META-ANALYSIS

Impact of different dietary approaches on blood lipid control in patients with type 2 diabetes mellitus: a systematic review and network meta-analysis

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Abstract

The aim of this study was to assess the effects of different dietary approaches on low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglyceride (TG) levels in patients with type 2 diabetes (T2D) by applying network meta-analysis (NMA). Systematic electronic and hand searches were conducted until January 2018. Randomized controlled trials (RCTs) with an intervention period of ≥ 12 weeks, focussing on adults with T2D, and comparing dietary approaches regarding LDL, HDL or TGs, were included. For each outcome measure, random effects NMA was performed in order to determine the effect of each dietary approach compared to every other dietary intervention. Mean differences (MDs) and 95% confidence intervals (95% CIs) were calculated, and for the ranking, the surface under the cumulative ranking curves (SUCRA) was determined. Additionally, the credibility of evidence was evaluated. 52 RCTs (44 for LDL, 48 for HDL and 52 for TGs) comparing nine dietary approaches (low fat, vegetarian, Mediterranean, high protein, moderate carbohydrate, low carbohydrate, control, low glycaemic index/glycaemic load and Palaeolithic diet) enrolling 5360 T2D patients were included. The vegetarian diet most effectively reduced LDL levels [MD (95% CI): -0.33 (-0.55, -0.12) mmol/L; compared to the control diet]. The Mediterranean diet beneficially raised HDL [MD (95% CI): 0.09 (0.04, 0.15) mmol/L] and decreased TG levels [MD (95% CI): -0.41 (-0.72, -0.10) mmol/L] compared to the control diet. The Mediterranean diet was the most effective dietary approach to manage diabetic dyslipidaemia altogether (SUCRA: 79%). The overall findings are mainly limited by low credibility of evidence.

Keywords Type 2 diabetes · Diet · Blood lipids · Systematic review · Network meta-analysis

Lukas Schwingshackl and Sabrina Schlesinger have contributed equally to this work.

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Introduction

Diabetes mellitus is a global health problem with a prevalence of worldwide 8.8% [1], which is associated with many other health problems and high health care costs [2, 3]. Patients with type 2 diabetes (T2D) typically show abnormalities in their lipid metabolism, such as higher levels of fasting and postprandial triglycerides (TGs), higher levels of small, dense low-density lipoprotein (LDL) particles as well as lower levels of high-density lipoprotein (HDL) cholesterol [4–7]. This state of dyslipidaemia can further worsen beta-cell dysfunction and insulin resistance and therefore increase the incidence for unfavourable macrovascular endpoints (e.g. cardiovascular disease (CVD), myocardial infarction and CVD mortality) [8, 9] and for microvascular complications [10–12]. Thus, it is important to improve the management of T2D in order to achieve glycaemic and lipid goals [5, 13]. Diet as modifiable lifestyle factor plays

an important role in that matter [14, 15]. Based on expert opinion, the American Diabetes Association (ADA) recommends an individualized medical nutrition therapy for glycaemic and lipid management, because there is no single dietary approach for patients with T2D [15]. In the literature, several dietary regimens, such as dietary patterns with different macronutrient compositions (e.g. low/high fat, low/high carbohydrate, high protein) [16–18], the Mediterranean diet [19] or the vegetarian diet [20], were investigated regarding their effects on the management of T2D. A recent review discussed the controversy of low fat versus low carbohydrate diets regarding their effects on chronic diseases, including diabetes [21]. However, no clear conclusion, which diet offers the greatest benefit regarding prevention or management of diabetes, was drawn. An emerging methodology in the field of nutrition research to compare multiple interventions lies in network meta-analysis (NMA). NMA is an extension of pairwise meta-analysis, which simultaneously compares direct effects between interventions within trials and indirect effects between trials, in order to estimate all possible relative effects in a network and to obtain a therapeutically meaningful ranking [22, 23]. A recent NMA comparing five dietary approaches (Mediterranean, high carbohydrate, low carbohydrate, low fat and control diet) found, that the Mediterranean diet showed the most beneficial effects regarding both glycaemic and blood lipid control [24]. However, this publication does not show the whole picture, since randomized controlled trials (RCTs) on additional dietary approaches and their effects on glycaemic and blood lipid control are available. Moreover, transitivity and credibility of evidence was not evaluated in this NMA. Thus, another recent NMA compared nine dietary approaches (low fat, vegetarian, Mediterranean, high protein, moderate carbohydrate, low carbohydrate, control, low glycaemic index/ glycaemic load (low GI/GL) and Palaeolithic diet) regarding glycaemic control in T2D and also found the Mediterranean diet to be the most effective dietary approach [25]. However, the question which of these nine diets offers the most benefits regarding blood lipid control in T2D was not investigated in that NMA. Therefore, a systematic review and NMA was conducted to answer this question.

Methods

The review was registered in PROSPERO International Prospective Register of Systematic Reviews (https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=47464) and our strategy for the systematic review and NMA was pre-defined in a published protocol [26]. This systematic review and meta-analysis was planned, conducted and reported according to standards of quality for reporting systematic reviews and NMA [27, 28].

Literature search

The literature search and screening of the studies was conducted by two reviewers (LS, MN). Pubmed and the Cochrane Central Register of Controlled Trials (CENTRAL) were searched using predefined search terms until January 2018 with no language restriction and no restriction on date (Supplementary Table S1).

Eligibility criteria

Studies were included if they were RCTs (cross-over or parallel) and if they compared different interventions of dietary approaches (energy restricted, isocaloric or ad libitum) regarding the outcomes LDL, HDL and/or TGs.

Eligible dietary interventions were:

- Low carbohydrate diet (< 25% carbohydrates of total energy intake; > 30% fat of total energy intake; high intake of animal and/or plant protein) [29]
- Moderate carbohydrate diet (25–45% carbohydrates of total energy intake; 10–20% protein intake) [29]
- High protein diet (> 20% protein intake of total energy intake; high intake of animal and/or plant protein; < 35% fat) [30]
- Low fat diet (< 30% fat of total energy intake; high intake of cereals & grains; 10–15% protein intake) [29]
- Low GI/GL diet [31]
- Vegetarian (no meat and fish) and vegan diet (no animal products) [32]
- Mediterranean diet: high intake of fruit, vegetables, olive oil, legumes, cereals, fish, and moderate intake of red wine during meals [33]
- Palaeolithic diet: high intake of lean meat, fish, fruit, vegetables, eggs and nuts; exclusion of dairy products, cereal grains, beans, refined fats, sugar, candy, soft drinks, beer and extra addition of salt [34]
- Control diet: no intervention or minimal intervention.

Whenever possible, the classification of dietary approaches was adopted from the original studies, as was always the case for qualitative dietary approaches, such as the low GI/GL diet, the vegetarian diet, the Mediterranean and the Palaeolithic diet. However, this was not always possible for dietary approaches defined by their nutrient composition, because of overlaps between the approaches. For example, we classified a high carbohydrate [35–42], a low protein [43, 44] and a low fat diet [45–57] from the original trials, all as low fat diet, because these approaches had the same nutrient composition. Each dietary approach was assigned to only one intervention arm and different

intervention arms of one study were classified as different dietary approaches. Nevertheless, in some cases, there was an overlap of dietary approaches. For example, a vegetarian diet [58] would also fulfil the criteria for a low fat diet [29], but in contrast, low fat diets could not always be classified as vegetarian diets.

The duration of the intervention had to be at least 3 months.

Studies were included if they focussed on participants older than 18 years old and diagnosed with T2D according to ADA criteria or other internationally recognized standards [59]. Studies including T2D patients with co-morbidities, including hypertension and hyperlipidaemia were also included. Studies on children and adolescents, pregnant women, participants with type 1 diabetes, impaired glucose metabolism and chronic renal disease were excluded.

Studies were excluded if the interventions focussed on supplements or single foods, if dietary supplements were used as placebo, or if exercise and/or medication as co-intervention were not applied in all groups. Studies including interventions with very low calorie diets (<600 kcal/d) were also excluded, because they are not recommended at all by the Canadian Diabetes Association [60] and not for longer than 3 months without clinical supervision by the ADA [61].

Data extraction

Data were extracted by two reviewers (LS, MN). Extracted data were: name of first author, year of publication, study origin (country), study design (RCT: parallel or cross-over), sample size, mean baseline age, mean baseline body mass index (BMI), mean differences in weight change, study duration, sex, description of dietary intervention arms and control group, type of diet (energy restricted, isocaloric, ad libitum) and drop-outs. Post-intervention values with corresponding standard deviations for LDL, HDL and TGs were extracted as outcome data.

Risk of bias assessment

Risk of bias assessment for each included trial was conducted by two reviewers (LS, GH) using the risk of bias assessment tool by the Cochrane collaboration [62]. This tool assesses the following sources of bias: selection bias (random sequence generation and allocation concealment), performance bias (blinding of participants and personnel), attrition bias (incomplete outcome data) and reporting bias (selective reporting). A trial was classified as low risk of bias if at least three out of five items were rated as low risk and maximally one item was rated as high risk of bias. A trial was classified as high risk of bias if at least two out of five

items were rated as high risk of bias. All other studies were classified as moderate/unclear risk of bias [62].

Dealing with missing data

We contacted authors to receive missing outcome data (two authors sent additional data [38, 63]). If no post-intervention values with their corresponding standard deviations (SDs) were available, change scores and corresponding SDs were used according to the Cochrane handbook [64].

Data synthesis

Description of available data

Available data on study and population characteristics and variables (age, length of follow-up, outcome relevant baseline risk factors) were described for all included studies. For each outcome (LDL, HDL, and TGs), available direct comparisons between different dietary interventions and the control group were displayed in a network diagram [65].

Assessment of transitivity

Transitivity is a fundamental assumption when conducting a NMA and means that available treatment comparisons do not differ in their distribution of effect modifiers. Its violation threatens the validity of the results [65]. We considered mean baseline age, BMI, sample size, and study length as possible effect modifiers in this NMA. Exercise was not considered as effect modifier, because we already defined it as exclusion criterion, if it was not the same in the intervention and control group. We assessed transitivity using box plots to examine the distribution of effect modifiers between available direct comparisons [66].

