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Is dieting a risk for higher weight gain in normal-weight individual? A systematic review and meta-analysis

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Abstract

While there is an increasing prevalence of dieting in the overall population, weight loss (WL) practices could be a risk factor for weight gain (WG) in normal-weight (NW) individuals. The aim of the present work was to systematically review all the studies implicating diet restriction and body weight (BW) evolution in NW people. The literature search was registered in PROSPERO (CRD42021281442) and was performed in three databases from April 2021 to June 2022 for articles involving healthy NW adults. From a total of 1487 records initially identified, eighteen were selected in the systematic review. Of the eight dieting interventional studies, only one found a higher BW after weight recovery, but 75 % of them highlighted metabolic adaptations in response to WL favouring weight regain and persisting during/after BW recovery. Eight of the ten observational studies showed a relationship between dieting and major later WG, while the meta-analysis of observational studies results indicated that 'dieters' have a higher BW than 'non-dieters'. However, considering the high methodological heterogeneity and the publication bias of the studies, this result should be taken with caution. Moreover, the term 'diet' was poorly described, and we observed a large heterogeneity of the methods used to assess dieting status. Present results suggest that dieting could be a major risk factor for WG in the long term in NW individuals. There is, however, a real need for prospective randomised controlled studies, specifically assessing the relationship between WL induced by diet and subsequent weight in this population.

Key words: **Food restriction: Lean: Weight loss: Weight gain: Obesity**

Obesity and its cardiometabolic consequences have become a worldwide preoccupation, given the rapid increase in its prevalence in recent years. The literature on weight loss (WL) management is well documented, dieting (*i.e.* 'restrict oneself to small amounts or special kinds of food in order to lose weight' – *Oxford Dictionary*) being one of the most popular strategies, leading to significant beneficial effects on patients' body weight (BW), body composition, and cardiometabolic health[1–3]. Interestingly, while there is a high prevalence of dieting among individuals with elevated BMI, WL attempts also concern a significant and increasing proportion of normal-weight (NW) individuals[4–6]. For example, among French dieters, for instance, 33 % of men and 57 % of women are NW subjects[6]. Indeed, the media and social pressure related to leanness, and the widespread notion of an ideal body shape may alter body perception, particularly among young adults[7–10]. As illustrated in a cohort of

Abbreviations: BW, body weight; FFM, fat-free mass; FM, fat mass; NW, normal weight; REE, resting energy expenditure; WG, weight gain; WL, weight loss.

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European university students, 60 % of women with an average BMI of 22·5 kg/m^2 considered themselves as 'a little too fat'[9]. Body image distortion is often associated with the desire to lose weight[11], and leads individuals to engage in dieting independently of any need for weight management in terms of cardiometabolic health. In contrast, while dieting is associated with health improvements in people with overweight or obesity, it conversely could constitute a potential risk factor for increased adiposity and cardiometabolic disease, particularly in NW people. Indeed, in 2015, Dulloo and collaborators suggested that NW individuals might have a higher risk to experience post-WL 'fat overshooting' than people with obesity[12]. This phenomenon is well illustrated in the Minnesota Starvation Experiment, in which thirty-two healthy NW males sustained a 24-week semi-starvation, and lost an average of 25 % of their initial BW[13–15]. During weight recovery, adaptive thermogenesis and hyperphagia induced a fast rebound of fat mass (FM) (*i.e.* preferential catch-up fat), resulting in FM being fully recovered earlier than fat-free mass (FFM). However, the hyperphagia was found to persist until FFM returned to its initial value, leading hence to higher FM values than those prior to semi-starvation (*i.e.* fat overshooting)[14,16]. Importantly, the leaner the participant at baseline, the higher proportion of FFM lost during WL and the greater the risk of fat overshooting during recovery[12,16].

Interestingly, a recent narrative review suggested some beneficial effects (*e.g.* increased insulin sensitivity) of interventional dietary-induced WL in NW subjects, still pointing the lack of evidence among healthy lean individuals, since most of the available evidence concerns people suffering from metabolic disorders (such hypertension or insulin resistance)[17]. In addition, some observational studies showed benefits of energetic restriction in healthy NW individuals such as an improvement of lipid and glycemic profiles[18], or an important decrease in global mortality[19,20]. However, it concerned specific populations (*e.g.* Okinawans) who sustain a low-energy intake during all their lifespan[18–20], and it was well established that the 'obesogenic environment' of our actual societies, including high food availability, makes it difficult to maintain a low-energy diet on the long term[21–23]. Thus, due to the high probability of regaining the weight lost after a diet, individuals engaging in dieting may experience repeated WL and regains during their lifespan, a phenomenon called 'weight cycling'. Weight cycling is associated with an increased risk of cardiometabolic impairments such as hyperinsulinemia, insulin resistance, hypertension, and hypertriacylglycerolaemia, specifically in NW individuals[4]. In addition, associated with the fat overshooting theory described above, weight fluctuations are also related to major long-term weight gain (WG)[4]. However, the long-term effects of dieting on BW and body composition changes in NW healthy people remain unclear. In their prospective observational study, Lowe *et al.* showed that NW current dieter women gained almost three times more BW than non-dieters after an 8-month period (5 kg *v.* 1·6 kg, respectively)[24]. In contrast, in a study performed in a large cohort (*n* 4159, aged 18–29 years), women who declared dieting at baseline gained as much BW as non-dieters after 15 years, while in young men, dieters tended to gain more BW than non-dieters[25]. However, the majority of longitudinal studies that concern weight evolution in the overall population have relied on self-reported data[25–27] and have not considered body composition variations (*i.e.* FM and FFM).

While there is an increasing concern for weight control in healthy NW individuals leading them to regularly engage in dietary WL strategies, there is a clear need to question the potential effects of dieting on their BW and body composition. Therefore, the aim of the present work was (i) to conduct a systematic overview of the available studies involving dieting and WL, BW, and body composition in NW healthy people, and (ii) to determine the effect of dieting on subsequent and/or long-term BW in NW healthy individuals using a meta-analytic approach. In doing so, the results may help to better decipher the risks and/or benefits to healthy NW individuals engaging in dieting and to promote adapted public health messaging related to diet, based on robust scientific evidence.

Methods

This systematic review and meta-analysis followed the recommendations from the PRISMA statement[28], and the protocol was prospectively registered in the International Prospective Register of Systematic Reviews database (registration number: CRD42021281442).

Literature search strategy and article selection

The following electronic bibliographic databases were searched from April 2021, and the last run was performed in June 2022: PubMed-Medline, Embase, and Google Scholar. The search terms were a combination of medical subject headings (MeSH terms) and text words (title and abstract) which were adapted for use in each database. The main keywords selected were 'weight gain', 'weight change', 'weight loss', 'body weight', 'body composition', 'normal-weight', 'healthy', 'non-obese', 'diet/ing/ietary', and 'caloric restriction'. Limits were set to include all papers in healthy human adults published after 1945. The search was followed by a careful selection of eligible studies according to the criteria outlined below. References from narrative or systematic reviews and eligible publications were also screened to find additional records.

Inclusion and exclusion criteria

The inclusion criteria were as follows: (1) healthy (without pathology or co-morbidity) participants with initial BMI between 18·5 and 24·9 kg/m^2 (for the group mean); (2) adult women and men (data available from 18 to 55 years); (3) subjects who dieted in the past or dieted (*i.e.* restrict food to lose weight) during the study; (4) any study that mentioned weight changes (or the terms 'weigh maintenance' or 'weight (re)gain') related to diet and WL, which included (i) dietary interventional studies with a follow-up after WL, (ii) longitudinal observational studies (over a few months to several years) with weight changes in dieters, and (iii) cross-sectional observational studies that compared BW and/or body composition between dieters and non-dieters; and (5) publications in English or French language. Studies involving animal models, athletes or trained individuals were not included nor were interventional protocols involving an imposed overfeeding (*i.e.* where subjects must ingest daily food

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intake higher than their daily requirement) before and/or after WL.

Data extraction and synthesis

Search results were exported to a spreadsheet, and duplicates were removed. The first selection was performed based on titles only and then based on abstracts. Finally, remaining full texts were screened to assess eligibility according to the inclusion and exclusion criteria above. The selection process was performed independently by two reviewers, and any discrepancies were collectively discussed amongst these reviewers until a consensus was reached. The third reviewer was consulted when necessary. The flow diagram presented in Fig. 1 illustrates this selection process. Then, the qualitative data were extracted using a spreadsheet containing the following items chosen collectively by the authors: (1) reference; (2) population characteristics ('*n*' sample size, age, BMI, unique sample or specific sample analysed in others studies); (3) design of the study (randomised and/or controlled or not, longitudinal or cross-sectional, interventional or observational); (4) total duration of the study, number of measurement point and duration between each measurement point for longitudinal studies; (5) details of the intervention protocol for the interventional studies; (6) type of measurements or parameters assessed with details on the methods used; (7) details on subjects' food intake and/or diet; (8) main results for BW, weight changes and/or body composition; and (9) additional results linked to dietary or weight changes. When results were available for several groups, only those of control groups (called 'non-dieters') and the groups that matched the inclusion criteria were extracted. In addition, when BW and/or body composition data were not detailed in the paper, authors were contacted by email to obtain raw data and/or more information. When the extracted results were expressed as mean ± SEM, SEM was converted into SD. The methods and main results of the selected interventional studies are displayed in Table 1. Table 2 details the methodological characteristics of the selected observational studies, while Table 3 presents its detailed main results.

Quality assessment

The quality assessment of the included studies was performed independently by two reviewers using the 'Quality Assessment Tool for Quantitative Studies' developed by the Effective Public Health Practice Project[29]. Six criteria were assessed: selection bias, study design, confounders, blinding, data collection method, and withdrawals dropouts. Each criterion was qualified as 'strong', 'moderate', or 'weak'. The overall methodological quality was then rated as strong if there were no weak rating criteria, moderate if there was one weak rating, and weak if there were two or more weak ratings[30]. Any discrepancies were collectively discussed among the two reviewers until a consensus was reached, and the third reviewer was consulted when necessary. The quality assessment described above was presented individually for each study and collectively represented using a summary bar plot (in %) for each criterion and the overall quality.

Meta-analysis procedure

Given that interventional studies presented a high heterogeneity in terms of WL protocol, a meta-analysis was performed from the quantitative data for the observational studies only. Publications were selected with more restrictive criteria, as follows: (i) comparison between groups considered like 'dieters' and 'non-dieters'; (ii) data on BW in kg in dieter and non-dieter groups; (iii) data in women-only considering the little number of available data in NW men; and (iv) data were estimated from graphs, as accurately as possible, if numerical values were not available in tables or text.

Statistical analysis

Statistical analyses were performed with Stata software (version 15, StataCorp). The meta-analysis considered between and within study variability. To address the non-independence of data due to the study effect, random effects model was preferred to the usual statistical tests to assess the standardised mean difference and their 95 % CI. More precisely, Hedge's g standardised mean difference was estimated to assess the BW difference between dieters and non-dieters. Means and standard deviation were compiled when available or were estimated when median and interquartile range were reported. When SD was not available, an estimation according to available SD, for other studies, was calculated. Hedge's g is considered to be a conservative estimate, which is useful for studies with small sample sizes, and the results may be interpreted as reflecting a small ($g = 0{\cdot}2$–$0{\cdot}5$), medium ($g = 0{\cdot}5$–$0{\cdot}8$) or large effect ($g > 0{\cdot}8$). Heterogeneity in the study results was assessed by forest plots and the I^2 statistic which is the most common metric for measuring the magnitude of between-study heterogeneity. I^2 values range between 0 and 100 % and are typically considered low for < 25 %, moderate for 25–50 %, and high for > 50 %[31]. Publication bias was assessed by funnel plot, CI and with the Egger's regression test as a formal statistical test. Two-sided type I error was fixed at 5 %.

