REVIEW

Food Groups and Risk of Overweight, Obesity, and Weight Gain: A Systematic Review and Dose-Response Meta-Analysis of Prospective Studies

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ABSTRACT

This meta-analysis summarizes the evidence of a prospective association between the intake of foods [whole grains, refined grains, vegetables, fruit, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and sugar-sweetened beverages (SSBs)] and risk of general overweight/obesity, abdominal obesity, and weight gain. PubMed and Web of Science were searched for prospective observational studies until August 2018. Summary RRs and 95% CIs were estimated from 43 reports for the highest compared with the lowest intake categories, as well as for linear and nonlinear relations focusing on each outcome separately: overweight/obesity, abdominal obesity, and weight gain. The quality of evidence was evaluated with use of the NutriGrade tool. In the dose-response meta-analysis, inverse associations were found for whole-grain ($RR_{overweight/obesity}$: 0.93; 95% CI: 0.89, 0.96), fruit ($RR_{overweight/obesity}$: 0.93; 95% CI: 0.86, 1.00; $RR_{weight\ gain}$: 0.91; 95% CI: 0.86, 0.97), nut ($RR_{abdominal\ obesity}$: 0.42; 95% CI: 0.31, 0.57), legume ($RR_{overweight/obesity}$: 0.88; 95% CI: 0.84, 0.93), and fish ($RR_{abdominal\ obesity}$: 0.83; 95% CI: 0.71, 0.97) consumption and positive associations were found for refined grains ($RR_{overweight/obesity}$: 1.05; 95% CI: 1.00, 1.10), red meat ($RR_{abdominal\ obesity}$: 1.10; 95% CI: 1.04, 1.16; $RR_{weight\ gain}$: 1.14; 95% CI: 1.03, 1.26), and SSBs ($RR_{overweight/obesity}$: 1.05; 95% CI: 1.00, 1.11; $RR_{abdominal\ obesity}$: 1.12; 95% CI: 1.04, 1.20). The dose-response meta-analytical findings provided very low to low quality of evidence that certain food groups have an impact on different measurements of adiposity risk. To improve the quality of evidence, better-designed observational studies, inclusion of intervention trials, and use of novel statistical methods (e.g., substitution analyses or network meta-analyses) are needed. *Adv Nutr* 2019;10:205–218.

Keywords: food groups, diet, meta-analysis, dose-response, adiposity, weight gain

Introduction

Worldwide, overweight and obesity have reached epidemic proportions. In 2016, the WHO reported that >1.9 billion adults were overweight, of whom >650 million were patients with obesity (1). The prevalence has more than doubled in recent decades, and a further increase is projected (2). Overweight and obesity are associated with many chronic diseases (3). Thus, overweight and obesity belong to the leading causes of death (4). For adults, BMI is commonly used for the definition of general overweight [BMI (kg/m^2) ≥25 and <30] and obesity (BMI ≥30), whereas abdominal obesity, an independent predictor of health risk, can be defined by elevated waist circumference (5). The major cause of overweight/obesity is a long-term imbalance between energy intake and energy expenditure, which leads to weight gain (6, 7). Such an imbalance can be counteracted by a diet characterized by a low intake of high-energy-dense foods [e.g., sugar-sweetened beverages (SSBs), processed foods] and a high intake of low-energy-dense foods (e.g. fruit, vegetables, and whole-grain products) (8). However, a clear public health plan has not been established. Although a large body of research is available regarding effects seen between dietary intake and weight reduction in patients with obesity from short-term intervention studies, less is known about the role of specific food groups and their optimal intakes regarding the prevention of becoming obese. Meta-analyses have summarized findings from short-term randomized controlled trials (RCTs) regarding whole grains, vegetables and fruit, nuts, legumes, dairy products, and SSBs on measurements of adiposity (9–17); and other meta-analyses

have been published summarizing long-term associations between intakes of vegetables and fruit, dairy products, red and processed meat, and SSBs and risk of adiposity among observational studies (18–22). In this context, the quality of evidence on the associations between food groups and risk of general and abdominal overweight and obesity, as well as weight gain, also needs to be taken into account and the optimal intakes associated with the greatest risk reduction need to be clarified.

Therefore, we conducted a systematic review and meta-analysis to investigate the potential associations between 12 predefined food groups, including whole grains, refined grains, vegetables, fruit, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and SSBs, and the risk of overweight/obesity, abdominal obesity, and weight gain, completing our previous summaries of evidence on optimal intakes of these food groups regarding risk associations with all-cause mortality (23), colorectal cancer (24), coronary heart disease, stroke and heart failure (25), type 2 diabetes (26), and hypertension (27). In high compared with low linear and nonlinear dose-response meta-analyses, we aimed to quantify the strengths of these associations, to explore their shape (dose-response gradients), and to identify optimal intakes of these food groups regarding risk reduction of each outcome separately. Finally, we evaluated the quality of evidence using the NutriGrade scoring system.

Methods

The planning and conduct of the present meta-analysis followed a strategy that has been implemented in 5 recently published meta-analyses on food groups and risk of mortality (23), colorectal cancer (24), coronary heart disease, stroke and heart failure (25), type 2 diabetes (26), and hypertension (27). The strategy has been published in a protocol (28) and was registered in the PROSPERO International Prospective Register of Systematic Reviews (www.crd.york.ac.uk/prospero/index.asp; identifier CRD42016037069). The present systematic review and meta-analysis is an extension, focusing on associations between 12 food groups and risk of different measurements of adiposity, including overweight/obesity, abdominal obesity, or weight gain. Each outcome was analyzed separately. As previously described, the 12 food groups were selected because most of the dietary pattern scores were based on these and they find application in food-based dietary guidelines (28). The advantage of the investigation at the food-group level includes easier interpretation and transfer to public health messages and recommendations compared with findings from analyses at the nutrient level, including macronutrients. This systematic review and meta-analysis was conducted according to the guidelines for reporting proposed by the Meta-Analysis of Observational Studies in Epidemiology Group (29).

Search strategy

PubMed and Web of Science were searched until August 2018 by 2 investigators (LS, SS). There were no restrictions regarding language or calendar date, and predefined search terms were used (**Supplemental Table 1**). Reference lists from all related systematic reviews, meta-analyses, and original primary studies were screened to check for further potential articles. Disagreement between the 2 investigators was resolved by discussion.

Study selection

Two investigators (LS, SS) reviewed the titles and abstracts of all articles. Studies were eligible for inclusion if they met the following criteria: *1*) the study design was prospective (cohort studies, case-cohort studies, follow-up of RCTs, and nested case-control studies); *2*) the study provided information about the relation for ≥1 of the following 12 predefined food groups—whole grains/cereals, refined grains/cereals, vegetables, fruit, nuts, legumes, eggs, dairy products, fish, red meat, processed meat, and SSBs; *3*) the outcome was described as general overweight/obesity, abdominal obesity, or weight gain (defined as gain in weight or waist circumference over a time period) as a dichotomous endpoint; *4*) the study participants were aged ≥18 y; and *5*) standardized risk estimates were reported as ORs, RRs, or HRs. The definitions of the food groups are described in **Supplemental Table 2**. Studies were excluded if they reported on mean differences or linear associations (expressed as β-coefficients) on the relation between food groups and risk of adiposity (investigated as a continuous trait).

