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

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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 increasing weight. 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

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 Ingrid Gunnarsdottir). 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) results. 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 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).

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Outcome variable</th>
<th>No. of participants</th>
<th>A</th>
<th>B</th>
<th>Strength of evidence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrates</td>
<td>Weight</td>
<td>39,275</td>
<td>2</td>
<td></td>
<td>No conclusion</td>
<td>17, 19</td>
</tr>
<tr>
<td>CHO from foods with simple sugars</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>CHO from fruit and vegetables</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>CHO from potatoes</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>CHO from refined grains</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>Fibre</td>
<td>Weight</td>
<td>270,307</td>
<td>I</td>
<td>4</td>
<td>Probable</td>
<td>14, 18, 19, 21, 26</td>
</tr>
<tr>
<td>Fruit fibre</td>
<td>WC</td>
<td>106,019</td>
<td>I</td>
<td></td>
<td>Suggestive</td>
<td>14, 20, 23</td>
</tr>
<tr>
<td>Cereal fibre</td>
<td>WC</td>
<td>116,514</td>
<td>I</td>
<td></td>
<td>Suggestive</td>
<td>14, 35</td>
</tr>
<tr>
<td>Protein</td>
<td>Weight</td>
<td>49,277</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>19</td>
</tr>
<tr>
<td>Protein</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>Fat</td>
<td>Weight</td>
<td>257,991</td>
<td>I</td>
<td>3</td>
<td>No conclusion</td>
<td>16–19, 25, 42</td>
</tr>
<tr>
<td>Fat</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>SFA</td>
<td>Weight</td>
<td>130,950</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>15, 16</td>
</tr>
<tr>
<td>SFA</td>
<td>WC</td>
<td>89,432</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>16</td>
</tr>
<tr>
<td>MUFA</td>
<td>Weight</td>
<td>130,950</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>15, 16</td>
</tr>
<tr>
<td>MUFA</td>
<td>WC</td>
<td>89,432</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>16</td>
</tr>
<tr>
<td>PUFA</td>
<td>Weight</td>
<td>130,950</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>15, 16</td>
</tr>
<tr>
<td>PUFA</td>
<td>WC</td>
<td>89,432</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>16</td>
</tr>
<tr>
<td>TFA</td>
<td>Weight</td>
<td>41,518</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>15</td>
</tr>
<tr>
<td>TFA substituted for CHO</td>
<td>WC</td>
<td>16,587</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>20</td>
</tr>
<tr>
<td>TFA substituted for PUFA</td>
<td>WC</td>
<td>16,587</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>20</td>
</tr>
<tr>
<td>Vegetable fat</td>
<td>WC</td>
<td>44,817</td>
<td>I</td>
<td></td>
<td>No conclusion</td>
<td>17</td>
</tr>
<tr>
<td>Energy density</td>
<td>Weight</td>
<td>141,220</td>
<td>I</td>
<td>2</td>
<td>No conclusion</td>
<td>12, 13, 19</td>
</tr>
<tr>
<td>Energy density</td>
<td>WC</td>
<td>138,063</td>
<td>2</td>
<td></td>
<td>Suggestive</td>
<td>13, 23</td>
</tr>
</tbody>
</table>

CHO, carbohydrates; SFA, saturated fatty acids; PUFA, polyunsaturated fatty acids; TFA, trans fatty acids; WC, 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).

Some 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 the 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-Banerhee 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).

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

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Outcome variable</th>
<th>No of participants</th>
<th>+</th>
<th>ns</th>
<th>–</th>
<th>Number of studies rated as A or B</th>
<th>Strength of evidence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>GI</td>
<td>WC</td>
<td>49,007</td>
<td>1</td>
<td>1</td>
<td>W</td>
<td>1M</td>
<td>B: 2</td>
<td>No conclusion 23, 34</td>
</tr>
<tr>
<td>GL</td>
<td>Weight</td>
<td>89,808</td>
<td>1</td>
<td>1</td>
<td></td>
<td>1M</td>
<td>B: 2</td>
<td>No conclusion 31, 34</td>
</tr>
<tr>
<td>GL</td>
<td>WC</td>
<td>49,383</td>
<td>1</td>
<td>1</td>
<td>W</td>
<td>1M</td>
<td>B: 2</td>
<td>No conclusion 23, 34</td>
</tr>
</tbody>
</table>

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.

---

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

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Outcome variable</th>
<th>No of participants</th>
<th>+</th>
<th>ns</th>
<th>–</th>
<th>Number of studies rated as A or B</th>
<th>Strength of evidence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mediterranean diet index</td>
<td>Weight</td>
<td>390,498</td>
<td>1</td>
<td>2</td>
<td></td>
<td>B: 3</td>
<td>No conclusion</td>
<td>47, 49, 50</td>
</tr>
<tr>
<td>Healthy/prudent diet index</td>
<td>Weight</td>
<td>7,158</td>
<td>2</td>
<td>1</td>
<td></td>
<td>A: 1, B: 1</td>
<td>Suggestive</td>
<td>48, 51</td>
</tr>
</tbody>
</table>
Table 4. Summary of studies on the association between weight-maintenance interventions (prevention of weight regain) and weight change (see Appendix 4).  

