diabetesresearchandclinicalpractice 191 (2022) 110071

Contents lists available at ScienceDirect

Diabetes Research and Clinical Practice

journal homepage: www.journals.elsevier.com/diabetes-research-and-clinical-practice

Consumption of different animal-based foods and risk of type 2 diabetes: An umbrella review of meta-analyses of prospective studies

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ARTICLE INFO

Keywords:
Diabetes incidence
Diabetes risk
Animal-based food
Meat
Dairy
Diet

ABSTRACT

We performed an umbrella review of dose–response meta-analyses of prospective studies reporting the incidence of type 2 diabetes associated with the consumption of animal-based foods. A systematic search was conducted in PubMed, Web of Science, Scopus, and Embase according to PRISMA. Thirteen meta-analyses are included in the study providing 175 summary risk ratio estimates. The consumption of 100 g/day of total or red meat, or 50 g/day of processed meat, were associated with an increased risk; RR and 95 % CI were respectively 1.20, 1.13–1.27; 1.22, 1.14–1.30 and 1.30, 1.22–1.39. White meat (50 g/day) was associated with an increased risk, but of lesser magnitude (RR 1.04, 95 % CI 1.00–1.08). A risk reduction was reported for 200 g/day of total dairy (RR 0.95, 95 % CI 0.92–0.98) or low-fat dairy (RR 0.96, 95 % CI 0.92–1.00) or milk (RR 0.90, 95 % CI 0.83–0.98), or 100 g/day of yogurt (RR 0.94, 95 % CI 0.90–0.98). No association with diabetes risk was reported for fish or eggs. In conclusions animal-based foods have a different association with diabetes risk. To reduce diabetes risk the consumption of red and processed meat should be restricted; a moderate consumption of dairy foods, milk and yogurt, can be encouraged; moderate amounts of fish and eggs are allowed.

1. Introduction

Despite the tremendous advances in all areas of diabetes care [1], according to the Global Burden Disease Study type 2 diabetes remains the third major cause of diet-related death (338.714 deaths) and DALYs (24 million) worldwide [2,3]. Given the projected increase in the incidence of this disorder, the need for effective prevention strategies at the population level is getting more and more urgent. Adverse lifestyle, including poor quality diet, are established risk factors for type 2 diabetes and there is compelling evidence that interventions aimed at improving diet and other lifestyle related habits are effective in diabetes prevention. So far, healthy eating patterns, such as the traditional Mediterranean diet and the DASH diet, have been clearly associated with the reduction of type 2 diabetes risk [4]. Although it is often difficult to disentangle the precise downstream mechanisms by which foods exposure impacts the development and progression of type 2 diabetes, this does not preclude the need for adopting optimal dietary habits for lifelong diabetes prevention.

Unfortunately, the current food environment does not favor improvements in diet quality and strongly affects the ability of individuals to effectively adopt and sustain healthy dietary changes. Focusing on specific food groups and defining the intake associated with the lowest risk of type 2 diabetes may be helpful for the formulation of easily received dietary advice to the general public and to guide the most suitable food choices. Long-term randomized trials on lifestyle interventions and prevention of type 2 diabetes have so far evaluated the impact of a multifactorial nutritional approach largely aimed at weight reduction [5,6]. Other lines of epidemiological evidence, including long-term prospective observational studies and short-term trials on intermediate outcomes, have produced a large body of evidence which supports the current recommendation to shift towards diets rich in healthy plant-based foods (i.e., whole grains, fruits, vegetables, nuts, legumes, non-tropical vegetable oils), with low intakes of less healthy items (i.e., fruit juices, sweetened beverages, refined grains, potatoes, pastries) [7–9]. As for foods of animal origin, recommendations are vague and limited to the indication of a global reduction, however foods of animal origin are not all the same; substantial differences are observed with regard to the fat content and the fatty acid profile (i.e. saturated vs unsaturated fat) and some of them contain variable amounts of bioactive components with beneficial impact on health, as it

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https://doi.org/10.1016/j.diabres.2022.110071
Received 21 June 2022; Received in revised form 29 July 2022; Accepted 31 August 2022
Available online 5 September 2022

is the case for fermented dairy products. Furthermore, we should not forget that animal foods are largely consumed – the daily protein supply from animal products is about 33 g per capita worldwide, reaching 50–60 g in Europe [10] and exceeding 70 g in North America.

In consideration of the heterogeneous nature of the different animal-based foods and given their extensive consumption, it is relevant to address the potential impact of the different animal foods on type 2 diabetes risk in order to support dietary recommendations for type 2 diabetes prevention with updated and reliable scientific evidence on the appropriate choices for animal-based foods. The aim of this study is to provide data to substantiate recommendations for the most appropriate frequency and amount of consumption of animal foods by the adult population based on the available evidence on the relationship between the consumption of animal foods and the incidence of type 2 diabetes. To this aim we performed a systematic umbrella review of meta-analyses of prospective cohort studies reporting the risk estimates for the incidence of type 2 diabetes associated with well-defined amounts of consumption of single food item /food group of animal origin (i.e., meat, fish, dairy, egg).