Data extraction

The following data were extracted independently by 2 investigators (LS, MN): name of the first author, year of publication, country, cohort name, age at entry, sex, sample size, total cases, dietary assessment, outcome, outcome assessment, type and specification of food group, adjustment factors, duration of follow-up, and most-adjusted risk estimate (ORs, RRs, HRs with corresponding 95% CIs). Any discrepancies were discussed and resolved by a third investigator (SS).

Quality of meta-evidence

The quality of evidence of each meta-analysis was evaluated by applying the NutriGrade scoring system. Details of the scoring system have been described previously (30) and are shown in **Supplemental Methods 1**. Briefly, NutriGrade

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Supplemental Tables 1–17, Supplemental Methods 1, and Supplemental Figures 1–24 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/advances.
Address correspondence to LS (e-mail: lukas.schwingshackl@dife.de).
PROSPERO-registration:
https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=37069
Abbreviations used: RCT, randomized controlled trial; SSB, sugar-sweetened beverage; WCRF, World Cancer Research Fund.

comprises 8 domains: *1*) risk of bias/study quality/study limitations (maximum of 2 points), *2*) precision (maximum of 1 point), *3*) heterogeneity (maximum of 1 point), *4*) directness (maximum of 1 point), *5*) publication bias (maximum of 1 point), *6*) funding bias (maximum of 1 point), *7*) effect size (maximum of 2 points), and *8*) dose-response (maximum of 1 point). In total, a maximum score of 10 points can be achieved and meta-evidence is graded as high ($\geq$8 points), moderate (6 to $<$8 points), low (4 to $<$6 points), or very low (0 to $<$4 points). High quality of evidence was interpreted as high confidence in the effect estimate, and further research probably will not change the confidence in the effect estimate. Moderate quality of evidence represented a moderate confidence in the effect estimate, and further research could add evidence on the confidence and may change the effect estimate. Low quality of evidence was interpreted as low confidence in the effect estimate, and further research will provide important evidence on the confidence and likely change in the effect estimate. And finally, very low quality of evidence was interpreted as very low confidence in the effect estimate and meta-evidence is very limited and uncertain.

Statistical analysis

The outcomes were defined as overweight/obesity (including outcomes that focused on overweight and/or obesity measured by BMI), abdominal obesity (defined by elevated levels of waist circumference), or weight gain (defined as gain in weight or waist circumference during the time period). Summary RRs and 95% CIs for the associations between the 12 predefined food groups and risk of each outcome separately were calculated with use of the random-effects meta-analysis by the DerSimonian and Laird method (31). In a first step, a high compared with low meta-analysis was conducted by comparing the highest category of dietary intake with the lowest. In a second step, a linear dose-response meta-analysis as described by Greenland and Longnecker (32) was performed by investigating the 12 predefined food groups as a continuous trait through the use of servings per day as described previously (23–27). If studies reported exposure in serving size but did not specify the amount, recommended conversions were used (**Supplemental Table 4**). For studies that already reported on linear dose-response trends, data were directly extracted and used. If no dose-response association was reported, the study-specific slope was calculated. For this method, information on the distribution of cases and person-years or noncases, the RRs with corresponding 95% CIs, and quantification for $\geq$ 3 exposure categories is required. If the distribution of person-years in single categories was not reported, but information on the number of cases plus total person-years or number of total participants plus follow-up period was available, missing data were calculated as previously described (33, 34). If mean values for the exposure category were missing but the range of the exposure category was reported, we calculated the midpoint between the lower and upper values. If categories were open-ended, it was assumed that the range was similar to the adjacent category.

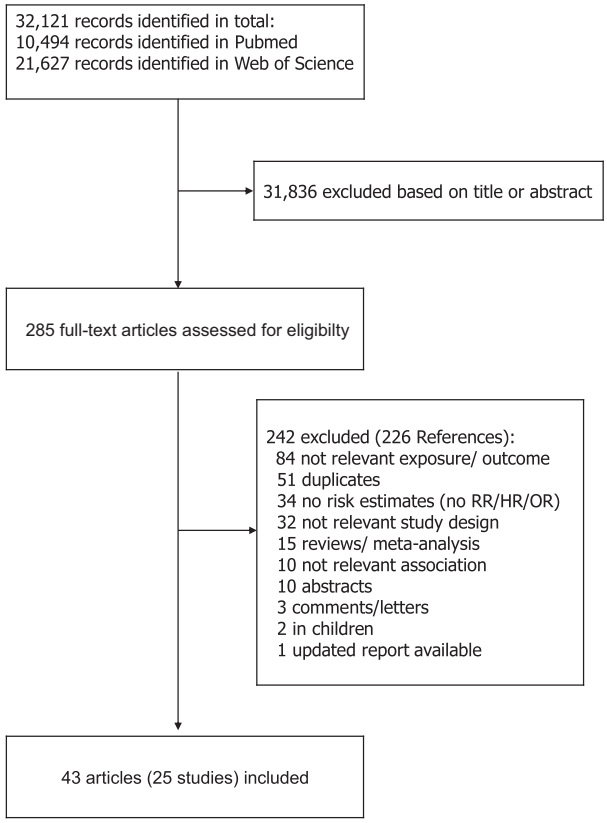

FIGURE 1 Flow diagram illustrating the identification and selection of studies.

Moreover, the potential of foods to reduce risk of overweight/obesity, abdominal obesity, or weight gain was calculated by multiplying the RR by selecting an optimal consumption of risk-reducing foods (calculated by $1 - \mathrm{RR}^{*}_{\mathrm{reduced}}$) and risk-increasing foods as noted (calculated by $1 - \frac{1}{\mathrm{RR}^{*}_{\mathrm{increased}}}$). Optimal intake of single food groups was defined as the serving category with the strongest inverse association with overweight/obesity, abdominal obesity, or weight gain risk with no further change in the risk association. Sensitivity analyses were performed excluding studies that did not adjust for energy intake.

Heterogeneity was evaluated by applying the Q test and I^2 statistic, with I^2 $>$50% regarded as having potentially important statistical heterogeneity (35). Publication bias was not assessed because all of the analyses were based on $<$10 studies and the Cochrane handbook recommends that $\geq$10 studies are necessary to achieve clear conclusions regarding publication bias (36). All statistical analyses were conducted using Stata version/SE 14.2 software (StataCorp) and Review Manager 5.3 (Nordic Cochrane Center).