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Outcome variable</th>
<th>No of participants</th>
<th>Strength of evidence</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP/LC (vs. LP/HC)</td>
<td>Weight</td>
<td>120</td>
<td>A: 1, B: 1</td>
<td>52, 59</td>
</tr>
<tr>
<td>HP/LC (vs. CON)</td>
<td>Weight</td>
<td>973</td>
<td>A: 1, B: 1</td>
<td>53, 55</td>
</tr>
<tr>
<td>HF/HC (vs. CON)</td>
<td>Weight</td>
<td>77</td>
<td>A: 1</td>
<td>54</td>
</tr>
<tr>
<td>HF/LC (vs. LP/HC)</td>
<td>Weight</td>
<td>99</td>
<td>A: 1</td>
<td>54</td>
</tr>
<tr>
<td>LF/HC (vs. CON)</td>
<td>Weight</td>
<td>175</td>
<td>A: 1, B: 1</td>
<td>54, 57</td>
</tr>
<tr>
<td>Low Gl vs. high Gl</td>
<td>Weight</td>
<td>773</td>
<td>A: 1</td>
<td>55</td>
</tr>
</tbody>
</table>

H, high; L, low; P, protein; F, fat; C, carbohydrate; CON, control – according to nutrition recommendations; Gl, 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
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).

---

**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).**

<table>
<thead>
<tr>
<th>Group name</th>
<th>Exposure variables</th>
<th>Effect</th>
<th>No of studies</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibre-rich foods</td>
<td>Fibre, vegetables, fruit, fruit fibre, carbohydrates from fruit and vegetables, whole grains, whole grain bread, nuts</td>
<td>+</td>
<td>5</td>
<td>14, 17–21, 23, 26, 30, 3M, 4W, 4M</td>
</tr>
<tr>
<td>Refined grains</td>
<td>Refined grains, carbohydrates from refined grains, refined bread</td>
<td>+</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Potatoes</td>
<td>Potatoes, carbohydrates from potatoes</td>
<td>+</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Dairy</td>
<td>Dairy general, high-fat dairy, low-fat dairy, milk and cheese, yoghurt</td>
<td>+</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Meat</td>
<td>Meat general, poultry, processed meat unprocessed or red meat</td>
<td>+</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Healthy diet</td>
<td>Index of Mediterranean diet, index of healthy/prudent diet</td>
<td>+</td>
<td>1</td>
<td>5</td>
</tr>
</tbody>
</table>

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.
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 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
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Ludwig DS. Dietary glycemic index and obesity. J Nutr 2000; 130: 280S–3S.


Appendix 1

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

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
## Appendix 2

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

<table>
<thead>
<tr>
<th>References</th>
<th>Reason for exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anderson et al. (62)</td>
<td>Macronutrient data not shown</td>
</tr>
<tr>
<td>Astrup (5)</td>
<td>Review, but concentrates on weight reduction only (not on weight management)</td>
</tr>
<tr>
<td>Astrup et al. (63)</td>
<td>Concentrates on weight reduction only</td>
</tr>
<tr>
<td>Ayyad et al. (64)</td>
<td>No macronutrient data, review on weight loss mainly</td>
</tr>
<tr>
<td>Azadbakht et al. (65)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Bes-Rastrollo et al. (66)</td>
<td>Cross-sectional study</td>
</tr>
<tr>
<td>Borg et al. (67)</td>
<td>Originally included in the evaluation but excluded from quality grading; no data on food vs. weight change in a prospective design</td>
</tr>
<tr>
<td>Brown et al. (68)</td>
<td>Originally included in the evaluation but excluded from quality grading; the review concentrated on weight reduction interventions with special diets</td>
</tr>
<tr>
<td>Burke et al. (70)</td>
<td>No macronutrient data</td>
</tr>
<tr>
<td>Burke et al. (69)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Burke et al. (71)</td>
<td>Physical activity and nutrition combined, not clear maintenance phase</td>
</tr>
<tr>
<td>Cardillo et al. (72)</td>
<td>Originally included in the evaluation but excluded from quality grading; weight loss was different between the groups initially</td>
</tr>
<tr>
<td>Carels et al. (73)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Carnethon et al. (74)</td>
<td>No results on weight change, MBO as an outcome</td>
</tr>
<tr>
<td>Carty et al. (75)</td>
<td>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</td>
</tr>
<tr>
<td>Chen et al. (76)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Cheskin et al. (77)</td>
<td>Meal replacements, weight reduction only, no dietary data</td>
</tr>
<tr>
<td>Clifton et al. (78)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Davis et al. (79)</td>
<td>Meal replacements, weight reduction only, follow-up less than 6 months</td>
</tr>
<tr>
<td>Ditschuneit et al. (80)</td>
<td>Meal replacements, weight reduction only</td>
</tr>
<tr>
<td>Djuric et al. (81)</td>
<td>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</td>
</tr>
<tr>
<td>Due et al. (82)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Duffey et al. (83)</td>
<td>Only eating patterns, no macronutrient data</td>
</tr>
<tr>
<td>Eckel et al. (84)</td>
<td>No dietary data</td>
</tr>
<tr>
<td>Farshchi et al. (85)</td>
<td>Experimental study, focused on meal pattern and thermic effect of food</td>
</tr>
<tr>
<td>Flechtners-Mors et al. (86)</td>
<td>Meal replacements, weight reduction only</td>
</tr>
<tr>
<td>Forshee et al. (10)</td>
<td>Originally included in the evaluation but excluded from quality grading; review</td>
</tr>
<tr>
<td>French et al. (87)</td>
<td>Originally included in the evaluation but excluded from quality grading; study on visits to fast food restaurants and dietary, behavioural and demographic correlates</td>
</tr>
<tr>
<td>Gibson (8)</td>
<td>Originally included in the evaluation but excluded from quality grading; review</td>
</tr>
<tr>
<td>Greene et al. (88)</td>
<td>Originally was included in the evaluation but excluded from SLR: weight loss was different between the groups initially</td>
</tr>
<tr>
<td>Hensrud (89)</td>
<td>Not a systematic review</td>
</tr>
<tr>
<td>Hoy et al. (90)</td>
<td>Study on cancer patients</td>
</tr>
<tr>
<td>John et al. (91)</td>
<td>Physical activity and nutrition combined</td>
</tr>
<tr>
<td>Karnehed et al. (92)</td>
<td>Originally included in the evaluation but excluded from quality grading; dietary data were collected only at follow-up, not at baseline</td>
</tr>
</tbody>
</table>
## Appendix 2 (Continued)

