thermogenesis

DIT approximately accounts for 10 % of TEE and is defined as the energy needed for digestion, absorption, and storage of nutrients from our food. The changes in any of the aforementioned processes justify the interest in DIT changes after surgery, particularly after GBP, where the anatomical and physiological functions of the gut undergo significant modifications. Postprandial physiological mechanisms have been shown to be involved in DIT. The response to a meal results in bile acid secretion as well as gut hormone release. Some of the gut hormones have been found to alter diet-induced energy expenditure. Although cholecystokinin (CCK) does not seem to affect DIT as reflected by a study based on CCK-KO mice [56], peptide YY (PYY) or glucagon-like peptide 1 (GLP-1) may do. Studies have effectively suggested the role of PYY in energy expenditure modulations [57, 58]. In the arcuate nucleus of the hypothalamus, PYY binds to inhibitory Y2 receptor (Y2R), where neuro-peptide Y (NPY) and pro-opiomelanocortine (POMC) neurons are located. PYY binds to Y2R on NPY neurons, inhibiting orexigenic NPY secretion, which in turn results in a greater POMC activation and thus secretion of anorexigenic hormones (alpha-melanocyte-stimulating hormone) [59–61] ultimately leading to an increase in total energy expenditure. In humans, correlations have been found between PYY concentration and REE [62, 63] and infusion of PYY have been shown to result in increased TEE in both lean and obese adult [64]. Polypeptide P has also been implicated in the regulation of energy expenditure in rodents, with peripheral administration favoring decreased total expenditure [65]. Data regarding the role of GLP-1 are inconsistent, with some animal studies showing increased TEE after both central and peripheral infusion [66, 67], whereas GLP-1 infusion in lean and/or obese humans led to reduced DIT and postprandial CHO oxidation [68, 69]. In 2006, Pannacciulli et al. found a positive association between fasting plasma GLP-1 concentrations and REE in humans, independent of body composition [70]. Bile acids (BA) have also been implicated in the regulation of oxygen consumption and energy expenditure [71]. So far, in vivo data on the relationships between BA and EE are mainly restricted to animal work. In humans, Brufau et al. did not find any association between bile acid and resting energy expenditure [71], contrary to Ockenga et al. who found a positive association between serum BA levels and EE (DIT) and to VO_2, in ten healthy individuals and eight patients with liver cirrhosis [72].

Bariatric surgery, particularly RYGBP, results in change of meal pattern and size, decrease energy intake during meals, change in food choices and taste, maldigestion, possible nutrient malabsorption [73–76], and enhanced postprandial release of GLP-1, PYY [77–79], and oxyntomodulin [80], all of which could impact DIT after GBP. To our knowledge, there are only two studies on DIT after GBP in humans [23, 81]. In one longitudinal study where DIT, measured by indirect calorimetry for 4 h after a 1.67-MJ meal (43.9 g carbohydrate= 44 % of energy/12.0 g protein=12 % of energy/19.9 g fat= 44 % of energy), did not differ 14 months after surgery compared to preoperative values (n=30 patients). On the contrary, a recent cross-sectional study suggests that DIT (assessed by indirect calorimetry) increased by 200 % 12 months after RYGBP, compared to a control group [81]. As previously underlined between human and animal studies on total energy expenditure, it has to be noticed that those two last studies did express EE differently which certainly explain their different conclusions.

Bueter et al. however found greater energy expenditure in rats that underwent GBP after a 5-g test meal compare to a control group, underlying the impact of surgery on DIT [20]. Further investigations are needed to know the exact impact of surgery on DIT and whether or not it can contribute to the decreased TEE observed in operated obese individuals and thus maybe play a role in weight regain.

Clinical Implications

Bariatric surgery is currently the best way to achieve significant and sustained weight loss. With weight loss, both fat mass and lean body mass decrease, which results in decreased REE. Such decreased REE may then limit weight loss over time and even favor weight regain in some patients. Diet-induced weight loss is also associated with long-term changes in hormonal profiles, i.e., leptin, ghrelin, peptide YY, gastric inhibitory polypeptide (this nomenclature is not sure for GIP anymore, should be glucose-dependent insulinotropic

polypeptide), amylin, pancreatic polypeptide, and cholecystokinin, which may together influence TEE and appetite control favoring a rapid weight regain [82]. Further studies are needed to establish whether or not interventional strategies (clinical or behavioral) may be a great solution to maintain energy expenditure and then limit weight regain.