Results

Of 32,121 identified records, 285 reports were retrieved for full-text review (**Figure 1**). Of these, 242 reports were

excluded for various reasons, as listed in **Supplemental Table 3**. In total, 43 reports (based on 25 prospective studies) were included in the systematic review and meta-analysis of the 12 predefined food groups and risk of overweight/obesity (defined by BMI), abdominal obesity (defined as elevated waist circumference), or weight gain (defined as gain in weight or waist circumference during the time period). In detail, 6 prospective studies were included in the meta-analysis for consumption of whole grains (37–42), 4 studies for refined grains (37, 38, 40, 41), 7 for vegetables (39, 43–48), 6 for fruit (39, 43, 45, 47–49), 4 for nuts (5 reports) (47, 50–53), 2 for legumes (45, 47), 2 for eggs (47, 54), 11 for dairy products (12 reports) (39, 47, 55–64), 4 for fish (47, 65–67), 4 for red meat (39, 68–70), 2 for processed meat (47, 68), and 9 for SSBs (11 reports) (56, 69, 71–79) (**Supplemental Tables 5–16**).

Whole grains

Five studies were included in the analysis on overweight/obesity (38–42) (Supplemental Table 5). Of these, 2 studies reported on overweight and obesity in combination (38, 40) and 3 reported on obesity (39, 41, 42). In high compared with low meta-analysis, the summary RR for overweight/obesity was 0.85 (95% CI: 0.79, 0.91) without indication for heterogeneity (I^2 = 0%) (**Supplemental Figure 1**). Three studies were included in the dose-response meta-analysis; and per each increase of 30 g whole-grain products/d, the risk of overweight/obesity decreased by 7% (RR: 0.93; 95% CI: 0.89, 0.96; I^2 = 0%, **Supplemental Figure 2**). There was no indication for a nonlinear association between whole-grain intake and risk of overweight/obesity (*P*-nonlinearity = 0.16; **Figure 2**A).

Three studies reported on intake of whole-grain products and the risk of weight gain, in which weight gain was defined as >2 kg during a mean period of 4 y (37), ≥10 kg during 13 y (38), or ≥25 kg during an average time of 12 y (41) (Supplemental Table 5). The summary RR (95% CI) for weight gain was 0.83 (0.70, 0.97), with I^2 = 16% in high compared with low analysis (Supplemental Figure 1), and 0.91 (0.82, 1.02), with I^2 = 69% for each increase of 30 g whole-grain products/d (Supplemental Figure 2). There was no indication of a nonlinear relation (*P*-nonlinearity = 0.10; **Figure 3**A).

Refined grains

Three studies reported on overweight/obesity, with 2 studies focusing on overweight and obesity combined (38, 40) and 1 focusing on obesity (41) (Supplemental Table 6). In high compared with low meta-analysis, the summary RR was 1.11 (95% CI: 0.85, 1.45), with high heterogeneity (I^2 = 84%) (**Supplemental Figure 3**). A positive association was observed in the dose-response meta-analysis (intake of refined grains per 30 g/d: 1.05; 95% CI: 1.00, 1.10; I^2 = 61%; **Supplemental Figure 4**). However, the nonlinear dose-response meta-analysis indicated that the association had a J-shape curve and a higher risk of overweight/obesity was identified for an intake of refined grains >90 g/d (*P*-nonlinearity < 0.001; Figure 2B).

The association between intake of refined grains and the risk of weight gain was investigated in 3 studies, defining weight gain as an increase in weight >2 kg (average time period: 4 y) (37), ≥10 kg (average time period: 13 y) (38), and >25 kg (average time period: 12 y) (41) (Supplemental Table 6). The summary RR for weight gain was 1.05 (95% CI: 0.78, 1.41), with I^2 = 66% when comparing a high intake of refined grains with a low intake (Supplemental Figure 3), and 1.01 (95% CI: 0.92, 1.12), with I^2 = 68% per each 30-g/d intake of refined grains in the linear dose-response meta-analysis (Supplemental Figure 4). There was no indication of a nonlinear relation between intake of refined grains and the risk of weight gain (*P*-nonlinearity = 0.11; Figure 3B)

Vegetables

Three studies reported on the associations between intake of vegetables and the risk of overweight/obesity, with 1 study focusing on overweight and obesity as a combined endpoint (45) and 2 on obesity (39, 43) (Supplemental Table 7). In high compared with low meta-analysis the summary RR for the association between intake of vegetables and the risk of overweight/obesity was 0.93 (95% CI: 0.83, 1.03), with I^2 = 66% (**Supplemental Figure 5**). Two studies were combined in the dose-response meta-analysis on vegetable intake and the risk of overweight/obesity; no association was observed (intake of vegetables per 100 g/d: summary RR: 0.98; 95% CI: 0.93, 1.03; **Supplemental Figure 6**). The test for nonlinearity was not significant (*P*-nonlinearity = 0.08), but the graph for the association between vegetable intake and risk of overweight/obesity indicated that a significant risk reduction was observed for an intake of ≤200–300 g/d, with more-imprecise results above that amount (Figure 2C).

For the association between intake of vegetables and the risk of weight gain, 5 studies were included (Supplemental Table 7). One study defined weight gain as an increase in weight of >25 kg during an average time period of 12 y (43), 1 study as >3 kg/y (46), 1 study as >2 kg/y (47), 1 study as >3.41 kg during an average time period of 10 y (48), and 1 study as any weight gain at the waist (44). A high intake of vegetables was associated with a reduced risk of weight gain (summary RR: 0.78; 95% CI: 0.62, 0.98; I^2 = 82%; Supplemental Figure 5). When we excluded the study focusing on weight gain only at the waist, the summary RR was 0.71 (95% CI: 0.47, 1.06; I^2 = 77%). The summary RR for weight gain per each 100-g/d intake of vegetables was 0.90 (95% CI: 0.81, 1.01), with I^2 = 60% (Supplemental Figure 6); a nonlinear relation was not evident (*P*-nonlinearity = 0.98; Figure 3C).

Fruit

Four studies investigated the association between fruit intake and the risk of overweight/obesity, with 2 studies focusing on overweight and obesity as a combined endpoint (45, 49) and 2 on obesity (39, 43) (Supplemental Table 8). Fruit intake was associated with a decreased risk of overweight/obesity in high