2. Methods

This umbrella review of meta-analyses was conducted according to the Preferred Reporting Items for Systematic Reviews and meta-Analyses (PRISMA) guidelines [11]. The study protocol is registered in PROSPERO (registration number: CRD42022306145).

2.1. Literature search

A systematic literature search was conducted in PubMed, Web of Science, Scopus, and Embase databases until December 2021 for meta-analyses of observational studies investigating the association between foods of animal origin and type 2 diabetes, using the following pre-defined search strategy restricted to articles published in English: (meat OR total meat OR processed meat OR red meat OR white meat OR poultry OR fish OR fatty fish OR lean fish OR dairy OR dairy products OR total dairy OR full fat dairy OR low fat dairy OR milk OR fermented dairy OR cheese OR yogurt OR eggs) AND (consumption OR intake OR serving OR eating) AND (diabetes OR type 2 diabetes mellitus). We applied humans and meta-analyses filters in order to restrict the search only to articles of interest for our work. We also hand-searched the reference lists of relevant studies and of previous reviews. The literature search was independently conducted by two authors (AG, IC). Disagreements were resolved through consultation with a third independent reviewer (MV).

2.2. Selection of meta-analyses

Studies were included if they met the following criteria: (1) consumption of foods of animal origin (i.e., dairy products, eggs, meat, fish) was assessed by established instruments of dietary evaluation (i.e., food frequency questionnaires, 24 h dietary recalls, and dietary records), (2) the study design was represented by meta-analyses of observational prospective cohort studies in adults with multivariable adjusted summary risk estimates and corresponding 95 % confidence intervals, (3) the incidence of type 2 diabetes represented the research outcome. Studies were excluded if no summary estimate was reported (i.e., systematic reviews without meta-analysis), if they were meta-analyses of RCTs or case-control/cross-sectional studies, if the meta-analyses considered children or ill populations, or type 1 diabetes or gestational diabetes as outcome. We also excluded publications reporting on exposure of plasma levels or biomarkers rather than dietary intake. If an included meta-analysis described both high vs low and dose–response analyses, only the data on dose–response analysis were selected. A detailed flow chart of the screening and selection process of eligible articles is presented in Fig. 1.