Physical activity represents the main tool that can be used to maintain energy expenditure after weight loss. Although physical activity is considered as a cornerstone in the nonsurgical treatment of obesity for weight loss and maintenance [83, 84], very few data are available on physical activity level (PAL) of patients that underwent bariatric surgery. Jacobi et al. have recently reviewed this topic and concluded that PAL tends to increase postoperatively [28]. However, they pointed out that one study assessed PAL 10 years postoperatively and observed a weight regain accompanied by declined PAL [1]. The increased PAL reported in most of the studies has to be considered with caution regarding the subjective nature of most of the results that are based on self-reported questionnaires. Overreporting is an inherent limitation of validity when using such PA questionnaires [85], particularly in obese people [86]. Few objective data are then available regarding the level of physical activity in such patients, and even less is known in terms of exercise prescription (frequency, intensity, and duration). Shang and Hasenberg randomly assigned 60 obese patients that underwent RYGBP to either a low aerobic exercise program (1 h/week) or a multiple aerobic session intervention (2×1 h/week) and found a lower decreased lean body mass in the multiple exercise group, underlying then the qualitative importance of physical activity to prevent the fat-free mass reduction that occurs after surgery [87]. Although aerobic exercise leads to improved type 1a (aerobic) muscle fibers which increases the patients aerobic capacities and activity and then favors a greater energy expenditure, further work are needed to question the impact of resistance training that should favor a higher muscle mass. Making people engage in physical activity remains difficult at a time where sedentary behaviors are prevalent and particularly in obese persons with comorbidities limiting their mobility. Recently, Vatier et al. described changes in both physical activity and sedentary behaviors of obese patients after GBP [88] Self-reported physical activity and time spent watching TV (as a typical sedentary behavior) and body composition (assessed by DXA) were assessed in 86 obese patients (BMI 41.3–53.5 kg m^{-2}) prior, 6, and 12 months after RYGBP. Their results pointed out that the increased leisure time physical activity is accompanied by a decrease in the time spent to sedentary activities, which is related to body composition improvements 1 year after surgery (mean loss of weight −37.1 kg, fat mass −25.7 kg, lean body mass −9.4 kg).

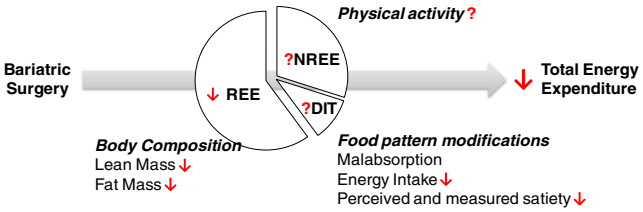

Fig. 1 Actual evidence regarding the impact of bariatric surgery on total (TEE), resting (REE), non-resting (NREE), and diet-induced (DIT) energy expenditure (*downward arrow* decrease; *upward arrow* increase; *question mark* remains unknown)

Dietary strategies, particularly with high protein diet [89], may be used to prevent the decline in lean mass and counteract the reduced energy expenditure after surgery. There is effectively increasing evidence to support that a high protein supplementation may promote weight loss and prevent weight regain thanks to its impact on diet-induced thermogenesis, satiety, and muscle mass conservation [90–92]. Faria et al. recently reviewed the implications of protein diet in bariatric patients [89]. They concluded that high protein supplementation can lead to increased satiety, weight loss enhancement, and improved body composition in such patients. According to their data, the quality and nature of the protein are as important as the quantity, with leucine favoring a better muscle mass maintenance. Indeed new concepts like the "slow/fast protein" concept could be applied to obese subjects especially after bariatric surgery [93]. More experimental studies are necessary to develop dietary recommendations to be done in bariatric patients who may be at risk for protein deficiency after surgery [94]. Indeed, these patients often have inadequate protein intake and/or absorption because of reduced energy intake and/or food intolerance [45, 94, 95]. As a result, bariatric patients have difficulties maintaining the recommended levels of protein consumption (expressed per kilogram of body weight) [45]. Dietary strategies are thus necessary, particularly in terms of protein intake, to avoid protein deficiency and prevent the decline of lean mass and resting energy expenditure.