Statistical analysis

For each outcome (LDL, HDL, and TGs), random effects NMA were conducted in order to determine the pooled effect of each intervention relative to every other intervention. A NMA is an extension of pairwise meta-analysis which enables a simultaneous comparison of multiple interventions while keeping the internal randomization of each trial and which allows for an estimation of all possible pairwise effects and for obtaining a clinically meaningful ranking [67]. Summary mean differences (MDs) and their corresponding 95% confidence intervals (95% CIs) were presented in a league table for each outcome (LDL, HDL, and TGs) and the relative ranking of different diets were estimated by distribution of the ranking probabilities and the surface under the cumulative ranking curve (SUCRA) [67]. Additionally, the SUCRA values of the three outcomes

(LDL, HDL, and TGs) were averaged into a summary SUCRA score for each dietary approach, in order to rank the treatments according to their overall effect on diabetic dyslipidaemia. Moreover, a common network-specific heterogeneity parameter was assumed. Calculations were fitted in a frequentist framework and conducted with STATA 14. The network plots were generated using the R package "netmeta" [68, 69].

Assessment of inconsistency

Valid results also depend on consistent results. Therefore, direct and indirect evidence should be in agreement [65, 70]. For the assessment of inconsistency, local and global approaches were used [70]. Specifically, we used the loop-specific approach that detects loops of evidence with important inconsistency [71] and the side-splitting approach that examines if direct and indirect estimates disagree [72]. As global approach, the design-by-treatment interaction model was applied, which investigates inconsistency from all possible sources in the entire network simultaneously [73, 74].

Subgroup and sensitivity analyses

Sensitivity analyses were conducted excluding trials with high risk of bias, including only long-term trials (≥ 12 months) and including only trials in patients with a mean diabetes duration of ≥ 5 years. We also ran an univariate meta-regression to investigate the association between mean differences in weight change and changes in LDL, HDL and TGs [75].

Small study effects and publication bias

If 10 or more studies were available, a comparison adjusted funnel-plot [65] and a contour-enhanced funnel plot were generated. The latter examines if asymmetry is likely to be explained by publication bias [76]. Inference on risk for publication bias was based on non-statistical considerations, namely how likely it is that studies have been conducted but not published, based on expertise of investigators in the field.

Credibility of evidence

The credibility of evidence was evaluated applying the Grading of Recommendations Assessment, Development and Evaluation (GRADE) systems extended for NMA [70]. For the evaluation we used the online tool CINeMA available from http://cinema.ispm.ch/, which is based on this methodological framework. The five domains "within-study bias", "indirectness", "imprecision", "heterogeneity", and "incoherence" are investigated for each pairwise comparison.

Based on the contribution matrix, which shows how much information comes from direct and indirect evidence, it is determined how much information comes from low, moderate or high risk of bias studies and "no concerns", "serious concerns" or "major concerns" are assigned to each domain. The overall confidence rating was determined according to pre-defined criteria (Supplementary Tables S17-19).

Results

Characteristics

Of the 3185 publications initially identified, 52 RCTs (44 for LDL, 48 for HDL and 52 for TGs) met the inclusion criteria and provided sufficient data to be included in the meta-analysis [34–58, 63, 77–102] (Supplementary Fig. S1). A list of excluded studies can be found in Supplementary Table S2. The trials were published between 1978 and 2016 and included a total of 5360 T2D patients. Sixteen trials were conducted in North America, 13 in Europe, 8 in Asia and 15 in Australia and New Zealand. The study duration ranged between 3 and 48 months. The enrolled patients were between 44 and 65 years old and had a BMI between 23 (Asian population) and 40 kg/m^2. The study characteristics are summarized in Supplementary Table S3. The dietary interventions have been described in detail in the previous NMA [25].

Risk of bias

Nineteen trials were assessed as low risk of bias, 7 as high risk of bias and 26 as moderate/unclear risk of bias. Concerning the specific items of the risk of bias assessment, there was indication for low risk of bias for 58% of the trials for random sequence generation, for 17% for the allocation concealment, for 2% for the blinding of participations and personnel, for 63% for incomplete outcome data and for 81% for selective reporting. The risk of bias assessment for the individual trials is summarized in Supplementary Fig. S2.

Network meta-analysis

Figure 1 shows the network diagrams for LDL (Fig. 1a), HDL (Fig. 1b), and TGs (Fig. 1c). The nodes represent the number of patients and the lines the number of trials. The highest number of trials were available for the comparison between a moderate carbohydrate diet and a low fat diet ($n_{LDL} = 10$, $n_{HDL} = 12$, $n_{TG} = 13$), and for a high protein diet compared to a low fat diet ($n_{LDL} = 9$, $n_{HDL} = 9$, $n_{TG} = 9$). The statistical contribution of direct and indirect evidence of the different comparisons is in line with the NMA by Schwingshackl et al. [25]. According to that

Fig. 1 Network diagrams for LDL (**a**), HDL (**b**) and TGs (**c**). The size of the nodes is proportional to the total number of participants allocated to each dietary approach and the thickness of the lines proportional to the number of studies evaluating each direct comparison. *GI/ GL* Glycaemic index/glycaemic load
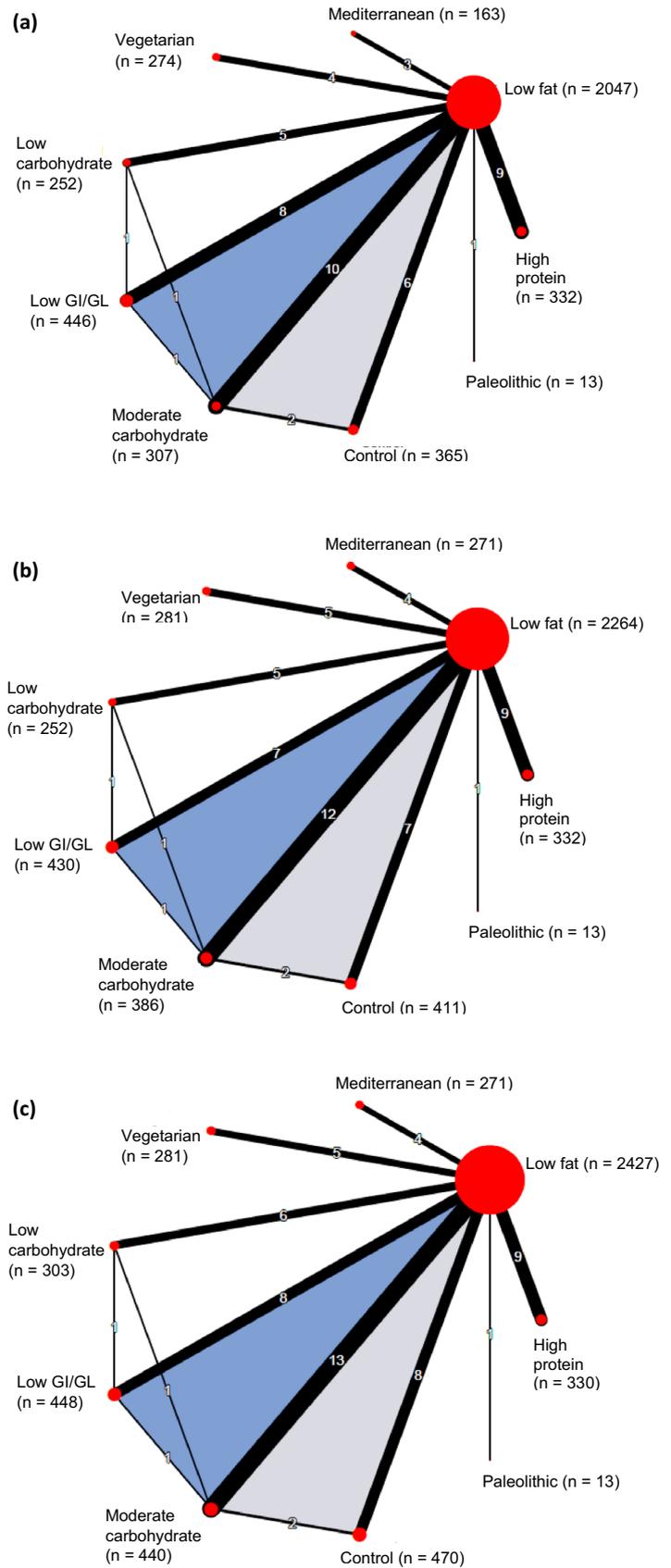
contribution matrix, most of the contribution to the study effects come from indirect comparisons. Direct comparisons were mostly available for the different dietary approaches compared to a low fat diet (Fig. 1). There was no evidence for transitivity (Supplementary Fig. S3-6).

LDL-cholesterol

Table 1 summarizes the effect size estimates (MDs with their 95% CI) for the comparison of every dietary approach compared with each other on LDL (mmol/L). The vegetarian diet, low GI/GL diet, and moderate carbohydrate diet were found to be more effective in reducing LDL than the control diet [MD (95% CI): −0.33 (−0.55, −0.12) mmol/L, −0.26 (−0.43, −0.09) mmol/L, and −0.21 (−0.38, −0.05) mmol/L, respectively]. Vegetarian diet intervention also decreased LDL more effectively compared to the low carbohydrate diet [MD (95% CI): −0.29 (−0.52, −0.06) mmol/L], the high protein diet [MD (95% CI): −0.31 (−0.53, −0.09) mmol/L] and the low fat diet [MD (95% CI): −0.23 (−0.40, −0.06) mmol/L]. The low GI/GL diet yielded a stronger reduction in LDL compared to the low carbohydrate diet [MD (95% CI): −0.21 (−0.40, −0.02) mmol/L], the high protein diet [MD (95% CI): −0.23 (−0.41, −0.05) mmol/L], and the low fat diet [MD (95% CI): −0.15 (−0.26, −0.04) mmol/L].

