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Other Reviews

Relative changes in resting energy expenditure during weight loss: a systematic review

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Summary

A more comprehensive understanding of the effects of weight loss on the changes in resting energy expenditure (EE) is relevant. A MEDLINE search was performed to identify studies with information relevant to this systematic review. From this search, the mean rate of resting EE decrease relative to weight loss was calculated from 90 available publications. A decrease of resting EE relative to weight loss of -15.4 ± 8.7 kcal kg^{-1} was observed from 2996 subjects. No sex differences were noted in the overall resting EE decrease relative to weight loss. However, a significant sex differences was seen with pharmacological interventions, which seemed to depress the resting EE relative to weight loss to a greater extent in men than in women ($P < 0.05$). A greater drop in resting EE relative to weight loss was observed for short interventions (more than 2 but less than 6 weeks) when compared with long interventions (<6 weeks) (-27.7 ± 6.7 vs. -12.8 ± 7.1 kcal kg^{-1}) ($P < 0.001$). Men and women have a similar decrease in resting EE relative to weight loss except in the case of pharmacological interventions. Short interventions also produced greater resting EE losses relative to weight loss.

Keywords: Energy expenditure, obesity, weight loss.

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Introduction

One of the better documented effects of weight loss is that it is accompanied by a decrease in resting energy expenditure (EE) (1,2). Resting EE, which represents the energy needed to support minimal daily functions (3), comprises two-thirds of total daily EE (4). As such, it is not surprising to note that the weight loss-induced decrease in resting EE has also been shown to be a determinant of weight regain (5).

Because successful long-term weight loss necessarily results from a matching of energy intake to the decrease of EE that occurs with weight loss, it is important to obtain values that accurately reflect changes of resting EE with respect to the lost weight. This is particularly relevant in the context where post-weight loss resting EE is lower than that values obtained with prediction equations (6,7). Indeed, Leibel *et al.* determined that maintenance of a body weight 10% or more below the initial body weight resulted in a resting EE of ~8 kcal kg^{-1} d^{-1} lower than the EE of the initial body weight in the obese (7). It thus becomes useful and relevant to gather information on resting EE relative to changes in body-weight loss resulting from interventions aimed at inducing prolonged negative energy balance. As such, the objective of this study was to perform a systematic review of peer reviewed studies including values pertaining to resting EE, resting metabolic rate, sleeping metabolic rate and basal metabolic rate before and after weight loss for both sexes, and for different weight-loss strategies. These values were in turn used to determine weighted mean rates of the decreases in resting EE relative to body-weight loss in response to different interventions.

Methods

The primary objective of this review was to determine the resting EE decrease relative to weight loss in obese

individuals (kcal kg^{-1}). Papers were identified from 1969 to 2008 through the MEDLINE database utilizing different combinations of the following keywords: resting metabolic rate, resting EE, sleeping metabolic rate, basal metabolic rate, weight loss, obesity, diet, exercise, drug therapy and bariatric surgery. Abstracts of papers that matched the initial criteria were carefully reviewed and of those, 156 were selected to be thoroughly analysed for relevance. It is important to note that the term resting EE will be used throughout this study to represent resting EE, resting metabolic rate, basal metabolic rate and sleeping metabolic rate although it is understood that by definition and in theory the latter two are different from resting EE. The selection of papers was carried out systematically through specific collection criteria. In order to be included in the study, the publications had (i) To include specific information on type of weight-loss interventions; (ii) To be performed on overweight or obese adults who were otherwise healthy, except in the case of surgical interventions where individuals were only considered candidates for some of the procedures if they had comorbidities such as diabetes and hypertension and (iii) To have values of resting EE or resting metabolic rate or basal metabolic rate or sleeping metabolic rate and body weight before and after the intervention. For studies dealing with more than one study group, all those groups that fit the inclusion criteria within that study were included and treated as individual sets of data. Reasons for exclusion are described in Table 1. Furthermore, the groups were categorized into the following (i) Participant sex; (ii) Type of intervention and (iii) The length of the study. Of the original 135 papers, 90 were included for the review and subsequent calculations.

Conversion to kcal d^{-1} was necessary for some of the studies where EE was expressed in kilojoules (kj) or mega joules (MJ); in such circumstances, 1 kcal = 4.184 kj = 0.004184 MJ. In the cases where studies reported oxygen consumption (8–10), the Weir formula (11) was used to determine the caloric equivalent of oxygen consumption. The respiratory exchange ratio for these calculations was assumed to be 0.78 for both men and women before weight loss and 0.79 after weight loss based on previous results (2).

Once all the relevant information for the 90 studies was retrieved, the relative changes of resting EE, basal metabolic rate, sleeping metabolic rate or resting metabolic rate (RMR) were calculated. This was done by dividing the absolute decrease in EE (kcal) by the absolute decrease in body weight (kg).

$$[\Delta \text{kcal d}^{-1} \text{ of RMR or REE}] \div [\Delta \text{kg}]$$

Statistical analyses

The means and standard deviations of the groups were determined by weighing the means from the different studies for all the group comparisons that were performed (Appendix 1). This process entailed pooling the number of subjects from each study as well as their means together and determining what percentage of the total each study contributed to the final group mean, which was considered as the weight of the study. All results are reported as the mean ± SD and range of values for each of the groups are reported in parenthesis. Groups were then compared by performing a two-tailed *t*-test designed specifically for groups with unequal *n* values. A series of correlation analyses were also performed between the relative changes in EE and the magnitude of the caloric restriction, and the changes in fat mass and fat-free mass. Differences were considered significant at a confidence interval of 95% ($P < 0.05$).

Table 1 Main reasons for exclusion of publications

Criteria for exclusion	% of all excluded papers ($n = 66$)
Compared obese vs. non-obese or post-obese	21.2
Not enough information provided (i.e. body weight not reported or omission of pre/post-intervention values, intervention not specified)	19.7
Subjects were not adults (under 18 years)	13.6
Subjects were unhealthy or diseased (i.e. ischaemia, diabetes etc.)	12.1
Intervention was less than 2 weeks	10.6
Intervention not specified or no control of intervention (too many different interventions between subjects)	6.1
Focused on the effect of overfeeding	3.0
Subjects were of regular weight for their age and sex	3.0
No intervention or weight loss and/or a comparison with a standard resting metabolic rate equation	3.0
Only provided values for total energy expenditure using doubly labelled water	1.5
Focused only on energy intake with resting energy expenditure (no weight loss)	1.5
Focused only on exercise energy expenditure without resting energy expenditure	1.5
Focused on weight gain	1.5
Only one subject used	1.5

Results

The summary of the results for each of the studies is reported in Table 2. In all, there were 2996 subjects when combining men and women from all of the selected studies. For all weight-loss interventions the mean decrease of resting EE was –15.4 ± 8.7 (–52.6–4.4) kcal kg^{-1} of weight