Conclusion

Patients who undergo bariatric surgery experience a decreased TEE, mainly due to reduced REE, explained by a decreased LBM, similarly to patients after diet-induced weight loss (Fig. 1). There is little evidence so far that surgical weight loss modifies the various components of EE differentially than dietary calorie restriction, and that altered EE may explain the sustained weight loss after surgery. However, there are numerous changes in hormones involved in the regulation of energy homeostasis. Moreover, assessment of body composition in severely obese patients

is not always optimal mainly due to methodological limitations. The anatomical changes resulting from intestinal modifications after bypass surgeries may modify DIT. For now, similarly to diet-induced weight loss, physical activity and dietary protein intake appear as the best strategies available to increase NREE and TEE, and to prevent the decline in LBM and REE after surgical weight loss.

Acknowledgments The Dr Laferrere and Miss Brakoniecki are supported through the NIH NIDDK ROI 067561 grant. The Dr Thivel is supported by the Clinical Nutrition and Metabolism Society through its 2011 Research Award.

References

1. Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351:2683–93.
2. Sjostrom L, Narbro K, Sjostrom CD, et al. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357:741–52.
3. Adams TD, Gress RE, Smith SC, et al. Long-term mortality after gastric bypass surgery. N Engl J Med. 2007;357:753–61.
4. Buchwald H, Estok R, Fahrbach K, et al. Weight and type 2 diabetes after bariatric surgery: systematic review and meta-analysis. Am J Med. 2009;122:248–56. e245.
5. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2008. Obes Surg. 2009;19:1605–11.
6. Pories WJ. Bariatric surgery: risks and rewards. J Clin Endocrinol Metab. 2008;93:S89–96.
7. Greenway FL. Surgery for obesity. Endocrinol Metab Clin North Am. 1996;25:1005–27.
8. Borg CM, le Roux CW, Ghatei MA, et al. Progressive rise in gut hormone levels after Roux-en-Y gastric bypass suggests gut adaptation and explains altered satiety. Br J Surg. 2006;93:210–5.
9. Olbers T, Bjorkman S, Lindroos A, et al. Body composition, dietary intake, and energy expenditure after laparoscopic Roux-en-Y gastric bypass and laparoscopic vertical banded gastroplasty: a randomized clinical trial. Ann Surg. 2006;244:715–22.
10. Scruggs DM, Buffington C, Cowan Jr GS. Taste acuity of the morbidly obese before and after gastric bypass surgery. Obes Surg. 1994;4:24–8.
11. Burge JC, Schaumburg JZ, Choban PS, et al. Changes in patients' taste acuity after Roux-en-Y gastric bypass for clinically severe obesity. J Am Diet Assoc. 1995;95:666–70.
12. Tichansky DS, Boughter Jr JD, Madan AK. Taste change after laparoscopic Roux-en-Y gastric bypass and laparoscopic adjustable gastric banding. Surg Obes Relat Dis. 2006;2:440–4.
13. Nyhlin H, Brydon G, Danielsson A, et al. Bile acid malabsorption after intestinal bypass surgery for obesity. A comparison between jejunoileal shunt and biliointestinal bypass. Int J Obes. 1990;14:47–55.
14. Anonymous. The SBU on overweight and obesity: Huge increase of overweight-related diseases. Lakartidningen. 2002; 99:3188–92
15. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. N Engl J Med. 1995;332:621–8.
16. Weigle DS, Sande KJ, Iverius PH, et al. Weight loss leads to a marked decrease in nonresting energy expenditure in ambulatory human subjects. Metabolism. 1988;37:930–6.
17. Bray GA, Smith SR, Dejonge L, et al. Effect of diet composition on energy expenditure during weight loss: the POUNDS LOST Study. Int J Obes (Lond). 2011;36:448–55.
