

Fogelholm, M., Anderssen, S. A., Gunnarsdottir, I., Lahti-Koski, M. (2012).
Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review.
Food & nutrition research, 56, 19103.

This journal is published under the terms of [the Creative Commons Attribution-Noncommercial 3.0 Unported License](https://creativecommons.org/licenses/by-nc/3.0/).

Dietary macronutrients and food consumption as determinants of long-term weight change in adult populations: a systematic literature review

Mikael Fogelholm^{1*}, Sigmund Anderssen²,
Ingibjörg Gunnarsdottir³ and Marjaana Lahti-Koski⁴

¹Department of Food and Environmental Sciences, University of Helsinki, Helsinki, Finland; ²Department of Sports Medicine, Norwegian School of Sport Sciences, Oslo, Norway; ³Faculty of Food Science and Nutrition, University of Iceland, Reykjavik, Iceland; ⁴Finnish Heart Association, Helsinki, Finland

Abstract

This systematic literature review examined the role of dietary macronutrient composition, food consumption and dietary patterns in predicting weight or waist circumference (WC) change, with and without prior weight reduction. The literature search covered year 2000 and onwards. Prospective cohort studies, case-control studies and interventions were included. The studies had adult (18–70 y), mostly Caucasian participants. Out of a total of 1,517 abstracts, 119 full papers were identified as potentially relevant. After a careful scrutiny, 50 papers were quality graded as A (highest), B or C. Forty-three papers with grading A or B were included in evidence grading, which was done separately for all exposure-outcome combinations. The grade of evidence was classified as convincing, probable, suggestive or no conclusion. We found probable evidence for high intake of dietary fibre and nuts predicting less weight gain, and for high intake of meat in predicting more weight gain. Suggestive evidence was found for a protective role against increasing weight from whole grains, cereal fibre, high-fat dairy products and high scores in an index describing a prudent dietary pattern. Likewise, there was suggestive evidence for both fibre and fruit intake in protection against larger increases in WC. Also suggestive evidence was found for high intake of refined grains, and sweets and desserts in predicting more weight gain, and for refined (white) bread and high energy density in predicting larger increases in WC. The results suggested that the proportion of macronutrients in the diet was not important in predicting changes in weight or WC. In contrast, plenty of fibre-rich foods and dairy products, and less refined grains, meat and sugar-rich foods and drinks were associated with less weight gain in prospective cohort studies. The results on the role of dietary macronutrient composition in prevention of weight regain (after prior weight loss) were inconclusive.

Keywords: *obesity; weight gain; weight maintenance; diet; fat; carbohydrates; protein; nutrition*

Received: 13 March 2012; Revised: 2 June 2012; Accepted: 29 June 2012; Published: 13 August 2012

The prevalence of obesity has increased globally during the past 30 y (1). According to the WHO statistics, 35% of adults aged 20 y and older were overweight (BMI ≥ 25 kg/m²) in 2008 (2). The worldwide prevalence of obesity has nearly doubled between 1980 and 2008. Moreover, WHO has estimated that worldwide 2.8 million people die each year as a result of being overweight or obese, and an estimated 35.8 million (2.3%)

of global disability-adjusted life-years are caused by overweight or obesity. A recent European study concluded that in a worst-case scenario almost every third European adult might be obese by year 2015 (3).

The total food supply has increased during the last decades (4). When compared against the secular trends in obesity, an increase in food supply and a concomitant increase in total energy intake are likely to be one of the

major drivers in the obesity epidemic (1). However, the role of dietary macronutrient composition, intake of specific food items or dietary patterns in development of obesity is not clear.

During the last decade, a few narrative reviews have addressed the role of diet in prevention of weight gain (5–7). Systematic reviews and meta-analyses have focused on specific issues, like the role of sugar-sweetened beverages (8–10). The results have been inconclusive. Moreover, we are not aware of any recent (last 5 y) and broad systematic reviews examining the associations of dietary macronutrients, food intake and dietary patterns vs. change in weight or waist circumference (WC) in adult populations. These data are needed to, e.g. give supporting evidence in formulating new nutrition recommendations. The present work was done in connection to the 2012 Nordic Nutrition Recommendations. The purpose of this systematic literature review was to examine the associations of dietary macronutrient composition, food consumption and dietary patterns in prevention of weight or WC gain, with and without prior weight reduction.

Methods

Research questions and definitions

The research questions were formulated separately for studies on primary prevention of weight gain and for studies addressing weight regain after prior weight reduction.

- (1) Primary prevention of obesity (maintenance of body weight and/or WC):

What is the effect of different *dietary macronutrient composition* on long-term (≥ 1 y) change in weight/WC/body fat in an adult population?

- (2) Prevention of weight regain after weight loss (or maintenance of reduced body weight):

What is the effect of different *dietary macronutrient composition* on long-term (≥ 1 y) change in weight/WC/body fat in individuals who have deliberately reduced their weight by at least 5%?

In the search, dietary macronutrient composition was defined as containing:

- (1) carbohydrates, fat and protein as % in energy intake
- (2) fat quality in diet: variation in saturated (SFA), monounsaturated (MUFA) or polyunsaturated (PUFA) fatty acids, as % in energy intake or g/day
- (3) sugar intake as g/day or % in energy intake
- (4) fibre (fiber) intake as g/day

Several of the papers selected for the review contained data on food consumption or dietary patterns. Consequently, the review was expanded to include different food items and food groups, such as cereal products, whole-grain cereals, fruit, vegetables, milk and milk products, meat, etc. Moreover, we also included studies using a whole-diet approach, such as the Mediterranean diet or an index for healthy eating (according to existing dietary recommendations).

The search terms are shown in Appendix 1. The databases used were PubMed and SweMed/SweMed+ (the latter was used to identify Nordic articles not published in PubMed).

Inclusion criteria

The *a priori* defined inclusion criteria were as follows:

Publication year

- year 2000 and later

Study type

- Cross-sectional: *excluded*
- Follow-up (cohort): *included* but minimum follow-up 1 y
- Case-control: *included*
- Weight-maintenance interventions: *included* with the following criteria: (1) intentional mean weight loss at least 5%; (2) at least 6 months follow-up. The follow-up (after weight reduction) could be non-randomised (observational cohort study) or a randomised intervention. In the latter case, the randomisation was done after weight loss, in the beginning of the weight-maintenance intervention. A further premise was that weight reduction was similar in different weight-maintenance groups. Weight loss interventions were also accepted if the total duration was longer than 3 y.

Age

- Inclusion criteria: adult. Age range 18–70 y.
- Exclusion: studies with >70 y participants only and those in which results were not separately analysed by age (i.e. >70 y participants in their own group)

Race/geographical location

- Studies without Caucasians or with Caucasians as minority group were excluded

Selection and evaluation of papers

The abstracts after the initial search were screened by two of the authors (Sigmund Anderssen and Ingibjörg Gunnarsdóttir). All articles suggested by at least one of the two were ordered as full papers. The two other authors (Mikael Fogelholm and Marjaana Lahti-Koski)

then screened the full papers. Again, papers suggested by at least one of them were at least preliminary included in the quality assessment (most careful scrutiny) and evaluation table. Also reviews were ordered as full papers. However, they were not eventually included in the quality grading, because of too much variation in, for example, inclusion criteria, years covered and age groups included.

The quality assessment of the papers was done according to the principles of the Nordic Nutrition Recommendation 2012 working group (11). In short, all papers were evaluated according to a three-scale grading: A = high quality studies with very low level of potential bias; B = some bias, but not enough to invalidate the results; C = significant bias and weaknesses that may invalidate the results. The preliminary quality assessments and construction of summary tables were done individually (Marjaana Lahti-Koski: macronutrients and weight change; SA and IG: food consumption and weight change, dietary patterns and weight change; MF: weight change after weight reduction), but the final product was cross-checked together by all authors.

After the quality grading, four summary tables (macronutrients, food consumption, dietary patterns and weight change after weight reduction) were formed from all studies quality graded A or B. In these tables, the results were arranged according to exposure and outcome variables. However, we did not separate unadjusted and adjusted (to BMI) WC. We always chose the model with most adjustments as the statistical outcome. Moreover, we used analyses with sexes combined, if possible. Otherwise the results of men and women are presented separately. We did not use any other stratification variables, such as prior weight change or smoking.

The grading of evidence was based on the summary tables and a four-class grading: convincing (high), probable (moderate), suggestive (low) and no conclusion (insufficient). The minimum requirement for 'suggestive' was two studies showing an association, and no conflicting results. If some studies showed ns (neither positive nor negative association), it was decided that for 'suggestive evidence', the number of results showing an association was required to be at least two higher than those showing no association.

Results

A total of 1,517 abstracts were initially screened for eligibility (Fig. 1). Out of these, 119 were selected and ordered as full papers. A total of 50 papers were quality graded (12–61). These include 41 papers identified through the original literature search and nine additional papers (17, 30, 31, 32, 36, 45, 47, 51, 55) found from the reference lists of the other publications or 'related citations' in PubMed. The reasons for excluding 78 full papers (5, 8–10, 62–135) are shown in Appendix 2. The number of studies with data on body composition was

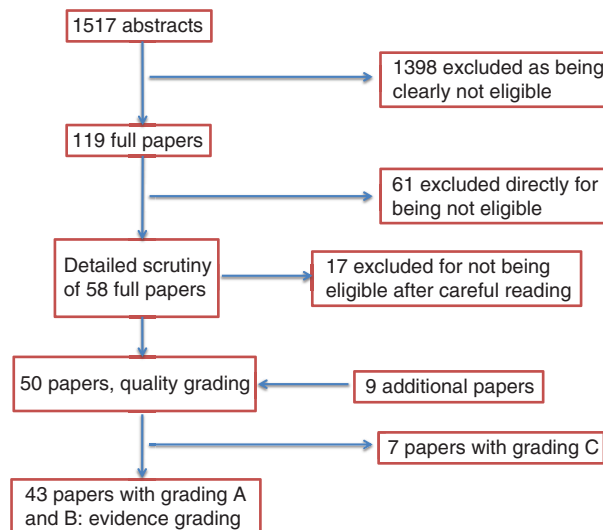


Fig. 1. Flow-chart of the systematic literature review process.

low and therefore our analyses are based only on weight (BMI) and WC.

The evidence tables (Appendix 3–6) present all studies with quality assessment. Studies on the association between macronutrients and weight change are presented in Appendix 3. Studies using energy density as an exposure were also included here. Studies on food consumption and weight change are presented in Appendix 4. Studies using glycaemic index (GI) or glycaemic load (GL) as the main exposure variable are also shown here. Appendix 5 presents the studies on dietary patterns and weight change, and Appendix 6 shows studies on weight change after prior weight reduction (studies on weight regain). The results are summarised for the grading of evidence in Tables 1–4 (in the text).

Macronutrients and change in weight or WC

Most of the studies used for the grading of evidence for the association between macronutrient intake and weight change were prospective cohort studies (Table 1 and Appendix 3). The spread of exposures against the two optional outcomes (change in weight or WC) was large, and most exposure-outcome combinations were assessed by only one or two studies. This leads inevitably to difficulties in finding any evidence for associations between macronutrient intakes and weight change.

The evidence linking high fibre intake to prevention of weight gain was considered probable. In addition, three suggestive associations were found, for cereal fibre against weight change, and for fibre and energy density against change in WC. Five studies assessed weight gain in relation to fibre intake. The association was negative (high fibre intake indicated smaller weight gain) in three studies (14, 18, 21, 26), while one (19) did not find an association. A similar, albeit slightly weaker conclusion was obtained

Table 1. Summary of studies on the association between dietary macronutrients and weight change (see Appendix 1).

Exposure	Outcome variable	No. of participants	Reported associations			Number of studies rated as A or B ¹	Strength of evidence	References
			+	ns	-			
Carbohydrates	Weight	39,275		2		A: 1, B: 1	No conclusion	17, 19
CHO from foods with simple sugars	WC	44,817	IW	IM	B: 1	B: 1	No conclusion	17
CHO from fruit and vegetables	WC	44,817		IM	IW	B: 1	No conclusion	17
CHO from potatoes	WC	44,817	IW	IM		B: 1	No conclusion	17
CHO from refined grains	WC	44,817	IW	IM		B: 1	No conclusion	17
Fibre	Weight	270,307		1	4	A: 3, B: 2	Probable	14, 18, 19, 21, 26
Fibre	WC	106,019		IM	1	B: 3	Suggestive	14, 20, 23
					IW			
					IM			
Fruit fibre	Weight	27,082			IM	B: 1	No conclusion	35
Cereal fibre	Weight	116,514			1	B: 2	Suggestive	14, 35
					IM			
Protein	Weight	49,277		1		A: 1	No conclusion	19
Protein	WC	44,817			1	B: 1	No conclusion	17
Fat	Weight	257,991	1	3		A: 2, B: 4	No conclusion	16–19, 25, 42
			2W	IM				
Fat	WC	44,817	1			B: 1	No conclusion	17
SFA	Weight	130,950	IW	1		B: 2	No conclusion	15, 16
SFA	WC	89,432		1		B: 1	No conclusion	16
MUFA	Weight	130,950		1	IW	B: 2	No conclusion	15, 16
MUFA	WC	89,432		1		B: 1	No conclusion	16
PUFA	Weight	130,950	IW	1		B: 2	No conclusion	15, 16
PUFA	WC	89,432		1		B: 1	No conclusion	16
TFA	Weight	41,518	IW			B: 1	No conclusion	15
TFA substituted for CHO	WC	16,587	IM			B: 1	No conclusion	20
TFA substituted for PUFA	WC	16,587	IM			B: 1	No conclusion	20
Vegetable fat	WC	44,817	IW			B: 1	No conclusion	17
Energy density	Weight	141,220	IW	2		A: 1, B: 2	No conclusion	12, 13, 19
Energy density	WC	138,063	2			B: 2	Suggestive	13, 23

CHO, carbohydrates; SFA, saturated fatty acids; PUFA, polyunsaturated fatty-acids; TFA, trans fatty acids; W, waist circumference; M, men; W, women; +, associated with increased weight gain; ns, no association with weight change; -, associated with decreased weight gain (prevention of weight gain).

¹Some studies included several analyses (e.g. separately for men and women). Therefore, the number of results may be greater than the number of studies.

for cereal fibre (14, 35). Also studies analysing the association between fruit fibre against weight change (35), or the association between total fibre and change in WC (14, 23), tended to favour a protective role of fibre intake.

The other suggestive evidence on the role of dietary macronutrients in development of obesity was observed for energy density (total energy intake divided by the weight of food consumed) against change in WC: both identified studies (13, 23) reported that higher energy density was associated with larger increase in WC. The results on energy density against weight change were less consistent. Bes-Rastrollo et al. (12) reported that an increase in energy density was associated with a simultaneous increase in weight, while two other studies (13, 19) did not find an association.

The intake of total carbohydrates, fats and proteins did not show consistent associations with weight gain. Especially in the case of fat intake vs. weight change, the number of studies (four) was in fact relatively high, but the results were quite evenly dispersed between a positive association (higher fat intake would increase weight gain) (25, 42) and no significant association (16, 17). Similarly, the results on intake of SFA or PUFA against development of obesity indicated either a positive (15) or no significant association (16). Field et al. (15) linked MUFA with protection of weight gain, but this finding was not confirmed in the study of Forouhi et al. (16). Koh-Banerjee et al. (20) investigated the role of trans-fatty acids (TFA): their results suggested that TFA, when substituted for carbohydrates or PUFA, are associated with increased WC. Also Field et al. (15) found a positive association between TFA intake and weight gain. Hence, all three analyses showed that high intake of TFA predicts weight gain. The lack of multiple data on specific combinations prevents us from making a stronger conclusion.

Howard et al. reported that higher intake of total carbohydrates protected against weight gain in women (18), but Halkjaer et al. (17) did not find an association between carbohydrate intake and change in weight or WC. The source of carbohydrates may be relevant, however, since Halkjaer et al. (17) reported a positive association between carbohydrates from foods with simple sugars, from potatoes and from refined grains, against change in WC in women. In contrast, they also found that high carbohydrates intake from vegetables (women only) and fruit protected against an increase in WC.

The role of protein in prevention of an increase in weight or WC was inconsistent: the two identified studies reported a neutral (19) or negative (17) association.

Foods and change in weight or WC

Compared with the association between macronutrients and weight change, a few more 'suggestive' associations were found (Table 2 and Appendix 4). According to the

data, high intake of whole grains, fruit, nuts and high-fat dairy protect against increasing obesity, whereas refined grains, white bread, meat and sweets and desserts seem to promote gains in weight or WC. Unfortunately, even here the main challenge in making broader conclusions was that the number of studies for a specific combination of exposure and outcome was limited (rarely more than two data points).

The suggestive association linking high intake of whole grains to lower weight gain was based on two cohort studies (35, 36). No other studies in this combination of exposure and outcome were found. However, Halkjaer et al. (32) did not find an association between the intake of wholegrain bread and change in WC. Two studies (33, 39) reported that a high intake of fruit predicted smaller increase in WC, with no conflicting results. On the other hand, studies linking fruit to changes in weight were not equally consistent (36, 45).

Three studies reported a negative association between intake of nuts and change in weight (30, 36, 60), and no conflicting data were found. The evidence was regarded as probable. Unfortunately, these studies are not fully independent, since two of them are partly or totally based on data from the Nurses' Health Study (30, 36).

Several studies have investigated the role of dairy products in prevention of weight gain. Again, the definition of exposure variable was inconsistent (dairy in general, high-fat dairy, low-fat dairy, etc.) and this left only a few relevant combinations for assessment in this review. Both studies examining the relationship between high-fat dairy and weight gain reported a negative association, that is, higher intake of these dairy products was associated with smaller gains in weight (38, 50). Also some other studies found a protective role for dairy products (33, 36, 39, 41), while others did not report any significant associations between dairy intake and change in weight or WC (32, 38). There were no studies with a positive association between any kind of dairy products and change in weight or WC.

The intake of refined bread was associated with an increase in WC in both studies identified for this review (32, 39). A similar supporting evidence was observed for the positive association between refined grain and weight change (21, 36).

Three studies reported a positive association between meat intake and weight change (40, 44, 50) and this evidence was regarded as probable. The studies of Rosell et al. (40) and Vergnaud et al. (44) are not, however, totally independent: the former was based on a subpopulation of the EPIC-cohort, while the latter used the entire cohort for analyses. Some other studies also linked higher intake of meat, poultry or processed meat with an increase in weight or WC (33, 36, 39). No association were reported by a few (28, 32, 33), whereas Halkjaer et al. (33)

Table 2. Summary of studies on the association between food consumption and weight change (see Appendix 2).