Results

Selection of the studies

The search yielded a total of 1320 publications identified from database searching and 167 records identified from additional sources, making a total of 1344 scientific articles after the removal of duplicates. Based on title and abstract screening, 1269 publications were excluded. A total of seventy-five records were fully read to assess the eligibility according to the inclusion and exclusion criteria. At this step of the selection process, fifty-seven articles were removed, as detailed in Fig. 1. Finally, a total of eighteen scientific papers remained in the qualitative analysis of the systematic review, published from 1990 to 2019.

Quality assessment

The quality of selection bias minimisation, study design, and blinding of participants and personnel was judged to be moderate for the majority of the studies. The quality of confounder

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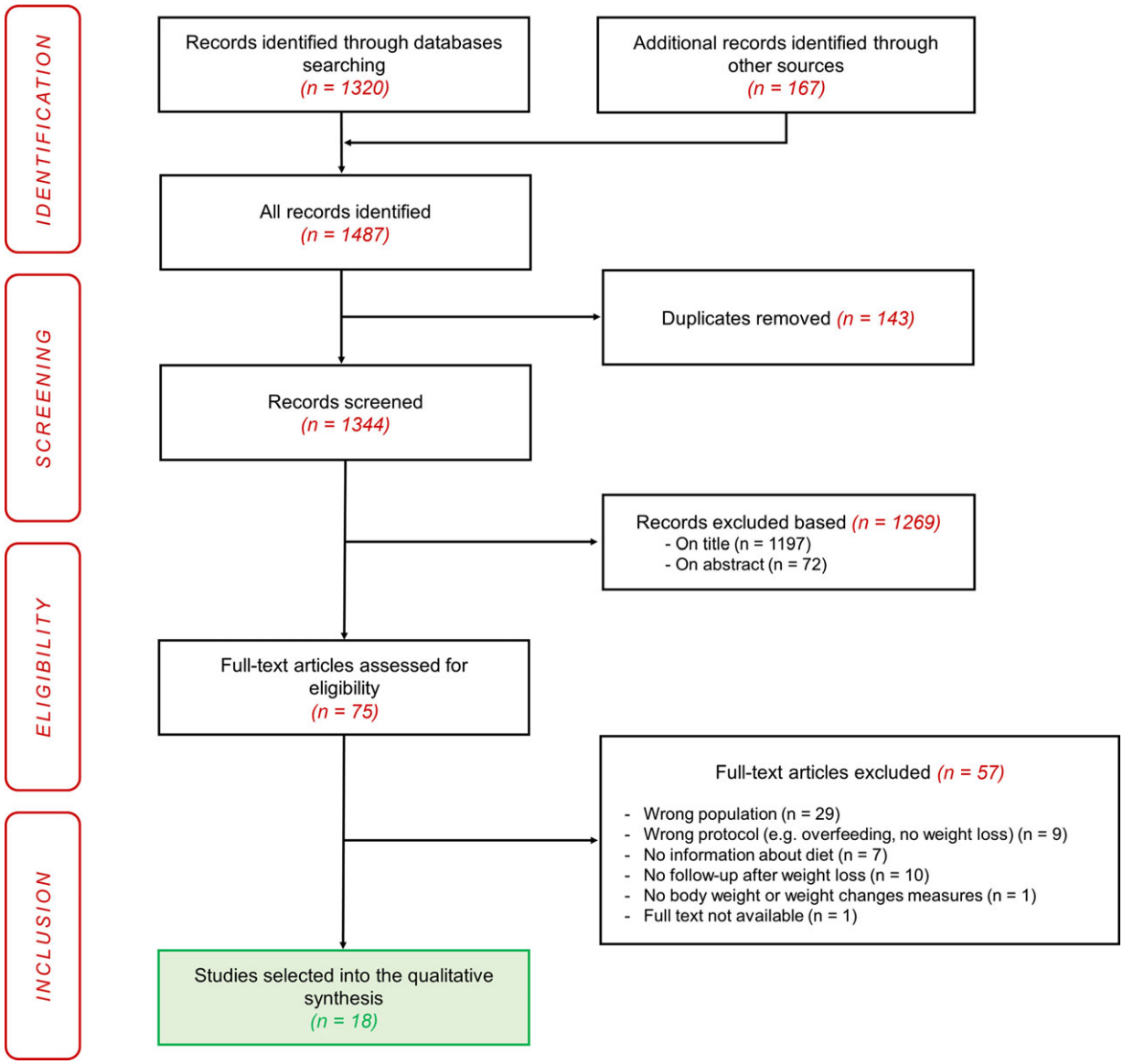

Fig. 1. Flow diagram of the identification, screening, eligibility, and inclusion of the studies in the systematic review.

reduction was considered as strong for 61 % (*n* 11) and weak for 39 % (*n* 7) of the studies. Data collection methods were judged strong for 56 % (*n* 10) of the articles, while withdrawals and dropout were considered as strong for 33 % (*n* 6). Finally, the global quality assessment was defined as strong for six studies[13–15,24,25,32], moderate for nine studies[27,33–40], and weak for three studies[26,41,42]. Individual quality assessment of the selected publications is detailed in Fig. 2.

Designs of the studies

Two main study designs were identified among the eighteen publications selected in the systematic review. Height studies[13–15,34,37–40] followed an interventional longitudinal protocol that involved a WL induced by diet from 3 weeks[34,37] to 24 weeks[13–15] duration in NW subjects, followed by a period of free-living or imposed recovery diet during 14 d[38] to 40 weeks[40] (Table 1). Only one[40] of these studies included a control group without WL intervention, but results were thus not extracted as the mean BMI for this cohort was 27 kg/m² (overweight subjects). Two studies[34,39] compared healthy young NW subjects with overweight and/or old ones (results were not extracted for these groups). Three studies[34,37,39] imposed an individualised energy deficit of about 3 to 4 MJ/d (deficit of 800–1000 kcal/d). In Siggaard *et al.*'s study[40], subjects received guidelines to set up an *ad libitum* high-carbohydrate and low-fat diet guarantying a minimum of 7 to 8 MJ/d (1670–1910 kcal/d). Fat was restricted to 20–25 % of daily energy intake, but subjects presented also an 11 % decrease in total energy intake[40]. Finally, the other studies imposed a restricted standard daily food intake of 6·6 MJ/d (1570 kcal/d) in male subjects[13–15] and below 5·0 MJ/d (1200 kcal/d) in female subjects[38]. During the first 12-week recovery period (prior to *ad libitum* access to food), the

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Table 1. Characteristics of the populations, description of the protocols, measurements and parameters, and main results linked to body weight changes of the interventional longitudinal studies selected in the systematic review

Reference	Characteristics of the groups	Interventional protocol	Measurements–parameters (method)	Main results
Dulloo *et al.* (1996)	*Minnesota cohort* *n* 32 (m) 25 ± 4 years (range: 20, 33) BMI NR	**Pre-WL (12 weeks):** weight maintenance with 14·6 MJ/d (12–13 % protein + 34–40 % fat + 47–54 % CHO) **WL-1 (12 weeks) + WL-2 (12 weeks):** 6·6 MJ/d (25 % protein + 17 % fat + 58 % CHO) **WG (12 weeks):** recovery-imposed diet of about 12·1 MJ/d (men were split into four groups of different degree of EI deficits) (12–17 % protein + 20 % fat + 63–68 % CHO) Subjects resided at the university during all the study with provided meals.	Anthropometry/body composition: BW; height; FM, FFM (hydrodensitometry); abdominal and arm circumferences Energy expenditure: REE (indirect calorimetry); ΔREE (= $REE_{MEASURED}$ – $REE_{PREDICTED}$)	**Pre-WL/WL-1/WL-2/WG** ΔREE (MJ/d): *−0·10 (range: −0·84, +0·40)/ NR/-1·96 (range: −2·72, −0·90)/-0·89 (range: −1·88, 0)* → Positive correlation between ΔREE and FM recovery → Reduced thermogenesis = accelerated FM recovery **Extracted from raw data§:** BW (kg): 69·2 ± 5·8/57·3 ± 4·8***/46·5 ± 3·6***/ 54·0 ± 3·7*** FM (kg): 9·7 ± 4·1/4·6 ± 3·3***/2·8 ± 2·3***/ 5·9 ± 2·6*** FM (%): 13·7 ± 5·0/7·8 ± 5·2***/5·8 ± 4·5***/ 10·9 ± 4·5*** FFM (kg): 59·5 ± 4·2/52·7 ± 4·3***/43·7 ± 3·1***/ 48·1 ± 3·3***
Dulloo *et al.* (1997)	*Minnesota cohort* *n* 12 (m) 25 ± 4 years (range: 20, 33) BMI NR	**Pre-WL (12 weeks):** weight maintenance with 15·1 MJ/d (13 % protein + 37 % fat + 50 % CHO) **WL-1 (12 weeks) + WL-2 (12 weeks):** 6·6 MJ/d (25 % protein + 17 % fat + 58 % CHO) **WG-1 (12 weeks):** recovery imposed diet with range of 9·9–14·2 MJ/d (men were split into four groups of different degree of EI deficits) (12–17 % protein + 20 % fat + 63– 68 % CHO) **WG-2 (8 weeks):** *ad libitum* EI (about 20·0 MJ/d) (14% protein + 35% fat + 51 % CHO) Subjects resided at the university during all the study with provided meals.	Anthropometry/body composition: BW; FM, FFM (hydrodensitometry); FFM and FM corrected for excess hydration (formula of Keys *et al.*) Energy intake: Food intake; control EI (= mean EI on the last 3 weeks of pre-WL period); weekly hyperphagia during WG-2 (= EI_{WG-2}–control EI)	**Pre-WL/WL-1/WL-2/WG-1/WG-2 (week 1/ week 2/. . ./week 8)** FM (% of FM_{pre-WL}): 100/*47*/*32*/*83* (range: 42, 148)/174 FFM (% of FFM_{pre-WL}): 100/*87*/83/88 (range: 84, 90)/98 FFM corrected (% of FFM_{pre-WL}): 100/NR/73/ 80/NR EI (% of EI_{pre-WL}): 100/*45/44/101/(148/165/149/ 141/119/108/106)* → Negative correlation of hyperphagia (EI during WG-2) with the degree of FM recovery, FFM recovery and EI deficit at WG-1 → The 6 men in the 2 lower-EI groups at WG-1 gained 4·6 kg more at WG-2 than the 6 men in the upper-EI groups at WG-1 **Extracted from raw data§:** BW (kg): 67·4 ± 5·1/55·8 ± 4·2***/45·7 ± 3·0***/ 53·5 ± 3·5***/70·5 ± 4·4*** FM (kg): 8·9 ± 4·6/4·5 ± 4·0***/2·8 ± 2·7***/ 6·4 ± 3·3***/13·5 ± 3·0*** FM (%): 12·9 ± 5·7/7·7 ± 6·1***/5·9 ± 5·0***/ 11·7 ± 5·5/19·0 ± 3·3*** FFM (kg): 58·5 ± 3·3/51·3 ± 2·9***/42·9 ± 2·0***/ 47·1 ± 2·7***/57·0 ± 3·2*

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Table 1. (*Continued*)