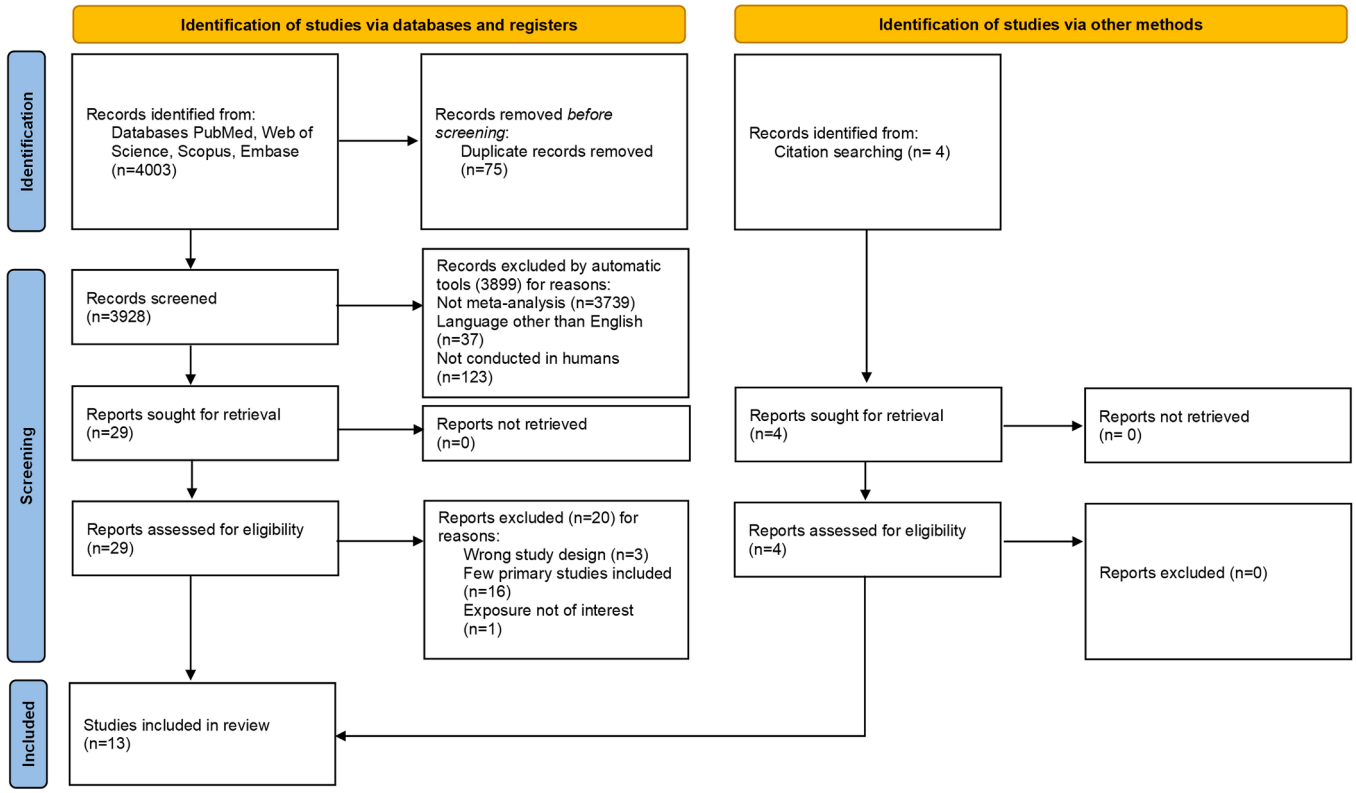

Fig. 1. PRISMA flowchart indicating the results of the search strategy.

2.3. Data extraction

Data were extracted by 2 investigators (AG, and IC), and any discrepancies were resolved through consultation with a third independent reviewer (MV). For each published meta-analysis, we extracted the following data: name of the first author, publication year, number of included studies, total number of cases and participants, type of comparison (including dose of exposure), type of metric (RR), effect sizes with corresponding 95 % CIs, I^2 value for between-study heterogeneity, P value for between-study heterogeneity, Egger's P value or other statistics for publication bias, and p value for nonlinearity.

2.4. Assessment of methodological quality

The methodological quality of each included meta-analysis was evaluated by two independent authors trough AMSTAR (A Measurement Tool to Assess Systematic Reviews) [12,13], a strict, reliable, and valid measurement tool to evaluate systematic reviews and meta-analyses. It includes 11 items about the conduct of a meta-analysis, including the methods for literature search, study selection and data extraction, reporting of included and excluded studies, quality assessment of the included studies, statistical methods for the meta-analysis, publication bias, and conflict of interest. Each question can be answered with "yes," "no," "can't answer," and "not applicable". A "yes" scores one point, whereas the other answers score 0 points. An overall score of at least 8 points was defined as the cutoff value for high quality, 4–7 points as moderate quality, and 3 points or less as low quality [14].

2.5. Evaluation of quality of evidence

The quality of evidence was evaluated by using a modified version of NutriGrade [15]. This is a numerical scoring system which includes eight items: (1) Risk of bias, study quality, or study limitations (0–2 points); (2) Precision, evaluated through the number of cases (events), sample size, and inspection of the 95 % CIs (0–1 points); (3) Heterogeneity, evaluated through the chi-square (Cochrane's Q) test and the I^2 statistic (0–1 point); (4) Directness (i.e., whether there were differences in the study populations or interventions or exposures of interest) (0–1 point); (5) Publication bias (0–1 point); (6) Funding bias (0–1 point); (7) Effect size, because it is generally assumed that very large effects are less likely driven by confounding (0–2 points); (8) Dose-response association (0–1 point).

High quality of evidence is defined by an overall score of 8 points or more which means that there is high confidence in the effect estimate and that further research probably will not change the effect estimate. An overall score of 6 to <8 points was assigned to moderate confidence in the effect estimate, where further research could add evidence on the confidence and could change the effect estimate. An overall score of 4 to <6 points meant that there was low confidence in the effect estimate, and that further research would provide important evidence and would likely change the effect estimate. Scores <4 points was assigned to very low quality of evidence, which meant that the quality of evidence was very limited and uncertain.

2.6. Data analysis

For each food item, we recalculated the meta-analysis using risk ratios of the primary studies included in the published meta-analyses that adjusted for most of the confounders. We applied the random effects model by DerSimonian and Laird [16] to obtain the adjusted summary risk ratios and corresponding 95 % confidence intervals. We used this approach to ensure that all adjusted summary risk ratios were calculated by a random effects model and to receive further information for the evaluation of the quality of evidence (including I^2, and publication bias). When the published meta-analyses presented risk ratios from the same cohort separately by sex or geographical area, we combined the risk ratios per cohort using the random effect methods before conducting the overall meta-analysis. Since all meta-analyses on fish consumption reported separate data for the two main geographical areas (America/Europe and Asia) we performed a subgroup analysis to obtain the adjusted summary risk ratios and corresponding 95 % confidence intervals for America/Europe and Asia, separately.