Table 2 Characteristics of included studies

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Women				
Auvichayapat *et al.* (43)	30 overweight women (BMI 27.42 ± 3.26)	Pharmacological/dietary: 250 mg green tea capsule and caloric restriction	12 weeks	22.6
Auvichayapat *et al.* (43)	30 overweight women (BMI 28 ± 3.51)	Diet: placebo and caloric restriction	12 weeks	19.3
Barnard *et al.* (44)	29 overweight women	Diet: low-fat vegan diet	14 weeks	–23.3
Bessard *et al.* (45)	6 obese women (Body fat 39.1 ± 1.1%)	Dietary: caloric restriction	11 weeks	–29.2
Bobbioni-Harsch *et al.* (24)	20 obese women (BMI 43.9 ± 1.3)	Surgical: Roux-en-Y gastric bypass surgery	12 months	–9.4
Bray (1)	6 obese women	Dietary: caloric restriction	31 d	–32.1
Brehm *et al.* (46)	20 obese women (BMI 33.5 ± 0.5)	Diet: low fat	4 months	–8.4
Brehm *et al.* (46)	20 obese women (BMI 32.8 ± 0.5)	Diet: low carbohydrate	4 months	–13.3
Burgess (47)	9 obese women	Diet: caloric restriction	12 weeks	–11.7
Busetto *et al.* (26)	15 obese women (BMI > 30)	Surgery: liposuction (ultrasound-assisted megalipoplasty)	180 d	–24.7
Cavallo *et al.* (48)	27 obese women	Diet: caloric restriction	15 d	–37.3
Coupaye *et al.* (49)	36 obese women (BMI 47.2 ± 8.5)	Surgical: adjustable gastric banding	1 year	–12.6
Coxon *et al.* (50)	12 overweight women (BMI 30 ± 7.1)	Diet: caloric restriction	8 weeks	–15.4
Coxon *et al.* (50)	14 overweight women (BMI 29.9 ± 4.8	Diet: caloric restriction	8 weeks	–10.3
de Boer *et al.* (51)	14 overweight women (BMI > 25)	Dietary: caloric restriction	10 weeks	–27.8
de Castro Cesar *et al.* (27)	21 obese women (BMI 47.31 ± 5.81)	Surgical: Roux-en-Y gastric bypass surgery	3 months	–11.8
den Besten *et al.* (52)	7 abdominally obese women (BMI 34.6 ± 1.7)	Diet: caloric restriction	8 weeks	–12.6
den Besten *et al.* (52)	8 gluteal-femoral obese women (BMI 31.6 ± 0.6)	Diet: caloric restriction	8 weeks	–3.5
Diepvens *et al.* (53)	23 overweight women (BMI 27.7 ± 1.8)	Pharmacological/dietary: 310 mg green tea capsule and caloric restriction	32 d	–12.8
Diepvens *et al.* (53)	24 overweight women (BMI 27.7 ± 1.8)	Diet: placebo and caloric restriction	32 d	–24.9
Diepvens *et al.* (54)	22 overweight women (BMI 28.9 ± 1.7)	Pharmacological/dietary: Olibra yogurt with caloric restriction	6 weeks	–9.2
Diepvens *et al.* (54)	28 overweight women (BMI 28.5 ± 2.2)	Diet: placebo and caloric restriction	6 weeks	–18.6
Dionne *et al.* (55)	10 overweight women (49.9 ± 8.1 kg fat mass)	Pharmacological/dietary: 60 mg fenfluramine & caloric restriction	15 weeks	–7.8
Donnelly *et al.* (56)	26 obese women (BMI 38.2 ± 5.9)	Diet: caloric restriction (liquid formula)	90 d	–6.8
Donnelly *et al.* (56)	16 obese women (BMI 37.5 ± 6.0)	Diet and exercise: caloric restriction and aerobic exercise	90 d	–7.4
Donnelly *et al.* (56)	18 obese women (BMI 38.2 ± 7.5)	Diet and Exercise: caloric restriction and resistance exercise	90 d	–8.9
Donnelly *et al.* (56)	9 obese women (BMI 38.3 ± 5.2)	Diet and Exercise: caloric restriction and resistance/aerobic combined	90 d	–9.5
Doucet *et al.* (2)	19 obese women (BMI 36.5 ± 0.8)	Pharmacological/dietary: 60 mg fenfluramine & non-macronutrient-specific caloric restriction	15 weeks	–3.6

Table 2 *Continued*

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Finer *et al.* (57)	5 obese women	Diet: caloric restriction via jaw wiring	90–250 d	–12.4
Foster *et al.* (58)	8 obese women	Diet: caloric restriction with balance deficit	8 weeks	–3.6
Foster *et al.* (58)	5 obese women	Diet: caloric restriction	8 weeks	–18.4
Foster *et al.* (39)	24 Overweight black women (mean BMI of 36.8 ± 4.4)	Dietary: caloric restriction	16 weeks	–12.7
Foster *et al.* (39)	85 overweight white women (mean BMI of 36.1 ± 5.1)	Dietary: caloric restriction	16 weeks	–6.6
Fricker *et al.* (59)	6 obese women (BMI 33.3 ± 2.6)	Diet: caloric restriction	3 weeks	–36.1
Froidevaux *et al.* (60)	17 obese women (BMI 29.8 ± 6.5)	Dietary: caloric restriction	12 ± 4 weeks	–12.8
Galtier *et al.* (61)	73 obese women (BMI 44.3 ± 7)	Surgical: laparoscopic adjustable banding	13.3 ± 6 months	–13.1
Garrow and Webster (8)	103 obese women (BMI 38 ± 8)	Dietary: caloric restriction	21 d	–31.4
Giese *et al.* (62)	14 overweight/obese women (BMI 29.1 ± 2.3)	Surgery: liposuction (large volume)	4 months	–0.5
Hainer *et al.* (63)	14 pairs of obese twins (28) (BMI 34.2 ± 7.8)	Diet and exercise: caloric restriction combined with aerobic exercise	4 weeks	–41.5
Hendler *et al.* (64)	6 obese women	Diet: high-protein, low-carbohydrate caloric restriction	30 d	–14.2
Hendler *et al.* (64)	4 obese women	Diet: pure sucrose caloric restriction	15 d	–17.8
Henson *et al.* (65)	7 moderately obese women (38.9 ± 1% fat mass)	Diet: caloric restriction & exercise: strenuous cycling 5 d week^{-1}	9 weeks	–19.3
Hill *et al.* (66)	3 obese women (BMI 35 ± 2)	Dietary: caloric restriction	5 weeks	–26.4
Hill *et al.* (66)	5 obese women (BMI 36 ± 1)	Exercise: daily walking	5 weeks	–30.7
Hill *et al.* (67)	7 obese women (43.8 ± 1.1% body fat)	Diet: caloric restriction	12 weeks	–26.0
Hill *et al.* (67)	16 obese women (43.9 ± 1.1% body fat)	Diet and Exercise: caloric restriction combined with aerobic walking programme	12 weeks	–11.5
Hunter *et al.* (68)	14 overweight women (BMI 29.1 ± 1.2)	Diet and exercise: caloric restriction and aerobic exercise	~25 weeks	–5.2
Hunter *et al.* (68)	16 overweight women (BMI 28.2 ± 1.5)	Diet and exercise: caloric restriction and aerobic exercise	~25 weeks	–6.1
Hunter *et al.* (68)	20 overweight women (BMI 27.9 ± 1.1)	Diet and exercise: caloric restriction and resistance exercise	~25 weeks	–5.5
Hunter *et al.* (68)	17 overweight women (BMI 28.2 ± 1.2)	Diet and exercise: caloric restriction and resistance exercise	~25 weeks	–3.5
Hunter *et al.* (68)	14 overweight women (BMI 28.3 ± 1.4)	Diet: caloric restriction	~25 weeks	–5.2
Hunter *et al.* (68)	13 overweight women (BMI 28.5 ± 1.3)	Diet: caloric restriction	~25 weeks	–10.1
Keim *et al.* (18)	5 obese women (36 ± 1% body fat)	Diet and exercise: caloric restriction combined with aerobic exercise	12 weeks	–10.6
Keim *et al.* (18)	5 obese women (35 ± 2% body fat)	Exercise: aerobic walking workout	12 weeks	3.2
Kempen *et al.* (69)	7 overweight/obese women (BMI 28–38)	Diet: caloric restriction (liquid formula)	4 weeks	–24.3
Kraemer *et al.* (70)	8 overweight women (BMI 27.3 ± 3.1)	Diet: caloric restriction	12 weeks	–12.1
Kraemer *et al.* (70)	9 overweight women (BMI 28.3 ± 4.2)	Diet and exercise: caloric restriction and aerobic exercise	12 weeks	–4.4