Reference	Characteristics of the groups	Interventional protocol	Measurements–parameters (method)	Main results
Dulloo *et al.* (1998)	*Minnesota cohort* *n* 32 (m) 25 ± 4 years (range: 20, 33) BMI NR	**Pre-WL (12 weeks):** weight maintenance with 15·1 MJ/d (13 % protein + 37 % fat + 50 % CHO) **WL-1 (12 weeks) + WL-2 (12 weeks):** 6·6 MJ/d (25 % protein + 17 % fat + 58 % CHO) **WG (12 weeks):** recovery-imposed diet with range of 9·9–14·2 MJ/d (men were split into four groups of different degree of EI deficits) (12–17 % protein + 20 % fat + 63–68 % CHO) Subjects resided at the university during all the study with provided meals.	Anthropometry/body composition: BW; FM, FFM (hydrodensitometry); FFM corrected for excess hydration (formula of Keys *et al.*) Energy expenditure: REE (indirect calorimetry); ΔAdjustedREE (for FM and FFM)	*See Dulloo et al. 1996 for BW (kg), FM (kg and %), and FFM (kg)* **Pre-WL/WL-1/WL-2/WG** REE (MJ/d): 6·73 ± 0·40/4·57 ± 0·44***/4·10 ± 0·41***/5·40 ± 0·47** ΔAdjustedREE (MJ): N/A/–1·49 ± 0·51/–1·71 ± 0·48/–0·69 ± 0·46 ΔAdjustedREE (% of pre-WL): N/A/–21·5 ± 6·9/24·7 ± 6·2/–9·7 ± 6·6
Heyman *et al.* (1992)	*n* 7 (m) 20·8 ± 1·3 years 22·6 ± 1·9 kg/m²	**Pre-WL-1:** baseline **Pre-WL-2 (10 d):** weight maintenance (protein: 1·5 g/d/kg of BW + 45 % fat and 55 % CHO for the non-protein macronutrients) including normal food items and a palatable low-protein liquid supplement (< 1 % protein + 45 % fat + 55 % CHO) **WL-1 (10 d) + WL-2 (10 d):** −3·35 MJ/d of $EI_{pre\text{-}WL}$ (EI similarly to pre-WL but without the palatable liquid supplement) **WG-1 (10 d):** *ad libitum* EI with specific instructions to consume as much or as little food and drink as they required to feel normally satiated and to not change their diet to gain the weight previously lost. Subjects were asked to not weigh themselves **WG-2 (37 d):** *ad libitum* EI without specific instructions Meals and energetic beverages were provided by the research centre (except for WG-2). They were encouraged to pursue a normal lifestyle during the study. Physical activity was monitored but not restricted.	Anthropometry/body composition: BW; height; BMI; waist, hip, mid-upper arm, thigh, and calf circumferences; skinfolds; FFM and FM (doubly labelled water; only for pre-WL and WL) Energy intake: Food intake (weighed food during each phase except WG-2); metabolisable energy (digestible EI from faecal samples); energy excess during WG (calculated as: Metabolisable energy–TEE) Energy expenditure (only at pre-WL and WL): TEE (doubly labelled water); fasting REE (indirect calorimetry); post-prandial REE (indirect calorimetry); respiratory quotient (indirect calorimetry); PA (trunk monitor, wrist motion sensor, and self-reported record for strenuous physical activity)	**Pre-WL-1/Pre-WL-2/WL-1/WL-2/WG-1/WG-2** Food intake (MJ/d): N/A/15·9 ± 4·1/13·1 ± 4·1***/13·1 ± 4·1***/18·1 ± 2·7**/NR Metabolisable energy (MJ/d): N/A/15·3 ± 3·6/12·0 ± 3·6***/12·0 ± 3·6***/16·9 ± 2·5/NR BW (kg): 69·2 ± 11·1/69·7 ± 10·6/NR/NR**/70·3 ± 10·9/70·5 ± 11·1 ΔBW (kg): N/A/*+0·65 ± 0·67/–0·23 ± 0·78/–1·41 ± 1·03/+1·19 ± 1·17/+1·43 ± 1·31* → Positive correlation between the WL (kg) and the energy excess during WG → FM and FFM stores were estimated to constitute 50 % and 10 %, respectively, of WL **Pre-WL/WL-1/WL-2 (statistical differences with pre-WL were measured from values for mean WL-1 + WL-2)** FM (%): 13·2 ± 4·0/11·9 ± 4·5/12·4 ± 4·0 Fasting REE (MJ/d): 7·22 ± 0·92/6·94 ± 0·96**/6·93 ± 0·90** Relative fasting REE (MJ/kg of FFM/d): 0·12 ± 0·01/0·12 ± 0·01*/0·12 ± 0·01* TEE (MJ/d): 15·35 ± 3·50/14·66 ± 3·81/13·56 ± 3·68 Reported strenuous PA (min/d): 40·8 ± 22·5/43·8 ± 11·9/36·2 ± 12·7
Kajioka *et al.* (2001)	*n* 5 (f) 24·6 ± 5·5 years (range: 22, 34) 20·5 ± 1·1 kg/m² Non-smokers, no oral contraceptive.	**Pre-WL:** baseline **WL-1 (30 d):** max 5·0 MJ/d **WG-1 (14 d):** *ad libitum* EI **WL-2 (30 d):** max 5·0 MJ/d **WG-2 (106 d):** *ad libitum* EI Free-living lifestyle. Subjects were supervised by a dietitian, cooked her meals herself and estimated their EI, notably with a nutritional balance. PA: regular exercise prohibited during all the study.	Anthropometry/body composition: BW; height; BMI; waist and hip circumferences; FM (plethysmography); FFM (BW–FM) Energy expenditure: REE (indirect calorimetry) Biological parameters: Lipoprotein lipase (only at pre-WL, WL-2 and WG-2), total cholesterol, TAG, HDL, LDL, T3, T4, thyroid-stimulating hormone (fasting blood samples); sitting systolic and diastolic blood pressure (sphygmomanometer)	**Pre-WL/WL-1/WG-1/WL-2/WG-2** BW (kg)‡: 52·1 ± 4·3/47·7 ± 4·3†/53·3 ± 4·0†/48·7 ± 4·0†/51·7 ± 4·3 ΔBW (kg) (%FM and %FFM $lost^{NAS}$): N/A/–4·42 kg (–72 % FM,–28 % FFM)/+5·61 kg (+55 % FM, +45 % FFM)/–4·55 kg (–59 % FM,–41 % FFM)/+2·94 kg (+95 % FM, +5 % FFM) FM (%): 23·6 ± 3·8/19·1 ± 4·9***/22·9 ± 2·9/19·5 ± 4·0***/23·8 ± 4·2 ΔFM (%): N/A/–3·1 ± 0·7/–0·1 ± 0·9/–2·7 ± 0·7/+0·1 ± 1·6 FFM: NR/NR***/NR/NR**/NR** ΔFFM (kg): N/A/–1·2 ± 0·7/+1·3 ± 0·7/–0·5 ± 0·7/–0·4 ± 0·7

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Table 1. (*Continued*)

Reference	Characteristics of the groups	Interventional protocol	Measurements–parameters (method)	Main results
				Waist and hip circumferences, ratio waist/hip‡: pre-WL = WG-2 REE: Pre-WL > (WL-1, WL-2, WG-2)***, Pre-WL < WG-1*** (absolute values NR) ΔREE (MJ): N/A/*–1·00 ± 0·26*/+*0·19 ± 0·62*/–*0·80 ± 0·52*/–*0·99 ± 0·80* (–15·8 %) Biological parameters: - Lipoprotein lipase: NS - Total cholesterol: pre-WL > WG-1, pre-WL < WL-2, pre-WL = WL-1 = WG-2 - TAG: pre-WL = WL, pre-WL < WG - LDL: pre-WL = WL-1 = WG, pre-WL < WL-2 - HDL: pre-WL = WL = WG-2, pre-WL < WG-1 - T3, T4: pre-WL > WL and WG - Thyroid-stimulating hormone: pre-WL > WL-1, pre-WL = WG = WL-2
Moriguti *et al.* (2000)	*n* 11 (5 m + 6 f) 25·7 ± 3·2 years 23·2 ± 1·6 kg/m² (range: 18, 24) Data were extracted only for the NW subjects' group.	**Pre-WL (2 weeks):** weight maintenance (13 % protein + 35 % fat + 52 % CHO) **WL (6 weeks):** –4·2 MJ/d from fat and CHO of EI_{pre-WL} **WG-1 (6 weeks) + WG-2 (6 weeks) + WG-3 (6 weeks) + WG-4 (6 weeks):** *ad libitum* EI without specific instructions Meals and energetic beverages were provided by the research centre (except for the last 22 weeks of WG). They were encouraged to pursue a normal lifestyle during the study and to maintain their PA level (no change was observed between pre-WL and WL).	Anthropometry/body composition: BW; height; BMI; FFM and FM (hydrodensitometry) Energy intake: Energy excretion (3-d faecal collection at pre-WL and WL); dietary compliance (osmolar excretion rate from urine samples at WL); frequency of hunger, satiety, thirst, and constipation (5-point scale, at pre-WL and WL) Energy expenditure: PA level (7-d waist monitor, and self-reported record for strenuous physical activity; at pre-WL and WL)	**Pre-WL/WL^{NAS}** Food intake (MJ/d): 11·6 ± 2·7/8·0 ± 3·2 Energy excretion (MJ/d): 0·33 ± 0·23/0·54 ± 0·36 Relative energy excretion (% of EI): 2·6 ± 3·7/5·5 ± 3·0 **WL/WG-1/WG-2/WG-3/WG-4** BW (kg): NR***/NR*/NR/NR ΔBW(kg): –3·13 ± 1·13/*–1·62 ± 1·36*/–*1·47 ± 1·44*/–*0·27 ± 1·97*/–0·28 ± 2·32 ΔFM(kg): –1·77 ± 1·29/NR/NR/NR/–0·05 ± 1·69 ΔFFM(kg): –1·35 ± 0·96/NR/NR/NR/–0·23 ± 1·56 → NW subjects regaining significantly more BW than OW (Time × Group effect, $P < 0·001$)
Roberts *et al.* (1994)	*n* 10 (m) 22·0 ± 3·0 years 22·8 ± 1·8 kg/m² Non-smokers, no history of endocrinopathy. Data were extracted only for the young subjects' group.	**Pre-WL (10 d):** weight maintenance (protein: 1·5 g/d/kg of BW + 45 % fat and 55 % CHO for the non-protein macronutrients) **WL-1 (9 d) + WL-2 (12 d):** –3·3 MJ/d of EI_{pre-WL} (protein: 1·5 g/d/kg of BW + 45 % fat and 55 % CHO for the non-protein macronutrients) **WG-1 (10 d):** *ad libitum* EI with specific instructions to not change the composition of food intake. Subjects were asked to not weigh themselves **WG-2 (36 d):** *ad libitum* EI without specific instructions Meals and energetic beverages were provided by the research centre, and for WG-2 phase, subjects were permitted to consume additional energetic beverages. They were encouraged to pursue a normal lifestyle during the study.	Anthropometry/body composition: BW; height; BMI; FFM (doubly labelled water); FM (BW-FFM) Energy intake: Food intake (weighed food during pre-WL, WL and WG-1); metabolisable energy (digestible EI from faecal samples) Energy expenditure: TEE (doubly labelled water); REE (indirect calorimetry); strenuous PA (self-reported on sheet)	**Pre-WL/WL (1 and 2)WG-1/WG-2** Metabolisable energy (MJ/d): 14·07 ± 2·20/NR/NR*/N/A BW (kg): NR/NR†/NR/NR FFM (kg): 58·4 ± 9·7/NR/NR/NR FM (%): 15·7 ± 6·2/NR/NR/NR TEE (MJ/d): 14·48 ± 2·68/NR/NR/NR Strenuous PA (min/d): 29·2 ± 24·5/NR/NR/NR **WL-1/WL-2/WG-1/WG-2** ΔBW (kg): *–0·7 ± 0·9*/*–1·6 ± 1·3*/+*0·5 ± 0·9*/+*1·1 ± 0·9* **WG-1 (days 1 to 10)** ΔFood intake (%): +*33·1 ± 22·1*/+*16·9 ± 18·7*/+*18·0 ± 24·3*/+*14·2 ± 20·2*/+*13·9 ± 17·4*/+*10·9 ± 12·3*/+*20 ± 18·3*/+*17·8 ± 14·2*/+*16·9 ± 25·3*/+*14·2 ± 20·6* → Negative correlation between initial FM (%) and EI at WG-1

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Table 1. (*Continued*)