18. Rosenbaum M, Hirsch J, Gallagher DA, et al. Long-term persistence of adaptive thermogenesis in subjects who have maintained a reduced body weight. Am J Clin Nutr. 2008;88:906–12.
19. le Roux CW, Aylwin SJ, Batterham RL, et al. Gut hormone profiles following bariatric surgery favor an anorectic state, facilitate weight loss, and improve metabolic parameters. Ann Surg. 2006;243:108–14.
20. Bueter M, Lowenstein C, Olbers T, et al. Gastric bypass increases energy expenditure in rats. Gastroenterology. 2010;138:1845–53.
21. Stylopoulos N, Hoppin AG, Kaplan LM. Roux-en-Y gastric bypass enhances energy expenditure and extends lifespan in diet-induced obese rats. Obesity Silver Spring. 2009;17:1839–47.
22. Tamboli RA, Hossain HA, Marks PA, et al. Body composition and energy metabolism following Roux-en-Y gastric bypass surgery. Obesity Silver Spring. 2010;18:1718–24.
23. Das SK, Roberts SB, McCrory MA, et al. Long-term changes in energy expenditure and body composition after massive weight loss induced by gastric bypass surgery. Am J Clin Nutr. 2003;78:22–30.
24. van Gemert WG, Westerterp KR, van Acker BA, et al. Energy, substrate and protein metabolism in morbid obesity before, during and after massive weight loss. Int J Obes Rel Metab Disord. 2000;24:711–8.
25. Ravussin E. SBA: energy metabolism. In: Stunkard AJ, editor. Obesity theory and therapy. New York: Raven; 1993.
26. Weigle DS, Brunzell JD. Assessment of energy expenditure in ambulatory reduced-obese subjects by the techniques of weight stabilization and exogenous weight replacement. Int J Obes. 1990;14 Suppl 1:69–77. discussion 77–81.
27. Peyrot N, Morin JB, Thivel D, et al. Mechanical work and metabolic cost of walking after weight loss in obese adolescents. Med Sci Sports Exerc. 2010;42:1914–22.
28. Jacobi D, Ciangura C, Couet C, et al. Physical activity and weight loss following bariatric surgery. Obes Rev. 2011;12:366–77.
29. Wang X, You T, Lenchik L, et al. Resting energy expenditure changes with weight loss: racial differences. Obesity Silver Spring. 2010;18:86–91.
30. Foster GD, Wadden TA, Swain RM, et al. Changes in resting energy expenditure after weight loss in obese African American and White women. Am J Clin Nutr. 1999;69:13–7.
31. van Gemert WG, Westerterp KR, Greve JW, et al. Reduction of sleeping metabolic rate after vertical banded gastroplasty. Int J Obes Relat Metab Disord. 1998;22:343–8.
32. Busetto E, Hrdy J. Compensation of aberration of inclined X-ray monochromators. J Synchrotron Radiat. 1995;2:288–91.
33. Buscemi S, Caimi G, Verga S. Resting metabolic rate and post-absorptive substrate oxidation in morbidly obese subjects before and after massive weight loss. Int J Obes Relat Metab Disord. 1996;20:41–6.
34. Bobbioni-Harsch E, Morel P, Huber O, et al. Energy economy hampers body weight loss after gastric bypass. J Clin Endocrinol Metab. 2000;85:4695–700.
35. Ott MT, Ott L, Haack D, et al. The MEE/PEE ratio as a predictor of excess weight loss for up to 1 year after vertical banded gastroplasty. Arch Surg. 1992;127:1089–93.
36. Camerini G, Adami GF, Marinari GM, et al. Failure of preoperative resting energy expenditure in predicting weight loss after gastroplasty. Obes Res. 2001;9:589–91.
37. Das SK. Body composition measurement in severe obesity. Curr Opin Clin Nutr Metab Care. 2005;8:602–6.
38. Wang Z, Ying Z, Bosy-Westphal A, et al. Evaluation of specific metabolic rates of major organs and tissues: comparison between nonobese and obese women. Obesity Silver Spring. 2012;20:95–100.