Exposure	Outcome variable	No of participants	Reported associations			Number of studies rated as A or B ¹	Strength of evidence	References
			+	ns	-			
Breakfast cereals	Risk of obesity	17,881			IM	B: 1	No conclusion	27
Whole grains	Weight	147,959			I IM	B: 2	Suggestive	35, 36
Wholegrain bread	WC	2,436		I		B: 1	No conclusion	32
Refined grains	Weight	194,968	2			B: 2	Suggestive	21, 36
Refined (white) bread	WC	51,067	2			B: 2	Suggestive	32, 39
Fruit	Weight	494,680		I	I	B: 2	No conclusion	36, 45
Fruit	WC	91,327			2	B: 1	Suggestive	33, 39
Fruit and vegetables	WC	2,436		I		B: 1	No conclusion	32
Vegetables	Weight	494,680		I	I	B: 2	No conclusion	36, 45
Vegetables	WC	91,327		IM	I	B: 2	No conclusion	33, 39
					IW			
Potato chips	Weight	120,877	I			B: 1	No conclusion	36
Potatoes	Weight	120,877	I			B: 1	No conclusion	36
Potatoes	WC	93,763	I	I		B: 2	No conclusion	32, 33, 39
			IW	IM				
Nut consumption	Weight	180,930			2	B: 3	Probable	30, 36, 60
					IW			
Olive oil	Weight	7,368		I		B: 1	No conclusion	29
Butter	Weight	120,877	I			B: 1	No conclusion	36
Butter and/or margarine	WC	93,763	I	I	IW	B: 3	No conclusion	32, 33, 39
				IM				
Dairy, general	Weight	42,856		IM	IW	B: 2	No conclusion	38, 41
Dairy, general	WC	48,631			I	B: 1	No conclusion	39
Dairy, high-fat	WC	42,696		IM	IW	B: 1	No conclusion	33
Dairy, high-fat/whole-fat	Weight	29,823			I	B: 2	Suggestive	38, 50
					IM			
Dairy, low-fat dairy	Weight	23,504		I		B: 1	No conclusion	38
Dairy, milk and cheese	WC	2,436		I		B: 1	No conclusion	32
Dairy, yoghurt	Weight	120,877			I	B: 1	No conclusion	36
Meat, general	Weight	380,122	3			B: 3	Probable	40, 44, 50
Meat, poultry	WC	42,696	IW	IM		B: 1	No conclusion	33
Meat, processed meat	Weight	120,877	I			B: 1	No conclusion	36
Meat, processed meat	WC	91,327	I	IM		B: 2	No conclusion	33, 39
			IW					
Meat, red (unprocessed) meat	Weight	128,071	I	I		B: 2	No conclusion	28, 36
Meat, red meat	WC	45,132		I	I	B: 2	No conclusion	32, 33
Hamburgers, pizza and sausages	Weight	7,194	I				No conclusion	28
Fish	WC	2,436		I		B: 1	No conclusion	32
SSSD	Weight	58,797	IW	I		B: 2	No conclusion	28, 43
SSSD	WC	48,631	I			B: 1	No conclusion	39
Sweetened fruit juice	Weight	7,194		I		B: 1	No conclusion	28
Sweets and desserts	Weight	138,246	2			B: 2	Suggestive	36, 42
Sugar and confectionary	WC	48,632	I			B: 1	No conclusion	39
Cakes and chocolate	WC	2,436		I		B: 1	No conclusion	32
Sauce	Weight	17,369	IW	IM		B: 1	No conclusion	42
Snack foods	WC	42,696	I			B: 1	No conclusion	33
GI	Weight	89,808	IW	I		B: 2	No conclusion	31, 34

Table 2 (Continued)

Exposure	Outcome variable	No of participants	Reported associations			Number of studies rated as A or B ¹	Strength of evidence	References
			+	ns	-			
				IM				
GI	WC	49,007	I, IW	IM	B: 2	No conclusion	23, 34	
GL	Weight	89,808	I	I	B: 2	No conclusion	31, 34	
GL	WC	49,383	IW	I	B: 2	No conclusion	23, 34	
				IM				

WC, waist circumference; M, men; W, women; GI, glycaemic index; GL, glycaemic load; SSSD, sugar-sweetened soft drink; +, associated with increased weight gain; ns, no association with weight change; -, associated with decreased weight gain (prevention of weight gain).

¹Some studies included several analyses (e.g. separately for men and women). Therefore, the number of results may be greater than the number of studies.

found that higher intake of red meat protected against an increase in WC, adjusted for BMI.

Two studies reported that a high intake of sweets and desserts, was associated with larger weight increases (36, 42). This association could be classified as suggestive. Two studies found a positive association between intake of sugar-sweetened soft drinks (SSSD) and weight or WC gain (39, 43), while such an association was not confirmed in a third study (28). However, there were no studies suggesting an inverse association of sugar-rich foods and change in weight or WC.

The few results linking GI or GL to changes in weight or WC were dispersed between a positive (23, 31, 34) and no association (23, 34). It may be worth noting that a positive association between GI/GL vs. change in weight or WC was more often observed in women than in men (23, 34).

Dietary patterns and weight change

We identified five studies with results on the relationship between dietary patterns and weight change (Table 3 and Appendix 5). Three of these used an index of the Mediterranean diet (47, 49, 50) and two others the American Diet Quality Index (48, 51). The index for Mediterranean diet is based on the consumption of 'positive' (e.g. fruit, vegetables, legumes, whole grains, fish, olive oil) and 'negative' (e.g. meat and dairy) food items. The Diet Quality Index is based on US dietary recommendations: it is a measure of how well an

individual meets the recommendations for SFA, cholesterol, sodium, total fat and total carbohydrate.

Both studies using the Diet Quality Index reported that meeting the recommendations was associated with less weight gain during the follow-up (48, 51). The evidence is suggestive. Two studies with the Mediterranean index supported this conclusion (47, 49), while the third study did not find an association between Mediterranean dietary patterns and weight change after all statistical adjustments (50).

Macronutrients and prevention of weight regain after weight loss

Only nine studies were identified with data on the association between dietary macronutrient composition and weight gain after prior weight reduction (Table 4 and Appendix 6). All six studies classified as A or B were randomised weight-maintenance interventions. Delbridge et al. (59) prescribed a weight-maintenance diet with energy intake corresponding to 1.3 × estimated resting energy expenditure, but all other studies used *ad lib* energy intake throughout the weight-maintenance phase. Overall, the results were inconclusive and it was not possible to make any conclusions.

A high-protein, low-carbohydrate diet protected against weight regain in one study (55), but no effects were observed in three other studies (52, 53, 59). Due et al. (54) found that both a high-fat, low-carbohydrate, and a low-fat, high-carbohydrate diet reduced weight regain,

Table 3. Summary of studies on the association between dietary patterns and weight change (see Appendix 3).

Exposure	Outcome variable	No of participants	Reported associations			Number of studies rated as A or B	Strength of evidence	References
			+	ns	-			
Mediterranean diet index	Weight	390,498		I	2	B: 3	No conclusion	47, 49, 50
Healthy/prudent diet index	Weight	7,158			2	A: I, B: I	Suggestive	48, 51

+, Associated with increased weight gain; ns, no association with weight change; -, associated with decreased weight gain (prevention of weight gain).

Table 4. Summary of studies on the association between weight-maintenance interventions (prevention of weight regain) and weight change (see Appendix 4).

Exposure	Outcome variable	No of participants	Reported associations			Number of studies rated as A or B	Strength of evidence	References
			+	ns	–			
HP/LC (vs. LP/HC)	Weight	120		2		B: 2	No conclusion	52, 59
HP/LC (vs. CON)	Weight	973		1W	1	A: 1, B: 1	No conclusion	53, 55
HF/LC (vs. CON)	Weight	77			1	A: 1	No conclusion	54
HF/LC (vs. LF/HC)	Weight	99			1	A: 1	No conclusion	54
LF/HC (vs. CON)	Weight	175	1	1		A: 1, B: 1	No conclusion	54, 57
Low GI vs. high GI	Weight	773				A: 1	No conclusion	55

H, high; L, low; P, protein; F, fat; C, carbohydrate; CON, control – according to nutrition recommendations; GI, glycaemic index; M, men; W, women; +, associated with increased weight gain; ns, no association with weight change; –, associated with decreased weight gain (prevention of weight gain).

when compared against a control diet with ‘normal’ macronutrient composition. Also in the study of Swinburn et al. (57), a low-fat, high-carbohydrate protected against weight regain at 2-y follow-up, but this effect was lost 2 y later.

Finally, Larsen et al. (55) found that a diet with low GI prevented weight regain, when compared against a high GI diet. This effect was observed regardless of the macronutrient composition. However, the most effective combination in terms of prevention of weight regain after weight reduction was high-protein, low-carbohydrate diet with low GI.

Discussion

Interpretation of results

The main findings of this systematic review on nutrients and foods in relation to weight change were the following: we found probable evidence for high intake of dietary fibre and nuts predicting less weight gain, and for high intake of meat in predicting more weight gain. Suggestive evidence was found for a protective role against increasing weight from whole grains, cereal fibre, high-fat dairy products and high scores in an index describing a prudent dietary pattern. Likewise, there was suggestive evidence for both fibre and fruit intake in protection against larger increases in WC. Also suggestive evidence was found for high intake of refined grains, and sweets and desserts in predicting more weight gain, and for refined (white) bread and high energy density in predicting larger increases in WC.

A major problem in assessing the grade of evidence was that similar combinations of exposure and outcome variables were eventually quite rare. Therefore, we decided to do a *post hoc* evidence analysis by first combining the outcome variables. Although WC, compared with BMI, may be a slightly stronger risk factor for cardiovascular diseases, Type 2 diabetes and breast and colorectal cancers, they both can be used as a measure of

obesity in population studies almost interchangeably (136, 137). Moreover, to get more studies into one evidence grading, we grouped foods by their closeness of nutrient composition. The results of these *post hoc* analyses are shown in Table 5. Since we may violate the strict rules of evidence grading by subjectively combining different exposure variables, this analysis is ‘unofficial’ and the grading of evidence is not shown in the table.

We combined studies with fibre, vegetables, fruit, fruit fibre, carbohydrates from fruit & vegetables, whole grains, whole grain bread or nuts as an exposure variable into one group called ‘fibre-rich foods’. Some studies included several analyses, either separately for men and women, or for different exposure and/or outcome variables. Hence, the identified 14 studies included a total of 28 analyses. Out of these, 21 results (13 with both sexes, 4 with only women and 4 with only men) indicated that a higher intake of at least one of these ‘fibre-rich foods’ is associated with prevention of obesity. Eight analyses did not find a significant association. In this light, the evidence for a protective role of fibre-rich foods in general might be considered moderately strong.

The use of fibre-rich products reduce dietary energy density by increasing the volume of food without bringing additional absorbable energy (12). Fruit and vegetables have a low GI, whereas fibre-rich bread may induce a lowered insulin response and delayed glucose decline (138). Both properties could increase satiety and reduce energy consumption (139). In addition, other biologically active compounds in fruit, vegetables and whole grain (e.g. phenolic compounds and phytoestrogens) may be related to weight control (35).

Nuts may be regarded as a ‘special case’ among fibre-rich products, not least because of their high fat content. Nevertheless, even earlier epidemiological evidence suggests an inverse association between nut consumption and body weight (140). The proposed mechanisms include increased energy expenditure due to high protein and

Table 5. *Post hoc* analyses: evidence for association between grouped exposure variables (taken from summary Tables 1 and 2) against grouped outcome variables (BMI and waist circumference not separated).

Group name	Exposure variables	Effect			No of studies ¹	References
		+	ns	–		
Fibre-rich foods	Fibre, vegetables, fruit, fruit fibre, carbohydrates from fruit and vegetables, whole grains, whole grain bread, nuts		5 3M	13 4W 4M	14	14, 17–21, 23, 26, 30, 35, 36, 39, 45, 60
Refined grains	Refined grains, carbohydrates from refined grains, refined bread	5			4	17, 21, 36, 39
Potatoes	Potatoes, carbohydrates from potatoes	1 1W	1 1M		3	17, 32, 36
Dairy	Dairy general, high-fat dairy, low-fat dairy, milk and cheese, yoghurt		2 2M	3 2W 1M	5	36, 38, 39, 41, 50
Meat	Meat general, poultry, processed meat unprocessed or red meat	6 2W	2 2M	1	8	28, 32, 33, 36, 39, 40, 44, 50
Healthy diet	Index of Mediterranean diet, index of healthy/prudent diet		1	4	5	47–51

M, men; W, women; +, associated with increased weight gain; ns, no association with weight change; –, associated with decreased weight gain (prevention of weight gain).

¹Some studies included several analyses, either separately for men and women, or for different exposure and/or outcome variables. Therefore, the number of results may be greater than the number of studies.

unsaturated fatty-acid content, enhanced satiety and ineffective absorption of fat (140). Short-term interventions have not shown any effects of nuts on body weight, whereas nut consumption seems to improve blood lipid levels in a dose-related manner (141).

Refined grains, carbohydrates from refined grains and refined bread formed a group called ‘refined grain foods’. Four studies included five analyses, and all of them showed an association between high intake of refined grains and increasing obesity. The level of evidence could be regarded as probable, but slightly weaker than the evidence seen for fibre-rich foods. Refined grain products have often high GI, high insulin response and a fast glucose decline even below baseline in an oral test (138). These properties could increase hunger and enhance lipogenesis, thereby promoting obesity (142). The different effects of whole-grain and refined cereals speak for separating different types of cereals in the food pyramid.

Also potatoes have high GI, and therefore it could be plausible to think that they – like refined grains – could induce obesity. The results of our review were not very convincing: two analyses supported the above hypothesis, while two other did not find an association between potato consumption and weight or WC change. It is possible that the way potatoes are prepared is important: Mozaffarian et al. (36) reported a positive association between potato consumption and weight gain, but in this study a majority of the potatoes was French fries.

All dairy products were combined to form a new group called ‘dairy foods’. In our ‘official’ analyses, we found suggestive evidence for a protecting role of high-fat dairy foods. The combined data did not strengthen this result. A total of four analyses showed a positive association between dairy food consumption and increasing obesity, whereas five analyses did not report any associations. If there indeed is an association between dairy products and prevention of weight gain, the proposed mechanisms might be related to calcium, protein or biopeptides (143). More research is needed to find out whether the mechanism could be related to milk fat. Earlier studies have, in contrast, indicated that unsaturated, rather than saturated, fatty acids may promote postprandial fat oxidation and stimulate diet-induced thermogenesis (144). The two studies showing an association between high-fat dairy and less weight gain (38, 50) did not very clearly specify their definition of dairy products, e.g. if only milk products were included. However, butter was apparently not included in either study.

A majority of the studies support the hypothesis that a high consumption of meat and meat products predict more weight gain. This finding might be considered confusing, because of the proposed satiating effects of protein (145). However, meat is energy dense and might thereby increase energy intake (44). It is also possible that meat intake only reflects some undetected dietary or lifestyle patterns that contribute to weight gain (44).

Yet another possibility is that meat increases fat-free mass and that BMI in this case would be misleading. Interestingly, the two studies showing a preventive role for protein or meat used WC as the outcome (17, 33). On the other hand, two studies identified poultry or processed meat as a predictor of larger gains in WC (33, 39).

We found suggestive evidence for an obesity-promoting role of sweets and desserts. Since the contribution of sweets to total energy intake is small (146), a likely explanation for this finding is residual confounding, that is, consumption of sweets probably mirror some other unhealthy dietary and/or physical activity patterns that lead to positive energy balance. In fact, we were rather expecting to find an association between the use of SSSD and weight gain. Out of the identified three studies, two suggested that SSSD predict weight or WC gain (39, 43), but the third (28) found an association only in a subgroup with prior weight gain. Hence, according to our strict rules we had to classify these data as inconclusive. Recent systematic reviews have also produced conflicting results on the association between SSSD and weight gain (8–10). A majority of the results suggesting a positive association between SSSD and weight gain have studied children and adolescents (8, 9). The compilation of different sugar-containing foods into one analysis did not bring any additional insights.

It is perhaps not a surprise that adherence to a presumed healthy diet predicts less weight gain. It is interesting that the Healthy Diet Index is in fact composed of items without any clear association with weight (total fat, saturated fat, dietary cholesterol, salt, carbohydrates) – and yet a diet fulfilling these requirements is at the same time suitable for weight control. The Mediterranean Diet Index is built from foods and many of the ‘positive’ foods are high in dietary fibre and these foods have in this review been identified as predictors of better weight control. Moreover, meat is considered a ‘negative’ item in the Mediterranean Diet Index and we found suggestive evidence for meat as a predictor for weight gain. The only discrepancy is related to dairy products which are ‘negative’ in the Mediterranean Diet Index, but, if anything, protective against weight gain in our review.

Methodological considerations

The criteria for A-grading were very strict. Because of the understandable crudeness of epidemiological methods, all really large studies (e.g. EPIC, Nurses’ Health Study, etc.) were classified as B, while some clearly smaller studies sometimes received an A-rating. In the end, this did not have an impact on the analyses, since all studies classified as A or B were included in the summary tables.

Most of the studies identified for this review were prospective cohort designs. Although interventions would be much stronger in identifying causal effects, the

possibility to study long-term (5–20 y) weight changes by using an intervention design would be extremely challenging and expensive. All prospective cohort studies need careful control for potential confounders. Although practically all A- and B-graded cohorts in our review were able to control for a multiple of potential confounding variables, residual confounding cannot be ruled out (147). Therefore, it is unclear whether the identified positive or negative associations really are effects of nutrients or foods vs. weight or WC.

One interesting point is whether energy intake should be included in the model. While adjusting for total energy intake may control for over- and under-reporting, energy intake is also a potential mechanism explaining the association between a nutrient/food and weight gain. Therefore, adjusting for energy intake might be regarded as overadjustment, which may dilute the real association between food/nutrient and weight change. For future studies, it would be recommendable to present models with energy intake as the only differing variable (to see if the inclusion of energy intake in the model has an effect on the results). We did not look for a potential association between total energy intake and weight change, since a positive energy balance is too much dependent on the level of total physical activity and energy expenditure. A scrutiny on the interaction between physical activity and diet, against weight change, was also outside the focus of this review.

Measurements of dietary intake and food consumption at baseline are usually inaccurate. Most of the population studies covered in this review used a food frequency questionnaire (FFQ). Although many of the FFQ’s have been validated (see Appendix 3–5), the validation was often restricted to certain nutrients. For instance, we are not aware of a FFQ planned to assess GI or dietary density. In addition to inaccurate baseline estimation, an individual’s dietary pattern may change during the follow-up. These lead to misclassifications of exposure and to at least some attenuation of association towards unity (type II error). In this light it is interesting to note that there were very few totally conflicting findings (same exposure showing both negative and positive association with the outcome). If some of the non-significant findings were indeed type II errors, there may be in reality more associations between diet and weight change than found in the present review.

Another point – which is in a way opposite to the previous – is that the large number of participants in several studies allows identification of even very small differences between groups (e.g. lowest vs. highest 25%). The practical significance of these differences is uncertain. Most studies have assessed the association between single nutrients and food items against weight change, but aggregating single foods into composite scores yields more robust estimations (36, 39). By combining exposure

variables (foods) into larger groups, as shown in Table 5, we wanted to improve the robustness of our analysis. To be meaningful, however, even these results should probably be translated into diet-level recommendations.