Table 2 *Continued*

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Kraemer *et al.* (70)	8 overweight/obese women (BMI 30.5 ± 5.1)	Diet and Exercise: caloric restriction and aerobic combined with strength	12 weeks	–20.4
Kucio *et al.* (71)	10 obese women (BMI 42.5 ± 2.7)	Pharmacological with diet: 5 mg yohimbine 4x per day with caloric restriction	6 weeks	–28.9
Kucio *et al.* (71)	10 obese women (BMI 40.3 ± 2.0)	Diet: placebo and caloric restriction	6 weeks	–52.6
Menozzi *et al.* (72)	71 obese women (BMI 40.3 ± 7)	Dietary: caloric restriction	20 d	–33.9
Mueller-Cunningham *et al.* (73)	54 overweight women (BMI 29.6 ± 6.3)	Diet: very low-fat diet	8 months	–12.2
Pasiakos *et al.* (74)	39 overweight and obese women (BMI 30.2 ± 0.5)	Diet and exercise: caloric restriction and aerobic stepping programme with pedometer	10 weeks	–9.0
Ravussin *et al.* (75)	5 obese women (BMI 34 ± 2.6)	Dietary: macronutrient-specific caloric restriction	10–16 weeks	–12.1
Refsum *et al.* (10)	34 obese women (BMI 40.9)	Surgical: gastric banding surgery	1 year	–10.9
Seagle *et al.* (76)	15 obese women (BMI 32.7 ± 0.9)	Pharmacological with diet: 10 mg sibutramine with caloric restriction	8 weeks	–27.6
Seagle *et al.* (76)	14 obese women (BMI 33.1 ± 1.0)	Pharmacological with diet: 30 mg sibutramine with caloric restriction	8 weeks	–24.3
Seagle *et al.* (76)	15 obese women (BMI 33.1 ± 1.0	Diet: placebo and caloric restriction	8 weeks	–19.7
Sheu *et al.* (77)	10 obese women (BMI 30.2 ± 0.6)	Diet and exercise: caloric restriction and non-restricted recreation	10–12 weeks	–10.1
Surwit *et al.* (78)	20 obese women (BMI 35.93 ± 4.8)	Diet: high-sucrose caloric restriction	6 weeks	–31.9
Surwit *et al.* (78)	22 obese women (BMI 40.3 ± 7.3)	Diet: low-sucrose caloric restriction	6 weeks	–25.8
Svendsen *et al.* (79)	49 overweight women (BMI > 25)	Diet: caloric restriction	12 weeks	–19.7
Svendsen *et al.* (79)	47 overweight women (BMI > 25)	Diet and exercise: caloric restriction and aerobic with resistance exercise	12 weeks	–19.5
Tagliaferri *et al.* (80)	10 obese women (BMI 36.3 ± 0.49)	Diet: placebo and caloric restriction	4 weeks	–41.3
Valtuena *et al.* (81)	9 obese women (BMI 43.6 ± 5.1)	Diet: caloric restriction	28 d	–27.2
Van Gaal *et al.* (82)	17 obese women (BMI 37.4 ± 1.2)	Pharmacological/dietary: 30 mg dexfenfluramine and caloric restriction	3 months	–4.9
Van Gaal *et al.* (82)	15 obese women (BMI 35.9 ± 0.9)	Diet: placebo and caloric restriction	3 months	–12.4
Vazquez and Kazi (83)	8 obese women (BMI 41 ± 5)	Diet: caloric restriction (ketogenic)	28 d	–13.8
Vazquez and Kazi (83)	8 obese women (BMI 37 ± 6)	Diet: caloric restriction	28 d	–31.3
Wadden *et al.* (21)	29 obese women (BMI 36.4 ± 5.5)	Dietary: caloric restriction	48 weeks	–8.9
Wadden *et al.* (21)	31 obese women (BMI 37.3 ± 5.1)	Exercise: aerobic step programme w/ progressive intensity increase	48 weeks	–4.6
Wadden *et al.* (21)	31 obese women (BMI 36.5 ± 6)	Exercise: strength training progressive resistance targeting large muscle groups	48 weeks	–10.8
Wadden *et al.* (21)	29 obese women (BMI 35.3 ± 4.4)	Combined exercise: aerobic step programme w/ progressive intensity increase (40% of time), strength training progressive resistance targeting large muscle groups (60% of time)	48 weeks	–5.7
Walsh *et al.* (84)	10 obese women (BMI 34.4 ± 3.9)	Pharmacological/dietary: 15 mg sibutramine & caloric restriction	12 weeks	–12.6
Walsh *et al.* (84)	9 obese women (BMI 34.5 ± 3.5)	Placebo/dietary: caloric restriction	12 weeks	–32.9