HDL-cholesterol

Table 2 summarizes the effect size estimates (MDs with their 95% CI) for the comparison of every dietary approach compared with each other on HDL (mmol/L). The only diet that effectively raised HDL was the Mediterranean diet. The MD (95% CI) was 0.09 (0.04, 0.15) mmol/L compared to the control diet, 0.12 (0.07, 0.18) mmol/L compared to the high protein diet, 0.08 (0.05, 0.12) mmol/L compared to the low fat diet, and 0.08 (0.03, 0.13) mmol/L compared to the moderate carbohydrate diet. The other diets showed either no effect or even decreased HDL, especially the vegetarian diet (Table 2).

Triglycerides

Table 3 summarizes the effect size estimates (MDs with their 95% CI) for the comparison of every dietary approach compared with each other on TGs (mmol/L). The Mediterranean diet showed reductions in TGs compared to the control diet, the low fat diet and the low GI/GL diet, [MD (95% CI): −0.41 (−0.72, −0.10) mmol/L, −0.33 (−0.59, −0.08) mmol/L, and −0.35 (−0.66, −0.05) mmol/L, respectively]. Additionally, the low carbohydrate diet was more favourable than the control diet [MD (95% CI): −0.36 (−0.62, −0.10) mmol/L], the low fat diet [MD (95% CI): −0.28 (−0.48, −0.09) mmol/L] and the low GI/GL diet [MD (95% CI): −0.30 (−0.55, −0.06) mmol/L]. The other diets showed no effect (Table 3).

Table 1 League table showing the results of the network meta-analysis comparing the effects (mean difference: MD) of all dietary approaches and 95% confidence intervals (95% CI) on LDL (mmol/L) in individuals with T2D (n = 44)

Vegetarian								
−0.08 (−0.28, 0.13)	**Low GI/GL**							
−0.14 (−0.44, 0.15)	−0.07 (−0.33, 0.20)	**Mediterranean**						
−0.13 (−0.97, 0.71)	−0.05 (−0.88, 0.78)	0.02 (−0.84, 0.87)	**Palaeolithic**					
−0.12 (−0.33, 0.08)	−0.05 (−0.19, 0.10)	0.02 (−0.25, 0.29)	0.01 (−0.83, 0.84)	**Moderate carbohydrate**				
−0.23 (−0.40, −0.06)	**−0.15 (−0.26, −0.04)**	−0.08 (−0.33, 0.16)	−0.10 (−0.92, 0.72)	−0.11 (−0.22, 0.01)	**Low fat**			
−0.31 (−0.53, −0.09)	**−0.23 (−0.41, −0.05)**	−0.17 (−0.45, 0.12)	−0.18 (−1.02, 0.65)	−0.19 (−0.37, 0.00)	−0.08 (−0.22, 0.06)	**High protein**		
−0.29 (−0.52, −0.06)	**−0.21 (−0.40, −0.02)**	−0.15 (−0.44, 0.15)	−0.16 (−1.00, 0.68)	−0.17 (−0.36, 0.02)	−0.06 (−0.22, 0.10)	0.02 (−0.19, 0.23)	**Low carbohydrate**	
−0.33 (−0.55, −0.12)	**−0.26 (−0.43, −0.09)**	−0.19 (−0.47, 0.08)	−0.21 (−1.04, 0.63)	**−0.21 (−0.38, −0.05)**	−0.11 (−0.24, 0.02)	−0.03 (−0.22, 0.17)	−0.05 (−0.25, 0.16)	**Control**

The values below the dietary approaches correspond to the mean difference in LDL (mmol/L) between the column and the row (e.g. the MD LDL between the Mediterranean diet and the control diet is −0.19 mmol/L). The effect was interpreted as statistically significant if the 95% CIs did not include the null value (highlighted in bold letters)

GI/GL Glycaemic index/glycaemic load; *LDL* low-density lipoprotein cholesterol

Table 2 League table showing the results of the network meta-analysis comparing the effects (mean difference: MD) of all dietary approaches and 95% confidence intervals (95% CI) on HDL (mmol/L) in individuals with T2D (n = 48)

Vegetarian								
−0.05 (−0.11, 0.01)	**Low GI/GL**							
−0.17 (−0.23, −0.10)	**−0.11 (−0.16, −0.06)**	**Mediterranean**						
−0.16 (−0.38, 0.05)	−0.11 (−0.32, 0.10)	0.00 (−0.21, 0.22)	**Palaeolithic**					
−0.09 (−0.15, −0.02)	−0.03 (−0.08, 0.01)	**0.08 (0.03, 0.13)**	0.08 (−0.13, 0.29)	**Moderate carbohydrate**				
−0.08 (−0.13, −0.03)	−0.03 (−0.06, 0.00)	**0.08 (0.05, 0.12)**	0.08 (−0.13, 0.29)	0.00 (−0.03, 0.04)	**Low fat**			
−0.04 (−0.11, 0.03)	0.01 (−0.04, 0.07)	**0.12 (0.07, 0.18)**	0.12 (−0.09, 0.33)	0.04 (−0.02, 0.10)	0.04 (0.00, 0.09)	**High protein**		
−0.13 (−0.20, −0.06)	**−0.08 (−0.13, −0.02)**	0.03 (−0.02, 0.09)	0.03 (−0.18, 0.24)	−0.05 (−0.11, 0.01)	−0.05 (−0.10, 0.00)	**−0.09 (−0.15, −0.02)**	**Low carbohydrate**	
−0.07 (−0.14, −0.01)	−0.02 (−0.07, 0.03)	**0.09 (0.04, 0.15)**	0.09 (−0.12, 0.30)	0.01 (−0.04, 0.06)	0.01 (−0.03, 0.05)	−0.03 (−0.09, 0.03)	0.06 (−0.01, 0.12)	**Control**

The values below the dietary approaches correspond to the mean difference in HDL (mmol/L) between the column and the row (e.g. the MD HDL between the Mediterranean diet and the control diet is 0.09 mmol/L). The effect was interpreted as statistically significant if the 95% CIs did not include the null value (highlighted in bold letters)

GI/GL Glycaemic index/glycaemic load; *HDL* high-density lipoprotein cholesterol

Table 3 League table showing the results of the network meta-analysis comparing the effects (mean difference: MD) of all dietary approaches and 95% confidence intervals (95% CI) on TGs (mmol/L) in individuals with T2D (n = 52)

Vegetarian								
−0.02 (−0.31, 0.27)	**Low GI/GL**							
0.33 (−0.02, 0.69)	**0.35 (0.05, 0.66)**	**Mediterranean**						
0.50 (−0.13, 1.13)	0.52 (−0.08, 1.12)	0.17 (−0.47, 0.80)	**Palaeolithic**					
0.13 (−0.15, 0.42)	0.15 (−0.05, 0.35)	−0.20 (−0.50, 0.10)	−0.36 (−0.96, 0.23)	**Moderate carbohydrate**				
0.00 (−0.24, 0.24)	0.02 (−0.14, 0.18)	**−0.33 (−0.59, −0.08)**	−0.50 (−1.08, 0.08)	−0.14 (−0.28, 0.01)	**Low fat**			
0.04 (−0.26, 0.34)	0.06 (−0.18, 0.30)	−0.29 (−0.61, 0.02)	−0.46 (−1.07, 0.15)	−0.09 (−0.33, 0.14)	0.04 (−0.14, 0.23)	**High protein**		
0.28 (−0.03, 0.59)	**0.30 (0.06, 0.55)**	−0.05 (−0.38, 0.28)	−0.22 (−0.83, 0.40)	0.15 (−0.09, 0.38)	**0.28 (0.09, 0.48)**	0.24 (−0.03, 0.51)	**Low carbohydrate**	
−0.08 (−0.38, 0.22)	−0.06 (−0.29, 0.18)	**−0.41 (−0.72, −0.10)**	−0.58 (−1.18, 0.03)	−0.21 (−0.43, 0.00)	−0.08 (−0.25, 0.10)	−0.12 (−0.37, 0.14)	**−0.36 (−0.62, −0.10)**	**Control**

The values below the dietary approaches correspond to the mean difference in TGs (mmol/L) between the column and the row (e.g. the MD TGs between the Mediterranean diet and the control diet is −0.41 mmol/L). The effect was interpreted as statistically significant if the 95% CIs did not include the null value (highlighted in bold letters)

GI/GL Glycaemic index/glycaemic load; *TG* triglycerides

SUCRA

The SUCRA-values for each outcome (LDL, HDL, TGs) and the summary SUCRA-scores for each diet are summarized in Table 4. For LDL, the vegetarian diet scored the highest SUCRA-value (88%), followed by the low GI/GL diet (77%) and the moderate carbohydrate diet (67%). For HDL, the Mediterranean diet reached the highest SUCRA-value (92%), followed by the low carbohydrate diet (80%) and the Palaeolithic diet (76%). The latter also achieved

Table 4 SUCRA ranking for the dietary approaches by outcome (LDL, HDL and TGs) and in summary

Rank	LDL	SUCRA (%)	HDL	SUCRA (%)	TG	SUCRA (%)	Summary of LDL, HDL and TG	SUCRA (%)
1	Vegetarian	88	Mediterranean	92	Palaeolithic	88	Mediterranean	79
2	Low GI/GL	77	Low carbohydrate	80	Mediterranean	84	Palaeolithic	73
3	Moderate carbohydrate	67	Palaeolithic	76	Low carbohydrate	80	Low carbohydrate	62
4	Mediterranean	61	Moderate carbohydrate	54	Moderate carbohydrate	61	Moderate carbohydrate	61
5	Palaeolithic	55	Low fat	53	High protein	39	Low GI/GL	43
6	Low fat	41	Control	44	Vegetarian	30	Low fat	41
7	Low carbohydrate	25	Low GI/GL	27	Low fat	29	Vegetarian	40
8	High protein	21	High protein	20	Low GI/GL	26	High protein	27
9	Control	15	Vegetarian	3	Control	13	Control	24

GI/GL Glycaemic index/glycaemic load; *LDL* low-density lipoprotein cholesterol; *HDL* high-density lipoprotein cholesterol; *TG* triglycerides

the highest SUCRA-score for TGs (88%), followed by the Mediterranean diet (84%) and the low carbohydrate diet (80%). According to the summary SUCRA ranking for the three outcomes combined, the most beneficial approach regarding the management of diabetic dyslipidaemia was the Mediterranean diet (79%), followed by the Palaeolithic (73%) and the low carbohydrate diet (62%). However, the results regarding the Palaeolithic diet need to be interpreted with caution, because only one trial was available for this diet.