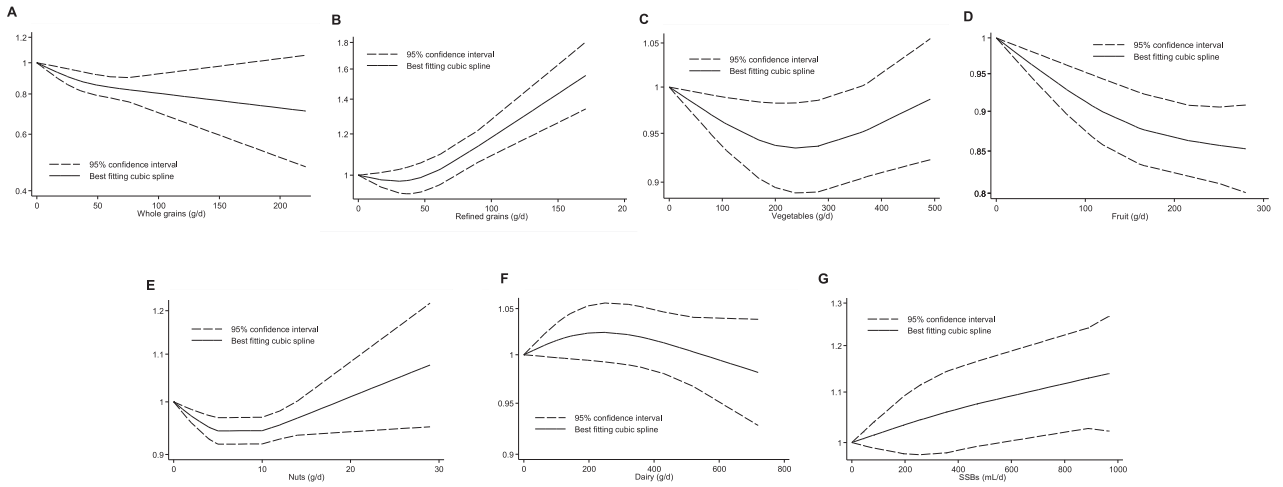

FIGURE 2 Nonlinear dose-response relation between daily intakes of whole grains (A) (*P*-nonlinearity = 0.16; *n* = 3 studies), refined grains (B) (*P*-nonlinearity < 0.001; *n* = 3 studies), vegetables (C) (*P*-nonlinearity = 0.08; *n* = 2 studies), fruit (D) (*P*-nonlinearity = 0.17; *n* = 2 studies), nuts (E) (*P*-nonlinearity < 0.001; *n* = 3 studies), dairy (F) (*P*-nonlinearity = 0.11; *n* = 3 studies), and SSBs (G) (*P*-nonlinearity = 0.82; *n* = 3 studies) and the relative risk (RRs and 95% CIs) of overweight/obesity. SSB, sugar-sweetened beverage.

compared with low (summary RR: 0.88; 95% CI: 0.80, 0.96; I^2 = 76%; **Supplemental Figure 7**) and linear dose-response (summary RR per each 100 g/d: 0.93; 95% CI: 0.86, 1.00; I^2 = 89%; **Supplemental Figure 8**) meta-analysis. There was no indication of a nonlinear relation (*P*-nonlinearity = 0.17; Figure 2D).

Three studies investigated the relation between intake of fruit and the risk of weight gain [defined as >2 kg/y (47), >3.41 kg during an average time period of 10 y (48), and >25 kg during an average time period of 12 y (43)] (Supplemental Table 8). The summary RRs for weight gain were 0.86 (95% CI: 0.70, 1.05), with I^2 = 46% in high compared with low meta-analysis (Supplemental Figure 7), and 0.91 (95% CI: 0.86, 0.97), with I^2 = 7% for increased intake of fruit per 100 g/d (Supplemental Figure 8). No evidence of a nonlinear relation was observed (*P*-nonlinearity = 0.14, Figure 3D). The graph indicated that findings after a daily intake of 300 g became more imprecise.

Nuts

Three studies were included in the meta-analysis on nut intake and the risk of overweight/obesity [2 focusing on overweight and obesity as a combined endpoint (50, 53) and 1 on obesity (51)] (Supplemental Table 9). The summary RR for overweight/obesity was 0.91 (95% CI: 0.80, 1.03), with I^2 = 25% when comparing a high intake of nuts with a low intake (**Supplemental Figure 9**). With each increase of 28 g/d, the risk of overweight/obesity was reduced by 22%, but the results were imprecise (summary RR per 28 g/d: 0.78; 95% CI: 0.58, 1.06; I^2 = 64%; **Supplemental Figure 10**). The shape of the relation between nut intake and the risk of overweight/obesity was J-shaped, showing a risk reduction for nut intake up to 10 g/d with more-imprecise findings above that (*P*-nonlinearity < 0.001; Figure 2E).

One study investigated the association between nut intake and the risk of abdominal obesity (defined by elevated waist circumference) (52) (Supplemental Table 9). This study reported a decreased risk between intake of nuts in high compared with low (RR: 0.76; 95% CI: 0.65, 0.89; Supplemental Figure 9) and dose-response (RR per 28 g/d: 0.42; 95% CI: 0.31, 0.57; Supplemental Figure 10) analysis.

The association between intake of nuts and the risk of weight gain was investigated in 2 studies, in which weight gain was defined as either >2 kg/y (47) or ≥5 kg (average time period: 2.3 y) (50) (Supplemental Table 9). The summary RRs for weight gain were 0.76 (95% CI: 0.58, 0.99), with I^2 = 0% in the high compared with low meta-analysis (Supplemental Figure 9), and 0.81 (95% CI: 0.64, 1.02), with I^2 = 0% in the dose-response meta-analysis (Supplemental Figure 10). Nonlinearity was not investigated because of lack of information.

Legumes

Only 1 study investigated the association between intake of legumes and the risk of overweight/obesity (45) (Supplemental Table 10). The RR was 0.87 (95% CI: 0.81, 0.94) in high compared with low analysis (**Supplemental Figure 11**). In dose-response analysis, the risk of overweight/obesity was reduced by 12% (RR: 0.88; 95% CI: 0.84, 0.93; **Supplemental Figure 12**) per each increase of 50 g of legumes/d.

In addition, only 1 study focused on intake of legumes and the risk of weight gain (defined as >2 kg/y) (47) (Supplemental Table 10). The RR was 0.89 (95% CI: 0.64, 1.24) in high compared with low analysis and per each 50 g/d (Supplemental Figures 11 and 12).