Table 2 *Continued*

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Warwick and Garrow (85)	2 obese women	Diet and exercise: caloric restriction with aerobic exercise	12–13 weeks	–7.4
Weinsier *et al.* (86)	24 overweight women (BMI 27.9 ± 1.8)	Diet: caloric restriction	15.4 ± 2.5 weeks	–15.6
Welle *et al.* (87)	6 obese women	Diet: caloric restriction	5 weeks	–13.1
Men				
Berube-Parent *et al.* (88)	8 obese men (BMI 30–40)	Pharmacological/dietary/exercise: 10 mg sibutramine with caloric restriction & aerobic exercise	12 weeks	–30.9
Burgess (47)	8 obese men	Diet: caloric restriction	12 weeks	–25.3
Chaput *et al.* (89)	11 obese men (BMI 33.5 ± 0.9)	Diet and exercise: caloric restriction combined with aerobic exercise	7.4 ± 1.9 months	–20.7
Chaput *et al.* (90)	11 obese men (BMI 31.3 ± 0.9)	Diet and exercise: caloric restriction and aerobic exercise	85.6 ± 46.4 d	–22.5
Dionne *et al.* (55)	10 overweight men (BMI 40.4 ± 9.8 kg fat mass)	Pharmacological/dietary: 60 mg fenfluramine & caloric restriction	15 weeks	–19.1
Doucet *et al.* (2)	16 obese men (BMI 33.9 ± 0.6)	Pharmacological/dietary: 60 mg fenfluramine & non-macronutrient-specific caloric restriction	15 weeks	–24.2
Finer *et al.* (57)	6 obese men	Diet: caloric restriction via jaw wiring	90–250 d	–16.4
Frey-Hewitt *et al.* (19)	36 overweight men (25.5 ± 5.8 kg fat mass)	Diet: caloric restriction	1 year	–22.3
Frey-Hewitt *et al.* (19)	44 overweight men (25.3 ± 6.3 kg fat mass)	Exercise: aerobic (walk/jog programme)	1 year	–5.6
Ravussin *et al.* (75)	2 obese men (BMI 36.3 ± 0.8)	Dietary: macronutrient-specific caloric restriction	10–16 weeks	–14.4
Sum *et al.* (91)	42 obese men (BMI 33.2 ± 0.8)	Exercise: aerobic and resistance combination (military)	5 months	–6.8
Tremblay *et al.* (92)	35 obese men (BMI 31.7 ± 2.6)	Pharmacological: placebo/Topiramate titrated to 200 mg	1 year	–10.6
Tremblay *et al.* (92)	33 obese men (BMI 31.8 ± 2.6)	Pharmacological: Topiramate titrated to 200 mg	1 year	–38.6
Verdich *et al.* (34)	21 obese men (BMI 38.6)	Diet: caloric restriction	8 weeks	–10.0
Weigle and Brunzell (93)	5 obese men	Diet: caloric restriction (liquid formula)	95 ± 14 d	–10.6
Weigle and Brunzell (93)	5 obese men	Exercise: weighted vest compensating for lost weight	95 ± 14 d	–9.3
Combined				
Abete *et al.* (94)	16 obese subjects (BMI 32.2 ± 4.4)	Diet: high glycaemic index caloric restriction	8 weeks	–22.8
Abete *et al.* (94)	16 obese subjects (BMI 32.8 ± 4.3)	Diet: low glycaemic index caloric restriction	8 weeks	–19.8
Amatruda *et al.* (95)	18 obese subjects (Body fat 41 ± 4%)	Dietary: caloric restriction, exercise encouraged but not prescribed	32–48 weeks	–8.1
Andersen *et al.* (96)	18 overweight subjects (BMI 30.8 ± 4.2)	Diet and exercise: caloric restriction and aerobic exercise	12 weeks	–24.3
Andersen *et al.* (96)	21 overweight subjects (BMI 31 ± 3.1)	Diet and exercise: caloric restriction and increases in lifestyle activity	12 weeks	–30.5
Ballor *et al.* (20)	18 obese subjects (BMI > 32)	Exercise: aerobic or resistance	12 weeks	–4.8
Ballor *et al.* (97)	20 obese subjects (BMI > 32)	Diet: caloric restriction	11 weeks	–29.0

Table 2 *Continued*

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Bryner *et al.* (98)	10 obese subjects (BMI 35.2 ± 3.9)	Diet and exercise: caloric restriction and aerobic exercise	12 weeks	–11.7
Bryner *et al.* (98)	10 obese subjects (BMI 35.5 ± 2.0)	Diet and exercise: caloric restriction and resistance exercise	12 weeks	4.4
Buscemi *et al.* (99)	10 obese subjects (BMI 53.7 ± 2.1)	Surgical: biliopancreatic bypass surgery	36–42 months	–11.4
Carey *et al.* (100)	19 obese subjects (BMI 48.7 ± 2.5)	Surgical: bariatric surgery	3–6 months	–11.1
Carrasco *et al.* (25)	38 obese subjects (34 women, 4 men) (BMI 44 ± 4.5)	Surgical: Roux-en-Y gastric bypass surgery	1 year	–11.9
Das *et al.* (101)	30 obese subjects (24 women, 6 men) (BMI 48.3 ± 8.2)	Surgical: gastric bypass surgery	14 ± 2 months	–10.7
Das *et al.* (101)	15 overweight subjects (BMI 27.5 ± 1.6)	Diet: high glycaemic index caloric restriction	12 months	–8.3
Das *et al.* (101)	14 overweight subjects (BMI 27.6 ± 1.2)	Diet: low glycaemic index caloric restriction	12 months	–5.8
del Genio *et al.* (102)	20 obese subjects (BMI 50.4 ± 6.5)	Surgical: laproscopic bariatric surgery	6 weeks	–13.0
del Genio *et al.* (102)	20 obese subjects (BMI 50.1 ± 8.5)	Diet and exercise: caloric restriction and walking	30 weeks	–14.9
Flancbaum *et al.* (28)	70 obese subjects (BMI 52 ± 10)	Surgical: Roux-en-Y gastric bypass surgery	12 months	–3.4
Geliebter *et al.* (103)	20 obese subjects (42.1 ± 14 kg fat mass)	Diet and exercise: caloric restriction and resistance exercise	8 weeks	–9.3
Geliebter *et al.* (103)	23 obese subjects (38.4 ± 12.5 kg fat mass)	Diet and exercise: caloric restriction and aerobic exercise	8 weeks	–16.3
Geliebter *et al.* (103)	22 obese subjects (33.8 ± 11.5 kg fat mass)	Diet: caloric restriction	8 weeks	–15.5
Kamphuis *et al.* (104)	14 overweight subjects (BMI 25–30)	Diet: caloric restriction	3 weeks	–24.3
Kamphuis *et al.* (104)	13 overweight subjects (BMI 25–30)	Diet: caloric restriction	3 weeks	–17.4
Kamphuis *et al.* (104)	13 overweight subjects (BMI 25–30)	Diet: caloric restriction	3 weeks	–26.6
Kamphuis *et al.* (104)	14 overweight subjects (BMI 25–30)	Diet: caloric restriction	3 weeks	–25.2
Karhunen *et al.* (105)	36 obese subjects (BMI 35.9 ± 3.9)	Pharmacological & dietary: Orlistat with mild caloric restriction	1 year	–10.1
Karhunen *et al.* (105)	36 obese subjects (BMI 35.9 ± 3.9)	Pharmacological & dietary: placebo with mild caloric restriction	1 year	–14.5
King *et al.* (106)	35 obese subjects (BMI 31.8 ± 4.1)	Exercise: aerobic	12 weeks	–40.1
Leibel *et al.* (7)	9 obese subjects (BMI > 28)	Dietary: caloric restriction (formula)	6–14 weeks	–16.3
Leibel *et al.* (7)	10 obese subjects (BMI > 28)	Dietary: caloric restriction (formula)	6–14 weeks	–13.8
Martin *et al.* (107)	12 overweight subjects (BMI 27.8 ± 1.4)	Diet: caloric restriction	6 months	–7.0
Martin *et al.* (107)	12 overweight subjects (BMI 27.5 ± 1.6)	Diet and exercise: caloric restriction and aerobic exercise	6 months	–12.2
Martin *et al.* (107)	12 overweight subjects (BMI 27.7 ± 1.8)	Diet: low-calorie diet	6 months	–11.7
Pasquali *et al.* (9)	5 obese subjects (BMI 45.2 ± 8.7)	Pharmacological with diet: 50 mg ephedrine 3x per day with caloric restriction	6 weeks	–12.8