39. Flancbaum L, Choban PS, Bradley LR, et al. Changes in measured resting energy expenditure after Roux-en-Y gastric bypass for clinically severe obesity. Surgery. 1997;122:943–9.
40. Flancbaum L, Verducci JS, Choban PS. Changes in measured resting energy expenditure after Roux-en-Y gastric bypass for clinically severe obesity are not related to bypass limb-length. Obes Surg. 1998;8:437–43.
41. Carey DG, Pliego GJ, Raymond RL. Body composition and metabolic changes following bariatric surgery: effects on fat mass, lean mass and basal metabolic rate: six months to one-year follow-up. Obes Surg. 2006;16:1602–8.
42. Sallet PC, Sallet JA, Dixon JB, et al. Eating behavior as a prognostic factor for weight loss after gastric bypass. Obes Surg. 2007;17:445–51.
43. Warde-Kamar J, Rogers M, Flancbaum L, et al. Calorie intake and meal patterns up to 4 years after Roux-en-Y gastric bypass surgery. Obes Surg. 2004;14:1070–9.
44. Brolin RE. Weight gain after short- and long-limb gastric bypass in patients followed for longer than 10 years. Ann Surg. 2007;246:163–4. author reply 164.
45. Faria SL, Faria OP, Lopes TC, et al. Relation between carbohydrate intake and weight loss after bariatric surgery. Obes Surg. 2009;19:708–16.
46. De Castro CM, de Lima Montebelo MI, Rasera Jr I, et al. Effects of Roux-en-Y gastric bypass on resting energy expenditure in women. Obes Surg. 2008;18:1376–80.
47. Harris J. BF: a biometric study of basal metabolism in man. Washington: Carnegie Institute of Washington; 1919.
48. WHO. Organization: energy and protein requirements. Report of a joint FAO/WHO/UNU expert consultation. vol. 206. Geneva: WHO; 1985
49. NR Council. Recommended dietary allowances. 10th edn. Washington: NR Council; 1989.
50. Ruiz JR, Ortega FB, Rodriguez G, et al. Validity of resting energy expenditure predictive equations before and after an energy-restricted diet intervention in obese women. PLoS One. 2011;6:e23759.
51. Mifflin MD, St Jeor ST, Hill LA, et al. A new predictive equation for resting energy expenditure in healthy individuals. Am J Clin Nutr. 1990;51:241–7.
52. Owen OE, Kavle E, Owen RS, et al. A reappraisal of caloric requirements in healthy women. Am J Clin Nutr. 1986;44:1–19.
53. Westerterp KR, Donkers JH, Fredrix EW, et al. Energy intake, physical activity and body weight: a simulation model. Br J Nutr. 1995;73:337–47.
54. Benedetti G, Mingrone G, Marcoccia S, et al. Body composition and energy expenditure after weight loss following bariatric surgery. J Am Coll Nutr. 2000;19:270–4.
55. Galtier F, Farret A, Verdier R, et al. Resting energy expenditure and fuel metabolism following laparoscopic adjustable gastric banding in severely obese women: relationships with excess weight lost. Int J Obes Lond. 2006;30:1104–10.
56. Lo CM, Samuelson LC, Chambers JB, et al. Characterization of mice lacking the gene for cholecystokinin. Am J Physiol Regul Integr Comp Physiol. 2008;294:R803–10.
57. Edelsbrunner ME, Herzog H, Holzer P. Evidence from knockout mice that peptide YY and neuropeptide Y enforce murine locomotion, exploration and ingestive behaviour in a circadian cycle- and gender-dependent manner. Behav Brain Res. 2009;203:97–107.
58. Sloth B, Holst JJ, Flint A, et al. Effects of PYY1-36 and PYY3-36 on appetite, energy intake, energy expenditure, glucose and fat metabolism in obese and lean subjects. Am J Physiol Endocrinol Metab. 2007;292:E1062–8.
59. Murphy KG, Bloom SR. Gut hormones in the control of appetite. Exp Physiol. 2004;89:507–16.