Inconsistency

The side-splitting approach showed inconsistency for the comparison of the low fat diet with the low carbohydrate diet ($p = 0.035$) (Supplementary Table S14) regarding LDL, and for the comparisons between the control diet with the low fat diet ($p = 0.031$) or the moderate carbohydrate diet ($p = 0.023$) regarding HDL, respectively, (Supplementary Table S15). For TGs, inconsistency was observed with the side-splitting approach for the comparison between the moderate carbohydrate and low carbohydrate diet ($p = 0.034$) (Supplementary Table S16). There was no indication for inconsistency with the design by treatment approach for any of the outcomes ($p_{LDL} = 0.517$, $p_{HDL} = p = 0.322$ and $p_{TG} = 0.451$). The loop-specific approach showed no inconsistency for LDL (Supplementary Fig. S7), borderline inconsistency for the loop between the low fat, moderate carbohydrate and, control diet regarding HDL (Supplementary Fig. S8), and inconsistency in the loop between the low carbohydrate, moderate carbohydrate, and low GI/GL diet regarding TGs (Supplementary Fig. S9).

Sensitivity analyses

The results for the sensitivity analyses are shown in Supplementary Tables S5-13. For LDL, when excluding studies with high risk of bias, the results of the main analysis were in general confirmed (Supplementary Table S5). When including only long-term trials (≥ 12 months), the effects pointed to the same directions, were stronger for the effects between the vegetarian diet and LDL, but were in general less precisely estimated (Supplementary Table S8). Additionally, including only trials in patients with a diabetes duration of ≥ 5 years, led to stronger LDL reductions of at least 25% for the vegetarian diet, for example compared to the control diet [$MD_{main\ analysis}$ (95% CI): -0.33 (-0.55, -0.12) mmol/L vs. $MD_{sensitivity\ analysis}$ (95% CI): -0.42 (-0.82, -0.02) mmol/L] (Supplementary Table S11). Regarding HDL, excluding studies with high risk of bias, attenuated the results regarding the vegetarian diet (Supplementary Table S6), while the direction of its effects changed, and the estimates were imprecise, when considering only long-term trials (≥ 12 months) (Supplementary Table S9). When including only trials in patients with a diabetes duration of ≥ 5 years, the beneficial effects of the Mediterranean diet regarding HDL increase were enhanced by at least 30%, for example compared to the control diet [$MD_{main\ analysis}$ (95% CI): 0.09 (0.04, 0.15) mmol/L vs. $MD_{sensitivity\ analysis}$ (95% CI): 0.16 (0.05, 0.27) mmol/L], but were attenuated and less precisely estimated for the vegetarian diet (Supplementary Table S12). Regarding TGs, excluding studies with high risk of bias generally confirmed the results of the main analysis (Supplementary Table S7), while the effects of the Mediterranean diet were stronger (change of > 15%, for example compared to the control diet [$MD_{main\ analysis}$ (95% CI): -0.41 (-0.72, -0.10) mmol/L vs. $MD_{sensitivity\ analysis}$ (95% CI): -0.48 (-0.82, -0.13) mmol/L]) in long-term trials (≥ 12 months) (Supplementary Table S10). Including

only studies in patients with a mean diabetes duration of ≥ 5 years, the effects pointed to the same direction compared to our main analysis, but were generally stronger (Supplementary Table S13).

In meta-regression models, a positive trend between differences in weight change and changes in TG was observed [0.039 mmol/L higher TG levels per 1 kg mean difference in weight change (95% CI: 0.004, 0.073 mmol/L) $p_{TG} = 0.030$] (Supplementary Fig. S12). For changes in LDL [0.008 mmol/L higher LDL per 1 kg mean difference in weight change (95% CI: − 0.023, 0.040 mmol/L), $p_{LDL} = 0.586$] (Supplementary Fig. S10), and HDL [0.002 mmol/L descrease in HDL per 1 kg mean difference in weight change (95% CI: − 0.012, 0.009 mmol/L), $p_{HLD} = 0.756$] (Supplementary Fig. S11), the picture was less clear.

Publication bias

No asymmetry was detected in the comparison adjusted funnel plots for any of the three outcomes (LDL, HDL, and TGs) (Supplementary Fig. S13-15).

Credibility of evidence

For the significant associations regarding LDL and HDL, the credibility of evidence was mainly low. Exceptions were the comparisons of the low GI/GL and the moderate carbohydrate diet with the control diet as well as for the high protein compared to the low GI/GL diet regarding LDL, and the comparisons of the Mediterranean diet with the low GI/GL, high protein, low fat, and moderate carbohydrate diet regarding HDL, where the credibility of evidence was moderate (Supplementary Tables S17 and S18). As for TGs, the credibility of evidence was moderate for the Mediterranean diet compared to the control, the low GI/GL and the low fat diet, and low for the low carbohydrate compared to the control, the low GI/GL and the low fat diet (Supplementary Table S19).

Discussion

In our NMA, we ranked nine dietary approaches (low fat, vegetarian, Mediterranean, high protein, moderate carbohydrate, low carbohydrate, control, low GI/GL, and Palaeolithic diet) according to their effects on blood lipid control (LDL, HDL and TGs) in patients with T2D. The dietary interventions showed slightly different effects regarding the three outcomes. The vegetarian, the low GI/GL, and the moderate carbohydrate diet yielded lower LDL levels compared to the control diet, with most robust findings for the vegetarian diet, for which stronger effects were observed

including only long-term trials (≥ 12 months) and studies in patients with a mean diabetes duration of ≥ 5 years. However, the vegetarian diet led to an adverse decrease in HDL levels, though this effect was attenuated excluding studies with high risk of bias and including only studies in patients with a mean diabetes duration of ≥ 5 years. In the other sensitivity analyses the results remained robst, with the Mediterranean diet leading to a beneficial increase of HDL levels compared to other dietary interventions, including the control diet. Strongest reductions of TG levels were seen for the Mediterranean and the low carbohydrate diet compared to the control diet. Furthermore, these reductions were higher in sensitivity analyses including only studies in patients with a mean diabetes duration of ≥ 5 years. The ranking according to the summary SUCRA for the three outcomes combined showed the highest score for the Mediterranean diet, followed by the Palaeolithic and the low carbohydrate diet, indicating that these three dietary interventions were the most effective ones regarding blood lipid control. The credibility of evidence for these effects was very low to moderate.

The ADA recommends "healthful eating patterns", for example a Mediterranean or a vegetarian diet, regarding the management of T2D, depending on each patient's individual goals [15]. However, no dietary intervention is specified and thus, it remains unclear which is the most beneficial dietary approach regarding the management of dyslipidaemia in T2D. It is in accordance with our results, that healthy dietary patterns like the Mediterranean and the vegetarian diet are effective in managing blood lipids in T2D. However, our overall SUCRA-ranking points towards the Mediterranean diet as the most beneficial diet to manage diabetic dyslipidaemia.

Several pairwise meta-analyses of RCTs examined the effect of different dietary approaches on blood lipid control. In these, the only effect on LDL was observed for a vegetarian diet compared to a control diet, which lowered LDL significantly in individuals with diabetes, obesity, or at risk for CVD, but not in healthy individuals [20, 103, 104]. This is in line with our results in T2D patients. While the meta-analyses of trials focussed on LDL as a surrogate marker for CVD, epidemiological studies also show that the vegetarian diet was favourably related to hard endpoints, such as CVD mortality [8, 105]. Our observed effects of the low GI/GL diet on LDL were confirmed in one meta-analysis [106], but not in another [16] including diabetes patients. However, the latter only included three trials (all of them showing no beneficial effects), as compared to eight trials in our NMA and 27 in the first meta-analysis, including trials which found significant beneficial effects of a low GI/GL diet on LDL [106]. A meta-analysis in overweight and obese individuals without diabetes also found a decrease in LDL for a low GI/GL diet [107]. Although our comparison of the low GI/GL

diet with the control diet is based solely on indirect evidence, the credibility of evidence for this association was moderate. As for HDL, previous pairwise meta-analyses observed a beneficial effect for a Mediterranean diet compared to a control diet [16, 19], which concurs with our results. However, a meta-analysis including trials in healthy individuals as well as participants with cardiovascular risk factors, such as diabetes and the metabolic syndrome, found no significant effect of the Mediterranean diet on HDL [108]. We also identified the Palaeolithic and low carbohydrate diet as effective dietary strategies to improve HDL. However, the estimates failed to reach statistical significance. For the Palaeolithic diet, this might be explained by the low numbers of studies (n = 1). In line with our findings, pairwise meta-analyses observed beneficial effects regarding HDL comparing a low carbohydrate diet to a control diet [16, 17] and a low fat, high carbohydrate diet [109] in diabetes patients. A meta-analysis including trials in individuals with and without T2D also found an increase in HDL for a low carbohydrate diet compared to a low fat diet [110]. In accordance with our data, previous meta-analyses of trials found that TG levels were lowered comparing a Mediterranean diet to a control diet [19] and to a low fat diet [16] as well as a low fat diet to a low carbohydrate diet [109] in T2D patients. This was also observed in a meta-analysis including studies in healthy individuals and participants with cardiometabolic risk factors when comparing the Mediterranean diet to a control diet [108]. In epidemiological studies, the Mediterranean diet was also found to be associated with reduced relative risk of harder CVD endpoints, such as CVD mortality in individuals with and without T2D [111, 112]. Additionally, we observed a reduction of TG levels for the low carbohydrate diet compared to the control diet. These findings were confirmed by two previous reports [17, 113], but not in another meta-analysis [16]. A meta-analysis including overweight and obese individuals with and without diabetes also found a significant reduction of TG levels comparing a low carbohydrate diet to a low fat diet [110]. Taken together, most of the evidence on dietary interventions and blood lipid control is derived from populations focussing on individuals with obesity, diabetes, or at cardiovascular risk, and limited evidence from healthy populations is available. The sparse evidence indicates that dietary interventions have a less important role in blood lipid metabolism in healthy individuals [114–116]. Thus, dietary interventions might have benefits especially for individuals with metabolic disorders, including diabetes. In the SUCRA ranking, the Mediterranean diet yielded the highest score, followed by the Palaeolithic and the low carbohydrate diet. In line with our results, Pan et al. [24] concluded in their NMA comparing five dietary approaches (Mediterranean, high carbohydrate, low carbohydrate, low fat, and control diet), that the Mediterranean diet was the best dietary approach to reduce

cardiovascular risk in T2D. In their report, the Mediterranean diet was ranked as the most effective dietary approach in reducing LDL and TGs. However, to increase HDL, the low carbohydrate and the low fat diet were more favourable than the Mediterranean diet, which does not concur with our findings. Discrepancies might be explained by the inclusion of more dietary approaches (nine in our NMA as opposed to five diets), the higher number of included studies (52 in our NMA compared to 10 trials) and the exclusion of short-term trials (< 3 months) in our NMA. Additionally, Pan et al. [24] did not calculate an overall ranking.