Table 2 *Continued*

Study	Participants	Intervention	Duration	ΔREE (kcal kg^{-1})
Pasquali *et al.* (9)	5 obese subjects (BMI 39.1 ± 3.5)	Pharmacological with diet: 50 mg ephedrine 3x per day with caloric restriction	6 weeks	–11.6
Pereira *et al.* (108)	17 overweight/obese subjects (BMI > 27)	Diet: low-fat caloric restriction	6–10 weeks	–18.5
Pereira *et al.* (108)	22 overweight/obese subjects (BMI > 27)	Diet: low glycaemic index caloric restriction	6–10 weeks	–10.0
Valtuena *et al.* (109)	8 morbidly obese subjects (BMI 45 ± 5.0)	Diet and exercise: caloric restriction (liquid) and aerobic exercise	28 d	–20.3
van Gemert *et al.* (110)	8 obese subjects (BMI 45.9 ± 6.6)	Surgical: vertical banded gastroplasty	12 months	–11.9
Vogels *et al.* (111)	91 overweight/obese subjects (BMI 30.2 ± 3.1)	Diet: caloric restriction	N/A	–18.2
Welle *et al.* (38)	5 obese subjects	Diet: caloric restriction	2 weeks	–41.8
Westerterp *et al.* (112)	5 obese subjects (BMI 42–62)	Surgical: vertical banded gastroplasty	54 weeks	–13.7
Westerterp-Plantenga *et al.* (113)	50 overweight/ obese subjects (BMI 29.3 ± 2.5)	Diet: caloric restriction	4 weeks	–27.0
Westerterp-Plantenga *et al.* (113)	53 overweight/ obese subjects (BMI 29.7 ± 2.6)	Diet: caloric restriction	4 weeks	–18.4
Zavala and Printen (114)	13 morbidly obese (BMI 54)	Surgical: gastric bypass surgery	6.9 months	–11.1
Zenk *et al.* (12)	19 overweight/obese subjects	Pharmacological with diet and exercise: 'Lean System 7' with caloric restriction and aerobic exercise	8 weeks	3.2
Zenk *et al.* (12)	16 overweight/obese subjects	Diet and exercise: placebo with caloric restriction and aerobic exercise	8 weeks	–0.3

BMI, body mass index; N/A, not available; REE, resting energy expenditure.

loss. No significant difference in the changes of resting EE decreases relative to weight loss were found when dividing these studies into sex irrespective of the intervention performed with values and ranges of –16.8 ± 9.2 (–52.6–3.2) and –16.5 ± 8.6 (–38.6–[–5.6]) kcal kg^{-1} for men and women respectively. Additionally, the studies that did not include information as to the sex of subjects ($n = 1040$) yielded a mean resting EE loss of –13.3 ± 8.8 (–41.8–22.6) kcal kg^{-1} of weight loss (Fig. 2).

Type of intervention

Figure 1 displays the results for the various weight-loss strategies. In total, 2977 subjects could be classified as receiving diet therapy, exercise intervention, the combination of diet and exercise, pharmacological therapy or surgical intervention. The 19 subjects in the experimental group of the Zenk *et al.* study (12) could not be included because the protocol combined pharmacological, dietary and exercise weight-loss strategies. Resting EE changes resulting from diet interventions generated the greatest losses of resting EE ranging from 19.3 to –52.6 kcal kg^{-1} weight loss with a mean change of –18.4 ± 8.4 kcal kg^{-1}, which was a statistically greater decline in comparison with exercise ($P < 0.05$), pharmacological intervention ($P < 0.05$), surgery ($P < 0.001$) and the combination of diet and exercise ($P < 0.05$). No other statistical differences were noted between groups.

When combining all types of weight-loss interventions, the overall decrease in resting EE relative to weight loss was not significantly different between men and women (Fig 2). Similarly, the decrease in resting EE decreases relative to weight loss of each of the interventions was not different between men and women, except for pharmaceutical interventions where a significantly greater relative decrease occurred in men when compared with women with changes of –24.2 ± 10.4 (–38.6–[–10.6]) vs. –13.5 ± 7.9 (–28.9–[–3.6]) kcal kg^{-1} respectively ($P < 0.05$, see Fig. 3).

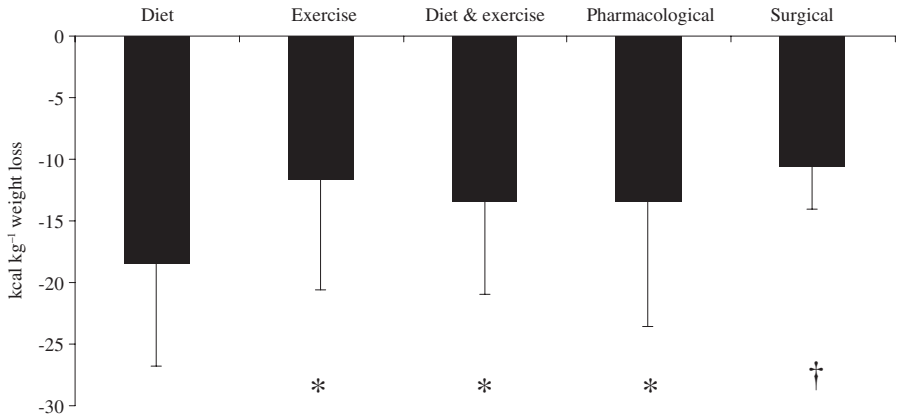

Figure 1 Comparison of the mean rate of changes in resting energy expenditure relative to weight loss with different weight-loss interventions in all men and women ($n = 2983$). * and † indicate significant difference from diet at $P < 0.05$ and $P < 0.001$ respectively.