Reference	Characteristics of the groups	Interventional protocol	Measurements–parameters (method)	Main results
Siggaard *et al.* (1996)	*n* 18 (17 m + 1 f) 34·7 ± 11·0 years 23·8 ± 1·3 kg/m² (range: 22·0, 24·9) *n* 3 smokers, *n* 10 with previous history of dieting, *n* 8 who exercised (2 times/week) Data were extracted only for the NW subjects' group.	**Pre-WL:** baseline (measured EI = 8·5 ± 1·3 MJ/d) **WL-1 (6 weeks) + WL-2 (6 weeks):** *ad libitum* high-CHO and low-fat diet. Subjects received a dietary guideline guaranteed a minimum of 7·0 to 8·0 MJ/d (15–20 % protein + 20–25 % fat + 60–65 % CHO) **WG-1 (12 weeks) + WG-2 (28 weeks):** subjects were encouraged to maintain the *ad libitum* high-CHO and low-fat diet and not to begin other diets No change of smoking status or PA level was observed during the study.	Anthropometry/body composition: BW; height; BMI; FFM and FM (bioimpedance, only at pre-WL, WL-1 and WL-2); waist and hip circumferences (only at pre-WL and WL-2) Energy intake: Food intake (4-d food record at pre-WL and probably at the beginning of WG-1); history of dieting (questionnaire) Energy expenditure (only at pre-WL and WL-2): PA level (questionnaire) Others (only at pre-WL and WL-2): Smoking status (questionnaire)	**WL-1/WL-2/WG-1/WG-2** [NAS] ΔBW (kg): *−1·86 ± 1·31*/–2·5 ± 1·7 (–3·7 % of BW_{pre-WL}) (NW < OW)/*–1·96 ± 1·31*/–*1·69 ± 1·26* (time effect: $P < 0·05$) WL > 5 kg (% of sample): NR/11 %/0 %/0 % 2·5–5 kg WL (% of sample): NR/44 %/25 %/25 % 0–2·5 kg WL (% of sample): NR/39 %/42 %/50 % WG (% of sample): NR/6 %/33 %/25 % ΔBMI (kg/m²): −0·7 ± 0·4 (NW = OW)/–0·9 ± 0·4 (NW < OW)/NR/NR ΔFM (kg): −1·9 ± 1·3 (NW < OW)/–2·8 ± 1·7 (NW < OW)/NR/NR ΔFM (%): −1·9 ± 2·1 (NW < OW)/–3·2 ± 1·7 (NW < OW)/NR/NR ΔFFM (kg): *+0·3 ± 1·2*/*+0·6 ± 1·4*/NR/NR ΔWaist-hip ratio: −0·01 ± 0·04/–0·01 ± 0·04/NR/NR No apparent statistical for pre-WL *v.* WL or WG measures

m, males; NR, not reported; WL, weight loss; CHO, carbohydrates; WG, weight gain; EI, energy intake; BW, body weight; FM, fat mass; FFM, fat-free mass; REE, resting energy expenditure; TEE, total energy expenditure; f, females; T3, triiodothyronine; T4, thyroxine; NAS, no apparent statistical between pre-WL and WL or WG measures; NS, no significant; NW, normal-weight group; OW: overweight group; PA, physical activity; *Italic*, values collected from graphs; Δ, change from pre-WL measure.
Results are expressed in mean and standard deviation.
* $P < 0·05$,
** $P < 0·01$,
*** $P < 0·001$, significantly different from pre-WL measure.
† Significantly different from pre-WL but *P*-value not reported.
‡ Statistical unclear.
§ Statistical performed from raw data (repeated-measures ANOVA).

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Table 2. Characteristics of the populations, time points assessments, measurements and parameters assessed in the observational studies

Reference	Characteristics of the groups (sample size (sex); initial age; BMI)	Time points measurements	Measurements–parameters (method)
Anastasiou *et al.* (2010)	*Cohort of adults from Greece* **High-body-fat (FM > 30 %) (HF)** *n* 15 (f); 21·1 ± 1·9 years; 22·7 ± 1·9 kg/m²** **Low-body-fat (FM < 30 %) (LF)** *n* 17 (f); 22·1 ± 3·3 years; 19·8 ± 0·8 kg/m²**	N/A (cross-sectional study)	Anthropometry/body composition: BW; height; waist and hip circumferences; FM, FFM (DXA) Energy intake, dietary and eating behaviours: Food intake (24-h food record); weight cycling (25-item Weight Cycling Questionnaire that includes the EAT-26 questionnaire); accuracy of reporting food intake (food intake/REE ratio, < 0·9 = under-reporter of self-reported food intake) Energy expenditure: PAEE (Harokopio Physical Activity Questionnaire); REE (equation of Schofield); TEE Physical condition: Aerobic capacity (Astrand test) Biological parameters: Glucose, TAG, FFA, total cholesterol, HDL, LDL, insulin (fasting blood samples); OGTT; HOMA
Colditz *et al.* (1990)	*Nurses' Health Study cohort (USA)* *n* 31 940 (f); range: 30, 54 years; NR Smokers, pregnant women, women who consumed > 15 g alcohol/d, women with special diets and women with prior diagnosis of cardiometabolic disease were excluded (during all the follow-up).	**Total: 8 years** T0: baseline T1: 2 years T2: 4 years T3: 6 years T4: 8 years	Anthropometry/body composition: BW (self-reported and measured for *n* 184); height (self-reported) Energy intake, dietary and eating behaviours (only at T2): Food intake (FFQ, 1-week food record for *n* 194)
Karkkainen *et al.* (2018)	*FinnTwin16 cohort (Finland)* **m maintainers** *n* 513 (m); 24 years; 24·2 ± 2·9 kg/m² **m gainers** *n* 1630 (m); 24 years; 23·7 ± 3·0 kg/m² **f losers** *n* 185 (f); 24 years; 23·9 ± 3·7 kg/m² **f maintainers** *n* 701 (f); 24 years; 21·8 ± 3·1 kg/m² **f gainers** *n* 1566; 24 years; 22·1 ± 3·5 kg/m² NAS for groups differences Participants are defined like 'maintainers' if their BW at T1 was within ± 5 % of their BMI at T0. Subjects suffering from chronic, potentially weight-affecting illness at T0 and T1 were excluded.	**Total: 10 years** T0: baseline T1: 10 years	Anthropometry/body composition: BW; height; waist circumference (self-reported); BMI Energy intake, dietary and eating behaviours (only at T0): Intentional WL ≥ 5 kg (1 question); regularity of eating and eating styles (self-reported questionnaire developed by Keski-Rahkonen, 2007); daily breakfast (1 question); food and fluid intake (FFQ); eating behaviours (bulimia subscale of EDI) Energy expenditure (only at T0): PAEE (self-reported) Others (only at T0): Life satisfaction (Allardt's four-item scale); self-rated health (one question); education level
Korkeila *et al.* (1999)	*FinnTwin16 cohort (Finland)* **m non-dieters** *n* 1731 (m) (T2: *n* 1120); range: 18, 29 years; 22·2 ± 2·1 kg/m²** **m dieters** *n* 142 (m) (T2: *n* 99); range: 18, 29 years; 24·3 ± 2·3 kg/m²** **f non-dieters** *n* 1811 (f) (T2: *n* 1294); range: 18, 29 years; 20·3 ± 2·1 kg/m²** **f dieters** *n* 475 (f) (T2: *n* 329); range: 18, 29 years; 22·6 ± 2·2 kg/m²** Pregnant women were excluded.	**Total: 15 years** T0: baseline T1: 6 years T2: 15 years	Anthropometry/body composition: BW; height (self-reported and measured for *n* 225 at T2); BMI Energy intake, dietary and eating behaviours (only at T0): WL attempts (questionnaire) Energy expenditure (only at T0): PAEE (self-reported) Others (only at T0): Smoking status, alcohol consumption, education level, social class and marital status (questionnaire)

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Table 2. (*Continued*)

Reference	Characteristics of the groups (sample size (sex); initial age; BMI)	Time points measurements	Measurements–parameters (method)
Lowe *et al.* (2006)	*Cohort of American first-year college students* **Dieters (past + current)** *n* 29 (f); NR; NR **Non-dieters** *n* 40 (f); NR; NR **Total groups** *n* 69 (f); 18·1 ± 0·2 year (range: 18, 19); 21·9 ± 2·4 kg/m² (range: 17·4, 26·6) Any medications known to affect appetite or weight excluded, no eating disorder in the year prior the study.	**Total: 8 months** T0: baseline T1: 3 weeks T2: 4 months T3: 8 months	Anthropometry/body composition: BW; height; BMI; highest BW in life (self-reported) Energy intake, dietary and eating behaviours (only at T1): Dieting history and restraint behaviours (10-item Restraint Scale, Classification of Lowe, 1993); BW suppression (= BW_{T0} – Highest BW in life); eating behaviours (TFEQ and DEBQ)
Palascha *et al.* (2015)	*Cohort of Dutch adults* *n* 241 (49 m + 192 f); 32·3 ± 15·3† years (range: 15, 74); 23 ± 4·5† kg/m² No specific inclusion or exclusion criteria.	N/A (cross-sectional study)	Anthropometry/body composition: BW, height (self-reported); BMI Energy intake, dietary and eating behaviours: Eating behaviours (Restraint Eating subscale of DEBQ); dichotomous thinking regarding food and dieting (eleven-item Dichotomous Thinking in Eating Disorders Scale); WL during the last 5 years (yes/no, no distinction between voluntary or unvoluntary WL); weight regain of 4 kg or more following WL (yes/no); dieting (yes/no) Others: Dichotomous thinking regarding general personality (fifteen-item Dichotomous Thinking Inventory); education level
Pietiläinen *et al.* (2012)	*FinnTwin16 cohort (Finland)* *n* 4129 (1922 m + 2207 f); 18 years; At 16 years; m: 20·4 kg/m², f: 20·2 kg/m² Study started at 16 years, but results were extracted from 18 years (T0) for subjects with intermediate basal BMI (25th percentile–75th percentile) Subjects with diabetes mellitus, systemic lupus erythematosus, inflammatory bowel disease, celiac disease, hyper- or hypo-thyroidism, malignancies, mobility disorders, eating disorders or with medication affecting BW were excluded.	**Total: 7 years** T0: baseline T1: 7 years	Anthropometry/body composition: BW, height (self-reported and measured for *n* 566); BMI; late physical maturity (growth in height between 16 and 18 years) Energy intake, dietary and eating behaviours: Number of intentional WL ≥ 5 kg (one question at T1); daily breakfast (one question) Energy expenditure: PAEE (questionnaire) Others: Smoking status (questionnaire); number of children (question at T1); parental BMI, father's socio-economic status (questionnaire at 16 years)
Sares-Jäske *et al.* (2019)	*Health Survey cohort (Finland)* *n* 2785 (1268 m + 1517 f); 47·2 ± 10·2 years (range: 30, 69); 26·5 ± 4·4 kg/m² Weight changes were analysed by BMI categories. Pregnant women at T0 and/or T1 were excluded.	**Total: 11 years** T0: baseline T1: 11 years	Anthropometry/body composition: BW; height; waist circumference Energy intake, dietary and eating behaviours: Dieting attempts, WL and WG history during the previous year (questionnaire at T0); food intake (FFQ); diet quality (Alternate Healthy Eating Index modified for Finnish dietary culture) Energy expenditure: PAEE (questionnaire) Biological parameters: Blood pressure Others: Mental disorders (DSM-IV and Interview); burnout (Maslach Burnout Inventory, in subjects who had been working during the last 12 months); marital status, education, smoking status, cancer diagnosis, self-rated health (interview); alcohol consumption (questionnaire); sense of coherence (Antonovsky's SOC-13 scale); type 2 diabetes diagnosis (interview, health examination and questionnaire); knee or hip arthrosis (health examination)

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Table 2. (*Continued*)