In each meta-analysis, we recalculated the between-study heterogeneity by using the I^2 statistic and the P value from the χ_2-based Cochran Q test [17]. The I^2 statistic describes the percentage of variation among studies that is due to heterogeneity rather than due to chance. $I^2 < 50$ % is considered as low-to-moderate heterogeneity between studies, whereas $I^2 > 50$ % and $I^2 > 75$ % are considered as moderate or high heterogeneity, respectively. However, I^2 is dependent on the study size (it increases with increasing study size), therefore, we also calculated $\tau 2$, which describes variability between studies, in relation to the risk estimates [18] independent of study size. Using the recalculated data, the 95 % prediction interval (PI) was also estimated. A 95 % PI represents the distribution of true effects in which 95 % of new and unique studies on the same subject will fall. Therefore, 95 % PI further signifies between-study heterogeneity, whereas a 95 % CI of each meta-analysis represents the accuracy of the summary effect size.

Publication bias and small study effects were assessed for each meta-analysis by graphical and statistical tests, namely the funnel plot and Egger's test [19]. Therefore, the primary studies from the meta-analyses included in our umbrella review were plotted. A P value <0.10 was taken as statistical evidence of the presence of small study effects (potential publication bias).

All statistical analyses were performed with RevMan 5.4 (Review Manager RevMan – Computer program; Version 5.4; The Cochrane Collaboration, 2020) and R 4.1.0 (The R Project for Statistical Computing; Version 4.1.0, 2021), and all tests were 2-sided with a significance level of 0.05 unless otherwise stated.

3. Results

3.1. Literature search

Of the 4003 publications initially identified, after eliminating duplicates, 3899 articles were excluded by automatic tools because they did not meet the inclusion criteria, since they were not meta-analyses (n = 3739), used a language other than English (n = 37), or were not conducted in humans (n = 123). Thirty-three meta-analyses were selected for eligibility, but 20 were excluded for: wrong study design (n = 3), <4 primary studies included (n = 16) or exposure not of interest (n = 1). Based on the inclusion criteria, we finally selected 13 published meta-analyses reporting 257 adjusted summary risk ratios (Fig. 1). The meta-analyses summarized the outcomes of prospective studies on the association with type 2 diabetes incidence of total meat (20 prospective studies), processed meat (36 prospective studies), red meat (31 prospective studies), white meat (10 prospective studies), total fish (41 prospective studies), total dairy (23 prospective studies), full-fat dairy (16 prospective studies), low-fat dairy (17 prospective studies), milk (12 prospective studies), cheese (11 prospective studies), yogurt (13 prospective studies), and egg (22 prospective studies) (Supplemental Tables S1–S4).

3.2. Study characteristics

Table 1 describes the characteristics of the meta-analyses included in the study. All together 34 meta-analyses met the criteria for inclusion in the review: 3 for total meat [20–22], 6 for processed meat [20–25], 4 for red meat [20–22,24], 2 for white meat [22,25], 5 for total fish [22,24–27], 3 for total dairy [24,28,29], 3 for full-fat dairy [28,30,31], 3 for low-fat dairy [29–31], 2 for milk [30,31], 1 for cheese [25], 1 for yogurt [29], and 1 for eggs [32]. The included meta-analyses comprised a number of incident diabetes cases equal to 24,465 for total meat,

Table 1
Summary of the results of meta-analyses of prospective cohort studies on the relationship between the consumption of food groups of animal origin and diabetes incidence.