60. Murphy KG, Dhillo WS, Bloom SR. Gut peptides in the regulation of food intake and energy homeostasis. Endocr Rev. 2006;27:719–27.
61. Stanley S, Wynne K, Bloom S. Gastrointestinal satiety signals III. Glucagon-like peptide 1, oxyntomodulin, peptide YY, and pancreatic polypeptide. Am J Physiol Gastrointest Liver Physiol. 2004;286:693–7.
62. Guo Y, Ma L, Enriori PJ, et al. Physiological evidence for the involvement of peptide YY in the regulation of energy homeostasis in humans. Obesity Silver Spring. 2006;14:1562–70.
63. Hill BR, De Souza MJ, Williams NI. Characterization of the diurnal rhythm of peptide YY and its association with energy balance parameters in normal-weight premenopausal women. Am J Physiol Endocrinol Metab. 2011;301:E409–15.
64. Sloth B, Davidsen L, Holst JJ, et al. Effect of subcutaneous injections of PYY1-36 and PYY3-36 on appetite, ad libitum energy intake, and plasma free fatty acid concentration in obese males. Am J Physiol Endocrinol Metab. 2007;293:E604–9.
65. Asakawa A, Inui A, Yuzuriha H, et al. Characterization of the effects of pancreatic polypeptide in the regulation of energy balance. Gastroenterology. 2003;124:1325–36.
66. Hwa JJ, Ghibaudi L, Williams P, et al. Differential effects of intracerebroventricular glucagon-like peptide-1 on feeding and energy expenditure regulation. Peptides. 1998;19:869–75.
67. Osaka T, Endo M, Yamakawa M, et al. Energy expenditure by intravenous administration of glucagon-like peptide-1 mediated by the lower brainstem and sympathoadrenal system. Peptides. 2005;26:1623–31.
68. Flint A, Raben A, Rehfeld JF, et al. The effect of glucagon-like peptide-1 on energy expenditure and substrate metabolism in humans. Int J Obes Relat Metab Disord. 2000;24:288–98.
69. Flint A, Raben A, Ersboll AK, et al. The effect of physiological levels of glucagon-like peptide-1 on appetite, gastric emptying, energy and substrate metabolism in obesity. Int J Obes Relat Metab Disord. 2001;25:781–92.
70. Pannacciulli N, Bunt JC, Koska J, et al. Higher fasting plasma concentrations of glucagon-like peptide 1 are associated with higher resting energy expenditure and fat oxidation rates in humans. Am J Clin Nutr. 2006;84:556–60.
71. Brufau G, Bahr MJ, Staels B, et al. Plasma bile acids are not associated with energy metabolism in humans. Nutr Metab (Lond). 2010; 7:73.
72. Ockenga J, Valentini L, Schuetz T, et al. Plasma bile acids are associated with energy expenditure and thyroid function in humans. J Clin Endocrinol Metab. 2012;97:535–42.
73. Brolin RE, Kenler HA, Gorman JH, et al. Long-limb gastric bypass in the superobese. A prospective randomized study. Ann Surg. 1992;215:387–95.
74. Crenn P, Morin MC, Joly F, et al. Net digestive absorption and adaptive hyperphagia in adult short bowel patients. Gut. 2004;53:1279–86.
75. Farrel J. Digestion and absorption of nutrients and vitamins. In: Brandt LJ, Eldman M, editors. Sleisenger & Fordtran's gastrointestinal and liver disease. Saunders Elsevier: Philadelphia; 2006. p. 2181–4.
76. Odstrcil EA, Martinez JG, Santa Ana CA, et al. The contribution of malabsorption to the reduction in net energy absorption after long-limb Roux-en-Y gastric bypass. Am J Clin Nutr. 2010;92:704–13.
77. Morinigo R, Moize V, Musri M, et al. Glucagon-like peptide-1, peptide YY, hunger, and satiety after gastric bypass surgery in morbidly obese subjects. J Clin Endocrinol Metab. 2006;91:1735–40.
78. Bose M, Teixeira J, Olivan B, et al. Weight loss and incretin responsiveness improve glucose control independently after gastric bypass surgery. J Diabetes. 2010;2:47–55.