Reference	Characteristics of the groups (sample size (sex); initial age; BMI)	Time points measurements	Measurements–parameters (method)
van Strien *et al.* (2014)	*Cohort of Dutch adults* Extracted from raw data at T0 for NW participants **m dieters** *n* 8; NR; 23·43 ± 1·60 kg/m² **m non-dieters** *n* 160; NR; 22·86 ± 1·64 kg/m² **f dieters** *n* 21; NR; 22·95 ± 1·39 kg/m² **f non-dieters** *n* 152; NR; 22·39 ± 1·65 kg/m² **Total groups** *n* 341 (168 m + 173 f); 47·8 ± 14·4 years; 22·67 ± 0·30 kg/m² Underweight subjects were excluded.	**Total: 1·5 years** T0: baseline T1: 4 months T2: 12 months T3: 16 months	Anthropometry/body composition: BW, height (self-reported) Energy intake, dietary and eating behaviours: Dieting status (Restraint Scale, nine-item) Energy expenditure: PAEE in summer and winter (self-reported)
van Wye *et al.* (2012)	*Aerobics Center Longitudinal Study cohort (USA)* **Weight cyclers (WC) (minimum 5 WL episodes of ≥ 2·3 kg)** *n* 68 (f); 45·4 ± 7·8 years; 23·4 ± 4·0 kg/m²* **Non-cyclers (NC)** *n* 73 (f); 47·9 ± 10·5 years; 21·4 ± 2·2 kg/m²* Subjects who reported unintentional WL at T0 were excluded.	**Total: 6 years** T0: baseline T1: 6 years	Anthropometry/body composition: BW; height; BMI; FM (method NR) Energy intake, dietary and eating behaviours: Weight cycling (one question adapted from the Brownell Weight Cycling Questionnaire at T0); dieting status (method NR, probably one question) Physical condition: Aerobic capacity (maximal exercise treadmill) Others: Smoking status (method NR, probably one question); marital status, chronic disease, depression and level of perceived tension/anxiety (questions)

FM, fat mass; HF, high-body fat group; f, females; LF, low-body fat group; BW, body weight; FFM, fat-free mass; DXA, dual-energy X-ray absorptiometry; EAT-26, 26-item Eating Attitudes Test; REE, resting energy expenditure; PAEE, physical activity energy expenditure; TEE, total energy expenditure; FFA, fat-free acids; OGTT, oral glucose tolerance test; HOMA, homeostatic model assessment of insulin resistance; NR, not reported; m, males; NAS, no apparent statistical; WL, weight loss; EDI, Eating Disorder Inventory; TFEQ, Three-Factors Eating Questionnaire; DEBQ, Dutch Eating Behavior Questionnaire; WG, weight gain; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; WC, weight-cyclers; NC, non-cyclers.
Results are expressed in mean and standard deviation.
* $P < 0.05$,
** $P < 0.001$, dieters *v.* non-dieters or HF *v.* LF or WC *v.* NC.
† No information about the nature of data (SD or SEM).

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Table 3. Main results of the observational studies selected in the systematic review, classified as three subgroups: results relative to dietary habits and weight loss (food intake, dietary and WL), main results on body weight and body composition, and additional results in link with body weight changes and/or diet

Reference	Food intake, dietary and WL	Main results on BW and/or body composition	Additional results
Anastasiou *et al.* (2010)	Food intake (MJ/d): **HF:** 7·3 ± 2·8* **LF:** 9·4 ± 3·7* Macronutrients: **HF = LF** Accuracy of reporting food intake (food intake/REE ratio): **HF:** 1·2 ± 0·4* (40 %* of under-reporters) **LF:** 1·8 ± 0·8* (6 %* of under-reporters) Dieting frequency (% sample): **HF:** 13 % Never, 53 % Seldom, 13 % Sometimes, 20 % Frequently **LF:** 65 % Never, 12 % Seldom, 18 % Sometimes, 6 % Frequently Interaction between group and diet frequency ($P < 0·05$) Number of WL per year: - 2–3 kg: **HF > LF** - > 5 kg: **HF = LF** Number of WL per lifetime: - 1·0–2·5 kg, 3·0–5·0 kg, > 6 kg: **HF = LF**	BW (kg); height (cm); FM (%): **HF:** 64·1 ± 7·4 kg*; 169 ± 15 cm*; 34·9 ± 3·9 %*** **LF:** 53·3 ± 4·1 kg*; 164 ± 13 cm*; 26·2 ± 2·5 %*** → Losing 1·0 to 2·5 kg per lifetime = ↗ the risk of high FM → Higher BW and TAG levels = ↘ insulin sensitivity	Eating behaviours: Satisfied body size and image: **HF < LF** Oral control score, bulimia scale, total EAT-26 score: **HF = LF** Dieting scale: **HF > LF** Energy expenditure: TEE: **HF > LF** Physical condition: Aerobic capacity: **HF < LF** Biological parameters: OGTT (glucose and insulin), TAG, FFA, total cholesterol, HDL, LDL: **HF = LF** Insulin/glucose: **HF > LF** Fasting insulin and glucose: **HF > LF****
Colditz *et al.* (1990)	**At T1** Food intake (*n* in sample)[NAS]: - ≤ 5·0 MJ/d: *n* 7578 -]5·0–7·7] MJ/d: *n* 15 938 - > 7·7 MJ/d: *n* 8399 CHO (*n* in sample)[NAS]: - ≤114 g/d: *n* 8336 - [115–18]) g/d: *n* 16 020 - > 189 g/d: *n* 7584 Fat (*n* in sample)[NAS]: - ≤ 49 g/d: *n* 8149 - [50–85] g/d: *n* 16 019 - > 85 g/d: *n* 7772 Protein (*n* in sample)[NAS]: - ≤ 56 g/d: *n* 8182 -]56–87] g/d: *n* 15 701 - > 87 g/d: *n* 8057	BW or ΔBW (kg): In function of weight change from T0 to T2 (**T2/ ΔT2-T4**)[NAS]: - WL > 5 kg (*n* 944): 66·16 ± 13·17/+5·79 ± 7·08 - WL = 3–5 kg (*n* 1300): 62·83 ± 10·35/+3·46 ± 4·62 - WL = 3 kg–WG = 3 kg (*n* 19 988): 61·68 ± 9·53/+1·54 ± 3·57 - WG = 3–4·9 kg (*n* 5178): 67·21 ± 10·36/+1·09 ± 4·36 - WG = 5–9·9 kg (*n* 3407): 74·19 ± 11·73/+1·02 ± 5·65 - WG > 10 kg (*n* 1123): 87·88 ± 14·74/–0·16 ± 9·06 In function of food intake at T0 (**T1/ ΔT1-T2/ ΔT2-T4**)[NAS]: - ≤ 5·0 MJ/d (*n* 8578): 63·4/+0·35 ± 1·8/+0·41 ± 1·2 -]5·0–7·7] MJ/d (*n* 15 938): 64·0/+0·44 ± 1·7/+0·38 ± 1·1 - > 7·7 MJ/d (*n* 8399): 65·1/+0·51 ± 1·8/+0·38 ± 1·2	N/A
Karkkainen *et al.* (2018)	**At T0 (recalculated data)** Participants with history of WL ≥ 5 kg (% of sample)[NAS]: **m maintainers:** 6 % **m gainers:** 12 % **f losers:** 27 % **f maintainers:** 14 % **f gainers:** 18 % Participants who daily breakfast (% of sample)[NAS]: **m maintainers:** 51 % **m gainers:** 48 % **f losers:** 61 %	BW or ΔBW (kg) (T0/ ΔT0–T1)[NAS]: **m maintainers:** 78·4 ± 10·9/+1·0 ± 2·3 **m gainers:** 76·2 ± 11·5/+10·3 ± 6·3 **f losers:** 66·0 ± 10·7/–5·6 ± 3·8 **f maintainers:** 60·4 ± 9·2/+0·4 ± 1·8 **f gainers:** 60·6 ± 10·4/+9·2 ± 6·3 Waist circumference or ΔWaist circumference (cm) (**T0/ ΔT0–T1**)[NAS]: **m maintainers:** 85·3 ± 8·8/+3·0 ± 5·2 **m gainers:** 85·1 ± 9·1/+10·1 ± 7·4 **f losers:** 78·7 ± 9·7/–2·4 ± 8·2 **f maintainers:** 73·4 ± 7·9/+3·3 ± 6·9 **f gainers:** 74·7 ± 9·6/+10·0 ± 8·3	Control of eating (% of sample at T0)[NAS]: **m maintainers:** 21 % overeating, 2 % restrictive, 1 % restrictive and overeating **m gainers:** 13 % overeating, 3 % restrictive, 1 % restrictive and overeating **f losers:** 30 % overeating, 17 % restrictive, 8 % restrictive and overeating **f maintainers:** 22 % overeating, 12 % restrictive, 6 % restrictive and overeating **f gainers:** 23 % overeating, 12 % restrictive, 6 % restrictive and overeating

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Table 3. (*Continued*)

Reference	Food intake, dietary and WL	Main results on BW and/or body composition	Additional results
	f maintainers: 64 % **f gainers:** 58 % Regularity of eating (% of sample)[NAS]: **m maintainers:** 13 % regular, 59 % quite regular, 24 % quite irregular, 4 chaotic % **m gainers:** 9 % regular, 55 % quite regular, 29 % quite irregular, 11 % chaotic **f losers:** 8 % regular, 52 % quite regular, 33 % quite irregular, 6 % chaotic **f maintainers:** 8 % regular, 59 % quite regular, 22 % quite irregular, 10 % chaotic **f gainers:** 6 % regular, 57 % quite regular, 27 % quite irregular, 9 % chaotic	Factors associated with weight maintenance or WG: **m:** OW at T0 = ↘ risk of WG; underweight at T0 = ↗ risk of WG; irregular eating, dieting and smoking were associated with WG; higher baseline BMI and higher education level were associated with weight maintenance. **f:** OW at T0 = ↗ risk of WG; sweet drinks, irregular eating, history of dieting, smoking, low satisfaction life, having two or more children were associated with WG; physical activity was associated with weight maintenance.	
Korkeila *et al.* (1999)	Frequency of dieting: - Smokers > non-smokers - Subjects with highest alcohol consumption > others - Men in upper social classes > men in lower social classes - High energy expenditure during leisure time and heavy PA work were associated with likelihood of dieting.	ΔBW(kg) (**ΔT0–T1/ ΔT0–T2**): **m non-dieters:** +3·60 ± 5·4/+7·45 ± 7·7**** **m dieters:** +3·65 ± 5·5/+8·89 ± 7·7**** **f non-dieters:** +2·26 ± 4·7/+6·23 ± 7·6 **f dieters:** +1·91 ± 4·8/+6·14 ± 7·6 Frequency (% of sample) of WG > 10kg (**T1/T2**): **m non-dieters:** 9 %/29 % **m dieters:** 12 %/36 % **f non-dieters:** 4 %/22 % **f dieters:** 7 %/25 % Factors associated with BMI at T0: - Smokers > non-smokers - Men with high alcohol consumption > men with low alcohol consumption - Married women > single women/married men = single men - Negative correlation of education level and social class with BMI - Positive correlation between work PA work and BMI - No correlation of PA during leisure time and BMI	Frequency (% of sample) of WG > 10 kg in function of BMI_{T0} (T1/T2): - Subjects with BMI_{T0} < 25 (NW): **m non-dieters (*n* 1551):** 9 %/29 % **m dieters (*n* 81):** 16 %/41 % **f non-dieters (= 1754):** 4 %/21 % **f dieters (*n* 392):** 6 %/25 % - Subjects with $BMI_{T0} \geq 25$ (OW): **m non-dieters (*n* 180):** 8 %/28 % **m dieters (*n* 61):** 7 %/30 % **f non-dieters (*n* 57):** 9 %/33 % **f dieters (*n* 83):** 12 %/27 % → At T1, ↗ risk of major WG in NW dieters' men but not in OW dieters' men → At T1, trend for ↗ risk of major WG in dieters' women (NW and OW) Pairwise analyses of twins on BMI: **Dieters** > **non-dieters** (at T0, T1 and T2)
Lowe *et al.* (2006)	BW suppression (kg): - Low WS (*n* 34): −0·77 ± 0·69 - High WS (*n* 35): −4·68 ± 3·65	BMI at T0: **dieters** = **non-dieters** (Δ = 0·9 kg/m²) BW or ΔBW (*v.* T0) (kg) (**T0/T1/T2/T3**): - In function of dieting status: **Total groups:** NR/+0·91/+1·90/+2·08 **Dieters:** **- Current dieters (*n* 7):** *61·4/NR/66/65·1* (+5·0) **- Past dieters (*n* 21):** *61·4/NR/62·9/63·9* (+2·5) **Non-dieters:** *57·4/NR/58·9/58·9* (+1·6) - In function of BW suppression status: **Low WS:** *59·1/NR/60·5/60·3* (+1·20) **High WS:** *59·1/NR/61·4/62·1* (+2·97) Time effect ($P < 0·01$), dieting status x time effect ($P < 0·05$), BW suppression effect ($P < 0·01$) on WG ([NAS] for *post hoc*) ΔBW (**ΔT0–T3**) (kg): **Low WS/non-dieters:** *+1·3 ± 0·7* **Low WS/dieters:** *+1·5 ± 0·7* **High WS/non-dieters:** *+2·1 ± 0·5* **High WS/dieters:** *+4·5 ± 0·7* → **High WS/dieters** > other groups (trend)	Predictors of weight changes: None of BMI_{T0} or parameters evaluated from questionnaires (Restraint Scale, TFEQ and DEBQ) predict weight changes.