Many cohorts were initiated more than 10 y ago. This is perhaps not very meaningful for analyses using foods, food groups or dietary patterns. However, since a certain macronutrient composition can be achieved by different food choices, the interpretation of the oldest studies should be done with care: for instance, a certain proportion of carbohydrates and fat in a diet in 1980s might be related to different food choices than a similar macronutrient distribution in 2012. This may also have a relevance to the association between macronutrients and weight gain. Finally, it may be relevant to repeat that the review covered publication years 2000–2012, and this may have excluded important older studies. Moreover, although PubMed is a very comprehensive database and it covers all major international medical journals, it is possible that some additional studies could have been identified by using, e.g. EMBASE or Scopus. The potential bias caused by using only PubMed and SweMed+ is, however, considered negligible.

Conclusion

In this systematic review covering publications from year 2000 onwards, we found probable evidence for high intake of dietary fibre and nuts predicting less weight gain, and for high intake of meat in predicting more weight gain. Suggestive evidence was found for a protective role against increasing weight from whole grains, cereal fibre, high-fat dairy products and high scores in an index describing a prudent dietary pattern. Likewise, there was suggestive evidence for both fibre and fruit intake in protection against larger increases in WC. Also suggestive evidence was found for high intake of refined grains, and sweets and desserts in predicting more weight gain, and for refined (white) bread and high energy density in predicting larger increases in WC. When foods with similar nutrient composition were combined for an unofficial analysis, fibre-rich foods in general predicted less weight gain and this association could be regarded as moderately strong (probably). The associations between foods and dietary patterns vs. weight gain were stronger compared to those between macronutrients vs. weight gain. In general, the results suggest that the proportion of macronutrients in the diet is not important in prevention of obesity. In contrast, plenty of fibre-rich foods and dairy products, and less refined grains, meat and sugar-rich foods and drinks were associated with less weight gain in prospective cohort studies.

Conflict of interest and funding

The review is part of the NNR 2012 project, with financial support from the Nordic Council of Ministers.

References

1. Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, et al. The global obesity pandemic: shaped by global drivers and local environments. *Lancet* 2011; 378: 804–14.
2. World Health Organization. WHO Global Status Report 2010. Geneva: World Health Organization.; 2011.
3. von Ruesten A, Steffen A, Floegel A, van der A DL, Masala G, Tjønneland A, et al. Trend in obesity prevalence in European adult cohort populations during follow-up since 1996 and their predictions to 2015. *PLoS ONE* 2011; 6: e27455.
4. Kearney J. Food consumption trends and drivers. *Philos Trans R Soc Lond B Biol Sci* 2010; 365: 2793–807.
5. Astrup A. The role of dietary fat in the prevention and treatment of obesity. Efficacy and safety of low-fat diets. *Int J Obes Relat Metab Disord* 2001; 25(Suppl 1): S46–50.
6. van Dam RM, Seidell JC. Carbohydrate intake and obesity. *Eur J Clin Nutr* 2007; 61(Suppl 1): S75–99.
7. Du H, Feskens E. Dietary determinants of obesity. *Acta Cardiol* 2010; 65: 377–86.
8. Gibson S. Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions. *Nutr Res Rev* 2008; 21: 134–47.
9. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* 2006; 84: 274–88.
10. Forshee RA, Storey ML, Allison DB, Glinsmann WH, Hein GL, Lineback DR, et al. A critical examination of the evidence relating high fructose corn syrup and weight gain. *Crit Rev Food Sci Nutr* 2007; 47: 561–82.
11. NNR5 working group. A guide for conducting systematic literature reviews for the 5th edition of the Nordic Nutrition Recommendations. <http://www.slv.se/upload/NNR5/A%20guide%20for%20conducting%20SLR%20for%20NNR5%20FINAL.pdf>. [cited 1 Aug 2012].
12. Bes-Rastrollo M, van Dam RM, Martinez-Gonzalez MA, Li TY, Sampson LL, Hu FB. Prospective study of dietary energy density and weight gain in women. *Am J Clin Nutr* 2008; 88: 769–77.
13. Du H, van der A DL, Ginder V, Jebb SA, Forouhi NG, Wareham NJ, et al. Dietary energy density in relation to subsequent changes of weight and waist circumference in European men and women. *PLoS ONE* 2009; 4: e5339.
14. Du H, van der A DL, Boshuizen HC, Forouhi NG, Wareham NJ, Halkjaer J, et al. Dietary fiber and subsequent changes in body weight and waist circumference in European men and women. *Am J Clin Nutr* 2010; 91: 329–36.
15. Field AE, Willett WC, Lissner L, Colditz GA. Dietary fat and weight gain among women in the Nurses' Health Study. *Obesity (Silver Spring)* 2007; 15: 967–76.
16. Forouhi NG, Sharp SJ, Du H, van der A DL, Halkjaer J, Schulze MB, et al. Dietary fat intake and subsequent weight change in adults: results from the European Prospective Investigation into Cancer and Nutrition cohorts. *Am J Clin Nutr* 2009; 90: 1632–41.
17. Halkjaer J, Tjønneland A, Thomsen BL, Overvad K, Sørensen TIA. Intake of macronutrients as predictors of 5-y changes in waist circumference. *Am J Clin Nutr* 2006; 84: 789–97.
18. Howard BV, Manson JE, Stefanick ML, Beresford SA, Frank G, Jones B, et al. Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. *JAMA* 2006; 295: 39–49.
19. Iqbal SI, Helge JW, Heitmann BL. Do energy density and dietary fiber influence subsequent 5-year weight changes

- in adult men and women? *Obesity* (Silver Spring) 2006; 14: 106–14.
20. Koh-Banerjee P, Chu N-F, Spiegelman D, Rosner B, Colditz G, Willett W, et al. Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16 587 US men. *Am J Clin Nutr* 2003; 78: 719–27.
 21. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz G. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. *Am J Clin Nutr* 2003; 78: 920–7.
 22. Mosca CL, Marshall JA, Grunwald GK, Cornier MA, Baxter J. Insulin resistance as a modifier of the relationship between dietary fat intake and weight gain. *Int J Obes Relat Metab Disord* 2004; 28: 803–12.
 23. Romaguera D, Angquist L, Du H, Jakobsen MU, Forouhi NG, Halkjaer J, et al. Dietary determinants of changes in waist circumference adjusted for body mass index – a proxy measure of visceral adiposity. *PLoS ONE* 2010; 5: e11588.
 24. Savage JS, Marini M, Birch LL. Dietary energy density predicts women's weight change over 6 y. *Am J Clin Nutr* 2008; 88: 677–84.
 25. Sherwood NE, Jeffery RW, French SA, Hannan PJ, Murray DM. Predictors of weight gain in the Pound of Prevention study. *Int J Obes Relat Metab Disord* 2000; 24: 395–403.
 26. Tucker LA, Thomas KS. Increasing total fiber intake reduces risk of weight and fat gains in women. *J Nutr* 2009; 139: 576–81.
 27. Bazzano LA, Song Y, Bubes V, Good CK, Manson JE, Liu S. Dietary intake of whole and refined grain breakfast cereals and weight gain in men. *Obes Res* 2005; 13: 1952–60.
 28. Bes-Rastrollo M, Sánchez-Villegas A, Gómez-Gracia E, Martínez JA, Pajares RM, Martínez-González MA. Predictors of weight gain in a Mediterranean cohort: the Seguimiento Universidad de Navarra Study 1. *Am J Clin Nutr* 2006; 83: 362–370; quiz 394–395.
 29. Bes-Rastrollo M, Sánchez-Villegas A, de la Fuente C, de Irala J, Martínez JA, Martínez-González MA. Olive oil consumption and weight change: the SUN prospective cohort study. *Lipids* 2006; 41: 249–56.
 30. Bes-Rastrollo M, Wedick NM, Martínez-González MA, Li TY, Sampson L, Hu FB. Prospective study of nut consumption, long-term weight change, and obesity risk in women. *Am J Clin Nutr* 2009; 89: 1913–9.
 31. Du H, van der A DL, van Bakel MME, Slimani N, Forouhi NG, Wareham NJ, et al. Dietary glycaemic index, glycaemic load and subsequent changes of weight and waist circumference in European men and women. *Int J Obes (Lond)* 2009; 33: 1280–8.
 32. Halkjaer J, Sørensen TIA, Tjønneland A, Togo P, Holst C, Heitmann BL. Food and drinking patterns as predictors of 6-year BMI-adjusted changes in waist circumference. *Br J Nutr* 2004; 92: 735–48.
 33. Halkjaer J, Tjønneland A, Overvad K, Sørensen TIA. Dietary predictors of 5-year changes in waist circumference. *J Am Diet Assoc* 2009; 109: 1356–66.
 34. Hare-Bruun H, Flint A, Heitmann BL. Glycemic index and glycemic load in relation to changes in body weight, body fat distribution, and body composition in adult Danes. *Am J Clin Nutr* 2006; 84: 871–879; quiz 952–953.
 35. Koh-Banerjee P, Franz M, Sampson L, Liu S, Jacobs DR Jr, Spiegelman D, et al. Changes in whole-grain, bran, and cereal fiber consumption in relation to 8-y weight gain among men. *Am J Clin Nutr* 2004; 80: 1237–45.
 36. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med* 2011; 364: 2392–404.
 37. Poddar KH, Hosig KW, Nickols-Richardson SM, Anderson ES, Herbert WG, Duncan SE. Low-fat dairy intake and body weight and composition changes in college students. *J Am Diet Assoc* 2009; 109: 1433–8.
 38. Rajpathak SN, Rimm EB, Rosner B, Willett WC, Hu FB. Calcium and dairy intakes in relation to long-term weight gain in US men. *Am J Clin Nutr* 2006; 83: 559–66.
 39. Romaguera D, Ångquist L, Du H, Jakobsen MU, Forouhi NG, Halkjaer J, et al. Food composition of the diet in relation to changes in waist circumference adjusted for body mass index. *PLoS ONE* 2011; 6: e23384.
 40. Rosell M, Appleby P, Spencer E, Key T. Weight gain over 5 years in 21,966 meat-eating, fish-eating, vegetarian, and vegan men and women in EPIC-Oxford. *Int J Obes (Lond)* 2006; 30: 1389–96.
 41. Rosell M, Håkansson NN, Wolk A. Association between dairy food consumption and weight change over 9 y in 19,352 perimenopausal women. *Am J Clin Nutr* 2006; 84: 1481–8.
 42. Schulz M, Kroke A, Liese AD, Hoffmann K, Bergmann MM, Boeing H. Food groups as predictors for short-term weight changes in men and women of the EPIC-Potsdam cohort. *J Nutr* 2002; 132: 1335–40.
 43. Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, et al. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA* 2004; 292: 927–34.
 44. Vergnaud A-C, Norat T, Romaguera D, Mouw T, May AM, Travier N, et al. Meat consumption and prospective weight change in participants of the EPIC-PANACEA study. *Am J Clin Nutr* 2010; 92: 398–407.
 45. Vergnaud A-C, Norat T, Romaguera D, Mouw T, May AM, Romieu I, et al. Fruit and vegetable consumption and prospective weight change in participants of the European Prospective Investigation into Cancer and Nutrition-Physical Activity, Nutrition, Alcohol, Cessation of Smoking, Eating Out of Home, and Obesity study. *Am J Clin Nutr* 2012; 95: 184–93.
 46. Vioque J, Weinbrenner T, Castelló A, Asensio L, Garcia de la Hera M. Intake of fruits and vegetables in relation to 10-year weight gain among Spanish adults. *Obesity* (Silver Spring) 2008; 16: 664–70.
 47. Beunza J-J, Toledo E, Hu FB, Bes-Rastrollo M, Serrano-Martínez M, Sánchez-Villegas A, et al. Adherence to the Mediterranean diet, long-term weight change, and incident overweight or obesity: the Seguimiento Universidad de Navarra (SUN) cohort. *Am J Clin Nutr* 2010; 92: 1484–93.
 48. Quatromoni PA, Pencina M, Cobain MR, Jacques PF, D'Agostino RB. Dietary quality predicts adult weight gain: findings from the Framingham Offspring Study. *Obesity* (Silver Spring) 2006; 14: 1383–91.
 49. Romaguera D, Norat T, Vergnaud A-C, Mouw T, May AM, Agudo A, et al. Mediterranean dietary patterns and prospective weight change in participants of the EPIC-PANACEA project. *Am J Clin Nutr* 2010; 92: 912–21.
 50. Sánchez-Villegas A, Bes-Rastrollo M, Martínez-González MA, Serra-Majem L. Adherence to a Mediterranean dietary pattern and weight gain in a follow-up study: the SUN cohort. *Int J Obes (Lond)* 2006; 30: 350–8.
 51. Zamora D, Gordon-Larsen P, Jacobs DR Jr, Popkin BM. Diet quality and weight gain among black and white young adults: the Coronary Artery Risk Development in Young Adults (CARDIA) Study (1985–2005). *Am J Clin Nutr* 2010; 92: 784–93.

52. Brinkworth GD, Noakes M, Parker B, Foster P, Clifton PM. Long-term effects of advice to consume a high-protein, low-fat diet, rather than a conventional weight-loss diet, in obese adults with type 2 diabetes: one-year follow-up of a randomised trial. *Diabetologia* 2004; 47: 1677–86.
53. Dale KS, McAuley KA, Taylor RW, Williams SM, Farmer VL, Hansen P, et al. Determining optimal approaches for weight maintenance: a randomized controlled trial. *CMAJ* 2009; 180: E39–46.
54. Due A, Larsen TM, Mu H, Hermansen K, Stender S, Astrup A. Comparison of 3 ad libitum diets for weight-loss maintenance, risk of cardiovascular disease, and diabetes: a 6-mo randomized, controlled trial. *Am J Clin Nutr* 2008; 88: 1232–41.
55. Larsen TM, Dalskov S-M, van Baak M, Jebb SA, Papadaki A, Pfeiffer AFH, et al. Diets with high or low protein content and glycemic index for weight-loss maintenance. *N Engl J Med* 2010; 363: 2102–13.
56. Phelan S, Wyatt HR, Hill JO, Wing RR. Are the eating and exercise habits of successful weight losers changing? *Obesity (Silver Spring)* 2006; 14: 710–6.
57. Swinburn BA, Metcalf PA, Ley SJ. Long-term (5-year) effects of a reduced-fat diet intervention in individuals with glucose intolerance. *Diabetes Care* 2001; 24: 619–24.
58. White C, Drummond S, De Looy A. Comparing advice to decrease both dietary fat and sucrose, or dietary fat only, on weight loss, weight maintenance and perceived quality of life. *Int J Food Sci Nutr* 2010; 61: 282–94.
59. Delbridge EA, Prendergast LA, Pritchard JE, Proietto J. One-year weight maintenance after significant weight loss in healthy overweight and obese subjects: does diet composition matter? *Am J Clin Nutr* 2009; 90: 1203–14.
60. Bes-Rastrollo M, Sabat e J, G omez-Gracia E, Alonso A, Mart inez JA, Mart inez-Gonz alez MA. Nut consumption and weight gain in a Mediterranean cohort: the SUN study. *Obesity (Silver Spring)* 2007; 15: 107–16.
61. Field AE, Wing RR, Manson JE, Spiegelman DL, Willett WC. Relationship of a large weight loss to long-term weight change among young and middle-aged US women. *Int J Obes Relat Metab Disord* 2001; 25: 1113–21.
62. Anderson JW, Konz EC, Frederich RC, Wood CL. Long-term weight-loss maintenance: a meta-analysis of US studies. *Am J Clin Nutr* 2001; 74: 579–84.
63. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* 2000; 24: 1545–52.
64. Ayyad C, Andersen T. Long-term efficacy of dietary treatment of obesity: a systematic review of studies published between 1931 and 1999. *Obes Rev* 2000; 1: 113–9.
65. Azadbakht L, Mirmiran P, Esmailzadeh A, Azizi F. Better dietary adherence and weight maintenance achieved by a long-term moderate-fat diet. *Br J Nutr* 2007; 97: 399–404.
66. Bes-Rastrollo M, Mart nez-Gonz alez MA, S anchez-Villegas A, de la Fuente Arrillaga C, Mart nez JA. Association of fiber intake and fruit/vegetable consumption with weight gain in a Mediterranean population. *Nutrition* 2006; 22: 504–11.
67. Borg P, Fogelholm M, Kukkonen-Harjula K. Food selection and eating behaviour during weight maintenance intervention and 2-y follow-up in obese men. *Int J Obes Relat Metab Disord* 2004; 28: 1548–54.
68. Brown T, Avenell A, Edmunds LD, Moore H, Whittaker V, Avery L, et al. Systematic review of long-term lifestyle interventions to prevent weight gain and morbidity in adults. *Obes Rev* 2009; 10: 627–38.
69. Burke LE, Warziski M, Styn MA, Music E, Hudson AG, Sereika SM. A randomized clinical trial of a standard versus vegetarian diet for weight loss: the impact of treatment preference. *Int J Obes (Lond)* 2008; 32: 166–76.
70. Burke LE, Hudson AG, Warziski MT, Styn MA, Music E, Elci OU, et al. Effects of a vegetarian diet and treatment preference on biochemical and dietary variables in overweight and obese adults: a randomized clinical trial. *Am J Clin Nutr* 2007; 86: 588–96.
71. Burke V, Mori TA, Giangiulio N, Gillam HF, Beilin LJ, Houghton S, et al. An innovative program for changing health behaviours. *Asia Pac J Clin Nutr* 2002; 11(Suppl 3): S586–97.
72. Cardillo S, Seshadri P, Iqbal N. The effects of a low-carbohydrate versus low-fat diet on adipocytokines in severely obese adults: three-year follow-up of a randomized trial. *Eur Rev Med Pharmacol Sci* 2006; 10: 99–106.
73. Carels RA, Darby LA, Douglass OM, Cacciapaglia HM, Rydin S. Education on the glycemic index of foods fails to improve treatment outcomes in a behavioral weight loss program. *Eat Behav* 2005; 6: 145–50.
74. Carnethon MR, Loria CM, Hill JO, Sidney S, Savage PJ, Liu K. Risk factors for the metabolic syndrome: the Coronary Artery Risk Development in Young Adults (CARDIA) study, 1985–2001. *Diabetes Care* 2004; 27: 2707–15.
75. Carty CL, Kooperberg C, Neuhauser ML, Tinker L, Howard B, Wactawski-Wende J, et al. Low-fat dietary pattern and change in body-composition traits in the Women’s Health Initiative Dietary Modification Trial. *Am J Clin Nutr* 2011; 93: 516–24.
76. Chen L, Appel LJ, Loria C, Lin P-H, Champagne CM, Elmer PJ, et al. Reduction in consumption of sugar-sweetened beverages is associated with weight loss: the PREMIER trial. *Am J Clin Nutr* 2009; 89: 1299–306.
77. Cheskin LJ, Mitchell AM, Jhaveri AD, Mitola AH, Davis LM, Lewis RA, et al. Efficacy of meal replacements versus a standard food-based diet for weight loss in type 2 diabetes: a controlled clinical trial. *Diabetes Educ* 2008; 34: 118–27.
78. Clifton PM, Keogh JB, Noakes M. Long-term effects of a high-protein weight-loss diet. *Am J Clin Nutr* 2008; 87: 23–9.
79. Davis LM, Coleman C, Kiel J, Rampolla J, Hutchison T, Ford L, et al. Efficacy of a meal replacement diet plan compared to a food-based diet plan after a period of weight loss and weight maintenance: a randomized controlled trial. *Nutr J* 2010; 9: 11.
80. Ditschuneit HH, Flechtner-Mors M. Value of structured meals for weight management: risk factors and long-term weight maintenance. *Obes Res* 2001; 9(Suppl 4): S284S–9.
81. Djuric Z, Poore KM, Depper JB, Uhley VE, Lababidi S, Covington C, et al. Methods to increase fruit and vegetable intake with and without a decrease in fat intake: compliance and effects on body weight in the nutrition and breast health study. *Nutr Cancer* 2002; 43: 141–51.
82. Due A, Toubro S, Skov AR, Astrup A. Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial. *Int J Obes Relat Metab Disord* 2004; 28: 1283–90.
83. Duffey KJ, Gordon-Larsen P, Jacobs DR Jr, Williams OD, Popkin BM. Differential associations of fast food and restaurant food consumption with 3-y change in body mass index: the Coronary Artery Risk Development in Young Adults Study. *Am J Clin Nutr* 2007; 85: 201–8.
84. Eckel RH, Hernandez TL, Bell ML, Weil KM, Shepard TY, Grunwald GK, et al. Carbohydrate balance predicts weight and fat gain in adults. *Am J Clin Nutr* 2006; 83: 803–8.
85. Farshchi HR, Taylor MA, Macdonald IA. Decreased thermic effect of food after an irregular compared with a regular meal