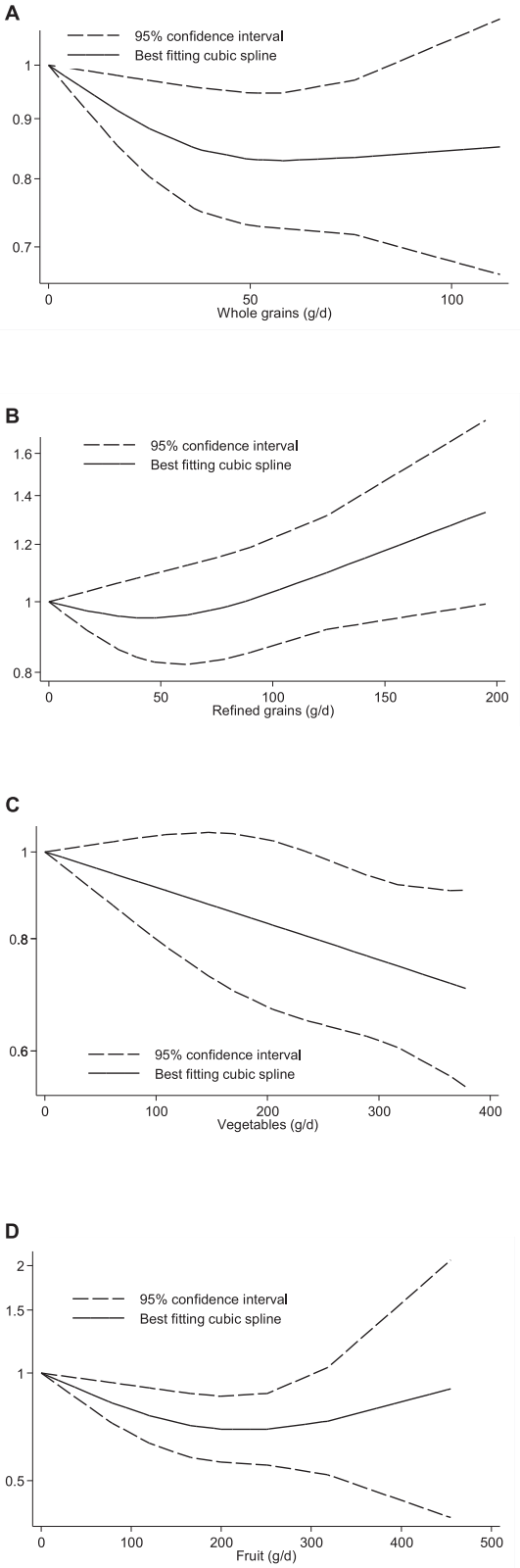

FIGURE 3 Nonlinear dose-response relation between daily intakes of whole grains (A) (*P*-nonlinearity = 0.10; *n* = 3 studies), refined grains (B) (*P*-nonlinearity = 0.11; *n* = 3 studies), vegetables (C) (*P*-nonlinearity = 0.98; *n* = 3 studies), and fruit (D) (*P*-nonlinearity = 0.14; *n* = 2 studies) and the relative risk (RRs and 95% CIs) of weight gain.

Eggs

There was only 1 study (54) that investigated the association between egg intake and the risk of abdominal obesity (defined by elevated waist circumference) (Supplemental Table 11), which did not find an association, either in the high compared with low (RR: 0.97; 95% CI: 0.59, 1.59; **Supplemental Figure 13**) or in the dose-response (RR: 0.95; 95% CI: 0.63, 1.43; **Supplemental Figure 14**) analysis (54).

Another study reported on intake of egg and the risk of weight gain (defined as >2 kg/y) (47) (Supplemental Table 11). This study indicated that egg intake was associated with an increased risk of weight gain. The RR for high egg intake compared with low intake was 1.54 (95% CI: 1.00, 2.37) (Supplemental Figure 13), and with each increase in egg intake of 50 g/d the RR was 1.24 (95% CI: 1.00, 1.54; Supplemental Figure 14).

Dairy products

Six studies investigated the association between dairy products and the risk of overweight/obesity [3 studies focusing on overweight and obesity in combination (59, 61, 62) and 3 on obesity (39, 58, 64)] (Supplemental Table 12). No association was observed for dairy products and the risk of overweight/obesity, either in high compared with low meta-analysis (summary RR: 0.96; 95% CI: 0.88, 1.06; I^2 = 82%; **Supplemental Figure 15**) or in dose-response meta-analysis (summary RR per 200 g/d: 0.97; 95% CI: 0.93, 1.01; I^2 = 79%; *P*-nonlinearity = 0.11; **Supplemental Figure 16**) (Figure 2F).

The association between dairy products and the risk of abdominal obesity (defined by elevated waist circumference) was examined in 5 studies (55–57, 63, 64) (Supplemental Table 12). The summary RR for abdominal obesity was 0.92 (95% CI: 0.82, 1.04), with I^2 = 34% for high intake of dairy products compared with low intake (Supplemental Figure 15); and for each increase of 200 g/d, corresponding results were 1.01 (95% CI: 0.95, 1.07), with I^2 = 0% (Supplemental Figure 16), with no indication for nonlinearity (P = 0.95; **Figure 4**A).

Two studies reported on the association between dairy products and the risk of weight gain [defined as >1 kg during an average time of 8.8 y (60) or >2 kg/y (47)] (Supplemental Table 12), and no associations were found in high compared with low meta-analysis (summary RR: 0.97; 95% CI: 0.91, 1.03; I^2 = 81%; Supplemental Figure 15) or in the dose-response meta-analysis (summary RR per 200 g/d: 0.99; 95% CI: 0.93, 1.05; Supplemental Figure 16). Nonlinearity was not explored because of a lack of information.

Fish

One study investigated intake of fish and the risk of overweight/obesity (66) (Supplemental Table 13) and did not find an association (**Supplemental Figures 17** and **18**). We identified 2 studies that reported on fish intake and the risk of abdominal obesity [defined by elevated waist circumference (65, 67)] (Supplemental Table 13), which indicated an inverse association. Summary RRs were 0.75 (95% CI: 0.62, 0.89),

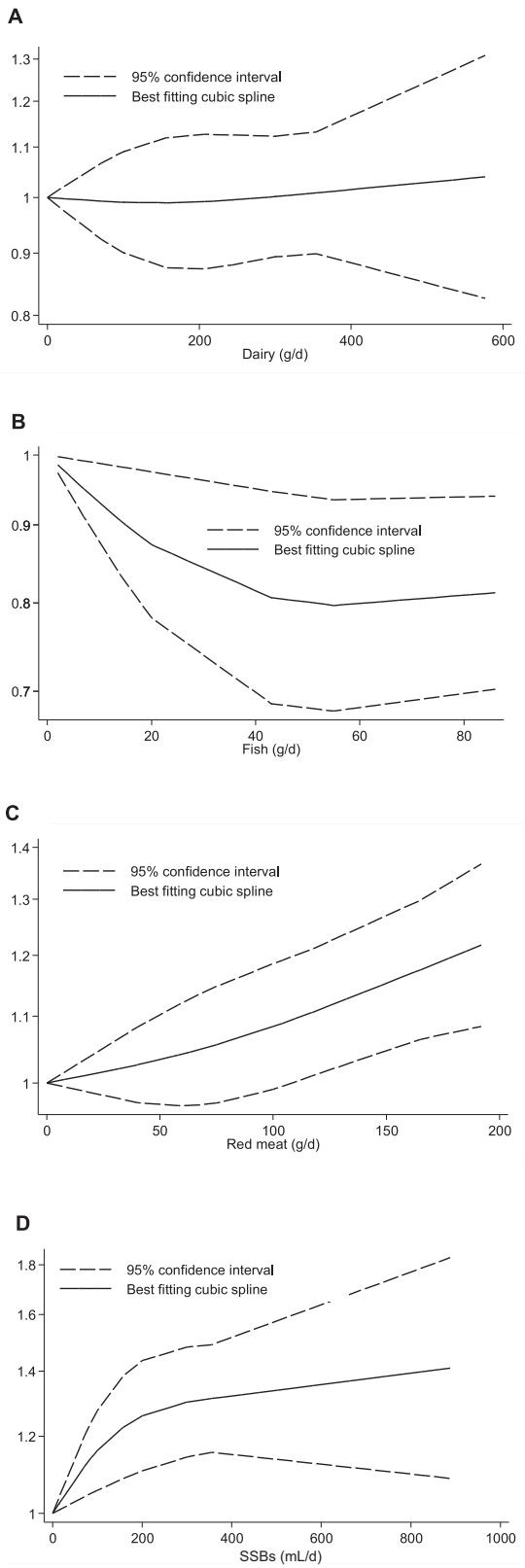

FIGURE 4 Nonlinear dose-response relation between daily intakes of dairy (A) (*P*-nonlinearity = 0.95; *n* = 3 studies), fish (B) (*P*-nonlinearity = 0.07; *n* = 2 studies), red meat (C) (*P*-nonlinearity = 0.57; *n* = 2 studies), and SSBs (D) (*P*-nonlinearity = 0.03; *n* = 4 studies) and the relative risk (RRs and 95% CIs) of abdominal obesity. SSB, sugar-sweetened beverage.

with I^2 = 0% in high compared with low meta-analysis (Supplemental Figure 17), and 0.83 (95% CI: 0.71, 0.97), with I^2 = 0% per increase of 100 g fish/d (Supplemental Figure 18). There was no indication of nonlinearity (*P*-nonlinearity = 0.07), but the graph indicated a stronger risk reduction at lower levels of fish intake and the curve reached a plateau at ∼40 g/d (Figure 4B). One study showed findings on fish intake and weight gain (>2 kg/y) (47) (Supplemental Table 13), where the RR was 1.06 (95% CI: 0.83, 1.35) in high compared with low and dose-response analysis (Supplemental Figures 17 and 18).