<table>
<thead>
<tr>
<th>References</th>
<th>Reason for exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaukua et al. (93)</td>
<td>No dietary data</td>
</tr>
<tr>
<td>Keogh et al. (94)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Kristal et al. (95)</td>
<td>No results on weight change</td>
</tr>
<tr>
<td>Kuller et al. (96)</td>
<td>Physical activity and nutrition combined</td>
</tr>
<tr>
<td>Lantz et al. (97)</td>
<td>Weight reduction only, comparisons between VLCDs</td>
</tr>
<tr>
<td>Layman et al. (98)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Lejeune et al. (99)</td>
<td>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</td>
</tr>
<tr>
<td>Leser et al. (100)</td>
<td>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</td>
</tr>
<tr>
<td>Lindstrom et al. (101)</td>
<td>Physical activity and nutrition combined</td>
</tr>
<tr>
<td>Macdonald et al. (102)</td>
<td>Macronutrient data not shown</td>
</tr>
<tr>
<td>Malik et al. (9)</td>
<td>Originally included in the evaluation but excluded from quality grading; review</td>
</tr>
<tr>
<td>Marinilli Pinto et al. (103)</td>
<td>Study on counseling, only weight loss results</td>
</tr>
<tr>
<td>McAuley et al. (104)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Moore et al. (105)</td>
<td>Description of a study, no results included</td>
</tr>
<tr>
<td>Moran et al. (106)</td>
<td>Meal replacements, weight reduction only</td>
</tr>
<tr>
<td>Mozaffarian et al. (107)</td>
<td>No results on weight change</td>
</tr>
<tr>
<td>Ohchner et al. (108)</td>
<td>Macronutrient data not shown, mixed race</td>
</tr>
<tr>
<td>Packianathan et al. (109)</td>
<td>No macronutrient data, meal replacements, weight reduction only</td>
</tr>
<tr>
<td>Palmer et al. (110)</td>
<td>Race: African-American, weight not an outcome</td>
</tr>
<tr>
<td>Poppitt et al. (111)</td>
<td>Weight reduction only, short follow-up (6 months)</td>
</tr>
<tr>
<td>Raynor et al. (112)</td>
<td>Exercise intervention, study on weight loss, no clear data on macronutrients</td>
</tr>
<tr>
<td>Razquin et al. (113)</td>
<td>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</td>
</tr>
<tr>
<td>Redman et al. (114)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Riebe et al. (115)</td>
<td>Physical activity and nutrition combined</td>
</tr>
<tr>
<td>Sacks et al. (116)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Sarris (117)</td>
<td>No dietary intake data</td>
</tr>
<tr>
<td>Sarris et al. (118)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Sasaki et al. (119)</td>
<td>No results on weight change</td>
</tr>
<tr>
<td>Schoellcr et al. (120)</td>
<td>Study on CLA treatment, no diet, weight reduction only</td>
</tr>
<tr>
<td>Sichieri et al. (121)</td>
<td>Originally included in the evaluation but excluded from quality grading; this is a weight reduction study</td>
</tr>
<tr>
<td>Sinkin-Silverman et al. (122)</td>
<td>Physical activity and nutrition combined</td>
</tr>
<tr>
<td>Sloth et al. (123)</td>
<td>Originally included in the evaluation but excluded from quality grading; same database as in Due et al. (82) but fewer cases</td>
</tr>
<tr>
<td>Steptoe et al. (124)</td>
<td>No results on weight change, multiple interventions</td>
</tr>
<tr>
<td>Stookey et al. (125)</td>
<td>Race: only Asian (Chinese)</td>
</tr>
<tr>
<td>Stote et al. (126)</td>
<td>No macronutrient data, study on meal frequency</td>
</tr>
<tr>
<td>Svekey et al. (127)</td>
<td>No macronutrient data, mixed race</td>
</tr>
<tr>
<td>Thorpe et al. (128)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>Turk et al. (129)</td>
<td>Originally included in the evaluation but excluded from quality grading; review</td>
</tr>
<tr>
<td>Turner-McGrievy et al. (130)</td>
<td>Weight reduction only</td>
</tr>
<tr>
<td>van de Vijver et al. (131)</td>
<td>Cross-sectional design</td>
</tr>
<tr>
<td>Vang et al. (132)</td>
<td>No results on weight change, no macronutrient data</td>
</tr>
<tr>
<td>Wang et al. (133)</td>
<td>Data on alcohol consumption only</td>
</tr>
<tr>
<td>Whigham et al. (134)</td>
<td>Study on CLA treatment, no diet, weight reduction only</td>
</tr>
<tr>
<td>Woo et al. (135)</td>
<td>Race: only Asian (Chinese)</td>
</tr>
</tbody>
</table>
## Appendix 3
### Evidence tables