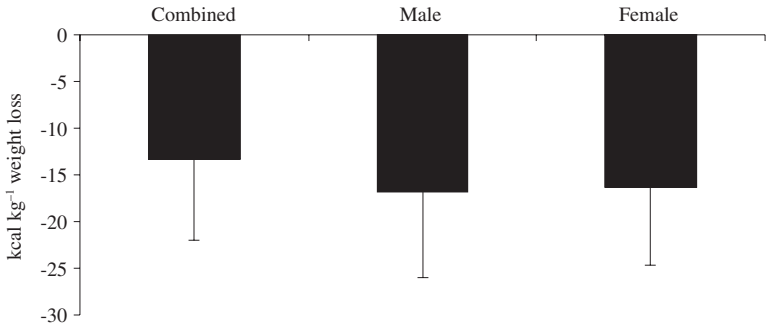

Figure 2 Mean rate of changes in resting energy expenditure relative to weight loss with all interventions divided by sex ($n = 2996$). The group labelled *Combined* refers to those studies that did not provide sex-specific information.

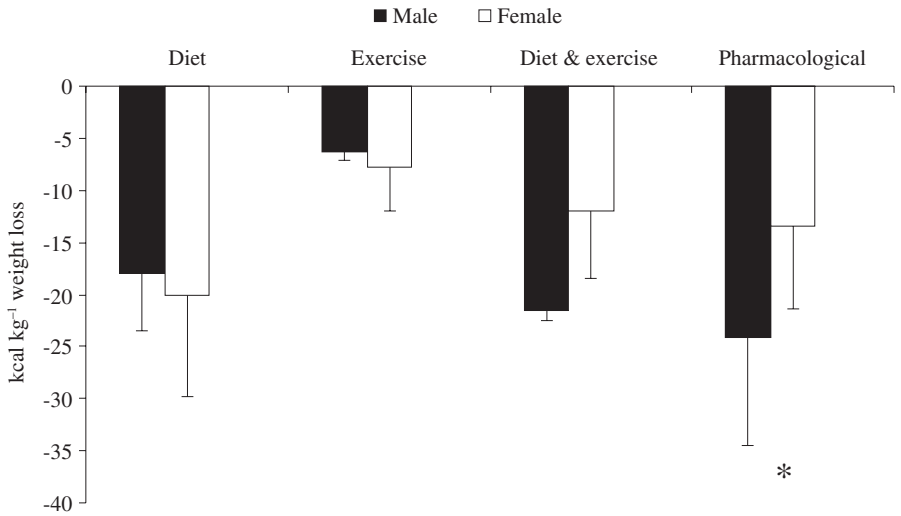

Figure 3 Comparison of men ($n = 293$) and women ($n = 1515$) with regards to the effects of different weight-loss interventions on the mean rate of changes in resting energy expenditure (EE) relative to weight loss. * indicates that the mean rate of changes in resting EE relative to weight loss in men is significantly different from that observed in women ($P < 0.05$). Women with surgically induced weight loss were not included in this analysis as no comparison group was available in men.

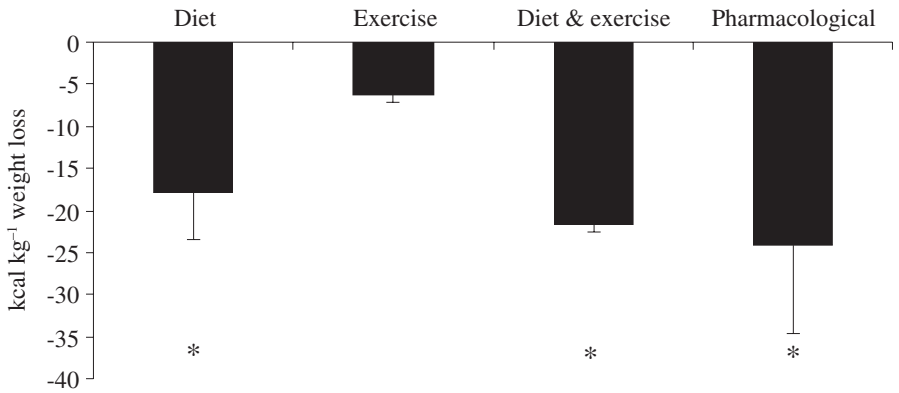

Figure 4 Comparison of the mean rate of changes in resting energy expenditure relative to weight loss with different weight-loss interventions in men ($n = 293$). * indicates significant difference from exercise ($P < 0.05$).

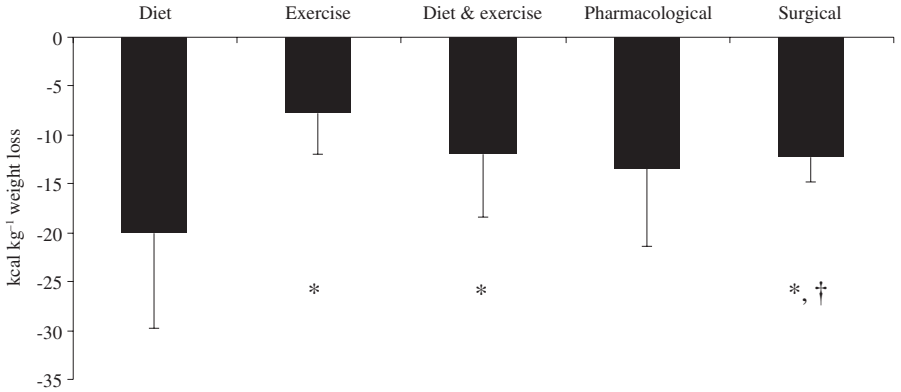

Figure 5 Comparison of the mean rate of changes in resting energy expenditure relative to weight loss with different weight-loss interventions in women ($n = 1728$). * and † indicate significant difference from diet and exercise respectively.

Men

Studies on the effects of various weight-loss strategies on resting EE in men had a combined total of 293 subjects, and the results can be seen in Fig. 4. Interventions utilizing only exercise had the lowest declines in resting EE relative to weight loss (–6.3 kcal kg^{-1} ranging from –9.3 to –5.6) in comparison with diet ($P < 0.05$), pharmaceuticals ($P < 0.05$) and the combination of diet and exercise ($P < 0.05$). No other significant differences were found between other interventions.

Women

The majority of studies looking at weight loss focused exclusively on women, with a total of 1662 participants (Fig. 5). Dietary interventions had a mean decrease of –20.1 ± 9.7 kcal kg^{-1} weight loss ranging from –52.6 to –3.5 kcal kg^{-1} weight loss. In addition to the decrease being greater than that for interventions combining all women, this decrease in the rate of resting EE relative to weight loss was greater than those seen in protocols that utilized surgery (–12.4 ± 2.6 [–24.7–{–0.5}] kcal kg^{-1} weight loss, $P < 0.05$), exercise (–7.7 ± 4.62 [–30.7–3.2] kcal kg^{-1} weight loss , $P < 0.01$) and the combination of diet and exercise (–12 ± 6.4 [–41.5–{–3.5}] kcal kg^{-1} weight loss , $P < 0.01$). Additionally, exercise interventions averaged a significantly smaller loss in resting EE relative to weight loss in comparison with those that used surgery (–7.7 ± 4.62 vs. –12.4 ± 2.6 kcal kg^{-1} weight loss, $P < 0.05$). There were no other significant group differences.