- pattern in healthy lean women. *Int J Obes Relat Metab Disord* 2004; 28: 653–60.
86. Flechtner-Mors M, Boehm BO, Wittmann R, Thoma U, Ditschuneit HH. Enhanced weight loss with protein-enriched meal replacements in subjects with the metabolic syndrome. *Diabetes Metab Res Rev* 2010; 26: 393–405.
 87. French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. *Int J Obes Relat Metab Disord* 2000; 24: 1353–9.
 88. Greene LF, Malpede CZ, Henson CS, Hubbert KA, Heimbarger DC, Ard JD. Weight maintenance 2 years after participation in a weight loss program promoting low-energy density foods. *Obesity (Silver Spring)* 2006; 14: 1795–801.
 89. Hensrud DD. Dietary treatment and long-term weight loss and maintenance in type 2 diabetes. *Obes Res* 2001; 9(Suppl 4): S348–53.
 90. Hoy MK, Winters BL, Chlebowski RT, Papoutsakis C, Shapiro A, Lubin MP, et al. Implementing a low-fat eating plan in the Women's Intervention Nutrition Study. *J Am Diet Assoc* 2009; 109: 688–96.
 91. Jehn ML, Patt MR, Appel LJ, Miller ER 3rd. One year follow-up of overweight and obese hypertensive adults following intensive lifestyle therapy. *J Hum Nutr Diet* 2006; 19: 349–54.
 92. Karnehed N, Tynelius P, Heitmann BL, Rasmussen F. Physical activity, diet and gene-environment interactions in relation to body mass index and waist circumference: the Swedish young male twins study. *Public Health Nutr* 2006; 9: 851–8.
 93. Kaukua JK, Pekkarinen TA, Rissanen AM. Health-related quality of life in a randomised placebo-controlled trial of sibutramine in obese patients with type II diabetes. *Int J Obes Relat Metab Disord* 2004; 28: 600–5.
 94. Keogh JB, Luscombe-Marsh ND, Noakes M, Wittert GA, Clifton PM. Long-term weight maintenance and cardiovascular risk factors are not different following weight loss on carbohydrate-restricted diets high in either monounsaturated fat or protein in obese hyperinsulinaemic men and women. *Br J Nutr* 2007; 97: 405–10.
 95. Kristal AR, Curry SJ, Shattuck AL, Feng Z, Li S. A randomized trial of a tailored, self-help dietary intervention: the Puget Sound Eating Patterns study. *Prev Med* 2000; 31: 380–9.
 96. Kuller LH, Simkin-Silverman LR, Wing RR, Meilahn EN, Ives DG. Women's healthy lifestyle project: a randomized clinical trial: results at 54 months. *Circulation* 2001; 103: 32–7.
 97. Lantz H, Peltonen M, Agren L, Torgerson JS. Intermittent versus on-demand use of a very low calorie diet: a randomized 2-year clinical trial. *J Intern Med* 2003; 253: 463–71.
 98. Layman DK, Evans EM, Erickson D, Seyler J, Weber J, Bagshaw D, et al. A moderate-protein diet produces sustained weight loss and long-term changes in body composition and blood lipids in obese adults. *J Nutr* 2009; 139: 514–21.
 99. Lejeune MPM, Kovacs EMR, Westerterp-Plantenga MS. Additional protein intake limits weight regain after weight loss in humans. *Br J Nutr* 2005; 93: 281–9.
 100. Leser MS, Yanovski SZ, Yanovski JA. A low-fat intake and greater activity level are associated with lower weight regain 3 years after completing a very-low-calorie diet. *J Am Diet Assoc* 2002; 102: 1252–6.
 101. Lindström J, Louheranta A, Mannelin M, Rastas M, Salminen V, Eriksson J, et al. The Finnish Diabetes Prevention Study (DPS): lifestyle intervention and 3-year results on diet and physical activity. *Diabetes Care* 2003; 26: 3230–6.
 102. Macdonald HM, New SA, Campbell MK, Reid DM. Longitudinal changes in weight in perimenopausal and early postmenopausal women: effects of dietary energy intake, energy expenditure, dietary calcium intake and hormone replacement therapy. *Int J Obes Relat Metab Disord* 2003; 27: 669–76.
 103. Marinilli Pinto A, Gorin AA, Raynor HA, Tate DF, Fava JL, Wing RR. Successful weight-loss maintenance in relation to method of weight loss. *Obesity (Silver Spring)* 2008; 16: 2456–61.
 104. McAuley KA, Smith KJ, Taylor RW, McLay RT, Williams SM, Mann JI. Long-term effects of popular dietary approaches on weight loss and features of insulin resistance. *Int J Obes (Lond)* 2006; 30: 342–9.
 105. Moore CS, Lindroos AK, Kreutzer M, Larsen TM, Astrup A, van Baak MA, et al. Dietary strategy to manipulate ad libitum macronutrient intake, and glycaemic index, across eight European countries in the Diogenes Study. *Obes Rev* 2010; 11: 67–75.
 106. Moran LJ, Noakes M, Clifton PM, Wittert GA, Williams G, Norman RJ. Short-term meal replacements followed by dietary macronutrient restriction enhance weight loss in polycystic ovary syndrome. *Am J Clin Nutr* 2006; 84: 77–87.
 107. Mozaffarian D, Marfisi R, Levantesi G, Silletta MG, Tavazzi L, Tognoni G, et al. Incidence of new-onset diabetes and impaired fasting glucose in patients with recent myocardial infarction and the effect of clinical and lifestyle risk factors. *Lancet* 2007; 370: 667–75.
 108. Ochner CN, Lowe MR. Self-reported changes in dietary calcium and energy intake predict weight regain following a weight loss diet in obese women. *J Nutr* 2007; 137: 2324–8.
 109. Packianathan I, Sheikh M, Boniface D, Finer N. Predictors of programme adherence and weight loss in women in an obesity programme using meal replacements. *Diabetes Obes Metab* 2005; 7: 439–47.
 110. Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L. Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med* 2008; 168: 1487–92.
 111. Poppitt SD, Keogh GF, Prentice AM, Williams DEM, Sonnemans HMW, Valk EEJ, et al. Long-term effects of ad libitum low-fat, high-carbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. *Am J Clin Nutr* 2002; 75: 11–20.
 112. Raynor HA, Jeffery RW, Tate DF, Wing RR. Relationship between changes in food group variety, dietary intake, and weight during obesity treatment. *Int J Obes Relat Metab Disord* 2004; 28: 813–20.
 113. Razquin C, Martinez JA, Martinez-Gonzalez MA, Mitjavila MT, Estruch R, Marti A. A 3 years follow-up of a Mediterranean diet rich in virgin olive oil is associated with high plasma antioxidant capacity and reduced body weight gain. *Eur J Clin Nutr* 2009; 63: 1387–93.
 114. Redman LM, Heilbronn LK, Martin CK, de Jonge L, Williamson DA, Delany JP, et al. Metabolic and behavioral compensations in response to caloric restriction: implications for the maintenance of weight loss. *PLoS ONE* 2009; 4: e4377.
 115. Riebe D, Blissmer B, Greene G, Caldwell M, Ruggiero L, Stillwell KM, et al. Long-term maintenance of exercise and healthy eating behaviors in overweight adults. *Prev Med* 2005; 40: 769–78.
 116. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med* 2009; 360: 859–73.
 117. Saris WH. Very-low-calorie diets and sustained weight loss. *Obes Res* 2001; 9(Suppl 4): S295–301.
 118. Saris WH, Astrup A, Prentice AM, Zunft HJ, Formiguera X, Verboeket-van de Venne WP, et al. Randomized controlled

- trial of changes in dietary carbohydrate/fat ratio and simple vs complex carbohydrates on body weight and blood lipids: the CARMEN study. *The Carbohydrate Ratio Management in European National diets*. *Int J Obes Relat Metab Disord* 2000; 24: 1310–8.
119. Sasaki S, Ishikawa T, Yanagibori R, Amano K. Change and 1-year maintenance of nutrient and food group intakes at a 12-week worksite dietary intervention trial for men at high risk of coronary heart disease. *J Nutr Sci Vitaminol* 2000; 46: 15–22.
 120. Schoeller DA, Watras AC, Whigham LD. A meta-analysis of the effects of conjugated linoleic acid on fat-free mass in humans. *Appl Physiol Nutr Metab* 2009; 34: 975–8.
 121. Sichieri R, Moura AS, Genelhu V, Hu F, Willett WC. An 18-month randomized trial of a low-glycemic-index diet and weight change in Brazilian women. *Am J Clin Nutr* 2007; 86: 707–13.
 122. Simkin-Silverman LR, Wing RR, Boraz MA, Kuller LH. Lifestyle intervention can prevent weight gain during menopause: results from a 5-year randomized clinical trial. *Ann Behav Med* 2003; 26: 212–20.
 123. Sloth B, Due A, Larsen TM, Holst JJ, Hedning A, Astrup A. The effect of a high-MUFA, low-glycaemic index diet and a low-fat diet on appetite and glucose metabolism during a 6-month weight maintenance period. *Br J Nutr* 2009; 101: 1846–58.
 124. Steptoe A, Kerry S, Rink E, Hilton S. The impact of behavioral counseling on stage of change in fat intake, physical activity, and cigarette smoking in adults at increased risk of coronary heart disease. *Am J Public Health* 2001; 91: 265–9.
 125. Stookey JD, Adair LS, Popkin BM. Do protein and energy intakes explain long-term changes in body composition? *J Nutr Health Aging* 2005; 9: 5–17.
 126. Stote KS, Baer DJ, Spears K, Paul DR, Harris GK, Rumpler WV, et al. A controlled trial of reduced meal frequency without caloric restriction in healthy, normal-weight, middle-aged adults. *Am J Clin Nutr* 2007; 85: 981–8.
 127. Svetkey LP, Stevens VJ, Brantley PJ, Appel LJ, Hollis JF, Loria CM, et al. Comparison of strategies for sustaining weight loss: the weight loss maintenance randomized controlled trial. *JAMA* 2008; 299: 1139–48.
 128. Thorpe MP, Jacobson EH, Layman DK, He X, Kris-Etherton PM, Evans EM. A diet high in protein, dairy, and calcium attenuates bone loss over twelve months of weight loss and maintenance relative to a conventional high-carbohydrate diet in adults. *J Nutr* 2008; 138: 1096–100.
 129. Turk MW, Yang K, Hravnak M, Sereika SM, Ewing LJ, Burke LE. Randomized clinical trials of weight loss maintenance: a review. *J Cardiovasc Nurs* 2009; 24: 58–80.
 130. Turner-McGrievy GM, Barnard ND, Scialli AR. A two-year randomized weight loss trial comparing a vegan diet to a more moderate low-fat diet. *Obesity (Silver Spring)* 2007; 15: 2276–81.
 131. van de Vijver LPL, van den Bosch LMC, van den Brandt PA, Goldbohm RA. Whole-grain consumption, dietary fibre intake and body mass index in the Netherlands cohort study. *Eur J Clin Nutr* 2009; 63: 31–8.
 132. Vang A, Singh PN, Lee JW, Haddad EH, Brinegar CH. Meats, processed meats, obesity, weight gain and occurrence of diabetes among adults: findings from adventist health studies. *Ann Nutr Metab* 2008; 52: 96–104.
 133. Wang L, Lee I-M, Manson JE, Buring JE, Sesso HD. Alcohol consumption, weight gain, and risk of becoming overweight in middle-aged and older women. *Arch Intern Med* 2010; 170: 453–61.
 134. Whigham LD, Watras AC, Schoeller DA. Efficacy of conjugated linoleic acid for reducing fat mass: a meta-analysis in humans. *Am J Clin Nutr* 2007; 85: 1203–11.
 135. Woo J, Cheung B, Ho S, Sham A, Lam TH. Influence of dietary pattern on the development of overweight in a Chinese population. *Eur J Clin Nutr* 2008; 62: 480–7.
 136. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. *Obes Rev* 2012; 13: 275–86.
 137. Freiberg MS, Pencina MJ, D'Agostino RB, Lanier K, Wilson PWF, Vasan RS. BMI vs. waist circumference for identifying vascular risk. *Obesity (Silver Spring)* 2008; 16: 463–9.
 138. Hätönen KA, Similä ME, Virtamo JR, Eriksson JG, Hannila M-L, Sinkko HK, et al. Methodologic considerations in the measurement of glycemic index: glycemic response to rye bread, oatmeal porridge, and mashed potato. *Am J Clin Nutr* 2006; 84: 1055–61.
 139. Isaksson H, Fredriksson H, Andersson R, Olsson J, Aman P. Effect of rye bread breakfasts on subjective hunger and satiety: a randomized controlled trial. *Nutr J* 2009; 8: 39.
 140. Sabaté J. Nut consumption and body weight. *Am J Clin Nutr* 2003; 78: 647S–50S.
 141. Sabaté J, Oda K, Ros E. Nut consumption and blood lipid levels: a pooled analysis of 25 intervention trials. *Arch Intern Med* 2010; 170: 821–7.
 142. Ludwig DS. Dietary glycemic index and obesity. *J Nutr* 2000; 130: 280S–3S.
 143. Zemel MB. Proposed role of calcium and dairy food components in weight management and metabolic health. *Phys Sportsmed* 2009; 37: 29–39.
 144. Soares MJ, Cummings SJ, Mamo JCL, Kenrick M, Piers LS. The acute effects of olive oil v. cream on postprandial thermogenesis and substrate oxidation in postmenopausal women. *Br J Nutr* 2004; 91: 245–52.
 145. Veldhorst MAB, Westerterp KR, Westerterp-Plantenga MS. Gluconeogenesis and protein-induced satiety. *Br J Nutr* 2012; 107: 595–600.
 146. O'Neil CE, Fulgoni VL 3rd, Nicklas TA. Association of candy consumption with body weight measures, other health risk factors for cardiovascular disease, and diet quality in US children and adolescents: NHANES 1999–2004. *Food Nutr Res* 2011; 55. <http://www.ncbi.nlm.nih.gov/pubmed/21691462> [cited 8 Feb 2012].
 147. Lawlor DA, Davey Smith G, Kundu D, Bruckdorfer KR, Ebrahim S. Those confounded vitamins: what can we learn from the differences between observational versus randomised trial evidence? *Lancet* 2004; 363: 1724–7.

***Mikael Fogelholm**

Department of Food and Environmental Sciences
 University of Helsinki
 POB 66
 00014 Helsinki
 Finland
 Email: mikael.fogelholm@helsinki.fi

Appendix I

Search terms:

Set I

- (1) Dietary carbohydrates.mesh. OR
- (2) Dietary fats.mesh. OR (as free text) 'saturated fats' OR 'monounsaturated fats' OR 'polyunsaturated fats' [TI, AB] OR
- (3) Fatty acids, unsaturated.mesh. OR
- (4) Proteins.mesh. OR
- (5) Dietary fiber.mesh. OR
- (6) Energy intake.mesh. OR
- (7) Diet, Carbohydrate-Restricted.mesh. OR
- (8) Diet, fat-restricted.mesh. OR
- (9) Diet, Mediterranean.mesh. OR
- (10) Diet, Protein-restricted.mesh. OR
- (11) Diet, vegetarian.mesh. OR
- (12) Ketogenic diet.mesh.

AND

Set II

- (1) Body weight.mesh. (narrower terms: overweight.mesh., including obesity.mesh.) OR
- (2) Waist-Hip Ratio.mesh. OR 'waist girth' OR
- (3) Waist Circumference.mesh. OR
- (4) Body composition.mesh. (incl. narrower term: body fat distribution.mesh. and adiposity.mesh.) OR
- (5) Adipose tissue.mesh. (incl. narrower term: abdominal fat.mesh.) OR 'body fat' OR
- (6) body mass index.mesh. OR 'fat mass'

AND

Set III

maintenance* OR gain* OR regain* (cannot use too common words, like: change OR changes OR changing)

Set I and Set II and Set III = Group I

Set IV

weight gain.mesh.

OR

'weight gain' OR 'Gain, Weight' OR 'Gains, Weight' OR 'Weight Gains' [TI, AB]

Set I AND Set IV = Group II

Group I

OR

Group II

AND

RCT. PT OR mesh

OR

cohort studies.mesh. (incl. term: longitudinal studies.mesh. OR prospective studies.mesh.)

OR

intervention studies.mesh.