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Table 3. (*Continued*)

Reference	Food intake, dietary and WL	Main results on BW and/or body composition	Additional results
Palascha *et al.* (2015)	Currents dieters (% of sample): 34 % (*n* 82)	Weight regain (4 kg or more) after WL: Yes = '**Weight regainers**' (*n* 55, 23 %) No = '**Weight maintainers**' (*n* 45, 19 %) (No WL for the other subjects) → Positive correlation of dieting with weight regain, restraint eating, BMI and dichotomous thinking regarding foods/dieting → Positive correlation of weight regain with restraint eating, BMI and dichotomous thinking regarding foods/dieting → No correlation of dieting and weight regain with general dichotomous thinking	Eating behaviours: → Positive correlation of restraint eating with BMI and dichotomous thinking (general personality and regarding food/dieting) → Positive correlation between BMI and dichotomous thinking regarding foods/dieting → Positive correlation between dichotomous thinking regarding general personality and dichotomous thinking regarding foods/dieting Restraint eating (score): **weight regainers** > **weight maintainers** Dichotomous thinking regarding foods/dieting (score): **weight regainers** > **weight maintainers**; **dieters** > **non-dieters**
Pietiläinen *et al.* (2012)	Intentional WL ≥ 5 kg (% of sample): **m**: 76 % never, 13 % once, 9 % at 2–4 times, 2 % at ≥ 5 times **f**: 62 % never, 23 % once, 13 % at 2–4 times, 2 % at ≥ 5 times → Higher BMI at 16 years, smoked daily, skipped breakfast and having a father in blue collar social class = ↗ risk having ≥1 intentional WL.	BMI (kg/m²) in function of number of intentional WL (**T0/T1**): **m:** - 0 WL: 21·4/23·2 - 1 WL: 22·1/24·7 - 2–4 WL: 22·5/25·0 - ≥ 5WL: 22·8/25·8 Effect of number of intentional WL ($P < 0·001$) (^{NAS} for *post hoc*) **f:** - 0 WL: 20·1/21·3 - 1 WL: 20·7/22·0 - 2–4 WL: 21·1/22·6 - ≥ 5 WL: 21·1/23·2 Effect of number of intentional WL ($P < 0·001$) (^{NAS} for *post hoc*) → The risk of becoming OW at T1 in the initially NW participants was proportional to the intentional WL frequency	→ Higher BMI at 16 years = ↗ risk of OW at T1 → In **females**, low PAEE at T0, father's low socioeconomic status and father's OW = ↗ risk of OW at T1 → WG in **males** > WG in **females** → WG in **passive females** > WG in **active females** → Parental BMIs were higher for subjects with the history of WL than subjects without history of WL Pairwise analyses of twins on BMI (kg/m²): In monozygotic twins, BMI at T1 (but not at 16 years and T0) is higher in twins with history of WL ($P < 0·05$) = dieting effect on WG In dizygotic twins, BMI is always higher in twins with history of WL (16 years, T0, T1) ($P < 0·001$) = genetic predisposition to higher BMI
Sares-Jäske *et al.* (2019)	N/A	ΔBMI (kg/m²) (**ΔT0–T1**): **- In dieters:** **NW (*n* 191):** 1·41 ± 2·42† **OW (*n* 409):** 1·17 ± 2·52 **With obesity (*n* 313):** 0·54 ± 3·14 **- In non-dieters:** **NW (*n* 934):** 0·76 ± 1·81† **OW (*n* 707):** 0·81 ± 2·12 **With obesity (*n* 231):** 0·69 ± 3·10 Dieting attempts x BMI interaction ($P < 0·01$) ΔWaist circumference (cm) (**ΔT0–T1**): **- In dieters:** **NW:** 4·12 ± 8·23 **OW:** 3·47 ± 8·18 **With obesity:** 1·65 ± 8·78 **- In non-dieters:** **NW:** 2·11 ± 6·18 **OW:** 2·58 ± 7·08 **With obesity:** 2·48 ± 8·76 Dieting attempts x initial BMI interaction ($P < 0·01$) (^{NAS} for *post hoc*) No interaction effect of previous weight changes at T0 (WL, WG or weight maintenance) x initial BMI on BMI change (ΔT0–T1).	N/A

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Table 3. (*Continued*)

Reference	Food intake, dietary and WL	Main results on BW and/or body composition	Additional results
van Strien *et al.* (2014)	Higher WL within 1 month (kg)‡: **m non-dieters:** 1·26 ± 0·72 **m dieters:** 1·63 ± 0·74 **f non-dieters:** 1·26 ± 0·61* **f dieters:** 1·57 ± 0·68* Higher BW in life (kg)‡: **m non-dieters:** 80·78 ± 10·28 **m dieters:** 83·13 ± 13·52 **f non-dieters:** 69·05 ± 9·43** **f dieters:** 75·14 ± 13·09**	BW (kg)‡: **m non-dieters:** 75·69 ± 7·90 **m dieters:** 73·38 ± 7·48 **f non-dieters:** 63·34 ± 6·88 **f dieters:** 65·33 ± 6·94 Higher WG in a week (kg)‡: **m non-dieters:** 1·67 ± 0·98 **m dieters:** 2·00 ± 1·07 **f non-dieters:** 1·71 ± 0·99** **f dieters:** 2·43 ± 1·21**	Feelings of guilt after overeating‡: **Non-dieters** < **dieters**
van Wye *et al.* (2012)	Dieting frequency (% of sample): **WC:** (35 % sometimes, 33 % often and 14 % always)* **NC:** (34 % sometimes, 16 % often and 7 % always)*	BW at T0 (kg); height (cm); FM(%); ΔWG(kg) (ΔT0–T1); estimated WG over 10 years (kg/year): **WC:** 62·1 ± 8·5 kg***; 163·3 ± 6·1 cm*; 25·7 ± 6·3 %; +2·4 ± 5·5****; +0·5 kg/year**** **NC:** 58·7 ± 6·6 kg***; 165·6 ± 5·6 cm*; 24·9 ± 6·5 %; +0·7 ± 4·0****; +0·25 kg/year**** → ↗ aerobic capacity at T0 = ↘ estimated WG	Others: Marital status, smoking status, history of chronic disease, tension and anxiety: **WC** = **NC**

WL, weight loss; BW, body weight; HF, high body-fat group; LF, low-body-fat group; REE, resting energy expenditure; FM, fat mass; EAT-26, 26-item Eating Attitudes Test; TEE, total energy expenditure; OGTT, oral glucose tolerance test; ; FFA, fat free acids; NAS, no apparent statistical test; CHO, carbohydrates; WG, weight gain; m: male; f, female; OW, overweight; PA, physical activity; NW, normal weight; WS, weight suppression; NR, not reported; TFEQ, Three-Factors Eating Questionnaire; DEBQ, Dutch Eating Behavior Questionnaire; IWL: intentional weight loss; PAEE, physical activity energy expenditure; WC, weight-cyclers; NC, non-cyclers.
Results are expressed in mean and standard deviation.
* $P < 0·05$.
** $P < 0·01$.
*** $P < 0·001$.
**** $P < 0·1$ (trend).
† Significant but *P*-value not reported, dieters *v.* non-dieters or HF *v.* LF or WC *v.* NC.
‡ Statistical performed from raw data (*t* tests).

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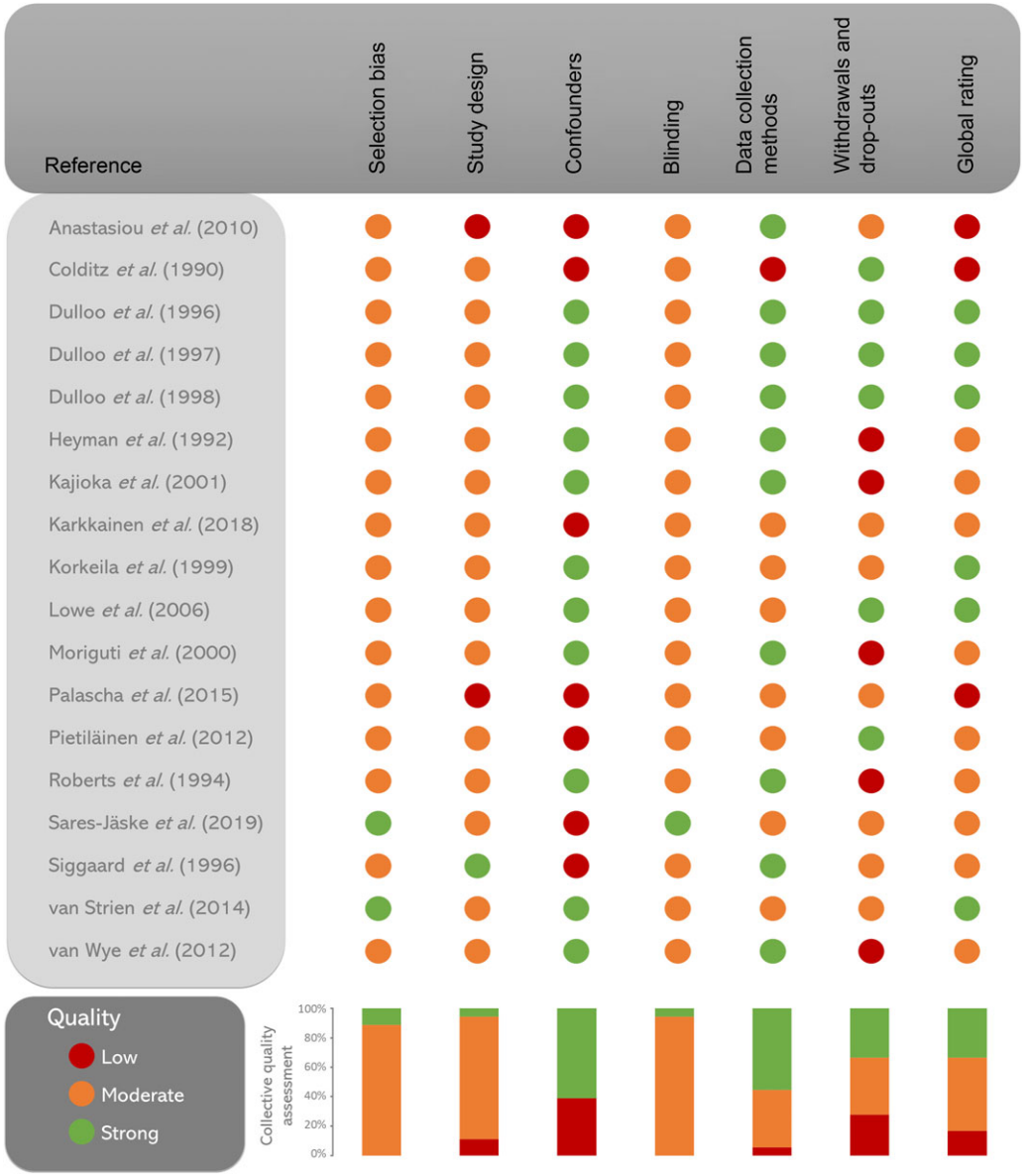

Fig. 2. Individual and collective quality assessment of the studies included in the systematic review.