Red meat

The association between intake of red meat and the risk of obesity was investigated in 1 study (Supplemental Table 14) (39), which showed an increased risk for high compared with low intake of red meat (RR: 1.23; 95% CI: 1.07, 1.41; **Supplemental Figure 19**). Two studies investigated the relation between intake of red meat and the risk of abdominal obesity (defined by elevated waist circumference; Supplemental Table 14) (68, 70). The summary RRs were 1.18 (95% CI: 1.06, 1.32) ,with I^2 = 0% in high compared with low meta-analysis (Supplemental Figure 19), and 1.10 (1.04, 1.16), with I^2 = 0% in dose-response meta-analysis (**Supplemental Figure 20**), without an indication for nonlinearity (*P*-nonlinearity = 0.57; Figure 4C).

One study reported an increased risk for weight gain (≥1 kg during a time period of an average of 28.5 mo) for intake of red meat (69) (Supplemental Table 14). The RRs for weight gain were 1.16 (95% CI: 0.99, 1.36) (Supplemental Figure 19) in high compared with low analysis and 1.14 (1.03, 1.26) per a 100-g/d increase in red meat (Supplemental Figure 20).

Processed meat

One study reported on intake of processed meat and the risk of abdominal obesity (defined by elevated waist circumference) (68) (Supplemental Table 15). The RR was increased in high compared with low analysis, but estimations were imprecise (RR: 8.80; 95% CI: 1.20, 64.28; **Supplemental Figure 21**). Another study investigated the association between processed meat intake and the risk of weight gain (>2 kg/y) (47) (Supplemental Table 15), where an increased risk in high compared with low and dose-response analysis was observed (RR: 1.18; 95% CI: 1.02, 1.36; Supplemental Figure 21 and **Supplemental Figure 22**).

SSBs

Three studies were identified that reported on risk of overweight/obesity [1 on overweight and obesity combined (76) and 2 on obesity (74, 79)] (Supplemental Table 16). The summary RR for risk of overweight/obesity was 1.20 (95% CI: 1.01, 1.43), with I^2 = 23% in high compared with low meta-analysis (**Supplemental Figure 23**), and for each increase of 250 mL SSBs/d was 1.05 (95% CI: 1.00, 1.11), with I^2 = 33% (**Supplemental Figure 24**). The relation between SSBs and

risk of overweight/obesity was linear (*P*-nonlinearity = 0.82; Figure 2G).

Seven studies investigated the association between SSBs and risk of abdominal obesity (defined by elevated waist circumference) (56, 71–75, 78) (Supplemental Table 16). In high compared with low meta-analysis, the summary RR was 1.34 (95% CI: 1.13, 1.59), with I^2 = 90% (Supplemental Figure 23), and in dose-response meta-analysis the risk increased by 12% with each increase of 250 mL SSBs/d (summary RR: 1.12; 95% CI: 1.04, 1.20; I^2 = 38%; Supplemental Figure 24). The increase in risk was stronger at lower amounts of SSB intake (until ∼300 mL/d), but an increase at higher intakes was still present (*P*-nonlinearity = 0.03; Figure 4D).

Two studies reported on SSB intake and risk of weight gain (Supplemental Table 16). In 1 study weight gain was defined as gain in weight of ≥1 kg during an average time of 28.5 mo (69) and in the other as any weight gain among individuals with overweight/obesity over a mean period of 5 y (77). The summary RR was 1.23 (95% CI: 1.11, 1.37), with I^2 = 0% in high compared with low meta-analysis (Supplemental Figure 23). For the dose-response analysis, information from only 1 study was available (RR per 250 mL/d: 1.12; 95% CI: 0.82, 1.53; Supplemental Figure 24) (69).

Sensitivity analyses

Excluding studies not adjusting for energy intake [1 study for whole grains and refined grains (38), 1 study for vegetables (47), 2 studies for fruit (47, 49), 1 study for nuts (47), 1 study for legumes (47), 1 study for eggs (47), 1 study for dairy (47), 1 study for fish (47), 1 study for processed meat (47), and 3 studies for SSBs (76, 77, 79)] confirmed all findings of the primary analysis.

Summary across food groups

Table 1 shows the summary risks for the 12 predefined food groups and risk of overweight/obesity from nonlinear dose-response meta-analyses. The optimal intake (strongest association for foods per serving per day with no further substantial change in risk for higher intake) of foods (5 servings of whole grains/d, 3 servings of vegetables/d, and 3 servings of fruit/d) resulted in a 38% reduction in risk of overweight/obesity compared with nonconsumption of these food groups. Risk-increasing foods (strongest risk for 5 servings of refined grains/d and 3 servings of SSBs/d) resulted in a 59% increased risk of overweight/obesity.

Table 2 summarizes the optimal intakes of foods regarding risk of abdominal obesity. Only intake of fish was associated with decreased risk of abdominal obesity and optimal intake was 1 serving/d. Foods associated with increased risk of abdominal obesity resulted in a 63% elevated risk (2 servings/d of red meat and 3 servings/d of SSB).

Table 3 shows the optimal intakes of food groups regarding risk of weight gain. Risk-reducing foods (optimal intakes: 2 servings/d of whole grains, 5 servings/d of vegetables, 3 servings/d of fruit) resulted in a 59% risk reduction of weight gain. No association was observed for foods with potential of increasing risk of weight gain in nonlinear dose-response meta-analysis.

Quality of evidence

For overweight/obesity, the quality of evidence, rated by NutriGrade (30), was very low for refined grains, eggs, fish, and SSBs and low for whole grains, vegetables, fruit, nuts, legumes, and dairy products. Regarding abdominal obesity, quality of evidence was graded as very low for dairy products and red meat and as low for nuts, fish, and SSBs. The quality of evidence for weight gain was graded as very low for whole grains, refined grains, vegetables, nuts, legumes, eggs, dairy products, fish, red meat, and processed meat and as low for fruit and SSBs. None of the associations was graded as moderate or high (**Supplemental Table 17**).