**Table 1. Macronutrients and prevention of weight gain**

<table>
<thead>
<tr>
<th>Reference details, First author, Year, Country</th>
<th>Population, subject characteristics</th>
<th>Study design (RCT, CT, cohort, case control etc.)</th>
<th>Outcome measures (Disease, biological measures)</th>
<th>Intervention/ exposure</th>
<th>Time between baseline exposure and outcome assessment</th>
<th>Dietary assessment method (FFQ, food record)</th>
<th>Internal validation (y/n)</th>
<th>No of subjects analysed</th>
<th>Intervention (I) (dose interval, duration), Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change</th>
<th>Follow-up period, drop-out rate (from baseline to follow-up) or from end of intervention to follow-up (Drop out (%))</th>
<th>Results (I, C)</th>
<th>Confounders adjusted for</th>
<th>Study quality and relevance, Comments (A-C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bes-Rastrollo, 2008, US (12)</td>
<td>Nurses’ health study, 116,671 women, age 36.5 (4.6) y</td>
<td>Cohort</td>
<td>No</td>
<td>Wt gain (self-report)</td>
<td>Change in dietary ED (defined as the amount of energy in a given weight of food)</td>
<td>8 y</td>
<td>133-item FFQ</td>
<td>n = 50,026</td>
<td>8 y. Dropout 57%</td>
<td>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 (p for trend &lt;0.001)</td>
<td>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</td>
<td>B</td>
<td>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.</td>
</tr>
</tbody>
</table>
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, presence of chronic diseases; baseline BMI 25.5-26.7 kg/m² for M and 24.4-25.8 kg/m² for W, WC 90-95 cm for men and 77-86 cm for women.

ED was calculated as EI from food divided by the weights of these foods. Drinks (water, alcohol, milk) not included.

Dietary ED 6.5 (1.9-12.5) y Country-specific FFQs, 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.

Change in wt and WC. Measured at baseline and two centres also at follow-up. Otherwise self report.

Dietary 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.

10 g fibre intake associated with -0.08 cm/year WC change (95% CI: -0.11, -0.05 cm) change in WC/y. 10 g cereal fibre associated with -0.10 cm (95% CI: -0.18, 0.02 cm) change in WC/y. 10 g fruit fibre associated with -0.10 cm (95% CI: -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 "Large multi-centre study with large variation in results between centres which are difficult to adjust for even though advanced statistical techniques are used. Variations due to non-stationary or follow-up time, EI/BMR in the top or bottom 1% of EPIC population, unrealistic anthropometric measures, presence of chronic diseases; baseline BMI 25.5-26.7 kg/m² for M and 24.4-25.8 kg/m² for W, WC 90-95 cm for men and 77-86 cm for women."
<table>
<thead>
<tr>
<th>Field 2007, US (15)</th>
<th>Cohort Registered nurses, W aged 41-68 y at baseline (1988), n = 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/m². Wt change, BMI in 1994; self-reported wt. Baseline fat intake (%), average intake and 8 y change in intake animal/vegetable fat = PUFA, SFA, trans fats.</th>
<th>Change in WC 8 y 136-item FFQ n = 41,518 For validation reference, see the original article. 16 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 B = 0.11 (p &lt; 0.0001), PUFA 0.42, SFA 0.40 and TFA 0.54. Baseline BMI, age, PA, time spent sitting, smoking, menopausal status and protein%.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forouhi, 2009, UK (total five countries) (16)</td>
<td>Cohort EPIC (see Du 2010), n = 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/m² for M and 25.3 kg/m² 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</td>
<td>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 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. 3.7-10.0 y. 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 BMI, age, PA, time spent sitting, smoking, menopausal status and protein%.</td>
</tr>
<tr>
<td>Halkjaer 2006 (17)</td>
<td>Cohort 50- to 64-y-old M and W living in greater Copenhagen or Aarhus area, random sample. Exclusion: cancer. Baseline n = 54,379, WC 80.0 cm for M and 95.0 cm for W, BMI 24.7 kg/m² for W and 26.1 kg/m² for M Total EI, EI from macronutrients, EI from macronutrient subgroups based on different food sources.</td>
<td>Change in WC 5 y 192-item FFQ n = 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. Baseline BMI, age, PA, time spent sitting, smoking, menopausal status and protein%.</td>
</tr>
</tbody>
</table>
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) n/C56,139 that provided consent and met/E% of fat criterion, of that 7,304 were excluded (e.g. nutritionist judgement, medical condition, eating out), n/C48,835 that were randomised to intervention (n/C19,541) and control (n/C29,294), 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/m² 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 every 3 y. n/C14,246 for I and n/C22,083 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.

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, p<0.01) maintained through year 9; W with the greatest reduction in fat intake had the largest wt loss (p for trend B0.001 both for I and C). Age, race, BMI at baseline, change in dietary intake and PA patterns; secondary analyses adjusted for EI.

A Iqbal 2006, Denmark (19) Cohort Danish census living in WC change in Dietary Y n/C862 M and n/C900 W. Weighed 7-day n/C57/44.7 E% at follow-up. Dietary components, EI in particular. 5 y Weighed 7-day n/C82.9 M and n/C50.0 W. Food record at baseline. No dietary record at follow-up. Only participation rate reported; we change in EI. Wt change; ht and wt measured at baseline and follow-up.