Author, year	*n* of studies (cohorts)	*n* of cases/total participants	Comparison	Amount	Type of metrics (RR)	Summary effect size (95 % CI)	I^2 (P-value)	Egger's p value	P-nonlinearity
Total Meat									
Micha et al., 2010	3	5,923/142,851	Dose-response	100 g/d	RR	1.12 (1.05, 1.19)	(0.29)	0.60	NA
Feskens et al., 2013	14	NA	Dose-response	100 g/d	RR	1.15 (1.07, 1.24)	54.0	0.10	NA
Yang et al., 2020	7	18,542/254,924	Dose-response	100 g/d	RR	1.36 (1.23, 1.49)	55.7 (0.035)	0.64	0.065
Red Meat									
Micha et al., 2010	5	7,349/298,982	Dose-response	100 g/d	RR	1.16 (0.92, 1.46)	(0.25)	0.62	NA
Feskens et al., 2013	14	NA	Dose-response	100 g/d	RR	1.13 (1.03, 1.23)	NA	NA	NA
Schwingshackl et al., 2017	14	43,781/520,342	Dose-response	100 g/d	RR	1.17 (1.08, 1.26)	83.0 (<0.0001)	NA	0.30
Yang et al., 2020	17	49086/663,144	Dose-response	100 g/d	RR	1.31 (1.19, 1.45)	76.2 (<0.001)	0.576	0.707
White Meat									
Fan et al., 2019	8	13,865/219,926	Dose-response	50 g/d	RR	1.02 (0.98, 1.07)	0.0 (0.685)	0.943	0.929
Yang et al., 2020	9	29,477/315,961	Dose-response	50 g/d	RR	1.04 (1.00, 1.09)	0.0 (0.812)	0.041	0.299
Processed Meat									
Aune et al., 2009	8	9456/372,205	Dose-response	50 g/d	RR	1.57 (1.28, 1.93)	74.0 (<0.0001)	0.69	NA
Micha et al., 2010	7	10,782/372,279	Dose-response	50 g/d	RR	1.19 (1.11, 1.27)	(0.03)	0.36	NA
Feskens et al., 2013	21	NA	Dose-response	50 g/d	RR	1.32 (1.19, 1.48)	89.0	NA	NA
Schwingshackl et al., 2017	14	43,781/520,342	Dose-response	50 g/d	RR	1.37 (1.22, 1.55)	87.0 (<0.0001)	NA	<0.001
Fan et al., 2019	16	33,916/582,920	Dose-response	50 g/d	RR	1.41 (1.24, 1.60)	85.6 (<0.0001)	0.208	<0.001
Yang et al., 2020	14	39,961/531,780	Dose-response	50 g/d	RR	1.46 (1.26, 1.69)	93.2 (<0.001)	0.002	0.004
Fish									
Wu et al., 2012	10 (13)	20,830/481,489	Dose-response	100 g/day	RR	1.12 (0.94, 1.34)	82.9 (<0.001)	0.45	NA
Schwingshackl et al., 2017	15	45,011/637,716	Dose-response	100 g/day	RR	1.09 (0.93, 1.28)	84 (<0.001)	NA	0.48
Fan et al., 2019	5 (8)	7,914/223,564	Dose-response	50 g/day	RR	0.99 (0.92, 1.07)	61.9 (0.010)	0.011	0.119
Yang et al., 2020	20	42,084/682,622	Dose-response	50 g/day	RR	1.03 (0.98, 1.09)	81.0 (<0.001)	0.368	0.448
Pastorino et al., 2021	28	48,084/956,122	Dose-response	100 g/day	RR	M: 1.00 (1.00, 1.01)F: 1.02 (1.00, 1.03)	52.3 (0.002) 33.6 (0.077)	NA	NA
Total Dairy									
Gao et al., 2013	13 (15)	27,095/457,893	Dose-response	200 g/day	RR	0.94 (0.91–0.97)	51.6 (0.02)	0.37	<0.001
Schwingshackl et al., 2017	19 (21)	44,474/566,782	Dose-response	200 g/day	RR	0.97 (0.94–0.99)	74 (<0.0001)	NA	0.89
Soedamah-Muthu et al., 2018	19 (21)	40,905/ 5,741,718	Dose-response	200 g/day	RR	0.97 (0.95–1.00)	62.8 (<0.001)	NA	Linear
Full-Fat Dairy									
Gao et al., 2013	8	9,398/260,700	Dose-response	200 g/day	RR	0.95 (0.88–1.04)	52.2 (0.04)	NA	NA
Aune et al., 2013	9	7,212/196,799	Dose-response	200 g/day	RR	0.98 (0.94–1.03)	7.6 (0.37)	0.77	0.57
Gijsbers et al., 2016	11 (13)	24,034/327,895	Dose-response	200 g/day	RR	0.98 (0.93–1.04)	0.016	NA	NA
Low-Fat Dairy									
Aune et al., 2013	9	10,775/274,571	Dose-response	200 g/day	RR	0.91 (0.86–0.96)	40.2 (0.10)	0.49	0.06
Gijsbers et al., 2016	5 (7)	19,889/262,025	Dose-response	200 g/day	RR	1.01 (0.97–1.06)	71.6 (0.002)	NA	NA
Soedamah-Muthu et al., 2018	13 (15)	28,531/ 5,313,782	Dose-response	200 g/day	RR	0.96 (0.92–1.00)	60.3 (<0.001)	NA	Linear
Milk									
Aune et al., 2013	7	14,393/167,982	Dose-response	200 g/day	RR	0.87 (0.72–1.04)	93.6 (<0.0001)	0.41	<0.0001
Gijsbers et al., 2016	11 (12)	17,241/145,472	Dose-response	200 g/day	RR	0.97 (0.93–1.02)	57.4 (0.007)	0.71	NA

(*continued on next page*)

Table 1 (*continued*)

Author, year	*n* of studies (cohorts)	*n* of cases/total participants	Comparison	Amount	Type of metrics (RR)	Summary effect size (95 % CI)	I^2 (P-value)	Egger's p value	P-nonlinearity
Cheese									
Fan et al., 2019	10 (11)	9479/186,941	Dose-response	30 g/day	RR	0.97 (0.93–1.03)	13.9 (0.312)	0.656	0.216
Yogurt									
Soedamah-Muthu et al., 2018	12 (14)	37,223/ 5,184,590	Dose-response	100 g/ day	RR	0.94 (0.91–0.97)	68.6 (<0.001)	NA	<0.0001
Eggs									
Drouin-Chartier et al., 2020	16 (22)	41,248/589,559	Dose-response	1 egg/ day	RR	1.07 (0.99–1.15)	69.8 (0.000)	0.03	NA

n represents the number of studies included in the meta-analysis. CI: Confidence Interval; g: grams; NA: Not Available; NS: Not Significant.