Discussion

This systematic review and meta-analysis summarizes the evidence on associations between 12 predefined food groups (whole grains, refined grains, vegetables, fruit, nuts, legumes, eggs, dairy, fish, red meat, processed meat, and SSBs) and risk of adiposity (defined as overweight/obesity, abdominal obesity, or weight gain) by conducting high compared with low, linear, and nonlinear dose-response meta-analyses of prospective observational studies. A reduced risk for overweight/obesity and weight gain was identified for intake of whole grains, vegetables, and fruit, whereas the intake of fish was associated with a reduced risk of abdominal obesity. Increased risk of adiposity was observed for refined grains (for overweight/obesity and weight gain), red meat (for abdominal obesity), and SSBs (for overweight/obesity and abdominal obesity). For the dose-response associations between the food groups and risk of measurements of adiposity, the quality of evidence was low to very low, implying that future research will add evidence to the current knowledge about relations between food groups and risk of adiposity.

To the best of our knowledge, this is the first systematic review and meta-analysis of prospective observational studies that summarized the evidence of food groups and risk of adiposity in linear and nonlinear dose-response meta-analyses. Previous meta-analyses of observational studies on intake of fruit and vegetables (18), dairy products (19, 20), red and processed meat (21), and SSBs (22) regarding risk of adiposity did not perform linear and nonlinear dose-response meta-analyses (18, 19) or included mainly cross-sectional studies (20, 21). In our meta-analysis, we observed similar findings to the previous meta-analyses for intake of vegetables and fruit regarding risk of adiposity (18) with an additional, inverse association for risk of weight gain in the present study. Furthermore, our findings on red meat and risk of adiposity pointed in the same direction as the previous report (21). Compared with the 2 previous meta-analyses reporting a decreased risk of adiposity for high intake of dairy products (20) or yogurt intake, respectively (19), we observed contradictory findings showing a null association. The meta-analysis by Wang et al. (20) included

TABLE 1 Relative risks from nonlinear dose-response meta-analysis of predefined food groups and risk of overweight/obesity according to intakes in servings per day[1]

	Servings/d						
Food group and daily serving size	**0**	**1**	**2**	**3**	**4**	**5**	**6**
Inverse association							
Whole grains (30 g)	1.00	0.89 (0.83, 0.95)	0.84 (0.78, 0.91)	0.81 (0.72, 0.92)	0.79 (0.66, 0.95)	0.77 (0.60, 0.98)	NA
Vegetables (80 g)	1.00	0.97 (0.95, 0.99)	0.94 (0.90, 0.98)	0.93 (0.89, 0.98)	0.94 (0.90, 0.99)	0.96 (0.91, 1.02)	NA
Fruit (80 g)	1.00	0.93 (0.90, 0.96)	0.88 (0.83, 0.92)	0.86 (0.81, 0.91)	NA	NA	NA
Positive association							
Refined grains (30 g)	1.00	0.97 (0.92, 1.03)	1.02 (0.95, 1.09)	1.14 (1.06, 1.22)	1.29 (1.18, 1.42)	1.43 (1.26, 1.63)	NA
SSBs (250 mL)	1.00	1.04 (0.98, 1.11)	1.08 (0.99, 1.17)	1.11 (1.02, 1.21)	NA	NA	NA
No association							
Dairy (200 g)	1.00	1.02 (0.99, 1.05)	1.01 (0.98, 1.05)	0.99 (0.95, 1.04)	NA	NA	NA
Nuts (28 g)	1.00	1.08 (0.95, 1.22)	NA	NA	NA	NA	NA

[1] Values are RRs (95% CIs). NA, not applicable; SSB, sugar-sweetened beverage.

only cross-sectional studies, which are more prone to recall bias, which might explain the discrepancies between our findings and the findings of that study. The other meta-analysis focused on mean differences of body weight rather than risk of overweight, obesity, or weight gain. This report did not find an association for total dairy products with body weight change, but there was an association for intake of yogurt and weight loss (19). In our study, we did not investigate specific subgroups such as yogurt because of the small number of studies. In addition, evidence from a recent meta-analysis of short-term RCTs suggests that higher dairy consumption reduces body weight only among adults with energy restriction (80). As we learned from this meta-analysis of RCTs, diet-adiposity associations could also be dependent on the energy balance. In observational studies, most risk estimates for foods are not only adjusted for energy intake at recruitment but also for initial BMI, which is a sensitive parameter for correcting imbalances between energy intake and expenditure. This means that the analytical strategy of the observational studies assumed that change in the food group of interest is compensated by comparable changes in other foods to keep the energy intake constant. However, the prospective nature of a cohort study also assumes that diet at baseline is influencing the energy balance during follow-up and consequently weight gain. Further, it would be important to discuss our results in the context of findings from RCTs, which have shorter duration and fewer participants but a higher internal validity. RCTs look at the impact of exchanging 1 food with other foods and could also have detailed data on macronutrient intake. One of the impacts could be a distorted energy balance with an increase in body weight. However, it is not initially clear from those studies whether the impact is directly from the food being intervened or from side effects of the intervention on overall food intake. If isocaloric food exchange in the arms of the RCTs is taken as the approach, impact on energy balance could be small and thus also differences in weight change between groups. Our meta-analysis showed a risk reduction of overweight/obesity and weight gain with a high intake of whole grains, whereas a meta-analysis of RCTs did not show an effect on weight or waist circumference but did show a reduction in total body fat (9). Comparable to our findings, in a meta-analysis of RCTs, a higher intake of fruit and vegetables was slightly effective in reducing body weight (10), whereas another meta-analysis of intervention trials did not support the proposition that recommendations to increase fruit and vegetable consumption will cause weight loss (81). According to our findings on inverse (but imprecise) associations between nut intake and risk of adiposity, a meta-analysis of 33 RCTs reported no effects of high nut intake on body weight, BMI, or waist circumference (11). For legumes, we identified only 1 study that investigated the outcome

TABLE 2 Relative risks from nonlinear dose-response meta-analysis of predefined food groups and risk of abdominal obesity according to intakes in servings per day[1]

	Servings/d						
Food group and daily serving size	**0**	**1**	**2**	**3**	**4**	**5**	**6**
Inverse association							
Fish (100 g)	1.00	0.81 (0.70, 0.94) (per 85 g/d)	NA	NA	NA	NA	NA
Positive association							
Red meat (85 g)	1.00	1.07 (0.98, 1.17)	1.18 (1.06, 1.30)	NA	NA	NA	NA
SSBs (250 mL)	1.00	1.28 (1.13, 1.45)	1.33 (1.13, 1.58)	1.38 (1.10, 1.70)	NA	NA	NA
No association							
Dairy (200 g)	1.00	0.99 (0.87, 1.13)	1.01 (0.88, 1.27)	1.04 (0.83, 1.31)	NA	NA	NA

[1] Values are RRs (95% CIs). NA, not applicable; SSB, sugar-sweetened beverage.