A Mikael Fogelholm et al. 2010, Denmark (20) Cohort Danish citizens living in WC change in Dietary Y n/C20,25 M and W aged 30, 40, 50 and 60 y at baseline, exclusion rate 21%. Only participation rate reported; we change in EI. Wt change; ht and wt measured at baseline and follow-up.

ED not associated with Wt change for either sex; in W, protein intake (E%) positively (B/C3.87, SE 1.91, p<0.04) and fibre intake (g) inversely (B/C22.8, SE 10.6, p<0.03) associated with Wt change in crude but not in adjusted (p<0.06/0.10) analyses.
information or extreme values, baseline BMI 25.1 kg/m² for M and 23.4 kg/m² for W. weight of CHO + prot + fat + alcohol + fibre + ash + water (g)

Koh-Banerjee, Cohort 2003, US (20)

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/m² (varied across age groups).

Changes 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

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.

n = 16,587 M

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, Cohort 2003, US (21)

Nurses’ health study, female nurses (n = 81,757) aged 38–63 y were followed from 1984 to 1996, exclusion because of diabetes, CVs or cancers, final baseline population 74,091, baseline BMI 24.5–24.9 kg/m² (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.

n = 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 (p 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, p < 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
Mosca, 2004, Cohort US (22)

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/m² for M and 24.3 kg/m² for W.

Wt change, measured wt and ht 11.2 y 24-h recall Nutrition Coordinating Center’s nutrient database at University of Minnesota, version 14 (1987)

Energy from fat (E%) 11.2 y 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).

Romaguera, 2010, Cohort Europe (23)

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. 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

Median follow-up 5.5 y. Drop-out 30.2% from baseline. I 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

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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 for mothers because of mixing data on wt. Final sample (n = 186), age range 14-48 y BMI 24.9 kg/m².

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 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.

Initial BMI, dietary fibre intake, caloric beverage intake not assessed at all, analyses not adjusted for age, total EI, fat E% and CHO E% varied across ED groups.


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.

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 and Drop-out rate 12%.

Increases in E% from fat associated with increases in body wt (coefficient 0.068, SE 0.034 in M, p = 0.045; coeff. 0.028, SE 0.014 in W, p = 0.042 in W); no sign differences in mean changes in dietary intake across wt change status (loser, gainer, maintainer).
Cohort Participants recruited via newspaper adds, flyers and company mass e-mail in two metropolitan areas in the US, eligibility tested by telephone interview (free from serious disease, non-smokers, premenopausal, not pregnant), at baseline n = 275 W, mean age 40.1 y, baseline BMI 24.0 kg/m².

Wt and body fat; measured wt at baseline and follow-up, body fat% measured by the BodPod.

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.

For each 1 g increase in fibre intake wt decreased by 0.25 kg (p < 0.006) and fat decreased by 0.25% (p < 0.005). Baseline fibre intake was not associated with wt change.

Age, season of assessment, baseline body fat, baseline and changes in fat intake, EI and PA.
### Evidence tables

#### Table 2. Foods and prevention of weight gain

| 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 | Study | Comments |
|-------|------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------|------------------------|------------------------------------------------|---------------------------------------------------------------|-----------------------------------------------|----------------------------------------------------------------------------------|------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------|-------------------------------------------------|
| Bazzano, 2005, Cohort | Male physicians, 40-84 y in 1982 n = 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. | | n = 17,881 | 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 (p for trend = 0.01). | Dropout n = 635 (illness), in addition 16.6% lack of breakfast cereal intake information. | 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 | B Semi quantitative FFQ assessed a limited number of foods. Unable to compare breakfast cereal intake to other types of breakfast foods or to skipping breakfast. | B | |
| Bes-Rastrollo, 2006, Spain (28) | 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 sausage (HPS). Analyses were also made for red | Median 28.5 months. | Semiquantitative FFQ (136 food items) Validated, see the original article for the reference. | | n = 7,194 | 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 (p for trend = 0.01). | 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 | B Weigh self-reported. Details of dietary assessments were lacking in this report, although they have been reported earlier. | B |
An increase in Nut consumption months.

Seems to be a difference in the consumption of nuts.

Participants who ate nuts two or more times per week had a significantly lower risk of weight gain (OR: 0.69; 95% CI: 0.53-0.90, p for trend = 0.05).

Red meat and sweetened fruit juice consumption were not significantly associated with weight gain.

Comparability of this population (students from Spain) and Nordic population is not clear.

B. Bes-Rastrollo, Cohort University graduates, 9,000 M (9-12) y

No significant association between baseline consumption of olive oil and subsequent weight change, nor between baseline PA, smoking, and subsequent weight change.

Participants who ate nuts two or more times per week had a significantly lower risk of weight gain (OR: 0.69; 95% CI: 0.53-0.90, p for trend = 0.05).

Comparability of this population (students from Spain) and Nordic population is not clear.
kcal/day for women). Overweight/obesity

Baseline $n = 11,714$

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.

Besarritzlo 2009, US (30) 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.

133-item FFQ Validated, see original article for the literature reference $n = 51,188$

8 y. Dropout 56%.

Greater nut consumption ($> 2$ 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).