The mediating effect of weight loss, and especially the reduction of abdominal obesity, is likely to explain part of the effects of the Mediterranean, the Palaeolithic and the low carbohydrate diet on diabetic dyslipidaemia [17, 34, 117]. These diets have been shown to promote weight loss by inducing satiety and increasing energy expenditure [118–120]. In addition, in our analyses, we observed a positive association between differences in weight change and changes in TG, and there was indication for a change in LDL, but not in HDL.

However, further mechanisms are probably attributable to individual components of the diets [17, 117]. The consumption of mono-unsaturated fatty acids (MUFAs) and omega-3 fatty acids is typically high in the Mediterranean diet because of high intake of extra virgin olive oil and fish [33] and has also been found to be increased in the low carbohydrate diet [121]. MUFAs have been shown to reduce TGs and increase HDL levels in patients with and without T2D, but had no significant effect on LDL levels [18, 122–124]. Supplementation with omega-3 fatty acids led to a, though only small, decrease in LDL and TG levels in patients with T2D [125]. This might partly explain the mainly beneficial effect of the Mediterranean and the low carbohydrate diet mainly on HDL and TGs, with non-significant effects on LDL. Moreover, inflammatory adipokines contribute to the genesis of dyslipidaemia in T2D [126]. Thus, the higher intake of anti-inflammatory and anti-oxidant compounds via high consumption of fruit and vegetables in all three diets is also likely to play a role in their beneficial effects on blood lipids [117]. In this context, especially the Mediterranean diet offers benefits through higher intake of dietary polyphenols found in olive oil, nuts, legumes and red wine [117]. Furthermore, since there is a reciprocal relation between dyslipidaemia and a disrupted glucose metabolism, beneficial effects of these dietary patterns on glycaemic control [25] may interact with blood lipid levels [4, 6, 7].

The role of HDL in patients with T2D is discussed controversially. Recent evidence suggests that HDL loses its anti-inflammatory properties in the pathological state of T2D [127]. Additionally, genetic information in the general population showed no association between HDL and coronary heart disease (CHD) [128]. However, other studies

indicate an inverse association between HDL levels and risk of chronic kidney disease in patients with T2D [11, 12]. In contrast, the role of LDL and TGs is less ambiguous. Higher values of LDL and TGs have been shown to be associated with increased risk for cardiovascular events and mortality [8, 9] as well as microvascular complications (diabetic neuropathy, nephropathy and retinopathy) [10] in patients with T2D. Additionally, genetic information in the general population showed a clear association of LDL and TGs with CHD [128].

Strengths and limitations

The strengths of this study include the use of novel NMA methods, which enabled us to compare and rank nine dietary approaches according to their efficacy regarding blood lipid control in T2D. Our NMA is the most comprehensive report, including more than 50 trials, which had an intervention period of at least 3 months or even more (22 trials with intervention period ≥ 12 months). Further strengths are the evaluation of inconsistency, the assessment of the credibility of evidence for each comparison, and the a priori published protocol [26].

A limitation of this study is the heterogeneous definition for the different dietary approaches. Whenever possible, we adopted the classifications from the original studies. However, because this was not always possible for dietary approaches defined by their nutrient composition due to overlaps between the approaches, they were categorized into a priori defined classes of dietary approaches as described previously [25]. Nevertheless, overlaps between some dietary approaches cannot be ruled out. For example, a vegetarian diet would also fulfil the criteria for a low fat diet, but in contrast, low fat diets could not be classified always as vegetarian diets. However, we did not recategorize the dietary approaches because each dietary approach could only be assigned to one intervention arm. Furthermore, the analyses of the trials were based on the intended randomization design and not on the actual adherence to the respective diets. Additionally, because only five RCTs with no reduced energy intake and weight loss were included in our NMA, no stratified analyses for energy balance were possible to examine the mediating effect of energy restriction on our results. However, we conducted univariate meta-regression analyses to investigate the association between differences in weight change and changes in blood lipids. Furthermore, for most RCTs, no information on existing co-morbidities and severity of diabetes was available and the information on the intake of hypoglycaemic drugs, antihypertensive, and lipid lowering medication differed between studies. Therefore, no sensitivity analysis considering these aspects was possible. Another limitation is the quality of the included studies. Seven out of the 52 included trials were of high risk of bias due to lack of allocation concealment and blinding. This was included in the credibility of evidence evaluation and lowered the confidence in the estimates, together with concerns due to indirectness (contribution to the study effects came mainly from indirect comparisons) and imprecision. Additionally, the results on the Palaeolithic diet need to be interpreted with caution because only one trial was available for this dietary approach. Furthermore, we only examined surrogate markers as endpoints and further research on hard endpoints, such as diabetic complications and mortality, is warranted.

Conclusions

According to our NMA, the Mediterranean diet was the most effective diet to manage diabetic dyslipidaemia. However, the credibility of evidence for the results was very low to moderate. Nevertheless, our findings could give a first direction towards dietary recommendations for the management of blood lipid control in patients with T2D.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

1. International Diabetes Federation. IDF Diabetes Atlas, 8th Edition2017 2017.
2. Jacobs E, Hoyer A, Brinks R, Icks A, Kuss O, Rathmann W. Healthcare costs of type 2 diabetes in Germany. Diabet Med. 2017;34(6):855–61. https://doi.org/10.1111/dme.13336.
3. WHO. Global report on diabetes. Geneva: World Health Organization 2016.
4. Jaiswal M, Schinske A, Pop-Busui R. Lipids and lipid management in diabetes. Best Pract Res Clin Endocrinol Metab. 2014;28(3):325–38. https://doi.org/10.1016/j.beem.2013.12.001.
5. Chahil TJ, Ginsberg HN. Diabetic dyslipidemia. Endocrinol Metab Clin North Am. 2006;35(3):491–510. https://doi.org/10.1016/j.ecl.2006.06.002 **vii–viii**.

6. Erion DM, Park HJ, Lee HY. The role of lipids in the pathogenesis and treatment of type 2 diabetes and associated co-morbidities. BMB Rep. 2016;49(3):139–48.

7. Bardini G, Rotella CM, Giannini S. Dyslipidemia and diabetes: reciprocal impact of impaired lipid metabolism and beta-cell dysfunction on micro- and macrovascular complications. Rev Diabet Stud. 2012;9(2–3):82–93. https://doi.org/10.1900/RDS.2012.9.82.

8. Wang Y, Lammi-Keefe CJ, Hou L, Hu G. Impact of low-density lipoprotein cholesterol on cardiovascular outcomes in people with type 2 diabetes: a meta-analysis of prospective cohort studies. Diabetes Res Clin Pract. 2013;102(1):65–75. https://doi.org/10.1016/j.diabres.2013.07.009.

9. Vazquez-Benitez G, Desai JR, Xu S, Goodrich GK, Schroeder EB, Nichols GA, et al. Preventable major cardiovascular events associated with uncontrolled glucose, blood pressure, and lipids and active smoking in adults with diabetes with and without cardiovascular disease: a contemporary analysis. Diabetes Care. 2015;38(5):905–12. https://doi.org/10.2337/dc14-1877.

10. Toth PP, Simko RJ, Palli SR, Koselleck D, Quimbo RA, Cziraky MJ. The impact of serum lipids on risk for microangiopathy in patients with type 2 diabetes mellitus. Cardiovasc Diabetol. 2012;11:109. https://doi.org/10.1186/1475-2840-11-109.

11. Russo GT, De Cosmo S, Viazzi F, Pacilli A, Ceriello A, Genovese S, et al. Plasma triglycerides and HDL-C levels predict the development of diabetic kidney disease in subjects with type 2 diabetes: the AMD annals initiative. Diabetes Care. 2016;39(12):2278–87. https://doi.org/10.2337/dc16-1246.

12. Zoppini G, Targher G, Chonchol M, Perrone F, Lippi G, Muggeo M. Higher HDL cholesterol levels are associated with a lower incidence of chronic kidney disease in patients with type 2 diabetes. Nutr Metab Cardiovasc Dis. 2009;19(8):580–6. https://doi.org/10.1016/j.numecd.2008.11.003.

13. Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care. 2014;37(Suppl 1):S120–43. https://doi.org/10.2337/dc14-S120.

14. Dyson PA, Twenefour D, Breen C, Duncan A, Elvin E, Goff L, et al. Diabetes UK evidence-based nutrition guidelines for the prevention and management of diabetes. Diabet Med. 2018;35(5):541–7. https://doi.org/10.1111/dme.13603.

15. American Diabetes A. 5. Lifestyle management: standards of medical care in diabetes—2019. Diabetes Care. 2019;42(Suppl 1):S46–60. https://doi.org/10.2337/dc19-s005.

16. Ajala O, English P, Pinkney J. Systematic review and meta-analysis of different dietary approaches to the management of type 2 diabetes. Am J Clin Nutr. 2013;97(3):505–16. https://doi.org/10.3945/ajcn.112.042457.