TABLE 3 Relative risks from nonlinear dose-response meta-analysis of predefined food groups and risk of weight gain according to intakes in servings per day[1]

	Servings/d						
Food group and daily serving size	**0**	**1**	**2**	**3**	**4**	**5**	**6**
Inverse association							
Whole grains (30 g)	1.00	0.87 (0.78, 0.97)	0.83 (0.73, 0.95)	0.84 (0.69, 1.00)	0.85 (0.66, 1.09)	NA	NA
Vegetables (80 g)	1.00	0.93 (0.83, 1.04)	0.86 (0.70, 1.05)	0.80 (0.65, 1.00)	0.75 (0.61, 0.92)	0.70 (0.55, 0.91)	NA
Fruit (80 g)	1.00	0.82 (0.72, 0.94)	0.71 (0.58, 0.88)	0.70 (0.55, 0.88)	0.73 (0.52, 1.03)	NA	NA
No association							
Refined grains (30 g)	1.00	0.96 (0.86, 1.06)	0.96 (0.82, 1.12)	1.01 (0.85, 1.19)	1.10 (0.92, 1.31)	1.18 (0.95, 1.50)	NA

[1] Values are RRs (95% CIs). NA, not applicable.

of overweight/obesity (inverse association) and weight gain (no association), respectively. Evidence from a recent meta-analysis of 21 RCTs indicated that higher intakes of legumes were effective in inducing weight loss over a median duration of 6 wk (17). Long-term studies are warranted to confirm these findings. Our meta-analysis indicated that fish intake was inversely associated with risk of abdominal obesity. These findings are confirmed by a meta-analysis of RCTs that investigated intake of fish or n–3 PUFAs and reported a reduction in body weight, waist circumference, BMI, as well as body fat for the intervention group compared with the controls (14). Higher intakes of SSBs were positively associated with risk of overweight and obesity in our meta-analysis. In addition, meta-analyses of intervention trials reported that higher intakes of SSBs induce weight gain (82). No previous meta-analyses, either of observational studies or of RCTs, were identified for associations on intake of refined grains and eggs with risk of adiposity.

The quality of evidence of our findings was graded as low to very low, indicating that future studies will add important evidence for associations between food groups and risk of overweight/obesity, abdominal obesity, or weight gain. This observation is in line with current dietary recommendations from different organizations [e.g., from the World Cancer Research Fund (WCRF)/American Institute for Cancer Research] (83). The WCRF also judged the evidence as "limited–suggestive" or "limited–no conclusion" for most of the associations between dietary factors and the risk of weight gain, overweight, and obesity. There was 1 exception regarding the judgment of evidence for SSBs. The WCRF concluded a strong quality of evidence for SSB intake and risk of adiposity ("strong evidence–convincing"), whereas in our meta-analysis, the quality of evidence for SSBs was graded as very low for general and abdominal obesity and as low for weight gain. The discrepancies can be explained by the WCRF deriving their conclusions from RCTs and prospective studies and grading the evidence for all outcomes (overweight, obesity, and weight gain) combined. A goal for the future is to have better-designed studies with repeated measurements of food intake and long-term follow-up periods on this issue. In the meantime, we have to cope with the observation that the intake of a food at baseline shows a relation to the probability of becoming obese during the follow-up period even after adjusting for energy intake. This indicates that intake of specific foods increases the probability of a positive energy balance or favors pathways that change the relation between energy expenditure for heat development and for fat storage. In the current stage, no definite conclusions can be drawn regarding the mechanisms because findings of clinical trials did not support the observational findings. Some of the food groups are markers of a healthy or unhealthy lifestyle per se. It is likely that individuals who consume higher amounts of whole-grain products, vegetables, fruit, and fish and lower amounts of refined grains, red meat, and SSBs are those who are more sensitive to weight gain. However, most of the studies adjusted for other lifestyle factors and partly for other dietary factors or dietary quality, reflecting that other mechanisms are conceivable. It has further been suggested that high intake of whole grains, fruits, and vegetables, characterized by low glycemic load and index, and high intake of refined grains or SSBs, containing high amounts of sugar, have effects on insulin secretion and resistance, which may have metabolic effects and contribute to weight gain and obesity (84–86). In this context, a meta-analysis of RCTs comparing diets with a low glycemic load and index with diets with a high glycemic load and index on anthropometric outcomes showed no effect (86). Furthermore, there might be a specific role of SSBs that impairs the regulation of hunger and satiety (87). The regulation of hunger and satiety is not only discussed regarding foods but also is an important topic regarding the macronutrient composition of the diet in general. Evidence from clinical trials suggested that the reduction in dietary fat was related to weight loss in the short term, but findings from observational studies were not consistent regarding longer duration (88). Also, high-protein diets were more effective in reducing body weight than were standard-protein diets in a meta-analysis of 24 trials (89), whereas in the DiOGenes cohort study, a higher intake of total and animal protein was associated with weight gain (90).

Strengths and limitations

Among the strengths of this systematic review and meta-analysis is the investigation of dose-response relations, which provided insights into the shape of the associations and enabled calculation of cutoffs for optimal intakes of food

groups. In addition, we graded the quality of evidence of identified associations through the use of an established tool. Finally, to reduce selection and recall bias from original studies, we included only prospective studies.

The major limitation is that some of the meta-analyses were based on small numbers of studies. One reason is that, according to our inclusion criteria, we excluded studies that did not report on risk estimates (RRs, HRs, ORs) between food groups and risk of adiposity, and thus several studies showing linear associations (expressed as regression coefficients) or mean differences for adiposity as a continuous outcome were not included in our report. However, the conduct, reporting, and findings from these studies were heterogeneous and not eligible for pooling in our meta-analysis. In addition, we decided to stratify the meta-analysis by measurements of adiposity to reduce heterogeneity between outcomes, and thus conducted meta-analysis for each outcome (overweight/obesity, abdominal obesity, and weight gain) separately. Because of the small number of studies, subgroup analyses (e.g., for sex, geographic location, etc.) were not conducted and publication bias and small-study effects were not assessed. Altogether, this leads to very low and low quality of evidence, indicating that findings should be interpreted with caution, especially findings from the nonlinear dose-response meta-analyses, which are based on a maximum of 4 studies. Another limitation related to the primary studies that were included in our meta-analysis is the lack of information regarding energy and macronutrient balance. However, most of the studies adjusted for total energy and some of them adjusted for intake of other foods, macronutrients, micronutrients, or dietary quality. In our sensitivity analysis, we excluded studies that did not adjust for energy intake. Our findings were robust; however, we cannot conclude how much of the effect between intakes of food groups and overweight, obesity, or weight gain was explained by energy intake. Future studies investigating the mediation effect might help to answer this question. Implementation of novel statistical methods such as substitution analyses (91), mediation analysis (92), or network meta-analyses of well-conducted intervention trials (93) is warranted to clarify the plausibility and nature of these associations.

Conclusions

The findings of the systematic review and meta-analysis indicate that high intakes of whole grains, vegetables, fruit, and probably fish as well as a low intake of refined grains, red meat, and SSBs are associated with a reduced risk of measures of adiposity, including overweight/obesity, abdominal obesity, or weight gain, respectively. These findings are in line with current public health recommendations regarding a health-promoting diet. However, with the current evidence rated as very low to low, findings should be interpreted with caution and better-designed observational studies, more evidence from intervention trials, and use of novel statistical methods (e.g., substitution analyses or network meta-analyses) are needed.

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