100,216 for red meat, 107,376 for processed meat, 43,342 for white meat, 163,923 for fish, 112,474 for total dairy, 34,154 for full-fat dairy, 59,195 for low fat dairy, 31,634 for milk, 14,311 for fermented dairy, 9,479 for cheese, 37,223 for yogurt, 41,248 for eggs.

All meta-analyses included primary studies from America, Europe, and Asia. The primary studies included provided multivariable adjusted risk estimates, adjustment was performed by age, sex, smoking, body mass index, physical activity, family history of diabetes, total energy intake, alcohol intake, and other dietary factors. Information on linearity of the dose–response relations (P non-linearity) were available for 46 % (n = 6) meta-analyses. The definition of the amount of consumption was generally consistent in the various studies; only for fish risk ratios were reported for a consumption of 50 g/capita/day in two meta-analyses [22,25], and for a consumption of 100 g/capita/day in three [24,26,27].

As for the quality of the meta-analyses, the average AMSTAR scores were 6.7 for total meat, 7.2 for processed meat, 7.0 for red meat, 8.5 for white meat, 8.0 for fish, 7.7 for total dairy, 7.7 for full fat dairy, 5.7 for low fat dairy, 7.5 for milk, 8.0 for fermented dairy, 9.0 for cheese, 3.0 for yogurt, and 7.0 for eggs (Supplemental Table 5). The most frequently detected flaws were that grey literature was not accounted for in the literature search, and no list of excluded studies was provided.

3.3. Association of animal foods consumption with the incidence of type 2 diabetes and quality of evidence

Fig. 2 shows the adjusted summary RR with their corresponding 95 % CI and the quality of evidence for the association between each food of animal origin and the incidence of type 2 diabetes. Details on the grading of every NutriGrade item is shown in Supplemental Table 6.

3.3.1. Meat

The consumption of 100 g/day of total meat or red meat, or 50 g/day of processed meat was associated with a significantly increases risk of type 2 diabetes; RR and 95 % CI are respectively 1.20, 1.13–1.27; 1.22, 1.14–1.30 and 1.30, 1.22–1.39 (Fig. 2). These analyses were characterized by a high heterogeneity among studies ($I^2 = 66$ %, $p < 0.0001$ for total meat, $I^2 = 77$ %, $p < 0.0001$ for red meat and $I^2 = 86$ %, $p < 0.0001$ for processed meat). The quality of evidence, evaluated by the NutriGrade scoring system, was moderate. The consumption of white meat, 50 g/day, was also associated with an increased risk of type 2 diabetes, but of lesser magnitude (RR 1.04, 95 % CI 1.00–1.08), with no significant heterogeneity ($I^2 = 0$ %, $p = 0.80$) and a low quality of evidence (Fig. 2).

3.3.2. Fish

No significant association with type 2 diabetes risk was found for a consumption of fish up to 100 g/day. The heterogeneity between studies was high ($I^2 = 81$ %, $p < 0.0001$), and the quality of evidence for these associations was low (Fig. 2). Dose-response data on the consumption of specific fish types (i.e., fatty fish and lean fish) were not available, but are relevant to investigate, due to the different nutritional composition of the two fish types and the possible implications for health outcomes.

3.3.3. Dairy products

A significant 5 % risk reduction of type 2 diabetes was found for the consumption of 200 g/day of total dairy products (including milk, cheese, and yogurt), the heterogeneity between studies was high ($I^2 = 80$ %, $p < 0.000$) and the quality of evidence was moderate. Separate analyses conducted for full fat or low-fat products showed a neutral association with diabetes risk for full fat products and a marginally significant risk reduction for low fat products; RR and 95 % CI were

Exposure	*n* of primary studies	*n* of cases	Comparison	Amount	Adjusted SRR random effects (95% CI)	Quality of evidence
Total Meat	13	28,644	Dose-response	100 g/day	1.20 (1.13, 1.27)	Moderate
Red Meat	21	62,352	Dose-response	100 g/day	1.22 (1.14, 1.30)	Moderate
White Meat	8	31,465	Dose-response	50 g/day	1.04 (1.00, 1.08)	Low
Processed Meats	24	64,900	Dose-response	50 g/day	1.30 (1.22, 1.39)	Moderate
Fish	12	43,688	Dose-response	50 g/day	1.04 (0.99, 1.09)	Low
Total Dairy	21	42,204	Dose-response	200 g/day	0.95 (0.92, 0.98)	Moderate
Full-Fat Dairy	14	28,817	Dose-response	200 g/day	0.98 (0.93, 1.03)	Low
Low-Fat Dairy	15	29,023	Dose-response	200 g/day	0.97 (0.93, 1.00)	Low
Milk	11	8,061	Dose-response	200 g/day	0.90 (0.83, 0.98)	Low
Cheese	10	9,479	Dose-response	30 g/day	0.97 (0.91, 1.04)	Moderate
Yogurt	10	37,223	Dose-response	100 g/day	0.94 (0.90, 0.98)	Moderate
Eggs	16	41,248	Dose-response	1 egg/day	1.07 (0.99, 1.15)	Low