17. Meng Y, Bai H, Wang S, Li Z, Wang Q, Chen L. Efficacy of low carbohydrate diet for type 2 diabetes mellitus management: a systematic review and meta-analysis of randomized controlled trials. Diabetes Res Clin Pract. 2017;131:124–31. https://doi.org/10.1016/j.diabres.2017.07.006.

18. Schwingshackl L, Hoffmann G. Comparison of the long-term effects of high-fat v. low-fat diet consumption on cardiometabolic risk factors in subjects with abnormal glucose metabolism: a systematic review and meta-analysis. Br J Nutr. 2014;111(12):2047–58. https://doi.org/10.1017/s0007114514000464.

19. Huo R, Du T, Xu Y, Xu W, Chen X, Sun K, et al. Effects of mediterranean-style diet on glycemic control, weight loss and cardiovascular risk factors among type 2 diabetes individuals: a meta-analysis. Eur J Clin Nutr. 2015;69(11):1200–8. https://doi.org/10.1038/ejcn.2014.243.

20. Viguiliouk E, Kendall CW, Kahleova H, Rahelic D, Salas-Salvado J, Choo VL, et al. Effect of vegetarian dietary patterns on cardiometabolic risk factors in diabetes: a systematic review and meta-analysis of randomized controlled trials. Clin Nutr. 2018. https://doi.org/10.1016/j.clnu.2018.05.032.

21. Ludwig DS, Willett WC, Volek JS, Neuhouser ML. Dietary fat: From foe to friend? Science. 2018;362(6416):764–70. https://doi.org/10.1126/science.aau2096.

22. Schwingshackl L, Buyken A, Chaimani A. Network meta-analysis reaches nutrition research. Eur J Nutr. 2018. https://doi.org/10.1007/s00394-018-1849-0.

23. Schwingshackl L, Schwarzer G, Rucker G, Meerpohl JJ. Perspective: network meta-analysis reaches nutrition research: current status, scientific concepts, and future directions. Adv Nutr. 2019. https://doi.org/10.1093/advances/nmz036.

24. Pan B, Wu Y, Yang Q, Ge L, Gao C, Xun Y, et al. The impact of major dietary patterns on glycemic control, cardiovascular risk factors, and weight loss in patients with type 2 diabetes: a network meta-analysis. J Evid Based Med. 2018. https://doi.org/10.1111/jebm.12312.

25. Schwingshackl L, Chaimani A, Hoffmann G, Schwedhelm C, Boeing H. A network meta-analysis on the comparative efficacy of different dietary approaches on glycaemic control in patients with type 2 diabetes mellitus. Eur J Epidemiol. 2018;33(2):157–70. https://doi.org/10.1007/s10654-017-0352-x.

26. Schwingshackl L, Chaimani A, Hoffmann G, Schwedhelm C, Boeing H. Impact of different dietary approaches on glycemic control and cardiovascular risk factors in patients with type 2 diabetes: a protocol for a systematic review and network meta-analysis. Syst Rev. 2017;6(1):57. https://doi.org/10.1186/s13643-017-0455-1.

27. Hutton B, Salanti G, Caldwell DM, Chaimani A, Schmid CH, Cameron C, et al. The PRISMA extension statement for reporting of systematic reviews incorporating network meta-analyses of health care interventions: checklist and explanations. Ann Intern Med. 2015;162(11):777–84. https://doi.org/10.7326/M14-2385.

28. Chaimani A, Caldwell DM, Li T, Higgins JPT, Salanti G. Additional considerations are required when preparing a protocol for a systematic review with multiple interventions. J Clin Epidemiol. 2017;83:65–74. https://doi.org/10.1016/j.jclinepi.2016.11.015.

29. Schwingshackl L, Hoffmann G. Comparison of effects of long-term low-fat versus high-fat diets on blood lipid levels in overweight or obese patients: a systematic review and meta-analysis. J Acad Nutr Diet. 2013;113(12):1640–61. https://doi.org/10.1016/j.jand.2013.07.010.

30. Schwingshackl L, Hoffmann G. Long-term effects of low-fat diets either low or high in protein on cardiovascular and metabolic risk factors: a systematic review and meta-analysis. Nutr J. 2013;12:48. https://doi.org/10.1186/1475-2891-12-48.

31. Schwingshackl L, Hoffmann G. Long-term effects of low glycemic index/load versus high glycemic index/load diets on parameters of obesity and obesity-associated risks: a systematic review and meta-analysis. Nutr Metab Cardiovasc Dis. 2013;23(8):699–706. https://doi.org/10.1016/j.numecd.2013.04.008.

32. Haider LM, Schwingshackl L, Hoffmann G, Ekmekcioglu C. The effect of vegetarian diets on iron status in adults: a systematic review and meta-analysis. Crit Rev Food Sci Nutr. 2018;58(8):1359–74. https://doi.org/10.1080/10408398.2016.1259210.

33. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348(26):2599–608. https://doi.org/10.1056/NEJMoa025039.

34. Jonsson T, Granfeldt Y, Ahren B, Branell UC, Palsson G, Hansson A, et al. Beneficial effects of a Paleolithic diet on cardiovascular risk factors in type 2 diabetes: a randomized

cross-over pilot study. Cardiovasc Diabetol. 2009;8:35. https://doi.org/10.1186/1475-2840-8-35.

35. Brehm BJ, Lattin BL, Summer SS, Boback JA, Gilchrist GM, Jandacek RJ, et al. One-year comparison of a high-monounsaturated fat diet with a high-carbohydrate diet in type 2 diabetes. Diabetes Care. 2009;32(2):215–20.

36. Heilbronn LK, Noakes M, Clifton PM. Effect of energy restriction, weight loss, and diet composition on plasma lipids and glucose in patients with type 2 diabetes. Diabetes Care. 1999;22(6):889–95.

37. Krebs JD, Elley CR, Parry-Strong A, Lunt H, Drury PL, Bell DA, et al. The diabetes excess weight loss (DEWL) trial: a randomised controlled trial of high-protein versus high-carbohydrate diets over 2 years in type 2 diabetes. Diabetologia. 2012;55(4):905–14.

38. Larsen RN, Mann NJ, Maclean E, Shaw JE. The effect of high-protein, low-carbohydrate diets in the treatment of type 2 diabetes: a 12 month randomised controlled trial. Diabetologia. 2011;54(4):731–40.

39. Milne RM, Mann JI, Chisholm AW, Williams SM. Long-term comparison of three dietary prescriptions in the treatment of NIDDM. Diabetes Care. 1994;17(1):74–80.

40. Shige H, Nestel P, Sviridov D, Noakes M, Clifton P. Effect of weight reduction on the distribution of apolipoprotein AI in high-density lipoprotein subfractions in obese non-insulin-dependent diabetic subjects. Metabolism. 2000;49(11):1453–9.

41. Tay J, Luscombe-Marsh ND, Thompson CH, Noakes M, Buckley JD, Wittert GA, et al. Comparison of low- and high-carbohydrate diets for type 2 diabetes management: a randomized trial. Am J Clin Nutr. 2015;102(4):780–90.

42. Watson N, Dyer K, Buckley J, Brinkworth G, Coates A, Parfitt G, et al. Effects of low-fat diets differing in protein and carbohydrate content on cardiometabolic risk factors during weight loss and weight maintenance in obese adults with type 2 diabetes. Nutrients. 2016;8(5):289.

43. Brinkworth GD, Noakes M, Parker B, Foster P, Clifton PM. Long-term effects of advice to consume a high-protein, low-fat diet, rather than a conventional weight-loss diet, in obese adults with type 2 diabetes: one-year follow-up of a randomised trial. Diabetologia. 2004;47(10):1677–86.

44. Parker B, Noakes M, Luscombe N, Clifton P. Effect of a high-protein, high-monounsaturated fat weight loss diet on glycemic control and lipid levels in type 2 diabetes. Diabetes Care. 2002;25(3):425–30.

45. Ceriello A, Esposito K, La SL, Pujadas G, De NV, Testa R, et al. The protective effect of the Mediterranean diet on endothelial resistance to GLP-1 in type 2 diabetes: a preliminary report. Cardiovasc Diabetol. 2014;13:140.

46. Daly ME, Paisey R, Paisey R, Millward BA, Eccles C, Williams K, et al. Short-term effects of severe dietary carbohydrate-restriction advice in type 2 diabetes—a randomized controlled trial. Diabet Med. 2006;23(1):15–20.

47. Davis NJ, Tomuta N, Schechter C, Isasi CR, Segal-Isaacson CJ, Stein D, et al. Comparative study of the effects of a 1-year dietary intervention of a low-carbohydrate diet versus a low-fat diet on weight and glycemic control in type 2 diabetes. Diabetes Care. 2009;32(7):1147–52.

48. de Bont AJ, Baker IA, St Leger AS, Sweetnam PM, Wragg KG, Stephens SM, et al. A randomised controlled trial of the effect of low fat diet advice on dietary response in insulin independent diabetic women. Diabetologia. 1981;21(6):529–33.

49. Esposito K, Maiorino MI, Ciotola M, Di PC, Scognamiglio P, Gicchino M, et al. Effects of a Mediterranean-style diet on the need for antihyperglycemic drug therapy in patients with newly diagnosed type 2 diabetes: a randomized trial. Ann Intern Med. 2009;151(5):306–14.

50. Fabricatore AN, Wadden TA, Ebbeling CB, Thomas JG, Stallings VA, Schwartz S, et al. Targeting dietary fat or glycemic load in the treatment of obesity and type 2 diabetes: a randomized controlled trial. Diabetes Res Clin Pract. 2011;92(1):37–45.

51. Guldbrand H, Dizdar B, Bunjaku B, Lindstrom T, Bachrach-Lindstrom M, Fredrikson M, et al. In type 2 diabetes, randomisation to advice to follow a low-carbohydrate diet transiently improves glycaemic control compared with advice to follow a low-fat diet producing a similar weight loss. Diabetologia. 2012;55(8):2118–27.

52. Li X, Cai X, Ma X, Jing L, Gu J, Bao L, et al. Short- and long-term effects of wholegrain oat intake on weight management and glucolipid metabolism in overweight type-2 diabetics: a randomized control trial. Nutrients. 2016;8(9):549.