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 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

$1.9 - 12.5$ y (mean = 6.5 y) $n = 89,432$

Median follow-up: 6.5 y (range: 1.9 to 12.5 yrs). Dropout 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 factors, 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, dropout 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/m² in M and 23.5 kg/m² in W. | Different food and beverage groups (11 groups). | 6 y | 26-item FFQ Validated against diet history. The results showed positive correlations. | n = 2,436 (1,200 W, 1,236 M). | 6 y | Drop out 36% |
|---|---|---|---|---|---|---|

**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% (n = 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. | n = 42,696 (22,570 W) | Drop out from baseline 24.5% |
|---|---|---|---|---|---|---|

**Age, other food groups than, B Food consumption was assessed with a very short questionnaire. Validity was only briefly described. The statistical power was not reported.**
diagnosed cancer.
Between follow-up and baseline 1,692 died, 435 emigrated, giving 54,379 participants for invitation to follow-up.

Hare-Bruun, Cohort
2006, Denmark (34)

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 352 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).

Diet history interview. 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 international table of GI and GL values and

6 y
Drop out 32%. n = 376 (185 men)

Positive associations between GI and changes in body wt (β-coefficient for log (body weight): 0.002, 95% CI: 0.0001–0.004), percent body fat and WC in W only. No associations between 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.

6 y
Power not reported.
by using mean values of different studies measuring the GI of similar foods. GI was expressed with white bread as reference.

Koh-Banerjee, 2004, USA (35)

Cohort study
Participants from Nurses’ Health Study and Health Professionals Follow-up Study at baseline in 1986, and for NHS and NHS II, n = 120,877. Initial BMI was 21.7 and 21.0 m².

Excluded were those who died, developed CVD, cancer or diabetes before 1994, had missing data on weight measures, dietary intake, PA and diabetes.

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.

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/m², 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.

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: 0.16–0.19).

Age, baseline BMI, sleep duration, changes in smoking, PA, television watching and alcohol use.

Self-reported body wt, reporting of dietary assessment tool and database inadequate.

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(32)
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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), and 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 to 0.53).

Poddar, 2009, US (37) Cohort. Freshmen-level in nutrition class in 2004. 362 eligible (sex NA). N = 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. n = 76 (65 W) (drop-out information not given) 6 months (drop-out reason not given). Total dairy intake was not associated with wt. Subjects with higher amount of low-fat dairy gained less body wt. Race, sex and percent intake of estimated energy requirement. Details about 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/m² (across quintiles).

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.

### Dairy intakes 12 y change (self reported).

<table>
<thead>
<tr>
<th>Foods</th>
<th>Baseline dairy and wt change (n = 23,504)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12 y. Drop out 17% from baseline measurements.</td>
</tr>
<tr>
<td></td>
<td>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).</td>
</tr>
<tr>
<td></td>
<td>Median 5.5%. Drop-out 30.2%.</td>
</tr>
<tr>
<td></td>
<td>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, smoking, alcohol intake, PA, GL, EI, and variety of food and nutrients.</td>
</tr>
<tr>
<td></td>
<td>B Slight variations in the anthropometric techniques between the centres and time-points. Statistical power was not calculated, but appears to be clearly adequate.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Foods</th>
<th>WC, adjusted to BMI by residuals.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Different food groups.</td>
<td>Median follow-up 5.5 y.</td>
</tr>
</tbody>
</table>

Pearson correlation between calcium intake from the FFQ and the average intake of two 1-week diet records was 0.53.
Rosell, 2006, Sweden (41) Cohort study
Subjects from the Mammography Chort, Vastmanland and Uppsala. W born 1914-1948, recruited in 1987-1990. Original sample of 90,669. 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/m² at baseline.

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 change during follow up.

Mean annual wt gain during follow up (self reported).

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

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).

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 studied categories of change in intake of the other dairy products.

EI rather low. Self reported wt at baseline and endpoint.

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.

Annual wt change during follow up.
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.

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 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.

Drop out 30%. Large wt gain (≥ 2 kg/y) was predicted by consumption of sweets. For each 100 g/day increment 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 at baseline. Vegetarians and vegans in the study. Wt self-reported.
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 in 1989. Excl. if they did not complete relevant dietary questions in 1991, had history of diabetes or CVD before 1995, were diagnosed with cancer, no data on body wt or had no data on PA. Baseline BMI 24.2 ± 2.4 kg/m² across SSSD consumption groups.

Mean wt changes from 1991 to 1995 and from 1995 to 1999 (self-reported) were assessed with a 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; 44% of those eligible after exclusion of 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 0.07 kg) during the two time periods, respectively.

Baseline age, alcohol intake, PA, smoking, postmenopausal hormone use, oral contraceptive use, total fat intake and BMI.

Drop-out rate exceeded 20%.

Vergnaud 2010, Europe (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/m², M 26.6 kg/m².

5 y wt change

Ranged from 25 y, 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, and for the region with the highest meat consumption. 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.

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.

Determinants of weight change in adult populations

Citation: Food & Nutrition Research 2012, 56: 19103 - http://dx.doi.org/10.3402/fnr.v56i0.19103
Vergnaud 2012, 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 consumption, education, PA, change in smoking, BMI at baseline, follow-up time, 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, 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/m² 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).

C Low participation rate, inclusion criteria were not clearly reported.