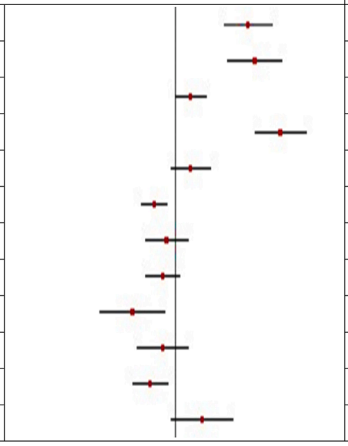

Fig. 2. Adjusted summary risk ratios (SRR) with 95% confidence intervals and quality of evidence for association between foods of animal origin and incidence of type 2 diabetes.

respectively 0.98, 0.93–1.03 and 0.96, 0.92–1.00, with a significant heterogeneity between studies ($I^2 = 50$ %, p = 0.01, for full fat dairy; $I^2 = 60$ %, p = 0.001, for low-fat dairy). The quality of evidence was low. As for individual dairy products, a significant risk reduction was associated with the consumption of 200 g/day of total milk (RR 0.90, 95 % CI 0.83–0.98) or low-fat milk (RR 0.93, 95 % CI 0.88–1.00), or 100 g/day of yogurt (RR 0.94, 95 % CI 0.90–0.98), whereas a neutral association was observed for full-fat milk (RR 0.96, 95 % CI 0.87–1.06, for a consumption of 200 g/day) or cheese (RR 0.97, 95 % CI 0.91–1.04, for a consumption of 30 g/day). The heterogeneity between studies was high for milk ($I^2 = 89$ %, p < 0.0001) and yogurt ($I^2 = 74$ %, p < 0.0001) and non-significant for cheese ($I^2 = 15$ %, p = 0.30). The quality of evidence was low for milk and moderate for cheese and yogurt (Fig. 2).

3.3.4. Eggs

A neutral association was found between the daily consumption of 1 egg and the risk of type 2 diabetes (RR 1.07, 95 % CI 0.99–1.15) though with high heterogeneity between studies ($I^2 = 70$ %, p = 0.001) (Fig. 2). The quality of evidence was graded as low.

3.4. Heterogeneity between primary studies

I^2, τ^2, and 95 % prediction intervals are reported in the Supplemental Figs. 1-13. For one exposure (eggs consumption), I^2, τ^2 and the 95 % prediction intervals could not be recalculated. As for the 95 % prediction intervals, only seven meta-analyses excluded the null value – this applies to the dose–response meta-analyses for total meat, red meat, white meat, processed meat, total dairy, milk, ad yogurt. These results suggest that in future studies, the true effect size of these exposures is expected to point to the same direction. However, it is of note that for most of the findings the I^2 describes a large or very large heterogeneity, except for cheese and white meat.

3.5. Publication bias and small study effects

Our results indicate the presence of small study effects (potential publication bias) according to funnel plot and Egger's test (P < 0.10) for total dairy, full-fat dairy, and eggs (Supplemental Fig. 14 a-m).

As for the primary studies available for the different funnel plots, >10 of them were available for total meat, red meat, and processed meat, and between five and 10 were available for white meat, fish, total dairy, full-fat dairy, low-fat dairy, milk, cheese, yogurt, and eggs.

4. Discussion

This work provides an overview of the existing evidence on the relationship between the consumption of individual foods of animal origin and the risk of type 2 diabetes, the quality of evidence for all the associations was also evaluated. Not all animal-based foods are associated with an increased risk of type 2 diabetes. In particular, while there is a consistently increased risk associated with the consumption of meat, particularly red and processed meat, the consumption of dairy foods, especially low-fat types, milk and yogurt, is associated with a reduced risk of type 2 diabetes. Eggs and fish show no association with the incidence of type 2 diabetes.

