Surgical Weight Loss: Impact on Energy Expenditure

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

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David Thivel, Katrina Brakonieki, Pascale Duché, Béatrice Morio, Yves Y. Boirie, et al.. Surgical Weight Loss: Impact on Energy Expenditure. Obesity Surgery, 2013, 23 (2), pp.255-266. ⟨10.1007/s11695-012-0839-1⟩. ⟨hal-02646060⟩

HAL Id: hal-02646060
https://hal.inrae.fr/hal-02646060v1

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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	
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Thivel, D., Brakonieki, K., Duche, P., Morio, B., Boirie, Y., Laferrere, B. (2013). Surgical Weight Loss: Impact on Energy Expenditure. Obesity Surgery, 23 (2), 255-266. DOI : 10.1007/s11695-012-0839-1

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	Received	
Schedule	Revised	
	Accepted	

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

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Thivel, D., Brakonieki, K., Duche, P., Morio, B., Boirie, Y., Laferrere, B. (2013). Surgical Weight Loss: Impact on Energy Expenditure. Obesity Surgery, 23 (2), 255-266. DOI : 10.1007/s11695-012-0839-1

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.

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

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

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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 Q3 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

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	Doubly labeled water (14 days)	↓
			3 months post		Preoperative: 9,400± 1,300 J/min
			12 months post		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	Metabolic chamber	↓ at 6 months
			6 months post		↓ at 12 months
			12 months post		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 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

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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	Biliopancreatic diversion (BPD)	Preoperative	Total body water	Indirect calorimetry	Preoperative, obese > lean
	15 lean/62.96±7.46 kg		30 months post	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)		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, 49 kg		12 months post, 1,475± 34 kcal/day
				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

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 >36 months post	12 months, 30.0±11.4/ 72.3±13.0 kg		REE G2 (>36 months) < 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)		

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

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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

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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 Q8 1 year after surgery (mean loss of weight −37.1 kg, fat mass −25.7 kg, lean body mass −9.4 kg).

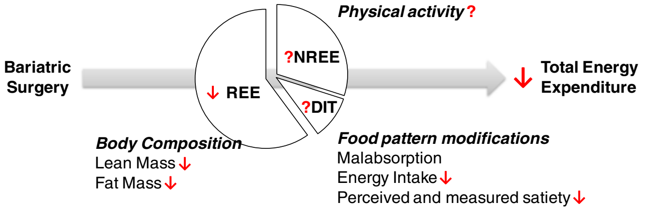

Fig. 1 Actual evidence regarding the impact of bariatric surgery on total (TEE), resting (REE), non-resting (NREE), and diet-induced (DIT) energy expenditure (*downward arrow* decrease; *upward arrow* increase; *question mark* remains unknown)

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 Q9 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.

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