Minnesota cohort was divided into four groups with an imposed recovery diet whose energy values varied from 9·95 MJ/d (2377 kcal/d) to 14·19 MJ/d (3389 kcal/d)[14]. In the other studies, diet interventions were immediately followed by a period of *ad libitum* energy intake. One study repeated, in the same participants, two 30-d energy-restricted dietary periods, separated by a free-living period of 14 d[38]. The ten remaining publications of the systematic review were observational studies, including two cross-sectional[41,42], and eight longitudinal studies that examined weight changes in the long term (8 months to 15 years) in participants representing the overall general population, with two to five measurement time points[24–27,32,33,35,36] (Table 2).

Characteristics of the participants

All the studies were performed in NW healthy individuals with a mean BMI between 19·5[41] and 24·3 kg/m²[25]. Among the eight interventional studies, three presented analyses using the same young male population (Minnesota study cohort, *n* 32, 25 ± 4 years)[13–15]. Three other studies enrolled only (or near, *n* 17 males and *n* 1 female for one study[40]) males[34,37,40], one study was performed in females[38], and the last interventional study was performed in both male and female subjects[39]. The majority of the observational studies involved the sampling from the general population, aged 16 years (but results extracted from 18 years)[27] to 54 years[26] at baseline, including large cohort

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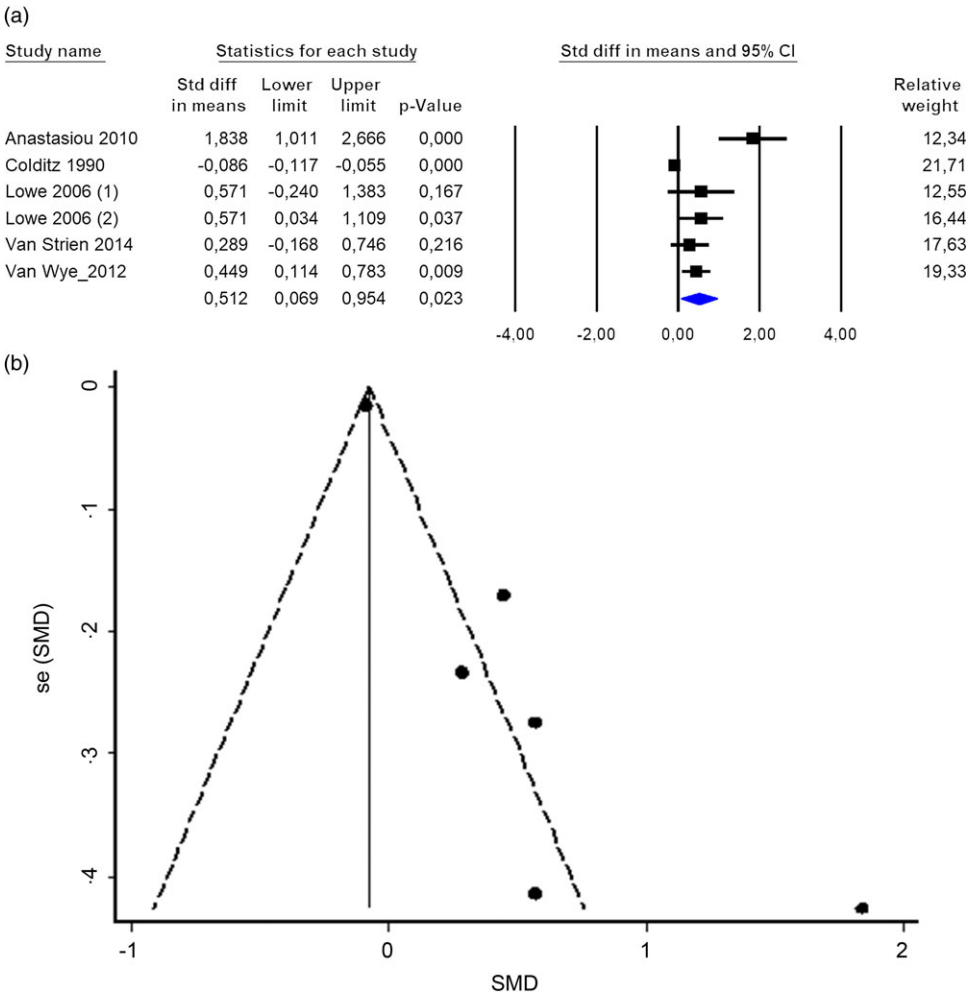

Fig. 3. Body weight comparison between dieters and non-dieters. (a) Forest plot of the meta-analysis. Data were obtained from observational studies and are expressed in standardised mean difference and their 95 %CI (lower and upper limits). (b) Funnel plot from the Egger's test. SMD, standardised mean difference.

studies such as the Finnish Twin cohort[25,27,33] and the Nurses' Health Study cohort[26]. Three observational studies were performed in women only[24,26,41], and only female participants were selected in one study since the males were overweight at baseline (BMI ≥ 25 kg/m²)[36]. In addition, one study selected two specific groups of women in function of their FM: the 'high-body-fat' group having a FM > 30 % and the 'low-body-fat' group having a FM < 30 %[41]. The remaining observational studies compared groups according to their dieting frequency[24,25,35] or their weight changes[26,33,36], resulting in groups of 'dieters' and 'non-dieters', or 'weight-cyclers' and 'non-cyclers'. For the study from van Strien *et al.* (2014)[32], NW dieter and non-dieter groups were identified based on the raw data that were available. Participants were defined as 'dieters' when they self-reported dieting 'sometimes', 'often', or 'always', and as 'non-dieters' when they reported having 'never' or 'rarely' done. Due to the low number of participants and the dropout rate during the long-term follow-up in the dieter groups (50 % of dropouts in males and 38 % in females), we extracted data at T0 only (Table 2).

Parameters assessed and methodological approaches

BW and body composition were assessed in all the interventional studies before WL ('pre-WL'), immediately after the WL phase ('WL'), and after a recovery diet period, termed 'weight (re)gain' ('WG') (except one study which assessed body composition only at pre-WL and WL[37]). FM and FFM were measured by hydrodensitometry[13–15,39], plethysmography[38], doubly labelled water[34,37], or bioimpedance[40]. Resting energy expenditure (REE) was evaluated by indirect calorimetry in six of the eight interventional studies[13–15,34,37,38]. Three studies analysed the *ad libitum* food intake during the recovery period[13,34,37]. In addition, one interventional and two observational studies performed biochemical analyses (*e.g.* total cholesterol, insulin, and thyroid hormones)[35,38,41] (Tables 1 and 2). Over half of the observational studies essentially used questionnaire measurements, with self-reported BW, height, and weight fluctuations[25–27,32,33,42]. In addition, body composition was assessed in only one study by dual-energy X-ray absorptiometry[41], and waist circumference was obtained in three

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studies[33,35,41]. Dieting status and food intake were measured by different techniques: 24-h or 1-week self-reported food record[26,41], 25-item Weight Cycling Questionnaire[41], FFQ[26,33,35], Restraint Scale[24,32], or self-reported dichotomous questions/no validated questionnaires[25,27,33,35,36,42]. In longitudinal studies, dieting status was evaluated once during the follow-up period, essentially at baseline (T0)[24,25,33,35,36], but also at the end of study[27] (Table 2). Van Strien and collaborators[32] measured it at each time point (but data were only extracted at T0 as explained above). One study did not assess the dieting status of subjects but measured food intake in the middle of the longitudinal follow-up[26]. Finally, several observational studies conducted energy expenditure assessments, including total energy expenditure and REE in one study[41], and self-reported physical activity energy expenditure in six studies[25,27,32,33,35,41]. Finally, aerobic capacity was evaluated in two observational studies by a maximal or submaximal cardiorespiratory test[36,41].

Main results from the interventional studies systematically reviewed

WL interventions resulted in a significant decrease of BW in all studies included[13–15,34,37–40] (Table 1). FM (% and/or kg) and FFM (kg) significantly decreased ($P < 0{\cdot}001$) in two cohorts after 30 d, 12 weeks, and 24 weeks of semi-starvation[13–15,38], with a reduction of REE also observed[13,38]. In the publication of Heyman *et al.* (1992), a decrease in absolute and relative (to FFM) REE was highlighted during and after WL ($P < 0{\cdot}01$)[37]. In the analyses of Dulloo and collaborators, most of the subjects of the Minnesota study did not regain their initial BW nor FM (kg), and none had regained FFM, after 12 weeks of imposed recovery diet[13–15]. However, following a subsequent 8 weeks of *ad libitum* energy intake, BW and FM (kg and %), but not FFM, were increased to higher values than those at baseline[14]. In one study[40], BW was not recovered after 40 weeks of a free-living period, but participants were encouraged to maintain the low-fat diet during this period. In three other studies, BW was restored as early as day 10 of *ad libitum* food intake[34,37,38], the regain being showed more rapid than the WL ($+257 \pm 120$ g/d *v.* -97 ± 79 g/d, $P < 0{\cdot}01$) in one publication[37]. In Moriguti *et al.*'s cohort, BW was restored after 6 weeks, and NW subjects regained significantly more than the group of overweight subjects[39]. In Kajioka's study, after the first recovery period (14 d), BW overshot baseline value while FM did not change (Table 1). Yet, after the second dieting phase and 106 d of free-living, BW and FM were similar to pre-WL values[38]. In addition, hyperphagia was observed in three studies relative to pre-WL energy intake[13,34,37]. Finally, after several weeks of recovery diet (12 to 15 weeks), REE remained at lower values than at baseline[13,38]. Results of biochemical parameters assessed in one study are detailed in Table 1. Finally, in Kajioka's study, blood pressure (both systolic and diastolic) was significantly reduced after the second WL phase but was significantly elevated compared with pre-WL after 15 weeks of free-living conditions[38].

Main results from the observational studies systematically reviewed and meta-analysed

Studies conducted in Greece and the USA observed that high-body-fat and weight-cycler groups had significantly higher BW and BMI and dieted more often compared with low-body-fat[41] and non-cycler[36] groups, respectively. In another such study conducted in Finland, BMI at baseline was higher ($P < 0{\cdot}001$) in male and female dieters compared with non-dieters[25], while a further study showed no difference in BMI and BW between these groups[32] (Table 3). Forty per cent of the studies reviewed observed a significant increase, or increasing trend, in BMI or BW in NW individuals engaging in diet/WL compared with those who did not diet and/or lose weight[25,32,35,36], while two others studies showed a positive association between WG and dieting[33,42]. Moreover, major WG was associated with a lower initial BW or BMI[25,27,33,35], irregular eating[33], dieting[24,25,27,33,42], smoking[33], and restrained eating[42]. Finally, five studies were selected for inclusion in the meta-analysis according to our selection criteria[24,26,32,36,41]. In the study of Anastasiou and collaborators, the high-body-fat group was considered analogous to 'dieters' and low-body-fat group to 'non-dieters', given that the high-body-fat group had a higher number of WL achieved per year and a higher total energy expenditure, while it had a reduced daily food intake and a higher score for the 26-item Eating Attitudes Test dieting subscale compared with the low-body-fat group[41]. For another study[26], we defined the 'dieters' as those who consumed less than 5·0 MJ/d (1205 kcal/d) (similar value to a semi-starvation), while 'non-dieters' were those who ingested more than 7·7 MJ/d (1844 kcal/d). Overall, the result of the meta-analysis showed that dieters had, on average, a higher BW compared with non-dieters (standardised mean difference: 0·512 [95 % CI: 0·069, 0·954]; $P = 0{\cdot}023$), as illustrated in Fig. 3(a). The standard differences in means ranged from –0·086 to 1·838 (n 5), and there was a high heterogeneity between studies ($I^2 = 87{\cdot}6$ %; $Q = 40{\cdot}42$; df = 5; $P < 0{\cdot}001$). The results of the Egger's test highlighted a publication bias (2·813 [95 % CI: 1·192, 4·434]; $P = 0{\cdot}009$), and the funnel plot is illustrated in Fig. 3(b).