53. Iqbal N, Vetter ML, Moore RH, Chittams JL, Dalton-Bakes CV, Dowd M, et al. Effects of a low-intensity intervention that prescribed a low-carbohydrate versus a low-fat diet in obese, diabetic participants. Obesity. 2010;18(9):1733–8.

54. McLaughlin T, Carter S, Lamendola C, Abbasi F, Schaaf P, Basina M, et al. Clinical efficacy of two hypocaloric diets that vary in overweight patients with type 2 diabetes: comparison of moderate fat versus carbohydrate reductions. Diabetes Care. 2007;30(7):1877–9.

55. Nicholson AS, Sklar M, Barnard ND, Gore S, Sullivan R, Browning S. Toward improved management of NIDDM: a randomized, controlled, pilot intervention using a lowfat, vegetarian diet. Prev Med. 1999;29(2):87–91.

56. Rock CL, Flatt SW, Pakiz B, Taylor KS, Leone AF, Brelje K, et al. Weight loss, glycemic control, and cardiovascular disease risk factors in response to differential diet composition in a weight loss program in type 2 diabetes: a randomized controlled trial. Diabetes Care. 2014;37(6):1573–80.

57. Walker KZ, O'Dea K, Nicholson GC, Muir JG. Dietary composition, body weight, and NIDDM. Comparison of high-fiber, high-carbohydrate, and modified-fat diets. Diabetes Care. 1995;18(3):401–3.

58. Kahleova H, Matoulek M, Malinska H, Oliyarnik O, Kazdova L, Neskudla T, et al. Vegetarian diet improves insulin resistance and oxidative stress markers more than conventional diet in subjects with type 2 diabetes. Diabet Med. 2011;28(5):549–59.

59. American Diabetes A. 2. Classification and diagnosis of diabetes: standards of medical care in diabetes—2018. Diabetes Care. 2018;41(Suppl 1):S13–27. https://doi.org/10.2337/dc18-s002.

60. Canadian Diabetes Association Clinical Practice Guidelines Expert C, Wharton S, Sharma AM, Lau DC. Weight management in diabetes. Can J Diabetes. 2013;37(Suppl 1):S82–6. https://doi.org/10.1016/j.jcjd.2013.01.025.

61. American Diabetes A. 7. Obesity management for the treatment of type 2 diabetes: standards of medical care in diabetes—2018. Diabetes Care. 2018;41(Suppl 1):S65–72. https://doi.org/10.2337/dc18-s007.

62. Higgins JP, Altman DG, Gotzsche PC, Juni P, Moher D, Oxman AD, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. BMJ. 2011;343:d5928. https://doi.org/10.1136/bmj.d5928.

63. Dyson PA, Beatty S, Matthews DR. A low-carbohydrate diet is more effective in reducing body weight than healthy eating in both diabetic and non-diabetic subjects. Diabet Med. 2007;24(12):1430–5.

64. Higgins JPT, Green S. Cochrane handbook for systematic reviewsof interventions version 5.1.0 [updated March 2011]. The Cochrane Collaboration, 2011. www.cochrane-handbook.org. Accessed 20 Aug 2016.

65. Chaimani A, Higgins JP, Mavridis D, Spyridonos P, Salanti G. Graphical tools for network meta-analysis in STATA. PLoS ONE.

2013;8(10):e76654. https://doi.org/10.1371/journal.pone.0076654.

66. Jansen JP, Naci H. Is network meta-analysis as valid as standard pairwise meta-analysis? It all depends on the distribution of effect modifiers. BMC Med. 2013;11:159. https://doi.org/10.1186/1741-7015-11-159.

67. Salanti G, Ades AE, Ioannidis JP. Graphical methods and numerical summaries for presenting results from multiple-treatment meta-analysis: an overview and tutorial. J Clin Epidemiol. 2011;64(2):163–71. https://doi.org/10.1016/j.jclinepi.2010.03.016.

68. Schwarzer G, Carpenter J, Ruecker G. Meta-analysis with R. 2015. http://meta-analysis-with-r.org. https://github.com/guido-s/netmeta. Accessed 20 May 2019.

69. Schwarzer G. netmeta: network meta-analysis using frequentist methods. 2019. https://github.com/guido-s/netmeta. Accessed 20 May 2019.

70. Salanti G, Del Giovane C, Chaimani A, Caldwell DM, Higgins JP. Evaluating the quality of evidence from a network meta-analysis. PLoS ONE. 2014;9(7):e99682. https://doi.org/10.1371/journal.pone.0099682.

71. Bucher HC, Guyatt GH, Griffith LE, Walter SD. The results of direct and indirect treatment comparisons in meta-analysis of randomized controlled trials. J Clin Epidemiol. 1997;50(6):683–91.

72. Dias S, Welton NJ, Caldwell DM, Ades AE. Checking consistency in mixed treatment comparison meta-analysis. Stat Med. 2010;29(7–8):932–44. https://doi.org/10.1002/sim.3767.

73. Higgins JP, Jackson D, Barrett JK, Lu G, Ades AE, White IR. Consistency and inconsistency in network meta-analysis: concepts and models for multi-arm studies. Res Synth Methods. 2012;3(2):98–110. https://doi.org/10.1002/jrsm.1044.

74. Jackson D, Barrett JK, Rice S, White IR, Higgins JP. A design-by-treatment interaction model for network meta-analysis with random inconsistency effects. Stat Med. 2014;33(21):3639–54. https://doi.org/10.1002/sim.6188.

75. Harbord RM, Higgins J. Meta-regression in Stata. Stata J. 2008;8(4):493–519.

76. Peters JL, Sutton AJ, Jones DR, Abrams KR, Rushton L. Contour-enhanced meta-analysis funnel plots help distinguish publication bias from other causes of asymmetry. J Clin Epidemiol. 2008;61(10):991–6. https://doi.org/10.1016/j.jclinepi.2007.11.010.

77. Andrews RC, Cooper AR, Montgomery AA, Norcross AJ, Peters TJ, Sharp DJ, et al. Diet or diet plus physical activity versus usual care in patients with newly diagnosed type 2 diabetes: the Early ACTID randomised controlled trial. Lancet. 2011;378(9786):129–39. https://doi.org/10.1016/S0140-6736(11)60442-X.

78. Barnard ND, Cohen J, Jenkins DJ, Turner-McGrievy G, Gloede L, Green A, et al. A low-fat vegan diet and a conventional diabetes diet in the treatment of type 2 diabetes: a randomized, controlled, 74-wk clinical trial. Am J Clin Nutr. 2009;89(5):1588S–96S.

79. Brand JC, Colagiuri S, Crossman S, Allen A, Roberts DC, Truswell AS. Low-glycemic index foods improve long-term glycemic control in NIDDM. Diabetes Care. 1991;14(2):95–101.

80. Brunerova L, Smejkalova V, Potockova J, Andel M. A comparison of the influence of a high-fat diet enriched in monounsaturated fatty acids and conventional diet on weight loss and metabolic parameters in obese non-diabetic and type 2 diabetic patients. Diabet Med. 2007;24(5):533–40.

81. Coppell KJ, Kataoka M, Williams SM, Chisholm AW, Vorgers SM, Mann JI. Nutritional intervention in patients with type 2 diabetes who are hyperglycaemic despite optimised drug treatment–Lifestyle Over and Above Drugs in Diabetes (LOADD) study: randomised controlled trial. BMJ. 2010;341:c3337. https://doi.org/10.1136/bmj.c3337.

82. Elhayany A, Lustman A, Abel R, Attal-Singer J, Vinker S. A low carbohydrate Mediterranean diet improves cardiovascular risk factors and diabetes control among overweight patients with type 2 diabetes mellitus: a 1-year prospective randomized intervention study. Diabetes Obes Metab. 2010;12(3):204–9.

83. Hockaday TD, Hockaday JM, Mann JI, Turner RC. Prospective comparison of modified fat-high-carbohydrate with standard low-carbohydrate dietary advice in the treatment of diabetes: one year follow-up study. Br J Nutr. 1978;39(2):357–62.

84. Huang MC, Hsu CC, Wang HS, Shin SJ. Prospective randomized controlled trial to evaluate effectiveness of registered dietitian-led diabetes management on glycemic and diet control in a primary care setting in Taiwan. Diabetes Care. 2010;33(2):233–9.

85. Itsiopoulos C, Brazionis L, Kaimakamis M, Cameron M, Best JD, O'Dea K, et al. Can the Mediterranean diet lower HbA1c in type 2 diabetes? Results from a randomized cross-over study. Nutr Metab Cardiovasc Dis. 2011;21(9):740–7.

86. Jenkins DJ, Kendall CW, McKeown-Eyssen G, Josse RG, Silverberg J, Booth GL, et al. Effect of a low-glycemic index or a high-cereal fiber diet on type 2 diabetes: a randomized trial. JAMA. 2008;300(23):2742–53.

87. Jenkins DJ, Kendall CW, Augustin LS, Mitchell S, Sahye-Pudaruth S, Blanco MS, et al. Effect of legumes as part of a low glycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus: a randomized controlled trial. Arch Intern Med. 2012;172(21):1653–60.

88. Jenkins DJ, Kendall CW, Vuksan V, Faulkner D, Augustin LS, Mitchell S, et al. Effect of lowering the glycemic load with canola oil on glycemic control and cardiovascular risk factors: a randomized controlled trial. Diabetes Care. 2014;37(7):1806–14.

89. Lee YM, Kim SA, Lee IK, Kim JG, Park KG, Jeong JY, et al. Effect of a brown rice based vegan diet and conventional diabetic diet on glycemic control of patients with type 2 diabetes: a 12-week randomized clinical trial. PLoS ONE. 2016;11(6):e0155918.

90. Liu H, Zhang M, Wu X, Wang C, Li Z. Effectiveness of a public dietitian-led diabetes nutrition intervention on glycemic control in a community setting in China. Asia Pac J Clin Nutr. 2015;24(3):525–32.