Trial length

Short interventions, defined as those that are over 2 weeks but less than 6 weeks in length, had greater resting EE decreases in comparison with longer trials. Figure 6 illustrates the differences, where long interventions had a mean resting EE decrease relative to weight loss of –12.8 ± 7.1 kcal kg^{-1} weight loss (22.6 to –52.6 kcal kg^{-1}) and in contrast, short interventions had an average fall in

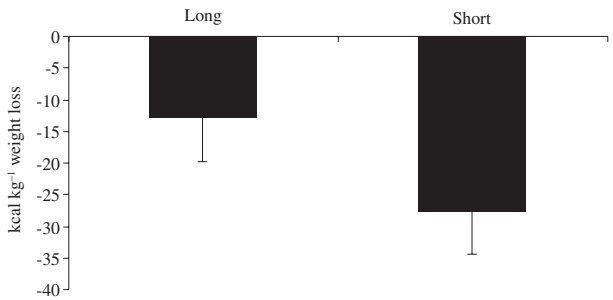

Figure 6 Comparison of the effect of time on the mean rate of changes in resting energy expenditure relative to weight loss with long (≥6 weeks) and short (<6 weeks) interventions. Differences were statistically significant ($P < 0.001$).

resting EE of –27.7 ± 6.7 kcal kg^{-1} weight loss (–12.8 to –41.8 kcal kg^{-1})($P < 0.001$).

Correlation analyses

We performed a series correlation analyses to determine the contribution of the magnitude of the caloric restriction and of the changes in body composition to the changes in EE during weight loss. For the magnitude of the caloric restriction, 62 groups ($n = 1075$) were included and a significant positive correlation with changes in EE was observed, indicating that a greater energy deficit lead to a greater decrease in EE ($r = 0.33$, $P < 0.05$). Changes in EE were also correlated with changes fat-free mass (68 groups, $n = 1456$) and changes in fat mass (70 groups, $n = 1501$). An inverse correlation between changes in fat mass and those in EE relative to weight loss was noted ($r = –0.28$, $P < 0.05$), which was not the case for fat-free mass ($r = –0.05$, NS).

Discussion

In summary, this review systematically analysed 90 publications from 1969 to 2008 in order to determine the change of EE as a function of weight loss. Recently, it was reported that the decline in EE, which favours weight regain (5), remains suppressed well beyond the period of the weight-loss intervention (13,14) indicating a potentially permanent depression of resting EE. We have in fact recently shown that prediction equations overestimate EE during and after weight loss (6,15). An important observation is that body weight regain after weight loss is rampant (16) and that, there even seems to be an overcompensation of fat deposition as a result of severe food restriction (17). As such, it would seem useful to carefully review existing literature on the weight loss-induced changes in EE, in hopes of improving guidance as to post-weight loss energy intake.

A good proportion of studies included in this review included only women in diet only interventions (31.7% or 950 of all subjects). Dietary restrictions generated the greatest drop in resting EE per kilogram of weight loss regardless of sex. Studies directly comparing the effects of exercise only interventions to dietary restrictions have shown that the decreases in resting EE per kilogram of weight loss seen with physical activity are lower than dietary restrictions (18–20). Along these lines, Wadden *et al.* argued that the actual exercise protocol may considerably affect the changes seen in resting EE in comparison with simply restricting calories. An aerobic step programme alone or in combination with resistance training showed lower decreases in the rate of resting EE change in comparison with caloric restriction, but the resistance training only group actually had a greater decrease in resting EE than the group using caloric restriction (21). Resistance training is often recommended as an important aspect of a weight-loss protocol as it should theoretically attenuate decreases in fat-free mass and subsequently diminish the decreases seen with resting EE during a weight-loss period. Although recent results by Hunter *et al.* support this perception, our own evidence indicates that in spite of the protective effects of resistance training on fat-free mass, resting and total EE did not differ from that observed in women who lost weight through caloric restriction only (22). The absence of a correlation between changes in relative resting EE in this study and the changes in fat-free mass lends support to these previous results.

An important sex difference in the changes in resting EE relative to weight loss was noted for pharmacotherapy. When considering this observation, it is important to think about the fact that one of the weaknesses of the results presented herein is the fact that different types of pharmaceutical compounds were pooled together. Indeed, some drugs present thermogenic and/or appetite suppressing properties, while others impact on nutrient absorption efficiency. As such, the outcome on EE may differ substantially from one study to the next and consequently, these results should be interpreted with caution. Despite the relatively small number of studies available for this analysis and studies that utilized drugs targeting different mechanisms were actually pooled together, the sex-related differences in the changes in resting EE that were observed are intriguing and should warrant further clarification.

Surgical interventions yielded a decrease in the resting EE per kilogram of weight lost of approximately –11 kcal kg^{-1} weight loss, and this difference was statistically different from that observed for diet interventions (Fig. 2). The reason underlying this observation falls beyond the scope of this review, but it is very likely that the weight losses following surgery are accompanied by a greater proportion attributable to fat losses. It has indeed been established that the energy consumption of fat is lower than that observed for lean tissue (23). Similarly to the pharmacological interventions, the wide array of surgical interventions

complicates the generalization of the changes in resting EE resulting from surgery-induced weight loss. The four studies utilizing Roux-en-Y procedures yielded a mean weighted change of approximately –10 kcal kg^{-1} weight loss while Busetto *et al.* demonstrated that large volume liposuction produced a mean change of approximately –25 kcal kg^{-1} weight loss (24–28). In spite of the fact that liposuction surgery immediately removes adipose tissue and prevents the loss of fat-free mass typically seen with other interventions, it still has a relatively high resting EE decrease per kilogram of weight loss implicating the potential importance of adipose tissue in regulating resting EE. Accordingly, leptin has already been suggested as a possible mediator of resting EE as fluctuations of this hormone occur at greater than expected levels with weight loss (29) and act to decrease sympathetic nervous system activity (30) thereby possibly lowering resting EE independently of changes in body mass (6). In fact, the restitution of pre-weight loss leptin levels with recombinant leptin has been shown to reverse the effects of weight loss on resting EE (31).