OR

meta-analysis, mesh OR pt
 OR
 'systematic review' OR 'systematic reviews' OR 'Cochrane database syst rev'
 OR 'randomised controlled' OR 'randomised controlled' OR meta-analysis
 human, 2000

Appendix 2

Reasons for excluding full papers ($n=78$) from the quality grading

References	Reason for exclusion
Anderson et al. (62)	Macronutrient data not shown
Astrup (5)	Review, but concentrates on weight reduction only (not on weight management)
Astrup et al. (63)	Concentrates on weight reduction only
Ayyad et al. (64)	No macronutrient data, review on weight loss mainly
Azadbakht et al. (65)	Weight reduction only
Bes-Rastrollo et al. (66)	Cross-sectional study
Borg et al. (67)	Originally included in the evaluation but excluded from quality grading: no data on food vs. weight change in a prospective design
Brown et al. (68)	Originally included in the evaluation but excluded from quality grading: the review concentrated on weight reduction interventions with special diets
Burke et al. (70)	No macronutrient data
Burke et al. (69)	Weight reduction only
Burke et al. (71)	Physical activity and nutrition combined, not clear maintenance phase
Cardillo et al. (72)	Originally included in the evaluation but excluded from quality grading: weight loss was different between the groups initially
Carels et al. (73)	Weight reduction only
Carnethon et al. (74)	No results on weight change, MBO as an outcome
Carty et al. (75)	Originally included in the evaluation but excluded from quality grading: same data as Howard et al. (18), but this is a subset with a smaller number of cases
Chen et al. (76)	Weight reduction only
Cheskin et al. (77)	Meal replacements, weight reduction only, no dietary data
Clifton et al. (78)	Weight reduction only
Davis et al. (79)	Meal replacements, weight reduction only, follow-up less than 6 months
Ditschuneit et al. (80)	Meal replacements, weight reduction only
Djuric et al. (81)	Originally included in the evaluation but excluded from quality grading: effects on body weight varied by groups during the first 3 months of the intervention; weight reduction study
Due et al. (82)	Weight reduction only
Duffey et al. (83)	Only eating patterns, no macronutrient data
Eckel et al. (84)	No dietary data
Farshchi et al. (85)	Experimental study, focused on meal pattern and thermic effect of food
Flechtner-Mors et al. (86)	Meal replacements, weight reduction only
Forshee et al. (10)	Originally included in the evaluation but excluded from quality grading: review
French et al. (87)	Originally included in the evaluation but excluded from quality grading: study on visits to fast food restaurants and dietary, behavioural and demographic correlates
Gibson (8)	Originally included in the evaluation but excluded from quality grading: review
Greene et al. (88)	Originally was included in the evaluation but excluded from SLR: weight loss was different between the groups initially
Hensrud (89)	Not a systematic review
Hoy et al. (90)	Study on cancer patients
Jehn et al. (91)	Physical activity and nutrition combined
Karnehed et al. (92)	Originally included in the evaluation but excluded from quality grading: dietary data were collected only at follow-up, not at baseline

Appendix 2 (Continued)

References	Reason for exclusion
Kaukua et al. (93)	No dietary data
Keogh et al. (94)	Weight reduction only
Kristal et al. (95)	No results on weight change
Kuller et al. (96)	Physical activity and nutrition combined
Lantz et al. (97)	Weight reduction only, comparisons between VLCDs
Layman et al. (98)	Weight reduction only
Lejeune et al. (99)	Originally included in the evaluation but excluded from quality grading: dietary intake not assessed, except for protein intake by urine analysis. Protein supplement used to increase protein intake
Leser et al. (100)	Originally included in the evaluation but excluded from quality grading: very small sample size, dietary intake assessed only in the end of the study, only fat-intake reported, PA assessed, but not used to adjust the results
Lindstrom et al. (101)	Physical activity and nutrition combined
Macdonald et al. (102)	Macronutrient data not shown
Malik et al. (9)	Originally included in the evaluation but excluded from quality grading: review
Marinilli Pinto et al. (103)	Study on counseling, only weight loss results
McAuley et al. (104)	Weight reduction only
Moore et al. (105)	Description of a study, no results included
Moran et al. (106)	Meal replacements, weight reduction only
Mozaffarian et al. (107)	No results on weight change
Ochner et al. (108)	Macronutrient data not shown, mixed race
Packianathan et al. (109)	No macronutrient data, meal replacements, weight reduction only
Palmer et al. (110)	Race: African-American, weight not an outcome
Poppitt et al. (111)	Weight reduction only, short follow-up (6 months)
Raynor et al. (112)	Exercise intervention, study on weight loss, no clear data on macronutrients
Razquin et al. (113)	Originally included in the evaluation but excluded from quality grading: the participants were mostly overweight and obese and had high-risk for cardiovascular diseases; e.g. Type 2 diabetes was an inclusion criteria
Redman et al. (114)	Weight reduction only
Riebe et al. (115)	Physical activity and nutrition combined
Sacks et al. (116)	Weight reduction only
Saris (117)	No dietary intake data
Saris et al. (118)	Weight reduction only
Sasaki et al. (119)	No results on weight change
Schoeller et al. (120)	Study on CLA treatment, no diet, weight reduction only
Sichieri et al. (121)	Originally included in the evaluation but excluded from quality grading: this is a weight reduction study
Simkin-Silverman et al. (122)	Physical activity and nutrition combined
Sloth et al. (123)	Originally included in the evaluation but excluded from quality grading: same database as in Due et al. (82) but fewer cases
Steptoe et al. (124)	No results on weight change, multiple interventions
Stookey et al. (125)	Race: only Asian (Chinese)
Stote et al. (126)	No macronutrient data, study on meal frequency
Svetkey et al. (127)	No macronutrient data, mixed race
Thorpe et al. (128)	Weight reduction only
Turk et al. (129)	Originally included in the evaluation but excluded from quality grading: review
Turner-McGrievy et al. (130)	Weight reduction only
van de Vijver et al. (131)	Cross-sectional design
Vang et al. (132)	No results on weight change, no macronutrient data
Wang et al. (133)	Data on alcohol consumption only
Whigham et al. (134)	Study on CLA treatment, no diet, weight reduction only
Woo et al. (135)	Race: only Asian (Chinese)

Appendix 3 Evidence tables

Table 1. Macronutrients and prevention of weight gain

Reference details, First author, Year, Country	Study design (RCT, CT, cohort, case control etc.)	Population, subject characteristics, Inclusion/exclusion criteria, setting, no. at baseline, male/female, age, ethnicity of the subjects, anthropometry, location	Outcome measures Disease, biological measures	Intervention/exposure	Time between baseline exposure and outcome assessment	Dietary assessment method FFQ, food record Internal validation (y/n)	No of subjects analysed	Intervention (I) (dose interval, duration), Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change	Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up) Drop out (%)	Results (I, C) (Absolute difference, RR, OR, <i>p</i> -value, confidence interval, sensitivity, specificity, observer reliability? etc.)	Confounders adjusted for	Study quality and relevance, Comments (A–C)
Bes-Rastrollo, 2008, US (12)	Cohort	Nurses' health study, 116,671 women, age 36.5 (4.6) y Excluded at baseline (1991) if did not complete FFQ, if they reported EI (<500 or >3,500 kcal/day), history of diabetes or CVD, cancer before 1999 (post test), pregnancy at any time from baseline to post test, no PA data assessed in 1991 and 1997, only baseline data, missing Wt data. Final <i>n</i> = 51,188.	Wt gain (self-report).	Change in dietary ED (defined as the amount of energy in a given weight of food).	8 y	133-item FFQ	<i>n</i> = 50,026		8 y. Dropout 57%.	W who increased dietary ED during follow-up the most had a significantly greater weight gain than those who decreased ED the most: 6.42 vs. 4.57 kg (<i>p</i> for trend <0.001).	Age, baseline alcohol intake, PA, smoking, postmenopausal hormone use, oral contraceptives, cereal fibre intake, TFA intake, baseline BMI, change in intake of SSSDs and changes in confounders between time periods.	B Weight self-reported. Details of dietary assessments were lacking in this report, although they have been reported earlier. The comparability of this population (nurses from the US) and Nordic population is not clear.

Du, 2009; Italy, UK, The Netherlands, Germany, Denmark (13)	Cohort	Eight cities/counties in Italy, UK, The Netherlands, Germany and Denmark (EPIC), age 20–78 y, $n = 146,543$ at baseline (1992–1998), $n = 102,346$ at follow up (1998–2005), excl. pregnancy, missing information on diet, anthropometry or follow-up time, EI/BMR in the top or bottom 1% of EPIC population, unrealistic anthropometric measures, history of cancer, diabetes or CVD at baseline.	Changes in wt and WC. Measured at baseline and two centres also at follow-up. Otherwise self report.	Dietary ED	6.5 (1.9–12.5) y	Country-specific FFQ, self-administered at baseline. Intake calculated using country-specific food composition tables. ED calculated as EI from food divided by the weights of these foods. Drinks (water, alcohol, milk) not included.	$n = 89,432$ (42% M)	6.5 (1.9–12.5) y	ED was not associated with weight change, but significantly with WC. For 1 kcal/g ED annual WC change was 0.09 cm/y (95% CI: 0.01–0.18)	Age, sex baseline wt, ht and WC, smoking, PA, education, alcohol, EI from beverages for women: also menopausal status and hormone use.	B Large multi-centre study with large variation in results between centres which are difficult to adjust for even though advanced statistical techniques are used. Variation between measured and self-reported body wt.
Du, 2010, the Netherlands (five countries) (14)	Cohort	Eight cities/counties in Italy, UK, The Netherlands, Germany and Denmark (DiOGenes), age 20–78 y, $n = 146,543$ at baseline (1992–1998), $n = 102,346$ at follow up (1998–2005), excl. pregnancy, missing information on diet, anthrop, or follow-up duration. EI/BMR in the top or bottom 1% of EPIC population, unrealistic anthrop measures, presence of chronic diseases; baseline BMI 25.5–26.7 kg/m ² for M and 24.4–25.8 kg/m ² for W, WC 90–95 cm for men and 77–86 cm for women.	Change in wt and WC; measured wt, ht and WC at baseline and follow-up, self-reported in six centres	Fibre intake: total, cereal fibre, and fruit and vegetable fibre	6.5 y (1.9–12.5) y	Country-specific FFQs at baseline. For validation reference, see the original article. Enzymatic-gravimetric method (AOAC) to define dietary fibre, except in UK where defined as non-starch polysaccharides using Englyst method	$n = 89,432$ (42% M)	6.5.y on average Drop-out 31.2%	10 g fibre intake associated with –39 g (95% CI: –71 to –7 g) wt change/year and –0.08 cm (–0.11, –0.05 cm) change in WC/y, 10 g cereal fibre assoc with –77 g (–127, –26 g) wt change per year, –0.10 cm (–0.18, 0.02 cm) change in WC/y.	Age, sex, baseline wt, ht and WC, smoking, PA, education, alcohol, GI, intake of protein, fat and CHD, total EI, in W menopausal status and hormone use.	B

Field 2007, US (15)	Cohort	Registered nurses, W aged 41–68 y at baseline (1988), <i>n</i> = 41,518, incl. free of CVD, cancer and diabetes at baseline, postal follow-up questionnaires every 2 y, race not reported, baseline BMI 25.0 kg/m ² .	Wt change, BMI in 1994; self-reported wt.	Baseline fat intake (E%), average intake and 8 y change in intake + animal fat/vegetable fat + PUFA, SFA, trans fats.	8 y	136-item FFQ For validation reference, see the original article.	<i>n</i> = 41,518	8 y. Drop out rates, or number of subjects that were excluded not reported.	beta for 1% difference (substituting 1% of calories from fat for 1% of calories from CHD) baseline fat intake <i>B</i> = 0.11 (<i>p</i> < 0.0001), PUFA 0.42, SFA 0.40 and TFA 0.54.	Baseline BMI, age, PA, time spent sitting, menopausal status and protein%.	B Number of subjects 1/3 of the original sample (1976), no data on representativeness of the data, dietary assessment methods poorly described.
Forouhi, 2009, UK (total five countries) (16)	Cohort	EPIC (see Du 2010), <i>n</i> = 146,543, eligible participants 89,432 (58% W), exclusion criteria see Du 2010, mean age 42.5–58.1 y in six cohorts, baseline BMI 26.3 kg/m ² for M and 25.3 kg/m ² for W, WC 94.4 cm for M and 80.3 cm for W	Annual change in wt (and WC); measured wt and ht at baseline and in 2 (out of 8) centres at follow-up, self-reported in six centres	Amount and type of dietary fat	3.7–10.0 y	Country specific FFQ, habitual intake of medium-sized serving of foods over the past year, in a subsample, also a standardised 24 h recall by using EPIC SOFT. For validation reference, see the original article.	<i>n</i> = 89,432 (58% W)	3.7–10.0 y. No follow-up data available	Weight change 0.90 g/y (95% CI: –0.54 to 2.34) for men and –1.30 g/y (–3.70 to 1.11) for women per 1 g/day energy-adjusted fat intake, a null association for PUFA; MUFA; WC and fat: no significant associations between any fat type and wt change	baseline wt and ht, EI follow-up period, PA, smoking, education, alcohol, protein	B Follow-up data
Halkjaer 2006 (17)	Cohort	50- to 64-y-old M and W living in greater Copenhagen or Aarhus area, random sample. Exclusion: cancer. Baseline <i>n</i> = 54,379, WC 80.0 cm for W and 95.0 cm for M, BMI 24.7 kg/m ² for W and 26.1 kg/m ² for M.	Change in WC	Total EI, EI from macronutrients, EI from macronutrient subgroups based on different food sources.	5 y	192-item FFQ	<i>n</i> = 44,817 (55% W)	5 y. Drop out rate 17.4%.	Neither total EI nor EI from each of the macronutrients was associated with changes in WC, except for an inverse association with protein, especially animal protein. In women, positive associations with changes in WC were seen for CHD from refined grains and potatoes and from foods with simple sugars, whereas	Baseline WC, BMI, age, smoking, alcohol, sporting activity, other macronutrients than the one analyzed, energy intake.	B Follow-up wt and WC were self-reported. Power not reported, but apparently adequate.

carbohydrate from fruit and vegetables was inversely associated and significantly different from any other CHD subgroup. Vegetable fat was positively associated with changes in WC for both M and W.

Howard, 2006, US (18)	RCT (intervention, trial)	<i>n</i> = 56,139 that provided consent and met >32 E% of fat criterion, of that 7,304 were excluded (e.g. nutritionist judgement, medical condition, eating out), <i>n</i> = 48,835 that were randomised to intervention (<i>n</i> = 19,541) and control (<i>n</i> = 29,294), aged 50–79 y. Mixed race reflecting the characteristics of the general population in US, (non-Hispanic white analysed separately). Baseline BMI 29.1 kg/m ² and WC 89.0 cm in both groups (I and C).	Mean wt change across follow-up; measured wt, ht, waist and hip.	Reduction of total fat to 20 E% and increase of vegetable and fruit intake to five or more servings and grains six or more servings daily.	Mean follow-up 7.5 y, randomisation between 1993–1998, anthropometric and nutrition data until August 2004.	Women's Health Initiative FFQ at baseline and I y+every 3 y.	<i>n</i> = 14,246 for I and <i>n</i> = 22,083 for C	See Carty 2010 for intervention; baseline 38.8 E% from fat in I and C, 29.8/38.1 E% at follow-up, SFA: 13.6 E% at baseline, 10.1/13.2 E% at follow-up, CHO: 44.5 E% at baseline, 52.7/44.7 E% at follow-up, fibre: 14.4 g at baseline, 16.9/14.4 g at follow-up.	Mean follow-up 7.5 y; 2,092 (4.3% of C group, 4.3% of I group) were deceased, 1,309 (2.5% C, 2.9% I) stopped follow-up, 670 (1.2% C, 1.6% I) were lost to follow-up.	Decrease in wt 2.2 kg in the I group at year 1 and mean wt 2.2 kg less than in C. A significant difference between I and C (0.5 kg, <i>p</i> = 0.01) maintained through year 9; W with the greatest reduction in fat intake had the argest wt loss (<i>p</i> for trend <0.001 both for I and C)	Age, race, BMI at baseline, change in dietary intake and PA patterns; secondary analyses adjusted for EI.
Iqbal 2006, Denmark (19)	Cohort	Danish citizens living in the western part of Copenhagen County, recruited and examined in 1976 (the 1936 cohort) and 1982 (MONICA), follow-up in 1981 and 1987, respectively, <i>n</i> = 20, 25 M and W aged 30, 40, 50 and 60 y at baseline, exclusion because of missing	Wt change; ht and wt measured at baseline and follow-up.	Dietary components, ED in particular.	5 y	Weighted 7-day food record at baseline. No data on database, ED calculated including the water content as follows: energy from CHO + prot + fat + alcohol (MJ) divided by	<i>n</i> = 862 M and <i>n</i> = 900 W.	Only participation rate reported: ≥79%; in that case assuming that drop- out rate must be less than 21%.	ED not associated with wt change for either sex; in W, protein intake (E%) positively (B = 3.87, SE 1.91, <i>p</i> = 0.04) and fibre intake (g) inversely (B = -22.8, SE 10.6, <i>p</i> = 0.03) associated with wt change in crude but not in adjusted (<i>p</i> = 0.06/0.10) analyses.	Age, BMI, PA, educational level, smoking, EI (baseline variables)	

Koh-Banerjee, 2003, US (20)	Cohort	information or extreme values, baseline BMI 25.1 kg/m ² for M and 23.4 kg/m ² for W. The Health Professionals' Follow-up Study with 51,529 male health professionals aged 40–75 y. At baseline in 1986, 17,584 excluded because of death or medical condition, 17,358 because of missing information, final sample 16,587, baseline BMI 24.9–25.2 kg/m ² (varied across age groups).	Change in WC, self-reported wt and ht (biannual questionnaires), self-reported WC with a sent tape measure in 1987 and 1996.	Changes in diet and macronutrients	9 y	weight of CHO + prot + fat + alcohol + fibre + ash + water (g) 131-item, semi-quantitative FFQ to assess typical food intake over the previous year, collected in 1986, 1990 and 1994. US Dept of Agriculture, Composition of foods – raw, processed and prepared 1963–1988. Validated among a subset of the study participants. See the original article for the literature reference.	<i>n</i> = 16,587 M	Reported follow-up rate 65%.	A 2% increment in EI from TFA substituted for PUFA of CHO associated with a 0.77 WC gain, an increase in fibre (12 g/day) predicted WC reduction of 0.63 cm.	Age, baseline WC and BMI, baseline and changes in total EI, alcohol consumption and PA, and changes in smoking; also changes in BMI for investigating associations independent of wt gain	B
Liu, 2003, US (21)	Cohort	Nurses' health study, female nurses (<i>n</i> = 81,757) aged 38–63 y were followed from 1984 to 1996, exclusion because of diabetes, CVDs or cancers, final baseline population 74,091, baseline BMI 24.5–24.9 kg/m ² (reported according to quintiles of intake of whole-grains at baseline).	Changes in body wt, self-reported wt every 2 y.	Fibre intake, consumption of whole-grain and refined-grain foods.	12 y	126-item semi-quantitative FFQ 1984, 1986, 1990 and 1994 (average consumption during the previous year). No information on database. See original article for the validation literature reference.	<i>n</i> = 74,091	Drop-out rates not reported.	Increase in whole grain intake (average wt gain in 2–4 y 1.23 ± 0.02 kg in the highest and 1.52 ± 0.02 kg in the lowest quintiles) and fibre intake (0.97 ± 0.02 kg and 1.73 ± 0.02 kg respectively) associated with less wt gain (<i>p</i> for trend < 0.0001), increase in refined grain intake associated with greater weight gain (1.57 ± 0.03 kg and 1.14 ± 0.03 kg, <i>p</i> < 0.0001); 12 y follow-up: greatest increase (the highest quintile of change)	Age, y of follow-up, change in PA, smoking status, hormone replacement therapy, intakes of alcohol, caffeine and total EI.	B The number of participants at follow-up not reported, physical activity level information from year 1982 but no information on the measurement

in fibre 1.52 kg
(5.16 ± 0.09 kg
vs. 3.64 ± 0.09
kg less wt gain
than those with
the smallest in-
crease (the
lowest quintile)