Sex, age, educational level, BMI, time spent watching TV, presence of disease, baseline ht, total EI, and energy-adjusted intakes of protein, SPA, MUFA, PUFA, fibre, caffeine and alcohol consumption.
### Appendix 5

**Evidence tables**

**Table 3. Diets and prevention of weight gain**

<table>
<thead>
<tr>
<th>Reference details, First author, Year, Country</th>
<th>Study design (RCT, CT, cohort, etc.)</th>
<th>Population, subject characteristics, Inclusion/exclusion criteria, setting, no at baseline, male/female, age, ethnicity of the subjects, anthropometry, location</th>
<th>Outcome measures</th>
<th>Intervention/ exposure</th>
<th>Time between baseline exposure and outcome assessment</th>
<th>Dietary assessment method FFQ, food record Internal validation (y/n)</th>
<th>No of subjects analysed</th>
<th>Intervention (I) (dose interval, duration) Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change</th>
<th>Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up)</th>
<th>Drop out (%)</th>
<th>Results (I, C) (Absolute difference, RR, OR, p-value, confidence interval, sensitivity, specificity, observer reliability/ etc)</th>
<th>Confounders adjusted for</th>
<th>Study quality and relevance, Comments (A–C)</th>
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<tr>
<td>Beunza 2010, Spain (47)</td>
<td>Cohort</td>
<td>University graduates Excl. those who reported total EI (&lt;800 or &gt;4200 kcal/day for M and &lt;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/m²</td>
<td>An increase in body wt of at least 5 kg during follow-up. Change in body wt during follow-up, Incident overweight/obesity</td>
<td>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.</td>
<td>Mean 5.7 y (median 6.2 y)</td>
<td>Semiquantitative 136-item FFQ. Validated, see original article for the literature reference</td>
<td>n = 10,376</td>
<td>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.</td>
<td>Participants with the lowest adherence (&lt;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).</td>
<td>Sex, age, baseline BMI, PA, sedentary behaviour, smoking, snacking, total EI.</td>
<td>B</td>
<td>Wt self-reported. The comparability of this population (students from Spain) and Nordic population is not clear.</td>
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<td>Quatromoni, Cohort</td>
<td>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.</td>
<td>8 y wt change, body wt measured</td>
<td>A five-point dietary quality index (DQI): Fat intake &lt; 30 E%, SAFA &lt; 10 E%, chol &lt; 300 mg/day, sodium &lt; 2,400 mg/day, CHO &gt; 50 E%</td>
<td>8 y (from examination 3 to examination 7, which took place in 1998–2001)</td>
<td>3-day dietary records at exam 3 (1984–1988) and exam 5 (1991–1996). Minnesota Nutrition Data System software (NDS 2.6)</td>
<td>n = 990 M and n = 1,255 W (1,847 female and 1,433 male observations, since most participant were assessed twice)</td>
<td>Not clearly reported, observation s include same individuals twice, yet reported as numbers</td>
<td>Higher DQI was associated with lower wt gain over 8 y (p for trend &lt; 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).</td>
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<td>Romaguera, Cohort study</td>
<td>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 El/ER. Thus n = 497,735 available for the baseline analysis. Baseline BMI not reported.</td>
<td>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.</td>
<td>Adherence to the Mediterranean diet (MED). Scores created from 0 to 18.</td>
<td>Ranged from 2 to 11 y.</td>
<td>Country specific validated dietary questionnaires (validation not reported here)</td>
<td>n = 373,803 (n = 103,455 M and n = 270,348 W)</td>
<td>Dropout 25%.</td>
<td>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.</td>
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*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.*
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 ($n = 7,908$) included, both M and W, extremely low/high values for total EI and subjects with missing values excl.

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.

$\text{n} = 6,319$

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).

Based on self-reported weight

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.

$\text{n} = 4,913$

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 (black and white) 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.
### Table 4. Prevention of weight regain after prior weight reduction