The fact that lean tissue is a major determinant of EE expenditure (4) and that it is also associated to a higher metabolic rate than fat (23) is well accepted. As such, it is rather intriguing to note that the changes in fat-free mass were not associated to the changes in resting EE relative to weight loss in our analyses. On the other hand, the changes in fat mass as well as the magnitude of the caloric restriction were both significantly associated to the changes in resting EE relative to weight loss. This prompted us to revisit some of our own data (2). A decrease in resting EE of approximately 140 kcal d^{-1} was noted after 15 weeks of intervention in this earlier study. The relatively large decrease in resting EE noted in this previous study was observed despite a relatively small contribution of fat-free mass during weight loss (–1.8 kg) and in the absence of an association between changes in resting EE and those in fat-free mass. Therefore, these observations suggest that predictors of resting EE obtained under conditions of energy balance, e.g. lean body mass, may not be accurate to predict changes in resting EE in response to prolonged caloric deficit aimed at inducing weight loss.

One of the important findings in this review was the substantial differences between interventions that were short (greater than 2 weeks but less than 6 weeks) and long (greater than 6 weeks). Short interventions showed a greater decrease in resting EE per kilogram of weight loss in comparison with longer interventions (Fig. 6). One might speculate that there may be an early and exaggerated depression in resting EE to buffer the decrease of energy reserves (32). Along these lines, we have evidenced a greater than expected decrease in resting EE maintaining the weight loss (5,15), which could be associated to the difficulty in maintaining weight stability after weight loss (5). This is in line with the idea proposed by Dulloo and Jacquet whereby there are controls of thermogenesis as a function of energy store and caloric imbalance. Decreases in body energy reserves and caloric deficit may result in a sudden decrease in thermogenesis in an effort to re-establish fat stores (33). Accordingly, as the rate of weight loss is reduced as the energy restriction progresses in time, it may also be possible that the difference between the short- and the long-term weight-loss interventions may be explained by the magnitude of the energy restriction. Indeed, as most energy restriction are fixed based on the pre-weight loss EE and that EE is reduced with changes in body mass, it would then be expected to note a narrowing of the gap between caloric intake and expenditure as the intervention progresses. In fact, the positive correlation that we observed between the magnitude of the caloric restriction and the changes in resting EE relative to weight loss lends support to this speculation.

Factors other than body weight and composition have been reported to modulate EE. Among these, the depression in leptin seen with the depletion of body fat stores has been shown to provide an independent contribution to the changes in EE that occur with weight loss (2,34). Leptin depletion has also been shown to down-regulate thyroid-stimulated hormone in the posterior ventricular nucleus affecting EE through changes in the cellular oxidation of glucose as a result of depleted peripheral thyroid hormones (6,35,36). In fact, the weight loss-induced changes in EE have been shown to be related to changes in thyroid function (37), even if administration of triiodothyronine and thyroxine during weight loss does note completely correct the decrease in EE observed under such conditions (38).

An intriguing observation that resulted from this review is the sometimes large inter-study variation in the weight loss-induced changes in EE. This is even more interesting as this variation in EE is also present when comparing studies that employed seemingly similar weight-loss interventions. The many factors discussed above and elsewhere likely contribute to the inter-individual variation in EE changes in response to weight loss within each of the groups. However, it is also probable that the inter-study variability in weight loss-induced EE is related to the intricacies of the interventions. For example, the fact that the caloric deficit was not the same across studies and that we have shown a significant correlation between the magnitude of the caloric deficit and the changes in EE may explain some of the noted variability. The results presented herein should thus be interpreted in light of the numerous factors that have been shown to modulate variations in EE during weight loss but that could only be partly explored in this review.

Because of some limitations of this review, many factors that influence resting EE and consequently the rate of resting EE change per kilogram of weight loss were not

taken into account in the analyses. One of these aspects is the race/ethnic-related differences in the response of resting EE to prolonged energy deficits. Indeed, it has been reported that individuals from African–American lineage tend to have lower resting EE values as well as smaller weight losses in comparison with Caucasian women (39,40). As such, the results from our analyses should be interpreted with caution as they did not take into consideration the demonstrated race/ethnic differences in energy metabolism.

Age also plays a pivotal role in determining resting EE and was not reported in this review. Although an argument can be made that as age increases, lean mass decreases and subsequently, resting EE is depressed, the changes seen with resting EE as a result of aging cannot be entirely explained by decreased fat-free mass alone (41). Cross-sectional and longitudinal evidence from Alfonzo-Gonzalez *et al.* confirms that resting EE is affected by age independent of changes in body composition and that this decrease is more pronounced in early adulthood in men but in the later years of women (42).

In conclusion, the combined resting EE changes regardless of intervention or sex was approximately –15 kcal kg^{-1} weight loss. The results of this review indicate that the mean decrease in relative EE change per kilogram of weight loss was not different between men and women when examining the combination of all of the interventions. When looking at all interventions, surgery, exercise, and the combination of diet and exercise seem to produce lower decreases in resting EE per kilogram of weight loss in comparison with dietary interventions. One interesting finding is that regardless of the intervention type, those that are shorter in length lasting less than 6 weeks illicit significantly greater decreases in resting EE relative to weight loss compared with longer studies. Results from this review indicate that although decreases in resting EE may be inevitable, combining diet therapy with other interventions such as exercise may assist overweight individuals in attenuating the reduction of resting EE per kilogram of weight lost. Given the important contribution of resting EE to total EE and that its reduction may be associated to body-weight relapse, results from this study warrant further research on weight loss modalities that may attenuate the decrease in resting EE.

Conflict of Interest Statement

No conflict of interest was declared.

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Appendix 1

Process of statistically weighing the studies.
The number of subjects in each study of a group were divided by the total number of subjects in all the studies comprising that specific group (i.e. men – exercise group, $n = 91$) to give a coefficient that signified the weight of each study.

Study	*n*	Mean decrease (kcal kg^{-1} body-weight loss)	Number coefficient (weight)	Actual
Sum *et al.* (1994) (91)	42	–6.8	0.46	3.133
Frey-Hewitt *et al.* (1990) (19)	44	–5.6	0.48	2.690
Weigle and Brunzell (1990) (93)	5	–9.3	0.05	0.513
			Mean ΔREE (kcal kg^{-1} body-weight loss)	–6.34

The coefficients of each study were multiplied by its own mean relative energy expenditure (EE) decrease as expressed in kcal kg^{-1} of body weight. This gave an actual number of what proportions of the total group mean each study contributed to. The sum of the 'actual' values determined the mean resting EE (REE) decrease of the group. In order to calculate the standard deviation of this group mean, it was necessary to individually subtract each of the study means from the group mean and this number was then squared. From there, the square root was determined and finally multiplied by the coefficient (SD actual).

Study	*n*	Group mean – study mean (kcal kg^{-1} body-weight loss)	Number coefficient (weight)	SD actual
Sum *et al.* (1994) (91)	42	0.45	0.46	0.207
Frey-Hewitt *et al.* (1990) (19)	44	–0.78	0.48	0.377
Weigle and Brunzell (1990) (93)	5	3.00	0.05	0.165
			Standard Deviation	0.7

The products of these were added up together to determine the weighted standard deviation of all studies.