79. Laferrere B. Effect of gastric bypass surgery on the incretins. Diabetes Metab. 2009;35:513–7.
80. Laferrere B, Swerdlow N, Bawa B, et al. Rise of oxyntomodulin in response to oral glucose after gastric bypass surgery in patients with type 2 diabetes. J Clin Endocrinol Metab. 2010;95:4072–6.
81. Faria SL, Faria OP, de Almeida Cardeal M, et al. Diet-induced thermogenesis and respiratory quotient after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2012;8:797–802.
82. Sumithran P, Prendergast LA, Delbridge E, et al. Long-term persistence of hormonal adaptations to weight loss. N Engl J Med. 2011;365:1597–604.
83. Jakicic JM, Clark K, Coleman E, et al. American college of sports medicine position stand. Appropriate intervention strategies for weight loss and prevention of weight regain for adults. Med Sci Sports Exerc. 2001;33:2145–56.
84. Wing RR, Phelan S. Long-term weight loss maintenance. Am J Clin Nutr. 2005;82:222S–5S.
85. Shephard RJ. Limits to the measurement of habitual physical activity by questionnaires. Br J Sports Med. 2003;37:197–206. discussion 206.
86. Lichtman SW, Pisarska K, Berman ER, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med. 1992;327:1893–8.
87. Shang E, Hasenberg T. Aerobic endurance training improves weight loss, body composition, and co-morbidities in patients after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6:260–6.
88. Vatier C, Henegar C, Ciangura C, et al. Dynamic relations between sedentary behavior, physical activity, and body composition after bariatric surgery. Obes Surg. 2012;22:1251–6.
89. Faria SL, Faria OP, Buffington C, et al. Dietary protein intake and bariatric surgery patients: a review. Obes Surg. 2011;21:1798–805.
90. Lorenzen J, Frederiksen R, Hoppe C, et al. The effect of milk proteins on appetite regulation and diet-induced thermogenesis. Eur J Clin Nutr. 2012;66:622–7.
91. Smeets AJ, Soenen S, Luscombe-Marsh ND, et al. Energy expenditure, satiety, and plasma ghrelin, glucagon-like peptide 1, and peptide tyrosine-tyrosine concentrations following a single high-protein lunch. J Nutr. 2008;138:698–702.
92. Lejeune MP, Westerterp KR, Adam TC, et al. Ghrelin and glucagon-like peptide 1 concentrations, 24-h satiety, and energy and substrate metabolism during a high-protein diet and measured in a respiration chamber. Am J Clin Nutr. 2006;83:89–94.
93. Boirie Y, Dangin M, Gachon P, et al. Slow and fast dietary proteins differently modulate postprandial protein accretion. Proc Natl Acad Sci U S A. 1997;94:14930–5.
94. Andreu A, Moize V, Rodriguez L, et al. Protein intake, body composition, and protein status following bariatric surgery. Obes Surg. 2010;20:1509–15.
95. Moize V, Geliebter A, Gluck ME, et al. Obese patients have inadequate protein intake related to protein intolerance up to 1 year following Roux-en-Y gastric bypass. Obes Surg. 2003;13:23–8.

AUTHOR QUERIES

AUTHOR PLEASE ANSWER ALL QUERIES.

Q1. Please check captured email address of the corresponding author if correct.
Q2. Please check the authors' affiliations if presented correctly.
Q3. The abbreviated term “EE” was provided with its expanded form “energy expenditure.” Please check if correct.
Q4. Please check the changes made in Tables 1 and 2 if correct.
Q5. “Carrasco et al. 2007” is cited in the body but its bibliographic information is missing. Kindly provide its bibliographic information. Otherwise, please delete it from the text/body.
Q6. The occurrences of “(9)” were changed to reference citation “[9].” Please check if correct.
Q7. The detail “(16)” was changed to reference citation “[16].” Please check if correct.
Q8. Please check the details in this sentence if presented correctly.
Q9. Missing citation for Fig. 1 was inserted here. Please check if correct.