91. Luger M, Holstein B, Schindler K, Kruschitz R, Ludvik B. Feasibility and efficacy of an isocaloric high-protein versus standard diet on insulin requirement, body weight and metabolic parameters in patients with type 2 diabetes on insulin therapy. Exp Clin Endocrinol Diabetes. 2013;121(5):286–94.

92. Ma Y, Olendzki BC, Merriam PA, Chiriboga DE, Culver AL, Li W, et al. A randomized clinical trial comparing low-glycemic index versus ADA dietary education among individuals with type 2 diabetes. Nutrition. 2008;24(1):45–56.

93. Mishra S, Xu J, Agarwal U, Gonzales J, Levin S, Barnard ND. A multicenter randomized controlled trial of a plant-based nutrition program to reduce body weight and cardiovascular risk in the corporate setting: the GEICO study. Eur J Clin Nutr. 2013;67(7):718–24.

94. Pedersen E, Jesudason DR, Clifton PM. High protein weight loss diets in obese subjects with type 2 diabetes mellitus. Nutr Metab Cardiovasc Dis. 2014;24(5):554–62.

95. Saslow LR, Kim S, Daubenmier JJ, Moskowitz JT, Phinney SD, Goldman V, et al. A randomized pilot trial of a moderate carbohydrate diet compared to a very low carbohydrate diet in overweight or obese individuals with type 2 diabetes mellitus or prediabetes. PLoS ONE. 2014;9(4):e91027.

96. Sato J, Kanazawa A, Makita S, Hatae C, Komiya K, Shimizu T, et al. A randomized controlled trial of 130 g/day

low-carbohydrate diet in type 2 diabetes with poor glycemic control. Clin Nutr. 2017;36(4):992–1000.

97. Uusitupa M, Laitinen J, Siitonen O, Vanninen E, Pyorala K. The maintenance of improved metabolic control after intensified diet therapy in recent type 2 diabetes. Diabetes Res Clin Pract. 1993;19(3):227–38.

98. Westman EC, Yancy WS Jr, Mavropoulos JC, Marquart M, McDuffie JR. The effect of a low-carbohydrate, ketogenic diet versus a low-glycemic index diet on glycemic control in type 2 diabetes mellitus. Nutr Metab (Lond). 2008;5:36.

99. Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG, et al. The Canadian trial of carbohydrates in diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. Am J Clin Nutr. 2008;87(1):114–25.

100. Wycherley TP, Noakes M, Clifton PM, Cleanthous X, Keogh JB, Brinkworth GD. A high-protein diet with resistance exercise training improves weight loss and body composition in overweight and obese patients with type 2 diabetes. Diabetes Care. 2010;33(5):969–76.

101. Yamada Y, Uchida J, Izumi H, Tsukamoto Y, Inoue G, Watanabe Y, et al. A non-calorie-restricted low-carbohydrate diet is effective as an alternative therapy for patients with type 2 diabetes. Intern Med. 2014;53(1):13–9.

102. Yusof BN, Talib RA, Kamaruddin NA, Karim NA, Chinna K, Gilbertson H. A low-GI diet is associated with a short-term improvement of glycaemic control in Asian patients with type 2 diabetes. Diabetes Obes Metab. 2009;11(4):387–96.

103. Yokoyama Y, Levin SM, Barnard ND. Association between plant-based diets and plasma lipids: a systematic review and meta-analysis. Nutr Rev. 2017;75(9):683–98. https://doi.org/10.1093/nutrit/nux030.

104. Wang F, Zheng J, Yang B, Jiang J, Fu Y, Li D. Effects of vegetarian diets on blood lipids: a systematic review and meta-analysis of randomized controlled trials. J Am Heart Assoc. 2015;4(10):e002408. https://doi.org/10.1161/JAHA.115.002408.

105. Archundia Herrera MC, Subhan FB, Chan CB. Dietary patterns and cardiovascular disease risk in people with type 2 diabetes. Curr Obes Rep. 2017;6(4):405–13. https://doi.org/10.1007/s13679-017-0284-5.

106. Goff LM, Cowland DE, Hooper L, Frost GS. Low glycaemic index diets and blood lipids: a systematic review and meta-analysis of randomised controlled trials. Nutr Metab Cardiovasc Dis. 2013;23(1):1–10. https://doi.org/10.1016/j.numecd.2012.06.002.

107. Fleming P, Godwin M. Low-glycaemic index diets in the management of blood lipids: a systematic review and meta-analysis. Fam Pract. 2013;30(5):485–91. https://doi.org/10.1093/fampra/cmt029.

108. Garcia M, Bihuniak JD, Shook J, Kenny A, Kerstetter J, Huedo-Medina TB. The Effect of the traditional mediterranean-style diet on metabolic risk factors: a meta-analysis. Nutrients. 2016;8(3):168. https://doi.org/10.3390/nu8030168.

109. Kodama S, Saito K, Tanaka S, Maki M, Yachi Y, Sato M, et al. Influence of fat and carbohydrate proportions on the metabolic profile in patients with type 2 diabetes: a meta-analysis. Diabetes Care. 2009;32(5):959–65. https://doi.org/10.2337/dc08-1716.

110. Mansoor N, Vinknes KJ, Veierod MB, Retterstol K. Effects of low-carbohydrate diets v. low-fat diets on body weight and cardiovascular risk factors: a meta-analysis of randomised controlled trials. Br J Nutr. 2016;115(3):466–79. https://doi.org/10.1017/s0007114515004699.

111. Bonaccio M, Di Castelnuovo A, Costanzo S, Persichillo M, De Curtis A, Donati MB, et al. Adherence to the traditional Mediterranean diet and mortality in subjects with diabetes. Prospective results from the MOLI-SANI study. Eur J Prev Cardiol. 2016;23(4):400–7. https://doi.org/10.1177/2047487315569409.

112. Grosso G, Marventano S, Yang J, Micek A, Pajak A, Scalfi L, et al. A comprehensive meta-analysis on evidence of Mediterranean diet and cardiovascular disease: are individual components equal? Crit Rev Food Sci Nutr. 2017;57(15):3218–32. https://doi.org/10.1080/10408398.2015.1107021.

113. Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS Jr, Brehm BJ, et al. Effects of low-carbohydrate versus low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. Arch Intern Med. 2006;166(3):285–93. https://doi.org/10.1001/archinte.166.3.285.

114. Goulet J, Lamarche B, Nadeau G, Lemieux S. Effect of a nutritional intervention promoting the Mediterranean food pattern on plasma lipids, lipoproteins and body weight in healthy French–Canadian women. Atherosclerosis. 2003;170(1):115–24.

115. Barnard ND, Scialli AR, Bertron P, Hurlock D, Edmonds K, Talev L. Effectiveness of a low-fat vegetarian diet in altering serum lipids in healthy premenopausal women. Am J Cardiol. 2000;85(8):969–72.

116. Ling WH, Laitinen M, Hänninen O. Shifting from conventional diet to an uncooked vegan diet reversibly alters serum lipid and apolipoprotein levels. Nutr Res. 1992;12:1431–40.

117. Kastorini CM, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. J Am Coll Cardiol. 2011;57(11):1299–313. https://doi.org/10.1016/j.jacc.2010.09.073.

118. Busetto L, Marangon M, De Stefano F. High-protein low-carbohydrate diets: What is the rationale? Diabetes Metab Res Rev. 2011;27(3):230–2. https://doi.org/10.1002/dmrr.1171.

119. Ebbeling CB, Feldman HA, Klein GL, Wong JMW, Bielak L, Steltz SK, et al. Effects of a low carbohydrate diet on energy expenditure during weight loss maintenance: randomized trial. BMJ. 2018;363:k4583. https://doi.org/10.1136/bmj.k4583.

120. Jonsson T, Granfeldt Y, Lindeberg S, Hallberg AC. Subjective satiety and other experiences of a Paleolithic diet compared to a diabetes diet in patients with type 2 diabetes. Nutr J. 2013;12:105. https://doi.org/10.1186/1475-2891-12-105.

121. Huntriss R, Campbell M, Bedwell C. The interpretation and effect of a low-carbohydrate diet in the management of type 2 diabetes: a systematic review and meta-analysis of randomised controlled trials. Eur J Clin Nutr. 2018;72(3):311–25. https://doi.org/10.1038/s41430-017-0019-4.

122. Ros E. Dietary cis-monounsaturated fatty acids and metabolic control in type 2 diabetes. Am J Clin Nutr. 2003;78(3 Suppl):617S–25S. https://doi.org/10.1093/ajcn/78.3.617S.

123. Qian F, Korat AA, Malik V, Hu FB. Metabolic effects of monounsaturated fatty acid-enriched diets compared with carbohydrate or polyunsaturated fatty acid-enriched diets in patients with type 2 diabetes: a systematic review and meta-analysis of randomized controlled trials. Diabetes Care. 2016;39(8):1448–57. https://doi.org/10.2337/dc16-0513.

124. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr. 2003;77(5):1146–55. https://doi.org/10.1093/ajcn/77.5.1146.

125. O'Mahoney LL, Matu J, Price OJ, Birch KM, Ajjan RA, Farrar D, et al. Omega-3 polyunsaturated fatty acids favourably modulate cardiometabolic biomarkers in type 2 diabetes: a meta-analysis and meta-regression of randomized controlled trials.

Cardiovasc Diabetol. 2018;17(1):98. https://doi.org/10.1186/s12933-018-0740-x.

126. Chehade JM, Gladysz M, Mooradian AD. Dyslipidemia in type 2 diabetes: prevalence, pathophysiology, and management. Drugs. 2013;73(4):327–39. https://doi.org/10.1007/s40265-013-0023-5.

127. Femlak M, Gluba-Brzozka A, Cialkowska-Rysz A, Rysz J. The role and function of HDL in patients with diabetes mellitus and the related cardiovascular risk. Lipids Health Dis. 2017;16(1):207. https://doi.org/10.1186/s12944-017-0594-3.

128. Holmes MV, Asselbergs FW, Palmer TM, Drenos F, Lanktree MB, Nelson CP, et al. Mendelian randomization of blood lipids for coronary heart disease. Eur Heart J. 2015;36(9):539–50. https://doi.org/10.1093/eurheartj/eht571.