Mosca, 2004, US (22)	Cohort	A geographically based (San Luis Valley, Colorado) sample ($n = 1,351$) aged 20–74 y, no history of diabetes, only subjects with normal glucose tolerance included ($n = 1,027$), exclusions during follow-up because of type 2 diabetes/IGT/IFG, pregnancy, change in smoking status, total $n = 782$ at baseline: non-Hispanic white M($n = 213$) and W ($n = 267$), Hispanic M ($n = 136$) and W ($n = 166$), baseline BMI 25.7 kg/m ² for M and 24.3 kg/m ² for W.	Wt change, measured wt and ht	Energy from fat (E%)	11.2 y	24-h recall Nutrition Coordinating Center's nutrient database at University of Minnesota, version 14 (1987)	$n = 782$ at baseline	The second visit after 4.9 y and the third visit after 11.2 y, visit 1: $n = 782$, visit 2: $n = 536$ (68.5%), visit 3: $n = 375$ (48%), i.e. drop-out 52%	Association between %FAT and estimated wt change was illustrated in a figure showing that wt gain was larger if E% fat 45 vs. 25 (interaction time* %fat from linear mixed model $B = 0.013$, $p = 0.0103$), the relationship stronger in W (interaction $p = 0.0002$) than in M ($p = 0.76$).	Gsex, ethnicity, baseline PA, baseline BMI, age, smoking, total energy intake	C Small sample size with a high drop-out rate, includes Hispanic subjects, dietary assessment based on 24 h recalls
Romaguera, 2010, Europe (23)	Cohort	EPIC participants who were involved in DiOGenes –project, eight centres from five countries (Italy, Netherlands, Germany, Denmark, UK). Exclusion: pregnancy, chronic diseases, age > 60 at baseline, smoking status changed during follow-up. Participants: 19,694 M and 28,937 W. These were selected from 102,346 participants with	WC, adjusted to BMI by residuals.	Dietary ED without drinks, GI and GL.	Median follow-up 5.5 y	Country-specific FFQ. National food composition tables. ED was calculated from solid, semi-solid and liquid foods, but not from drinks. GI database was specially developed using mainly published information. FFQ validation has been	$n = 19,694$ M and $n = 28,937$ W.	Median follow-up 5.5 y. Drop-out 30.2% from baseline.	1 kcal/g greater ED predicted a increase in WC of 0.09 cm (95% CI: 0.05–0.13) in M and 0.15 cm (0.09, 0.21) in W; 10 units greater GI predicted an increase in WC of 0.07 cm (0.03, 0.12) in M and 0.06 cm (0.03, 0.10) in W. Among W, lower fibre intake, higher GL, and higher alcohol consumption also predicted a higher WC.	All models: age, baseline wt, ht, and WC, smoking, alcohol, PA, education, menopausal status, etc. Further: Energy from drinks (in the model with ED as the independent variable), total EI (macronutrients), fibre and macronutrients	B Slight variations in the anthropometric techniques between the centres and time-points. Statistical power was not calculated, but appears to be clearly adequate.

		results on both baseline and follow-up.				reported earlier (see the original article for the literature reference)				(GI), fibre, fat, protein and E (GL), GI and macronutrients (fibre).		
Savage, 2008, US (24)	Cohort	White non-Hispanic W ($n=192$) living in Pennsylvania recruited as part of a longitudinal study designed to examine parental influences, eligibility criteria focused on daughters' characteristics, none for mothers (=participants), exclusions because of missing data on wt, final sample ($n=186$), age range 24.1–48.8 y at baseline, baseline BMI 26.9 kg/m ² .	Wt and BMI change, wt and ht measured at each occasion (4 ×, 2 y intervals).	Dietary ED, kcal/g; excluding beverages.	6 y	3 × 24 h recall interviews by telephone within a 2- to 3-week period at each occasion. Nutrition data system for research, University of Minnesota (version 4.01_30); ED (kcal/g) = total energy intake from the food (beverages excluded) divided by the total weight of food.	$n=186$ W	Data collected on four occasions across a 6 y period, at study entry 192 W of whom 183, 177 and 168 reassessed at y 2, 4 and 6. Drop out rate 12%.	ED* time interaction ($p<0.01$): W consuming higher ED diets ($ED>1.85$ kcal/g) gained more wt (on average 6.4 ± 6.5 kg over 6 y) than W with lower ED diets ($ED<1.5$ kcal/g) 2.5 ± 6.8 kg.	Initial BMI, dietary fibre intake, caloric beverage intake	C PA not assessed at all, analyses not adjusted for age, total EI, fat E% and CHO E% varied across ED groups.	
Sherwood, 2000, US (25)	Randomised trial, but data analysed as a cohort.	Participants for the Pound of Prevention study recruited by direct mailing, newspaper and radio ads etc, free of major chronic diseases, aged 20–45 y, predominantly white. Data derived from 826 W and 218 M (93% of total sample enrolled at baseline) who completed the baseline and at least one of the 3 annual follow-up assessments, baseline BMI 28.0 kg/m ² for M and 26.8 kg/m ² for W.	Wt change, body wt and ht measured at baseline and annually.	Macronutrient intake (and PA); total EI, E% from fat and from alcoholic beverages presented in this paper.	3 y	60-item version of Block FFQ to estimate usual dietary intake during the past y. Validation has been reported earlier (see the original article for the literature reference)	$n=826$ W, $n=218$ M	Participants randomised to one of two mail-based educational programs or to a no-contact control group; however, in this paper data analyses as one cohort, in analyses subjects were divided in weight gainers (>5 lb wt gain), wt maintainers	826 W, 218 M at baseline, 759 W and 198 M at y 3.	Increases in E% from fat associated with increases in body wt (coefficient 0.068, SE 0.034, $p=0.045$ in M and coeff. 0.028, SE 0.014, $p=0.042$ in W); no sign differences in mean changes in dietary intake across wt change status (loser, gainer, maintainer).	Age, smoking status, treatment group, baseline wt (and baseline value on respective dependent variables).	B Unable to assess the quality of dietary assessment method without original references for the method.

Tucker, 2009, US (26)	Cohort	Participants recruited via newspaper adds, flyers and company mass e-mail in two metropolitan areas in the Mountain West, US, eligibility tested by telephone interviews (free from serious diseases, non-smokers, premenopausal, not pregnant), at baseline $n=275$ W, mean age 40.1 y, baseline BMI 24.0 kg/m ² .	Wt and body fat; measured wt at baseline and follow-up, body fat% measured by using air displacement plethysmography the Body Pod.	Fibre intake.	20 months	7-day weighted food records at baseline and follow-up. USDA database and other food databases using ESHA Research software (version 7.6). Women were weighed before and after the week of diet recording to make sure that there was no significant weight change during the week of recording	$n=252$	(lost/gain ≤ 5 lb) and wt losers (lost > 5 lb).	Complete follow-up data available from 252 W. Drop out 8%.	For each 1 g increase in fibre intake wt decreased by 0.25 kg ($p=0.0061$) and fat decreased by 0.25%-point ($p=0.0052$). Baseline fibre intake was not associated with wt change.	Age, season of assessment, baseline body fat and fibre intake, baseline and changes in fat intake, EI and PA.	A
-----------------------	--------	--	--	---------------	-----------	--	---------	--	--	--	---	---

W, Women; M, Men; Wt, Weight; Ht, Height; WC, Waist circumference; PA, Physical activity; BMI, Body mass index; EI, Energy intake; ED, Energy density; TFA, Trans fatty acids; CHO, Carbohydrates; MUFA, Monounsaturated fatty acids; PUFA, Poly-unsaturated fatty acids; GI, Glycemic index; GL, Glycemic load; Y, Years.

Appendix 4 Evidence tables

Table 2. Foods and prevention of weight gain

Reference details, First author, Year, Country	Study design (RCT, CT, cohort, case control etc.)	Population, subject characteristics, Inclusion/exclusion criteria, Setting, No at baseline, Male/Female, Age, Ethnicity of the subjects, Anthropometry, Location	Outcome measures Disease, biological measures	Intervention/exposure	Time between baseline exposure and outcome assessment	Dietary assessment method FFQ, food record Internal validation (y/n)	No of subjects analysed	Intervention (I) (dose interval, duration) Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change	Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up) Drop out (%)	Results (I, C) (Absolute difference, RR, OR, <i>p</i> -value, confidence interval, sensitivity, specificity, observer reliability? etc)	Confounders adjusted for	Study quality and relevance, Comments (A–C)
Bazzano, 2005, US (27)	Cohort	Male physicians, 40–84 y in 1982 <i>n</i> = 22,066. Free of CVD, DM and cancer at baseline.	Risk of overweight and wt gain.	Whole and refined grain breakfast cereal intakes.	8 and 13 y	Semiquantitative FFQ.	<i>n</i> = 17,881		8 and 13 y. Dropout <i>n</i> = 635 (illness), in addition 16.6% lack of breakfast cereal intake information.	RR: 0.78 (8 y) and 0.88 (13 y) M who never or rarely consumed breakfast cereals versus those who consumed >= 1 serving per day. Consumers of breakfast cereal consistently weighed less than those who consumed cereals less often (<i>p</i> for trend = 0.01).	Age, smoking, baseline BMI, alcohol, PA, history of hypertension, high cholesterol and use of multivitamins. Also adjustment to fruit, vegetables and dairy: no change in results.	B Semi qualitative FFQ assessed a limited number of foods. Unable to compare breakfast cereal intake to other types of breakfast foods or to skipping breakfast.
Bes-Rastrollo, 2006, Spain (28)	Cohort	University graduates, 7,194 M and W 37 (± 12) y Excl. those who reported total EI (< 800 or > 4,200 kcal/day for men and < 600 or 3,500 kcal/day for women).	Wt change (self-reported). Validated self-report 1.5% mean relative error compared to objective measurement.	Sugar-sweetened soft drinks (SSSD) or consumption of hamburger, pizza, and sausages (HPS). Analyses were also made for red	Median 28.5 months.	Semiquantitative FFQ (136 food items) Validated, see the original article for the reference.	<i>n</i> = 7,194		28.5 month follow-up with > 90% follow-up rate.	SSSD was associated with wt gain only in subgroup assessment: those who had reported a previous wt gain (> = 3 kg; during the 5 y before this study baseline). Consumption	Sex, total EI from non-SSD sources, fibre, alcohol, milk, PA, smoking, snacking, TV, and baseline wt	B Weigh self-reported. Details of dietary assessments were lacking in this report, although they have been reported earlier. The

				meat and sweetened fruit juice.					of HPS was associated with higher wt gain, independent of consumption of SSSD and of previous wt gain. Fifth compared with the first quintile: OR 1.2 (1.0–1.4; <i>p</i> for trend = 0.05) Red meat and sweetened fruit juice consumption were not significantly associated with wt gain.	comparability of this population (students from Spain) and Nordic population is not clear.	
Bes-Rastrollo, 2006, Spain (29)	Cohort	University graduates, 9,000 M and W37 (± 12) y Excl. those who reported total EI (<800 or >4,200 kcal/day for men and <600 or 3,500 kcal/day for women).	Wt gain or likelihood of becoming overweight/obese.	Olive oil consumption.	Median 28.5 months.	Semiquantitative FFQ (136 food items). Validated, see original article for the reference.	<i>n</i> = 7,368	28.5 month follow-up with >90% follow-up rate.	No significant association between baseline consumption of olive oil and subsequent wt change, nor to the risk of developing overweight and obesity.	Age, sex, total EI, vegetable consumption, PA, smoking, snacking between meals, TV viewing, and baseline BMI.	B Wt self-reported. Details of dietary assessments were lacking in this report, although they have been reported earlier. The comparability of this population (students from Spain) and Nordic population is not clear.
Bes-Rastrollo 2007, Spain (60)	Cohort	University graduates, 9,000 M and W37 (± 12) y Excl. those who reported total EI (<800 or >4,200 kcal/day for men and <600 or 3,500	An increase in body wt of at least 5 kg during follow-up. Change in body wt during follow-up. Incident	Nut consumption = walnuts, almonds, hazelnuts, and peanuts.	Median 28 months.	Semiquantitative FFQ (136 food items) Validated, see original article for the reference.	<i>n</i> = 8,865	Median 28 months., Drop-out 24.3%.	Participants who ate nuts two or more times per week had a significantly lower risk of wt gain (OR: 0.69; 95% CI: 0.53–0.90, <i>p</i> for	Sex, baseline BMI, PA, smoking, snacking, television watching.	B Wt self-reported. Details of dietary assessments were lacking in this report,

		kcal/day for women). Baseline $n = 11,714$.	overweight/ obesity						trend = 0.006) than those who never or almost never ate nuts. Participants with little nut consumption (never/almost never) gained an average of 424 grams (102,746) more than frequent nut eaters.		although they have been reported earlier. The comparability of this population (students from Spain) and Nordic population is not clear.
Bes-Rastrollo 2009, US (30)	Cohort	Nurse's health study, 116,671 W, age 36.5 (± 4.6) y Excl. at baseline (1991) if did not complete FFQ, if they reported EI (<500 or >3,500 kcal/day), history of diabetes or CVD, cancer before 1999 (post test), pregnancy at any time from baseline to post test, no PA data assessed in 1991 and 1997, only baseline data, missing wt data.	Weight gain (self-report)	Total nut consumption = sum of intakes for peanuts, including peanut butter, and other nuts.	8 y	133-item FFQ Validated, see original article for the literature reference	$n = 51,188$	8 y. Drop out 56%.	Greater nut consumption (> or = 2 times/week compared with never/almost never) was associated with a slightly lower risk of obesity (hazard ratio: 0.77; 95% CI: 0.57–1.02; p for trend = 0.003).	Age, alcohol, PA, smoking, postmenopausal hormone use, oral contraceptives, baseline BMI, GL, intakes of several dietary components at baseline.	B Wt self-reported. Details of dietary assessments were lacking in this report, although they have been reported earlier. The comparability of this population (nurses from the US) and Nordic population is not clear
Du 2009 (31)	Cohort	Five European countries (Denmark, Germany, Italy, The Netherlands and the UK; DioGenes). A total of 89,432 participants, aged 20–78 y (mean = 53 y) at baseline.	Wt and WC.	Dietary GI and GL	1.9–12.5 y (mean = 6.5 y)	Country-specific FFQs at baseline. Enzymatic-gravimetric method (AOAC) to define dietary fibre, except in UK where defined as non-starch polysaccharides using Englyst method	$n = 89,432$	Median follow-up 6.5 y (range: 1.9 to 12.5 yrs). Drop out 30.2%.	With every 10-unit higher in GI, wt increased by 34 g/y (95% CI: -47 to 115) and WC increased by 0.19 cm/y (0.11, 0.27). With every 50-unit higher in GL, wt increased by 10 g/y (-65, 85) and WC increased by 0.06 cm/y (-0.01, 0.13).	Baseline anthropometrics, demographic and lifestyle factors, follow-up duration and other dietary factors.	B Variation in methods to measure wt and WC (partly self-assessed or reported, variation between the centres), drop-out exceeding 20%.

Validated earlier for total energy, carbohydrates, dietary fibre and main carbohydrate-containing foods, reported in several earlier papers. See original article for the literature reference

Halkjær 2004, Denmark (32)	Cohort	Danish M and W, aged 30, 40, 50 or 60 y, randomly selected and representative of Copenhagen County. Attendance at baseline 3,875 (1,845 W, 1,940 M) and at follow-up 2,436 (1,200 W, 1,236 M). Median BMI at baseline 25.2 kg/m ² in M and 23.5 kg/m ² in W.	WC	Different food and beverage groups (11 groups).	6 y	26-item FFQ Validated against diet history. The results showed positive correlations.	<i>n</i> = 2,436 (1,200 W, 1,236 M).	6 y. Drop out 36%	Intake of refined bread was positively associated with change in WC, with (β = 0.29 (95% CI: 0.07–0.51) or without (β = 0.42, 95% CI: 0.11–0.73) further adjustment for BMI. Spread on bread, milk and cheese, meat, fish, potatoes, rice, pasta, wholegrain bread, fruits and vegetables, cakes and chocolate were not related to WC.	Age, other food groups than, smoking, alcohol.	B Food consumption was assessed with a very short questionnaire. Validity was only briefly described. The statistical power was not reported.
Halkjær, 2009, Denmark (33)	Cohort	All M and W (in Copenhagen and Aarhus) aged 50–64 y invited with no previous history of cancer. 35% (<i>n</i> = 57,053) of the invited participated. In addition 547 were excl. because of newly	Changes in WC.	Different food and beverage groups (21 groups)	5 y	192 semi-quantitative FFQ. Validated against two 7-day weight diet records.	<i>n</i> = 42,696 (22,570 W)	Drop out from baseline 24.5%.	The β -coefficients (95% CI) were assessed against 60 kcal of each food item. Both M and W: Red meat (inverse: W: –0.13, 95% CI: –0.24 to –0.03; M: –0.06, 95% CI: –0.11 to	Baseline WC, BMI, age, smoking, PA, alcohol, EI per day from the 21 food and beverage groups.	B Follow-up weight was self-reported. Drop-out exceeded 20%.

diagnosed cancer.
Between follow-up and
baseline 1,692 died,
435 emigrated, giving
54,379
participants for
invitation to
follow-up.

–0.003), fruit
(inverse: W:
–0.07, 95% CI:
–0.13 to 0.004; M:
–0.10, 95% CI:
–0.15 to –0.04)
and snack food
(positive: W: 0.06,
95% CI: 0.003–
0.11; M: 0.09, 95%
CI: 0.05–0.13)
significantly
associated with
WC. W only:
Inverse; vegetables
(–0.36, 95% CI:
–0.51 to –0.21),
high-fat dairy
(–0.09, 95% CI:
–0.15 to –0.03),
butter (–0.12,
95% CI: –0.20 to
–0.04). Positive;
processed meat
(0.20, 95% CI:
0.04 –0.36),
potatoes (0.10,
95% CI: 0.0006–
0.19), poultry
(0.19, 95% CI:
0.01–0.37).

Hare-Bruun, 2006, Denmark (34)	Cohort	Random sample of adults drawn in 1982. N = 3,608 (79% of sample) participated at original baseline. Follow-up 1987/1988 with a dietary survey in a subset of 552 subjects aged 49 y (baseline in this study). A follow up in 1993/ 1994. Excl. those with missing data on wt, ht, WC, HC, body fat mass or lean body mass or lean body mass or age,	Changes in body wt, body fat distribution and body composition	Baseline GI and GL (calculated with white bread as the reference food).	6 y	Diet history interview. n = 376 (185 men) Average daily intake based on intakes during the previous month. A weighed GI and overall GL were assigned to the diet with the use of values from the 2002 interna- tional table of GI and GL values and	6 y. Drop out 32%.	Positive associations between GI and changes in body wt (β -coefficient for log (body weight): 0.002, 95% CI: 0.0001–0.004), per- cent body fat and WC in W only. No associations be- tween GI for M and no for GL either sex.	Baseline body wt, age, smoking, years of education, PA, EI, E% from protein, fat and fibre intake.	B Power not reported.
---	--------	---	---	---	-----	--	-----------------------	--	--	-----------------------------

by using mean values of different studies measuring the GI of similar foods. GI was expressed with white bread as reference.

education, smoking, PA, and diabetes.