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study design</th>
<th>Population, subject characteristics, Inclusion/exclusion criteria, Setting, No at baseline, Male/Female, Age, Ethnicity of the subjects, Anthropometry, Location</th>
<th>Outcome measures</th>
<th>Intervention/exposure</th>
<th>Time between baseline exposure and outcome assessment</th>
<th>Dietary assessment method FFQ, food record Internal validation (y/n)</th>
<th>No of subjects analysed</th>
<th>Intervention (I) (dose interval, duration)</th>
<th>Control (C) (active, placebo, usual care etc), compliance, achieved dietary change, adherence to dietary targets, actual dietary change</th>
<th>Follow-up period, drop-out rate (from baseline to follow-up, or from end of intervention to follow-up)</th>
<th>Drop out (%)</th>
<th>Results (I, C) (Absolute difference, RR, OR, p-value, confidence interval, sensitivity, specificity, observer reliability? Etc.)</th>
<th>Confounders adjusted for</th>
<th>Study quality and relevance, Comments (A–C)</th>
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<tr>
<td>Brinkworth, 2004, Australia (52)</td>
<td>RCT</td>
<td>Incl: BMI 27–40 kg/m², Type 2 diabetes, Excl: proteinuria, liver disease, CVD, gastrointestinal disease of a malignancy. Setting: outpatients. Baseline: low-protein (LP): n = 31, high-protein (HP): n = 33 Age 62 y (SD 2 y). Caucasian. Body composition by DXA.</td>
<td>Wt, fat-free mass, fat mass (DXA)</td>
<td>HP vs. LP diet for 12 weeks + 52 weeks follow-up. Only the changes during follow-up are assessed here.</td>
<td>12 + 52 weeks follow-up. Not reported. Biomarker assay: 24 h urinary urea/creatinine</td>
<td>LP: n = 19 n = 7 M, n = 12 W; HP n = 19 (n = 8 M, n = 11 W).</td>
<td>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.</td>
<td>Follow-up: 52 weeks. Drop out 39% in LP 42% in HP.</td>
<td>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 (p &gt; 0.05). Same result for FFM and FM.</td>
<td>No adjustment. ANOVA used for statistical comparison.</td>
<td>Statistical power calculation not reported, however the size seemed adequate; dietary assessment database not reported.</td>
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<td>Dale, 2009, New Zealand (53)</td>
<td>RCT</td>
<td>Incl: W who had lost &gt;5% body wt in the previous 6 months. Excl: chronic physical or psychiatric illness (e.g., diabetes, CVD, etc.), medications which affect wt, pregnancy. n = 200 at baseline, age 45 y (SD 10 y). 91% white</td>
<td>Wt, fat-free mass, fat mass (by BIA)</td>
<td>2 × 2 factorial design: supporting program: intensive or nurse; diet: high-MUFA or high-CHO. Ad lib energy intake.</td>
<td>104 months (2 y).</td>
<td>3-day diet record.</td>
<td>200 (in Intention-to-treat analysis).</td>
<td>High-MUFA: CHO 42%, protein 21%, fat 32%; High-CHO: CHO 47%, protein 19%, fat 30%.</td>
<td>n = 174 (87%) were followed for 2 y.</td>
<td>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).</td>
<td>Mixed analytical models accounting, e.g. baseline values. The models included terms, e.g. support program etc.</td>
<td>Statistical power calculation not reported, however the size seemed adequate; dietary assessment database not reported.</td>
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<td>Reference</td>
<td>Year</td>
<td>Country</td>
<td>Design</td>
<td>Inclusion criteria</td>
<td>Exclusion criteria</td>
<td>Number at baseline</td>
<td>Number randomised</td>
<td>Mean age (SD)</td>
<td>Wt and body composition methods</td>
<td>Wt change phase I</td>
<td>Wt change RCT</td>
<td>Statistical power calculation</td>
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<td>Delbridge, 2009</td>
<td>Australia (59)</td>
<td>RCT</td>
<td>Incl.: Age 18-75 y, BMI &gt; 30 or &gt; 27 kg/m² + co-morbidities.</td>
<td>Excl.: Several diseases, alcohol and drug abuse, lactation, pregnancy.</td>
<td>n = 179 at baseline, n = 141 randomised, mean age 44 y (SD 3 y).</td>
<td>Wt, waist, WC, body composition (BIA)</td>
<td>Wt-loss diet for 3 months, followed by 12 months RCT</td>
<td>3 + 12 months wt-maintenance intervention.</td>
<td>3 days food records, internal validation by 24 h urine urea excretion.</td>
<td>HP n = 71, HC n = 70</td>
<td>HP: Protein 30 E%, fat &lt; 30 E%, CHO &gt; 40%, HC: Protein 15%, fat &lt; 30%, CHO &gt; 55%.</td>
<td>No adjustments. A</td>
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<td>Due, 2008</td>
<td>Denmark (54)</td>
<td>RCT</td>
<td>Incl.: 18-35 y, BMI 28-36 kg/m², lost wt &gt; 8% during phase 1</td>
<td>Excl.: Numerous criteria related to pregnancy, health status, PA etc.</td>
<td>n = 131 randomised, age 28 y (SD 5 y).</td>
<td>Wt and body composition by DXA.</td>
<td>Wt-loss diet for 8 weeks, followed by 6 months RCT MUFA-diet, low-fat diet (LF), or control diet (C).</td>
<td>2 + 6 months wt-maintenance intervention.</td>
<td>Supermarket model: all foods were collected at ‘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).</td>
<td>MUFA: n = 52; LF: n = 47; C: n = 25</td>
<td>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%.</td>
<td>No adjustments. A</td>
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<td>Field, 2001</td>
<td>USA (61)</td>
<td>Cohort study</td>
<td>Incl.: W, participant in nurses’ health study; excl: numerous criteria related to pregnancy, health status, PA etc. n = 47,515 at baseline (1989), age 25-43 y. Wt maintenance analyses were</td>
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<td>Wt change 1989-1991, weight-loss maintenance 1991-1995</td>
<td>Wt change 1989-1991, weight-loss maintenance 1991-1995</td>
<td>116-item FFQ, validated previously, see original article for reference.</td>
<td>n = 3,916</td>
<td>Wt who had lost wt at least 5% between 1989 and 1991.</td>
<td>No data</td>
<td>Fat E% was not associated with wt change. There was a modest positive association between protein E% and weight gain.</td>
<td>No data</td>
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Cohort study 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/m², wt-loss >8% during phase 1. n = 773, age 42 y (SD 6 y), sex-distribution was not given.

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.

Intention-to-treat: Wt-regain was 0.93 kg less (0.5% 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.

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, W78.4%, total n = 2,266. 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).

Block Food-frequency questionnaire.

Baseline energy intake (β = 0.10, p = 0.002), fast food consumption (β = 0.1, p = 0.001) 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.

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)
### 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 year $n = 136$ ($n = 101$ 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 ad libitum (RF) versus usual diet, follow-up for 4-y.
  - 3-day food diary before randomisation and after 1 y. New Zealand database (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%.

#### Results:
- 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.

### 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 weight 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%.

#### Results:
- 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.

### Notes:
- European race only 70% and results were not presented separately for these participants.
- 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.