Discussion

While rising rates of overweight and obesity have led to an increasing pre-occupation around weight control and WL in the recent years[43], dieting also became quite common among NW individuals[4–6,44]. Social pressure relative to thinness, particularly among women, can drive them to lose weight, despite the absence of medical necessity contrary to people suffering from overweight or obesity[7–9]. Although frequent, the effects of WL in healthy NW individuals remain poorly investigated. While a sustainable energetic restriction seemed to show some beneficial effects in NW populations[18–20], the high availability of fatty, sweet and ultra-processed food in the occidental diet leads to overconsumption and it became difficult to maintain low-energy intake on the long term[21–23]. Consequently, the probability of weight regain after a WL induced by diet is high, and few

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studies and reviews suggested negative impacts of dieting, in particular an increased risk factor for later obesity[4,12,27]. In this context, the present work undertook a systematic analysis of the available studies questioning diet-induced WL and subsequent or long-term BW changes in healthy NW individuals. After a detailed and strict selection process, eighteen publications met the inclusion criteria for review. Importantly, our systematic approach clearly highlighted a high level of methodological heterogeneity between studies, which was certainly the first and maybe main result of the present work. Indeed, while eight of the included papers were interventional, ten were observational; six of the eight interventional studies being conducted among males only (or near, 94·4 % of men in one study[40]) [13–15,34,37,40], and four of the ten observational studies involved females only[24,26,36,41]. As detailed in Table 1, the duration, the nature of diets (*e.g.* proportion of macronutrients and magnitude of energy deficit), and even the methods used to assess WL itself were highly heterogeneous, leading to a complex comparison of study results.

Observational studies

In observational studies, the dieting status of the participants was assessed using different self-reported methods, which considerably limits the reliability of results. In addition, information about the practice of dieting or food intake was available at only one time point for 88 % of the longitudinal studies, and thus there was no indication of the persistence or interruption of the diet during the follow-up (Table 2). Importantly, the term 'diet' was poorly defined in all the observational studies, and the characteristics to determine the dieting status of subjects were various. While we defined diet as restricting energy intake or certain kinds of food in order to lose weight, dieting can be also described as maintaining, gaining or losing weight by the regulation of food intake[4,44]. Therefore, if 'dieting' includes the control of food intake to maintain a stable weight, it appears crucial to 'diet' in an obesogenic environment to prevent WG, without any WL induced by restricted food. Regarding the lack of consensus on the definition of dieting and the subjectivity and heterogeneity of methods to assess this status, the results have to be interpreted with caution. Finally, concerning the BW assessment, data were self-reported in 60 % of the included observational studies, with only two measuring body composition[36,41]. While there was a large heterogeneity in these observational studies, all of them seem to show a negative effect of dieting on BW, body composition and/or long-term BW changes. Despite the absence of statistical analysis, Colditz and collaborators observed that individuals who previously lost more than 5 kg gained about fourtimes as much weight over an 8-year period than those with lower weight variations ($\leq$ 3 kg)[26]. Moreover, 80 % of these observational studies showed a relationship between dieting and long-term weight change, with a major risk of WG related to diet[24,25,27,32,33,35,36,42]. Finally, three studies found a significantly higher BMI or BW in dieters than in non-dieters[25,36,41], that was confirmed by the results of our meta-analysis (Fig. 3). However, this result is questionable: is higher BW in dieters the cause or the consequence of dieting? Indeed, as suggested by Heberbrand & Hinney[45], initial BW and the tendency to easily gain weight are in part imputable to genetics as a risk factor for obesity. Consequently, people with higher initial BW, while still being within the NW range, are more likely to engage in diet compared with those with a lower BW, all the more if dieting can be defined as control of food intake to maintain weight or limit WG without necessary WL (in link with the definition of diet discussed above). Although our systematic and meta-analytic approaches both suggest that regular NW dieters might be at risk for later extra weight regain compared with non-dieters, it remains to be clearly determined (using a controlled interventional study design) whether WL *per se* is the cause of this subsequent higher BW. Importantly, Pietiläinen and collaborators[27] questioned the role of genetics in long-term WG in twins. They suggested an important effect of genetic predisposition illustrating by a higher BW in dizygotic twins trying to lose weight, but they also highlighted an effect of dieting *per se*, with larger long-term WG in dieters compared with non-dieters in monozygotic twin pairs[27]. However, objective measures of WL and weight regain are lacking in these observational studies, and interventional studies are therefore necessary to better understand the consequences of dieting on later weight changes.

Interventional studies

The classic Minnesota study remains, to date, the only energy restriction study to show a higher BW and FM after recovering from 24 weeks of drastic (semi-starvation) WL relative to baseline in NW healthy subjects[14]. However, weight recovery after 8 weeks of energy deficit (and about 14 % WL) induced by intense physical activity in young men undergoing the US Army Ranger training has also been reported to be accompanied by higher BW and FM values than at baseline; reviewed in reference[14]. In their work on dieting and later WG, Kajioka *et al.*[38] only observed a lower FFM in NW individuals, without any change in BW or FM, after two repeated cycles of WL and weight regain. Importantly, both this study and the Minnesota study observed a lower REE after WL and weight regain compared with pre-WL[13,38], highlighting an adaptive reduction of thermogenesis. This phenomenon is well documented in the literature in response to WL both in NW people[46–49] and people with obesity[46,50–52]. While a decrease in FM and FFM could partly explain the decrease in REE, cellular adaptations are made by the organism to promote energy conservation. In fact, a reduction of REE relative to FFM was observed after WL, both in male[37] and female[49] NW subjects. Although some studies reported that this 'thrifty metabolism' in REE seems to be attenuated or to disappear after a period of weight maintenance in individuals with obesity[47,51], there is evidence that adaptive thermogenesis in the compartments of REE and/or in non-REE may persist for several months to a year during the weight-reduced state[53–57]. Importantly, the suppression of adaptive thermogenesis has been shown to persist in NW individuals, with a reduction of 15 to 20 % in REE over 15 weeks after weight recovery[13,38]. In addition, Kajioka *et al.*[38] observed a decrease of triiodothyronine and thyroxine blood concentrations after WL intervention and recovery, both being implicated in energy

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metabolism and adaptive thermogenesis[58]. Semi-starvation and refeeding experimentations in rats have also highlighted cellular adaptations leading to an energy economy in skeletal muscles that persists during refeeding, including decreased muscle thyroid hormone levels[59].

Beyond compensatory adaptations in energy expenditure, an increase in energy intake above baseline has been observed during weight recovery in NW individuals, illustrating post-WL hyperphagia[14,34,37]. This increase in energy intake was shown in NW people after acute energy deficits induced by diet[60] and in subjects with overweight or obesity after dietary WL intervention[61]. Overall, hyperphagia and adaptive thermogenesis are compensatory adaptations that aim to resist WL during energy deficit and to facilitate weight regain. However, these modifications seem to persist after weight regain in NW individuals[14,34,37] and could explain the increase in BW above baseline values during post-dieting recovery. Indeed, in the Minnesota study, weight recovery was driven by persistent slowed metabolic rate and concomitant hyperphagia, resulting in more rapid restoration of FM than FFM. The hyperphagic state persisted beyond complete recovery of FM, until FFM has returned to baseline values[14,15,62], thereby resulting in an increase in FM beyond pre-WL values (*i.e.* fat overshooting)[14,16], and consequently a higher BW than baseline.

Importance of body weight status

In the Minnesota study, initial FM (%) was negatively associated with individuals' *P*-ratio, an index of body energy partitioning[15], suggesting that leaner subjects are more susceptible to mobilise energy from protein (FFM) during WL and to experience a FM rebound during weight regain[12,14]. The recent narrative review of Magkos (2022) supports this major loss of lean tissue in response to diet in NW people compared with subjects with obesity[17]. In addition, Roberts *et al.* observed a negative correlation between initial FM and hyperphagia during recovery[34], corroborating the hypothesis that leaner subjects were at higher risk of weight regain. Besides the dieting effect, several other studies of the systematic review highlighted the potential influence of weight status on BW evolution, whereby NW dieters are at higher risk for long-term WG than dieters suffering from overweight or obesity[25,27,35,39]. According to the theory of fat overshooting, habitual dieters, and specifically NW individuals, could be subject to catch-up fat resulting in higher BW than pre-WL[12,15]. Consequently, if NW dieters try again to lose weight by repeating WL and weight regains (*i.e.* weight cycling), the cumulative amount of fat overshoot over several weight cycles could ultimately result in obesity, as recently illustrated using a mathematical model of weight cycling that integrates the relationship between post-dieting fat overshooting with initial adiposity[63]. Independently of the gain in BW, weight cycling induced by diet seems to be associated with increased cardiometabolic risk, particularly in NW people[4], and even in athletes despite their high physical activity levels[64,65]. In that context, it appears important to prevent these potential risks associated with diet and weight cycling in initially healthy NW people.

Which role for physical activity?

Although physical activity and/or fitness levels were assessed in 70 % of the included observational studies[25,27,32,33,35,36,41], there was limited information about the relationship between WG, diet, and physical activity level. Nevertheless, physical activity has been suggested to limit the compensatory adaptations in response to energy deficits induced by diet in NW people[60]. Physical activity may therefore serve as a preventive strategy against fat overshooting thanks to the maintenance of FFM (or in minimising its loss) during WL, as demonstrated in people with obesity[66,67] and suggested in athletes[64]. However, this potential protector effect may depend on the degree of energy deficit and nature of physical activity performed. Indeed, while participants of the Minnesota study were asked to walk 35·4 km each week[62], they were also confronted with a semi-starvation diet, which might have surpassed the preventive effect of physical activity on FFM and other compensatory mechanisms.

Limitations

Several limitations must be considered in the present study. First, we use three important and relevant databases (*i.e.* PudMed-Medline, Embase, and Google Scholar) for our literature search strategy, but missing to include other databases, such as Web of Sciences or Scopus, might represent an important limitation of the present work that needs to be considered. Further, the systematic approach revealed an important heterogeneity between studies in terms of methodology, which somewhat restrict our analyses and conclusions. Then, in the observational studies, dieting status was not clearly defined by authors and not ascertained by objective measurements of WL and food intake in individuals reporting to diet, and our results must therefore be considered with precaution. Concerning the interventional studies, sample sizes remain modest and the majority relied on very-low energetic diets (semi-starvation). However, Julia and collaborators reported in the general population a high prevalence of consumption of non-individualised commercial diets (and low prevalence of diets prescribed by professionals), which are generally low- to very-low-energy diets[6]. Finally, the interventional studies were not controlled by a group of NW subjects without WL intervention. These limitations highlight the need to conduct studies to standardise methodological approaches with investigator-verified, objective measurements of BW and body composition throughout WL – WG cycles in NW individuals.

Conclusion

A significant proportion of NW individuals are engaged in dieting to control their weight, notably because of body image distortion and social pressure. The present systematic review seems to indicate that both interventional and observational studies, separately, suggest potential negative effects of dieting on BW and body composition in this population. Despite the observed high methodological heterogeneity and the significant publication bias, the meta-analysis of observational

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studies underlines a higher BW in dieters than in non-dieters. However, the results do not allow to confirm that higher BW and higher WG observed in dieters is the consequence or the origin of the practice of dieting. Similarly, the present systematic analysis of the included interventional studies suggests that negative energy balance induced by dieting generates compensatory adaptations, which may persist during refeeding and even after BW recovery in NW people, but leading to increased FM and BW relative to pre-WL only in one study. Importantly, the present systematic and meta-analytic approaches clearly add to the existing literature that the available evidences suffer from high methodological heterogeneity (both for interventional and observational studies), deeply limiting the formulation of strong conclusions and calling for further well-designed studies. While dieting could be a controversial risk factor for later obesity in NW individuals, there is a clear need to better understand the physiological adaptations occurring during WL and weight regain in this population *via* randomised controlled interventional studies.

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