Koh-Banerjee, 2004, US (35)	Cohort	51,529 male health professionals 40–75 y at baseline in 1986. Excluded were those who died, developed CVD, cancer or diabetes before 1994, had missing data on weight measures, dietary intake, PA	Wt gain	Whole-grain, and fibre.	8 y	Semi-quantitative FFQ. Validated among a subset of participants.	$n = 27,082$	8 y. Drop-out 47.4%.	Whole-grain intake inversely associated with wt gain, with an observed dose-response relation. For every 40 g/day increment in whole-grain intake – wt gain was reduced by 0.49 kg. Changes in cereal and fruit fibre were inversely related to wt gain.	Age, respective baseline exposure, smoking, baseline wt, and baseline values and changes in refined grains, EI, PA, alcohol, protein, TFA, SFA, MUFA and PUFA.	B Changes against changes. Wt was self-reported.
Mozaffarian, 2011, USA (36)	Cohort study	Participants from Nurses' Health Study, Nurses' Health Study II and Health Professionals Follow-up Study, total $n = 120,877$. Initial BMI for NHS and NHS II was 23.7 and 23.0 kg/m ² , and for HPFS 24.7.	Wt change (mean of 4 y periods)	Change in food consumption at baseline of each 4 y period.	NHS: 20 y; NHS II: 12 y; HPFS: 20 y. Analyses were done within 4 y periods covering the above time-period.	FFQ	$n = 120,877$	NHS: 20 y; NHS II: 12 y; HPFS: 20 y. Analyses were done within 4 y periods covering the above time-period.	The average 4 y wt gains in kg, against changes in servings, were positively associated with potato chips (0.55, 95% CI: 0.59–0.95), potatoes (0.58, 95% CI: 0.39–0.77), processed meats (0.42, 95% CI: 0.36–0.49), unprocessed meat (0.43, 95% CI: 0.25–0.61), butter (0.14, 95% CI: 0.07–0.20), sweets and desserts (0.19, 95% CI: 0.07–0.30), and refined grains (0.18, 95% CI:	Age, baseline BMI, sleep duration, changes in smoking, PA, television watching and alcohol use.	B Self-reported body wt, reporting of dietary assessment tool and database inadequate.

0.10–0.26). Negative associations were found with vegetables (–0.10, 95% CI: –0.15 to –0.05), nuts –0.26, 95% CI: –0.44 to –0.08), whole grains –0.17, 95% CI: –0.22 to –0.12), fruits –0.22, 95% CI: –0.29 to –0.16) and yoghurt –0.37, 95% CI: –0.45 to –0.30). Sugar-sweetened beverages were also positively associated with weight change (0.45, 95% CI: 0.38–0.53).

Poddar, 2009, US (37)	Cohort	Freshmen-level in nutrition 2004. 362 eligible (sex NA). <i>N</i> = 76 completed data collection in 2004 and 2005. Age 19.2 (SE 0.1) y.	Body wt and composition changes	Total and low-fat dairy intake.	6 months	7-day food record.	<i>n</i> = 76 (65 W)	6 months (drop-out information not given) Drop out 79% (conservative calculation)	Total dairy intake was not associated with wt. Subjects with higher amount of low-fat dairy products gained less body wt.	Race, sex and percent intake of estimated energy requirement	C Details about the recruitment procedure is missing. The students were on average 'normal wt' (BMI 23) and had already a healthy eating habits. Adjustment for PA is not done even though they have the information. Drop-out reason not given
-----------------------	--------	---	---------------------------------	---------------------------------	----------	--------------------	----------------------	---	---	--	--

Rajpathak SN, 2006, USA (38)	Cohort study	The Health Professionals Follow-up Study ($n = 51,529$). M subjects, 40–75 y. Subjects excl. if: <20 y ($n = 52$), unreasonable EI ($n = 1,596$), cancer, CVD or diabetes at baseline ($n = 3,571$) or endpoint ($n = 11,027$), no wt data in either 1986 or 1998 ($n = 11,779$), no calcium intake data in 1998 ($n = 3,889$). BMI at baseline 25.1–25.3 kg/m ² (across quintiles).	12 y wt change (self reported).	Dairy intakes	12 y	Semiquantitative FFQ, validated against 1 week diet records ($n = 127$) (coefficients reported, $r = 0.53$ for calcium). US Department of Agriculture, supplemented with information from manufacturers. Pearson correlation between calcium intake from the FFQ and the average intake of two 1-week diet records was 0.53.	Baseline dairy and wt change ($n = 23,504$)	12 y. Drop out 17% from baseline measurements.	Small difference in mean wt gain between extreme quintiles of high-fat dairy intake (3.24 ± 0.11 for the lowest quintile compared with 2.86 ± 0.11 for the highest quintile, p for trend = 0.03).	Age, baseline wt, smoking, alcohol intake, PA, GL, EI, and variety of food and nutrients.	B Self reported weight.
Romaguera 2011, Europe (39)	Cohort	EPIC participants who were involved in DiOGenes –project, eight centres from five countries (Italy, Netherlands, Germany, Denmark, UK). Exclusion: pregnancy, chronic diseases, age > 60 at baseline, smoking status changed during follow-up. Participants: 19,694 M and 28,937 W. These were selected from 102,346 participants with results on both baseline and follow-up.	WC, adjusted to BMI by residuals.	Different food groups.	Median follow-up 5.5 y.	Country-specific FFQ.	$n = 19,694$ M and $n = 28,937$ W, total $n = 48,631$.	Median 5.5%. Drop-out 30.2%.	The results were shown as β -coefficients and 95% CI. Negative associations with annual change in WC, adjusted for BMI, were seen for vegetables (–0.08, 95% CI: (–0.11 to –0.03), fruit (–0.04, 95% CI: –0.05 to –0.03), dairy (–0.01, 95% CI: –0.02 to –0.01). Positive associations were reported for potatoes (0.04, 95% CI: 0.01–0.06), white bread	total EI, age, baseline wt, baseline ht, baseline WC(BMI), smoking, alcohol intake, PA, education, follow-up duration, menopausal status (W only), and hormone replacement therapy use (W only),	B Slight variations in the anthropometric techniques between the centres and time-points. Statistical power was not calculated, but appears to be clearly adequate.

Rosell, 2006, Sweden (41)	Cohort study	Subjects from the Swedish Mammography Chort, Västmanland and Uppsala. W born 1914–1948, recruited in 1987–1990. Original sample of 90,069. 74% had dietary info ($n=66,651$). Follow up in 1997, excl. those who moved away, resulting in $n=56,030$. Of those 38,984 completed a FFQ. For the current study subjects were excl. if data on body wt or ht were missing at baseline or follow up ($n=1,783$), had a disease ($n=8,643$) and extreme changes in BMI ($n=12$). Cohort was restricted to W aged 40–55 at baseline. BMI 23.7 kg/m ² at baseline.	Annual wt change during follow up.	Dairy food consumption.	9 y.	67-item FFQ in 1987. A 96-item FFQ was used in 1997, and the frequency of dairy products during the previous years was assessed by open ended questions requesting participants to report the number of servings per day or week. Validation against 1 week diet records ($n=129$), coefficients for dairy ranged from 0.33–0.64.	$n=19,352$	Dropout 32% from baseline measurements (based on the assumption that the eligible sample was 28,546 incl. only women 40–55 at baseline – not clear in the text).	(0.01, 95% CI: 0.01–0.02), processed meat (0.04, 95% CI: 0.02–0.06), margarine (0.03, 95% CI: 0.01–0.05), sugar and confectionary (0.01, 95% CI: 0.00–0.01) and soft-drinks (0.04, 95% CI: 0.02–0.07).	Women consuming ≥ 1 serving/day whole milk and sour milk or cheese at baseline and did not change their consumption during follow up had decreased risk of mean wt gain of ≥ 1 kg/y compared with those consuming < 1 serving/day with no change in follow up (OR 0.85; 95% CI: 0.73–0.99 and OR 0.7; 95% CI: 0.59–0.84, respectively).	Age, ht and wt at baseline, education, parity, intakes at baseline: EI, fat, CHO, protein, fibre and alcohol and the absolute change in intakes of these nutrients during follow-up, and the categories of change in intake of the other dairy products.	B EI rather low. Self reported wt at baseline and endpoint.
Rosell M, 2006, UK (40)	Cohort study	Subjects from the EPIC-Oxford ($n=65,500$). Age ≥ 20 y M and W. The aim was to recruit participants with a wide range of diets by targeting vegetarians and vegans	Annual wt gain during follow up (self reported).	Meat-eating, fish-eating, vegetarian and vegan.	Median follow-up 5.3 y (range 3.2–9.1 y).	A 130-item FFQ was also used to assess intake in the previous 12 months (validation not reported).	$n=21,966$ ($n=5,373$ M and $n=16,593$ W)	The number of subjects eligible at baseline (after excl.) not available	Mean annual wt gain (g/y) was lower in vegans (284 g, 95% CI: 178, 390 and 303 g, 95% CI: 211, 396, in M and W, respectively)	PA, smoking, marital status, current paid job, age at leaving school, age at menarche, and	B Might not be representative to the Nordic population due to high proportion of	

as well as the general UK population. The current study is based on subjects who completed follow-up questionnaire and had no prevalent malignant neoplasm at baseline ($n = 36,956$). Excl. if wt was not self reported ($n = 1,389$), missing data or reporting error ($n = 2,267$), ≥ 70 y or had suffered from heart attack, stroke, angina or diabetes at baseline ($n = 4,625$), unclear diet group at baseline ($n = 529$) or missing values ($n = 6,180$). BMI at baseline: M 24.1 kg/m², W 23.4 kg/m².

Classification of diet groups was based on four questions: Do you eat any meat? Do you eat any fish? Do you eat any eggs? and Do you eat any dairy products? In addition to the questions used to classify the participants dietary intake was assess by a 130-item FFQ. No information on the internal validity of the four questions reported.

compared with meat eaters (406, 95% CI: 373, 439 and 423, 95% CI: 403, 443, in M and W, respectively). Fish eaters (W only) had also lower annual wt gain (338 g, 95% CI: 300–376) than meat eaters.

age, ht, wt at baseline.

vegetarians and vegans in the study. Wt self-reported.

Schulz 2002, Germany (42)	Cohort study	Subjects for the analysis were selected from the EPIC cohort in Potsdam, Germany ($n = 27,548$). M 24–69 y and W 19–70 with complete data on body wt and disease status at baseline and the first follow-up examination were eligible ($n = 24,950$). Smokers excl. (and those who had quit <2 y prior to baseline), subjects using appetite-suppressing drugs and with diseases were excl., as were pregnant or lactating women. BMI at baseline: W 25.8 kg/m ² , M 27.1 kg/m ² .	Annual wt change (baseline weight measured, follow-up wt self reported). Large wt gain defined as ≥ 2 kg/y.	Food groups (intake of food from different food groups).	Mean follow-up time 2.2 y (range 0.6–5.4 y)	148-item self-administered, validated (validation not reported here) questionnaire for assessment of habitual intake at baseline. At follow up subjects were asked whether they changed their dietary habits (profoundly, partly or not) after baseline.	$n = 17,369$ ($n = 11,005$ W and $n = 6,364$ M)	Drop out 30%.	Large wt gain (≥ 2 kg/y) was predicted by consumption of sweets. For each 100 g/day increase in sweets intake, the likely hood of observing a large weight gain increased by 48% (OR 1.48; 95% CI: 1.03, 2.13). In W large wt gain was predicted by reported higher fat, sauce and meat (OR 1.75, 95% CI: 1.01–3.06; OR 2.12, 95% CI: 1.17–3.82 and OR 1.36, 95% CI: 1.04–1.79, respectively).	Age, initial body wt and ht, education, weight history (cycling, previous wt loss or gain), medication, menopausal status, life and health contentment, dietary change, PA, prevalent diabetes and thyroid disease.	B Follow up questionnaire limited to changes. Self reported wt at endpoint but measured at baseline.
---------------------------	--------------	--	--	--	---	--	--	---------------	--	---	--

Schulze 2004, USA (43)	Cohort study	Subjects from the Nurses' Health Study II ($n = 116,671$), female US Nurses aged 24–44 y at study initiation 1989. Excl. if they did not complete relevant dietary questions in 1991, had history of diabetes or CVD before 1995 or reported diagnosis of cancer, no report on body wt or had no data on PA. Baseline BMI 24.2–24.80 kg/m ² across SSSD consumption groups.	Mean wt changes from 1991 to 1995 and from 1995 to 1999 (self reported)	SSSD	4 and 8 y	133-item validated semi quantitative FFQ. Correlation coefficients between the FFQ and multiple dietary records ranged from 0.36 to 0.89. See original article for the literature reference.	$n = 51,603$ W	Drop out during follow up 66% from the original sample; drop out during follow up 44% of those eligible after excl.	W who increased their consumption of SSSD from low to high (≤ 1 /week to ≥ 1 /day) had significantly larger increases in wt (4.69 kg (SE 0.20 kg) during 1991–1995 and 4.2 kg (SE 0.22 kg) during 1995–1999, than W who maintained a low (3.21 kg SE 0.03 kg and 2.04 kg, SE 0.03 kg) or a high (3.12 kg, SE 0.13 kg and 2.21 kg, SE 0.13 kg) intake or substantially reduced their intake (1.34 kg, SE 0.07 kg and 0.15 kg, SE 0.18 kg), during the two time periods, respectively. $p < 0.00$.	Baseline age, alcohol intake, PA, smoking, postmenopausal hormone use, oral contraceptive use, total fat intake and BMI.	B Drop-out rate exceeded 20%
Vergnaud 2010, Europé (44)	Cohort study	EPIC (PANACEA), 521,448 apparently healthy volunteers, 25–70 y from 23 European centres. Individuals with missing information excl., along with subjects with extreme values on anthropometry, pregnant women and extreme EI/ER. $N = 497,735$ available for the baseline analysis. BMI at baseline: W 25.1 kg/m ² , M 26.6 kg/m ² .	5 y wt change (follow-up range 2–11 y). Measured or self reported at baseline, self reported at endpoint.	Meat consumption (red meat, processed meat and poultry).	Ranged from 2 to 11 y, adjusted to 5 y.	Country specific validated dietary questionnaires (validation not reported here). EPIC Nutrient Database. Dietary calibration study completing an additional 24-h recall (EPIC-SOFT). See original article for the literature reference.	$n = 373,803$ ($n = 103,455$ M and $n = 270,348$ W)	Drop out 25%.	A 100 kcal/day increase in meat consumption was associated with 30 g (95% CI: 24–36) annual increase in wt. Significant for all types of meat, strongest association found for poultry.	Sex, age, indicator of meat consumption, educational level, PA, smoking status, initial BMI, follow-up time, total EI, E from alcohol, and plausible total EI reporting.	B Sample not intended to be representative of each region. Mixed methods of assessing wt as well as dietary intake. Follow-up period different between centres.

Vergnaud 2012, Europe (45)	cohort study	EPIC (PANACEA), 521,448 apparently healthy volunteers, 25–70 y from 23 European centres. Individuals with missing information excl., along with subjects with extreme values on anthropometry, pregnant W and extreme EI/ER. N = 497,735 available for the baseline analysis. After the follow-up, 373,803 participants (103,455 M and 270,348 W) were included in the analyses.	5 y wt change estimated from the available data (follow-up range 2–11 y). Measured or self reported at baseline, self reported at endpoint.	Fruit and vegetable consumption	Ranged from 2 to 11 y	Country specific validated dietary questionnaire. See original article for the literature reference. EPIC Nutrient Database Biomarkers: Spearman's correlation coefficient between total plasma carotenoids and total fruit and vegetable intakes	n = 373,803 (n = 103,455 M and n = 270,348 W)	Dropout 25%.	Baseline fruit and vegetable intakes were not associated with wt change overall.	Age, vegetable (or fruit) consumption, PA, change in smoking, BMI at baseline, EI, alcohol, plausibility of total EI.	B Sample not intended to be representative of each region. Mixed methods of assessing weight as well as dietary intake. Follow-up period different between centres.
Vioque, 2008, Spain (46)	Cohort study	Random sample of 1,799 M and W ≥ 15 y from Valencia. For the follow up 407 subjects were contacted. Average BMI at baseline was 25.8 kg/m ² in both the original sample and the analysed subjects (n = 206).	Changes in body wt (measured). Main outcome defined as wt gain ≥ 3.41 kg over the 10 y follow-up period.	Fruit and vegetable intake	10 y	Semiquantitative FFQ, 10 fruit items and 12 vegetable items. Average correlation coefficients with 1-week dietary records, for 1-y validity and reproducibility of nutrient intakes were 0.47 and 0.40 respectively. See original article for the literature reference	n = 206	Drop out from the original sample 89%, but 51% if based on the eligible sample.	OR (95% CI) of ≥ 3.41 kg wt gain in 10 y was 0.21 (0.06, 0.79) in quartile 4 of fruit and vegetable intake compared with the lowest quartile (p for trend 0.024).	Sex, age, educational level, BMI, time spend watching TV, presence of disease, baseline ht, total EI, and energy-adjusted intakes of protein, SFA, MUFA, PUFA, fibre, caffeine and alcohol consumption.	C Low participation rate, inclusion criteria were not clearly reported.

W, Women; M, Men; Wt, Weight; Ht, Height; WC, Waist circumference; PA, Physical activity; BMI, Body mass index; EI, Energy intake; ED, Energy density; TFA, Trans fatty acids; CHO, Carbohydrates; MUFA, Monounsaturated fatty acids; PUFA, Poly-unsaturated fatty acids; GI, Glycemic index; GL, Glycemic load; Y, Years; SSSD, sugar-sweetened soft drink.

Appendix 5 Evidence tables

Table 3. Diets and prevention of weight gain

Reference details, First author, Year, Country	Study design (RCT, CT, cohort, case control etc.)	Population, subject characteristics, Inclusion/exclusion criteria, setting, no at baseline, male/female, age, ethnicity of the subjects, anthropometry, location	Outcome measures Disease, biological measures	Intervention/exposure	Time between baseline exposure and outcome assessment	Dietary assessment method FFQ, food record Internal validation (y/n)	No of subjects analysed	Intervention (I) (dose interval, duration) Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change	Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up) Drop out (%)	Results (I, C) (Absolute difference, RR, OR, p-value, confidence interval, sensitivity, specificity, observer reliability? etc)	Confounders adjusted for	Study quality and relevance, Comments (A-C)
Beunza 2010, Spain (47)	Cohort	University graduates Excl. those who reported total EI (<800 or >4,200 kcal/day for M and <600 or 3,500 kcal/day for W), pregnancy, CVD at baseline, no wt data. Baseline $n = 15,339$, age 38 y, BMI 24.0 kg/m ²	An increase in body wt of at least 5 kg during follow-up. Change in body wt during follow-up, Incident overweight/obesity	Mediterranean dietary Score (MDS), range 0–9: positive items: vegetables, fruit and nuts, legumes, MUFA: SFA, moderate alcohol consumption, fish; negative: meat and poultry, dairy. See original article for reference.	Mean 5.7 y (median 6.2 y)	Semi-quantitative 136-item FFQ. Validated, see original article for the literature reference	$n = 10,376$		Mean 5.7 y. Drop out (did not participate in follow-up) was 8%, but a further 24% were excl. due to missing information etc.	Participants with the lowest adherence (≤ 3 points) to MDS had the highest average yearly wt gain, whereas participants with the highest (≥ 6 points) adherence exhibited the lowest wt gain (adjusted difference: -0.059 kg/y; 95% CI: 0.008 kg/y; p for trend = 0.02).	Sex, age, baseline BMI, PA, sedentary behaviour, smoking, snacking, total EI.	B Wt self-reported. The comparability of this population (students from Spain) and Nordic population is not clear.

Quatromoni, 2006, US (48)	Cohort	The Framingham Offspring cohort, baseline at examination 3 (1984–1988) $n=3,873$ of whom 2/3 contributed dietary data, incl. those who contributed one or two 8-y follow-up periods ($n=2,245$), excl. cancer; average age, 49–56 y) ethnicity not reported. Baseline mean BMI varied from 26.9 to 27.4 in men, and from 25.1 to 25.8 in women, according to different groups of DQI.	8 y wt change, body wt measured	A five-point dietary quality index (DQI): Fat intake <30 E%, SAFA <10 E%, chol <300 mg/day, sodium <2,400 mg/day, CHO >50 E%	8 y (from examination 3 to examination 7, which took place in 1998–2001)	3-day dietary records at exam 3 (1984–1988) and exam 5 (1991–1996). Minnesota Nutrition Data System software (NDS 2.6)	$n=990$ M and $n=1,255$ W (1,847 female and 1,433 male observations, since most participant were assessed twice)	Not clearly reported, observations include same individuals twice, yet reported as numbers	Higher DQI was associated with lower wt gain over 8 y (p for trend <0.01 for M and W), higher DQI associated with less wt gain: beta for 1-unit diff in DQI –0.48 for M and 0.60 for W (Note: wt expressed as pounds).	Age, BMI, smoking cessation, alcohol, PA, intentional changes in eating behaviour, menopausal status (W).	B Drop out rates not reported, ethnicity not known
Romaguera, 2010, Europé (49)	Cohort study	EPIC (PANACEA), $n=521,448$ apparently healthy volunteers, 25–70 y from 23 European centres. Individuals with missing information excl., along with subjects with extreme values on anthropometry, pregnant women and extreme EI/ER. Thus $n=497,735$ available for the baseline analysis. Baseline BMI not reported.	5 y wt change estimated from the available data (follow-up range 2–11 y). Measured or self reported at baseline, self reported at endpoint.	Adherence to the Mediterranean diet (MED). Scores created from 0 to 18.	Ranged from 2 to 11 y.	Country specific validated dietary questionnaires (validation not reported here)	$n=373,803$ ($n=103,455$ M and $n=270,348$ W)	Dropout 25%.	Two point increase in MED predicted –0.05 kg (95% CI: –0.07 to –0.02 kg) less wt gain in 5 y. High adherence (11–18 points) –0.16 kg (–0.24, –0.07 kg) less wt gain in 5 y than people with low adherence (0–6 points). Protective effects stronger in younger and non-obese.	Sex, age, baseline BMI, follow-up time, educational level, PA, smoking, menopausal status, total EI, and misreporting of EI.	B Sample not intended to be representative of each region. Mixed methods of assessing wt as well as dietary intake. Follow-up period different between centres.

Sanchez-Villegas 2006, Spain (50)	Cohort	Participants in the SUN cohort study, the recruitment started in December 1999 (ongoing as a dynamic cohort study), for this study participants followed >2 y (<i>n</i> =7,908) included, both M and W, extremely low/high values for total EI and subjects with missing values excl.	Change in wt and BMI. Wt self-reported.	Adherence to a Mediterranean dietary pattern (MDP).	28 months	A validated semi quantitative 136-item FFQ. Food composition tables for Spain; MDP defined by scores according to the tertile distribution of several components of Mediterranean diet. For validation, see original article for the literature reference.	<i>n</i> =6,319	Drop out 20%	Lowest baseline MDP-scores showed a higher wt gain, but the inverse association did not remain significant after adjusting for confounders, higher meat consumption at baseline associated with greater wt gain (0.41 kg vs. 0.85 kg in lowest vs. highest third), higher consumption of whole-fat dairy products assoc. with lower wt gain (0.64 vs. 0.28 kg in lowest vs. highest third).	Age, sex, baseline BMI, PA during leisure time, smoking, alcohol, EI, change in dietary habits and change in PA.	B Based on self-reported weight
Zamora, 2010, USA (51)	Cohort study	Subjects from the CARDIA study (<i>n</i> =5,115), Birmingham AL; Chicago IL; Minneapolis MN; and Oakland CA. Black (<i>n</i> =2,786) and white (<i>n</i> =2,427) M (47%) and W, 18–30 y at baseline. Baseline BMI 23.7 kg/m ² (whites). Eligibility criteria, freedom from chronic disease or disability.	Wt gain, 10 kg wt gain (measured)	Diet Quality Index (DQI) as an estimate of adherence to the Dietary Guidelines for Americans. Three categories created, low, medium and high diet quality.	7 and 20 y	Interview-administered questionnaire regarding usual dietary practices and a validated quantitative diet-history questionnaire that assessed consumption of foods over the past month.	<i>n</i> =4,913 (<i>n</i> =2,427 white) at baseline and <i>n</i> =3,739 (<i>n</i> =2,014 white) at 7 y	Drop out 19% at 7 y and 28% at 20 y.	High diet quality associated with significantly less wt gain than low diet quality (11.2 vs. 13.9). Overall HR for risk of 10 kg wt gain was 0.75 (95% CI: 0.65–0.87) for high DQI compared with low DQI.	PA, EI, smoking, sociodemographic characteristics.	The number of white subjects included in the 20 y follow up is missing.

W, Women; M, Men; Wt, Weight; Ht, Height; WC, Waist circumference; PA, Physical activity; BMI, Body mass index; EI, Energy intake; ED, Energy density; TFA, Trans fatty acids; CHO, Carbohydrates; MUFA, Monounsaturated fatty acids; PUFA, Poly-unsaturated fatty acids; GI, Glycemic index; GL, Glycemic load; Y, Years.

Appendix 6 Evidence tables

Table 4. Prevention of weight regain after prior weight reduction

Reference details, First author, Year, Country	Study design (RCT, CT, cohort, case control etc.)	Population, subject characteristics, Inclusion/exclusion criteria, Setting, No at baseline, Male/Female, Age, Ethnicity of the subjects, Anthropometry, Location	Outcome measures Disease, biological measures	Intervention/exposure	Time between baseline exposure and outcome assessment	Dietary assessment method FFQ, food record Internal validation (y/n)	No of subjects analysed	Intervention (I) (dose interval, duration) Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change	Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up) Drop out (%)	Results (I, C) (Absolute difference, RR, OR, <i>p</i> -value, confidence interval, sensitivity, specificity, observer reliability? Etc.)	Confounders adjusted for	Study quality and relevance, Comments (A–C)
Brinkworth, 2004, Australia (52)	RCT	Incl: BMI 27–40 kg/m ² , Type 2 diabetes. Excl: proteinuria, liver disease, CVD, gastrointestinal disease of a malignancy. Setting: outpatients. Baseline: low-protein (LP): <i>n</i> = 31, high-protein (HP): <i>n</i> = 33 Age 62 y (SD 2 y). Caucasian. Body composition by DXA.	Wt, fat-free mass, fat mass (DXA)	HP vs. LP diet for 12 weeks +52 weeks follow-up. <i>Ad lib</i> energy intake.	12+52 weeks follow-up. Only the changes during follow-up are assessed here.	Not reported. Biomarker assay: 24 h urinary ureal creatinine	LP: <i>n</i> = 19 <i>n</i> = 7 M, <i>n</i> = 12 W); HP <i>n</i> = 19 (<i>n</i> = 8 M, <i>n</i> = 11 W).	LP-diet: 15% protein, 55% CHO, 30% fat. HP-diet: 30% protein, 40% CHO, 30% fat. The diets were supervised for 12 weeks. No measurement of dietary intake.	Follow-up: 52 weeks. Drop out 39% in LP 42% in HP.	Initial wt loss in both groups was 5.3 kg. Wt gain during follow-up: LP: 3.3 kg; HP: 1.5 kg. Difference ns (<i>p</i> > 0.05). Same result for FFM and FM.	No adjustment. ANOVA used for statistical comparison.	B Small sample size, change of outcomes were not presented, although statistically analysed, no markers of dietary exposure. Note that LP-diet was close to normal dietary recommendations. HP-diet was also a moderately low-CHO-diet.
Dale, 2009, New Zealand (53)	RCT	Incl: W who had lost >5% body wt in the previous 6 months. Excl: chronic physical or psychiatric illness (e.g. diabetes, CVD, etc.), medications which affect wt, pregnancy. <i>n</i> = 200 at baseline, age 45 y (SD 10 y). 91% white	Wt, fat-free mass, fat mass (by BIA)	2 × 2 factorial design: supporting program: intensive or nurse; diet: high-MUFA or high-CHO. <i>Ad lib</i> energy intake.	104 months (2 y).	3-day diet record.	200 (in Intension-to-treat analysis).	High-MUFA: CHO 42%, protein 21%, fat 32%; High-CHO: CHO 47%, protein 19%, fat 30%.	<i>n</i> = 174 (87%) were followed for 2 y.	Difference between the diet-groups in change from baseline to 2 y: Wt 0.7 kg (95% CI: –1.1 to 2.4), fat mass 0.4 kg (95% CI: –0.3 to 1.1).	Mixed analytical models accounting, e.g. baseline values. The models included terms, e.g. support program etc.	B Statistical power calculation not reported, however the size seemed adequate; dietary assessment database not reported.

Delbridge 2009; Australia (59)	RCT	Incl.: Age 18–75 y, BMI >30 or >27 kg/m ² + co-morbidities. Excl.: Several diseases, alcohol and drug abuse, lactation, pregnancy. <i>n</i> = 179 at baseline, <i>n</i> = 141 randomised, mean age 44 y, (SD 3 y).	Wt, waist WC, body composition (BIA)	Wt-loss diet for 3 months, followed by 12 months RCT (high-protein, HP, or high-carbohydrate, HC, diets). Aim for energy intake during weight maintenance: 1.3 × estimated resting energy expenditure	3+12 months wt-maintenance intervention. Only the changes during intervention are assessed here.	3 days food records, internal validation by 24 h urine urea excretion. Foodworks Professional Edition version 3.02.581	HP <i>n</i> = 71, HC <i>n</i> = 70	HP: Protein 30 E%, fat <30 E%, CHO >40%. HC: Protein 15%, fat <30%, CHO >55%.	<i>n</i> = 82 completed the RCT. Drop out 40%.	Wt loss during phase I (3 months) was 16.5 kg (ns between HP vs. HC). Change during RCT: HP: wt +3.0 kg, FM +4.2 kg; HC: wt +4.3 kg, FM: +3.2 kg; ns for all measured variables; results not different for completers only or by ITT analysis).	No adjustments.	A Only concern: statistical power calculation not reported, however the size seemed adequate.
Due, 2008; Denmark (54)	RCT	Incl.: 18–35 y, BMI 28–36 kg/m ² , lost wt >8% during phase I (more details in another paper). <i>n</i> = 131 randomised, age 28 y (SD 5 y).	Wt and body composition by DXA.	Wt-loss diet for 8 weeks, followed by 6 months RCT: MUFA-diet, low-fat diet (LF), or control -diet (C):. <i>Ad lib</i> energy intake.	2+6 months wt-maintenance intervention. Only the changes during intervention are assessed here.	Supermarket model: all foods were collected at a 'supermarket' established at the department. The nutrient contents were analysed from a database. Compliance assessed by fatty acid analyses, biopsy from subcutaneous adipose tissue at screening and 6 months. Biomarkers: fat biopsy (fatty acid composition)	MUFA: <i>n</i> = 52; LF: <i>n</i> = 47; C: <i>n</i> = 25	Actual E% in each diet: MUFA-diet: Fat 38%, SFA 7%, MUFA 20%, PUFA 8%, CHO 43%, protein 15%. LF-diet: Fat 24%, SFA 8%, MUFA 8%, PUFA 5%, CHO 56%, protein 16%. C-diet: Fat 32%, SFA 15%, MUFA 10%, PUFA 4%, CHO 50%, protein 16%.	<i>n</i> = 106 completed the RCT. Drop out 15%.	Wt regains: MUFA 2.5 kg, LF 2.2 kg, CON 3.8 kg (ns). Regain in FM: MUFA 2.2 kg, LF 1.3 kg, C 3.5 kg. Differences (95% CI): MUFA vs. C: 1.9 (0.1–3.7) kg, LF vs. C: 2.5 (0.7–4.4) kg, MUFA vs. LF: 0.7 (–0.9 to –2.2) kg.	No adjustments.	A
Field, 2001, USA (61)	Cohort study	Incl.: W, participant in nurses' health study; excl.: numerous criteria related to pregnancy, health status, PA etc. <i>n</i> = 47,515 at baseline (1989), age 25–43 y. Wt maintenance analyses were	Self-reported wt (validated against measured weight at baseline, <i>r</i> = 0.97).	Wt change 1989–1991, weight-loss maintenance 1991–1995	116-item FFQ, validated previously, see original article for reference.	<i>n</i> = 3,916 W who had lost wt at least 5% between 1989 and 1991.	No data	Fat E% was not associated with wt change. There was a modest positive association between protein E% and weight gain.	Age, smoking, PA, wt cycling history, EI, BMI at age 18, wt change between age 18 and y 1989, wt change between 1989 and 1991	C Dietary data were not reported in details, e.g. no indication whether the data were adjusted, only small number of the original cohort included in the analysis, dietary intake assessed only once, self-reported wt.		

		done with 3,916 women who had lost wt at least 5% between 1989 and 1991.										
Larsen, 2010, eight European countries (55)	RCT	Incl.: Families with one healthy child between 5–17 y, parent 18–65 y, BMI 27–45 kg/m ² , wt-loss >8% during phase 1. <i>n</i> = 773, age 42 y (SD 6 y), sex-distribution was not given.	Wt	Wt-reduction for 8 weeks (800 kcal/day), followed by randomisation in one of five groups: low-protein, low-GI (LP-LGI), low-protein, high GI (LP-HGI), high-protein, low-GI (HP-LGI), high-protein, high-GI (HP-HGI) and control (C).	8 weeks + 26 weeks wt-maintenance intervention (RCT)	3 days food record at screening, 4 weeks after randomisation and at the end of the intervention. Local food databases, detailed report not in this paper GI was calculated by using glucose as reference, separately from other nutrient analyses. Adherence to diet was verified by urinary nitrogen analyses.	<i>n</i> = 773	E%, GI and fibre content of the diets at week 26: <u>LP-LGI</u> : CHO 51%, fat: 30%, protein: 18%, GI 56, fibre 21 g/day. <u>LP-HGI</u> : CHO 51%, fat 31%, protein 17%, GI 62, fibre 20 g/day. <u>HP-LGI</u> : CHO 45%, fat 32%, protein 22%, GI 56, fibre 21 g/day. <u>HP-HGI</u> : CHO 45%, fat 31%, protein 23%, GI 61, fibre 19 g/day. <u>C</u> : CHO 46%, fat 34%, protein 19%, GI 59, fibre 20 g/day.	26 weeks, drop-out 29%	Intention-to-treat: Wt-regain was 0.93 kg less (05% CI: 0.31, 1.55) in groups assigned to HP (regardless of GI), and 0.95 kg less (0.33, 1.57) in groups assigned to LGI (regardless of protein). No interaction between HP and LGI.	Centre, type of centre (shop or intervention), sex, age at screening, BMI at time of randomisation, body wt lost during wt reduction, family type.	A
Phelan, 2006, USA (56)	Cohort study	Individuals registered at National Weight Control Registry (NWCR) between 1995 and 2003, they had lost >13.4 kg wt, W 78.4%, total <i>n</i> = 2,708. Mean age 46.9 y (SD 12.6 y).	Wt (self-reported)	1-y follow-up (no specification for prior wt loss, other than amount >13.4 kg).	1 y	Block Food-frequency questionnaire.	<i>n</i> = 2,266		1 y. Drop-out 16.3%.	Baseline energy intake (β = 0.10, p = 0.002), fast food consumption (β = 0.1, p = 0.0001) and exercise (β = -0.10, p = 0.02) and 1-y increase in energy intake (β = 0.05, p = 0.04), fat E% (β = 0.10, p = 0.0001) and fast food	EI, fat, CHO, protein, exercise, breakfast consumption, fast food consumption.	C Power size not calculated, although probably adequate, main part of the study concentrated on differences between different recruitment years, self-reported body wt, initially unclear inclusion criteria (who could register?), selected group (prior wt loss substantial and this was even maintained for at least 1 y)

										($\beta=0.10$, $p=0.01$) or 1-y decrease in chol E% ($\beta=-0.12$, $p=0.0001$) or exercise ($\beta=-0.12$, $p=0.0001$) were associated with weight regain.		
Swinburn, 2001, New Zealand (57)	RCT	Incl.: Adults with impaired glucose tolerance or otherwise abnormal B-glucose, but not type 2 diabetes. At baseline $n=176$ (sex-distribution not reported) and at 1 y $n=136$ ($n=101$ M, $n=35$ W; European race 97 (76%), Maori, pacific Islanders and other 24%. Mean age 52.5 y (RF) and 52.0 y (control)	Wt, BMI	1-y RCT: reduced-fat <i>ad libitum</i> (RF) versus usual diet, follow-up for 4-y.	1-y intervention +4 y follow-up (total study duration 5 y)	3-day food diary before randomisation and after 1 y. New Zealand data base (Nutritionist III software)	$n=99$ at 2-y follow-up, $n=103$ at 4-y follow-up.	RF diet at 1 y: fat 26 E%, CHO 55 E%, protein 19 E%. Usual diet at 1 y: fat 34 E%, CHO 45 E%, protein 17 E%.	Drop-out at 1 y (end of intervention), 23%, at 2-y follow-up 44% and at 4-y follow-up 42%.	2-y follow-up: RF: -1.6 (SD 0.8) kg, usual diet: $+2.1$ (SD 0.7) kg, $p < 0.01$. At 4-y follow: RF $+1.6$ (SD 0.6) kg, usual diet: $+1.3$ (SD 0.7) kg, ns.	Age, sex, ethnicity	B Note: European race only 70% and results were not presented separately for these participants.
White, 2010, UK (58)	RCT	Incl.: BMI between 25 and 35 kg/m ² , free from illness, not on a specific diet or medication affecting wt, no wt-reduction for past 3 months, intention to lose wt. $n=169$, W, age 37, SD 1.3 y, Scottish (Caucasian).	Wt, WC, body composition (BIA)	3-month intervention: G1: reduced EI, fat and sugar; G2: reduced EI and fat only; G3: control (no reduction in EI), followed by 6 months wt-maintenance follow-up.	3-month intervention and 6-month follow-up (all together 9 months)	7-day unweighed dietary record at baseline, 3 months and 9 months.	$n=126$ (drop-out 25%)	Composition for intervention diets at 3 months: G1: protein 19 E%, CHO 51 E%, fat 25 E%, sucrose 5 E% G2: Protein 18 E%, CHO 50 E%, fat 27 E%, sucrose 7 E%	6 months	Change in body wt during the 6-month follow-up: G1: -0.1 kg (SD not reported); G2: 0.0 (SD not reported). Similarly: body fat-% was unchanged during follow-up in all groups.	Not indicated	C Rather short follow-up, no power calculations, randomisation not explained, no indications of comparability of the groups, results not adjusted for EI, very low sucrose intakes, lack of clear statistics for wt change.