In relation to meat consumption, 100 g/day of total or red meat or 50 g/day of processed meat, increase the risk of type 2 diabetes by 20 %, 22 % and 30 % respectively. These findings are coherent with those of a prior umbrella review of meta-analyses of prospective studies [33], reporting a 17 % and 37 % risk increase associated with the consumption of red meat or processed meat. For white meat, at variance with the neutral association found in a prior umbrella review, the present study, based on a larger number of primary studies, provides evidence that the daily consumption of 50 g is associated with a significant 4 % risk increase of type 2 diabetes. Considering the composition of red and processed meat in terms of nutrients and other relevant components, some plausible mechanisms through which their consumption may increases the risk of type 2 diabetes can be hypothesized. In particular, red and processed meat are important sources of dietary cholesterol, saturated fatty acids (SFAs), advanced glycation end products (AGEs) and heme–iron. Dietary cholesterol and SFAs promote chronic subclinical inflammation which in turn may contribute to impair insulin sensitivity [34,35]. Besides, AGEs and heme–iron can induce oxidative stress and lipid peroxidation, as well as protein modification and DNA damage [21,36]. Processed meats are also rich in nitrates, nitrites and sodium; the formers, by interacting with amines at the gastric level, can contribute to pancreatic β-cells damage and increase oxidative stress and inflammation [37], while sodium can induce endothelial dysfunction, which, in turn worsens insulin sensitivity [21]. White meat consumption is also associated with an increased risk of type 2 diabetes, but of lesser magnitude, this may be partly explained by the different nutritional composition of red or white meat. Poultry (white meat), as compared with beef, pork or lamb (red meat), is, in fact, characterized by a lower fat content, a more favorable fatty acid profile (i.e., a higher unsaturated/saturated fatty acid ratio) and a reduced amount of heme iron.

As for dairy foods, the consumption of 200 g/day of total dairy products is associated with a significantly reduced risk of type 2 diabetes. This may be partly explained considering the number of nutrients, vitamins and other components of dairy foods (e.g., calcium, vitamin D, proteins, peptides, etc.) with potential beneficial effects on glucose metabolism [38,39]. Distinguishing between low-fat and full-fat dairy, only the formers were associated with a significant, tough marginal, risk reduction (-4%). This seems to suggest that the beneficial relationship between total dairy consumption and the risk of type 2 diabetes may be driven by the low-fat types, particularly milk; however, such assumption should be taken with caution, considering both the extremely small magnitude of risk reduction and the low-quality evidence, as well as the presence of residual confounding factors like the different background diet and lifestyle between people consuming preferably low-fat dairy products and those habitually choosing the full-fat ones.

Interestingly, milk is among all dairy the item associated with the greatest risk reduction (−10 %); this may be partly due its content in whey proteins, which represent 20 % of all milk proteins [40,41] and which have a known effect on the regulation of postprandial glycemia, as well as to the control of appetite [41,42] and body weight [43] trough insulin-dependent and non-insulin-dependent mechanisms [44,45].

The other food item showing an inverse association with type 2 diabetes risk is yogurt. The possible mechanisms for this association remain insufficiently investigated; among others, yogurt is an important source of probiotics that can exert beneficial effects on glucose metabolism [46]. However, the contribution of probiotic bacteria *per se* to the improvement of blood glucose control remains controversial, since it depends on the bacterial strains present in each type of yogurt and on their amount [47]. Yet, protective effects of probiotics and probiotic-microbiome interactions have been consistently reported in relation to body weight gain and obesity, the major drivers of type 2 diabetes development [48]. This effect may justify the inverse association between habitual yogurt consumption and the risk of type 2 diabetes. Nevertheless, the structural change of lipids and proteins due to fermentation can also contribute, at least in part, to this favorable relationship [49].

The consumption of fish and eggs showed a neutral association with the risk of diabetes. Focusing on fish intake, it is of note that when the meta-analyses are grouped according to the geographical areas (America/Europe and Asia), the consumption of 50 g per day of fish was significantly associated with an increased risk of type 2 diabetes in studies from America/Europe (+7%) and to a lower risk in Asian studies (-5%). Yet, no significant differences emerged for a higher intake of fish (100 g/day) in either Europe/America or Asia. Preparation methods may play a role in this regional difference, since raw as well as boiled and steamed fish are the major preparation methods in Eastern cuisine, while hard cooking procedures like deep frying and grilling are more

popular in Western regions. Early studies have suggested that frying can modify the lipid profile through a decrease in long chain n-3 PUFA content [50]; deep frying (e.g., fried fast food etc.) can also cause the production of *trans*-fatty acids and oxidized lipid products as well as AGEs, that, in turn, may increase the risk of developing type 2 diabetes [51] by promoting systemic subclinical inflammation [52] and oxidative stress [53]. Also the prevalent type of fish consumed (i.e., fatty or lean fish, with diverse long chain n-3 PUFA content) may contribute to the different relationship with the risk of type 2 diabetes we have observed among the two regions, but the available studies did not report separate data for fatty or lean fish.

However, the background diet more plant-based in Asian regions and more animal-based in Europe and America may also partly confound this observation.

The major strength of this work is that it provides a systematic and comprehensive overview of the available evidence from all published meta-analyses on the dose–response relationship between the consumption of the single food items of animal origin and the incidence of type 2 diabetes.

Moreover, we carefully evaluated the quality of the methodology employed by the included meta-analysis and the overall quality of the evidence, with particular emphasis on the utilization of validated tools for the dietary behaviors assessment and of standardized statistical methods including the use of random-effects analysis and various measures of heterogeneity and publication bias. We are therefore confident that the evidence here provided is solid and may substantially facilitate the translation of scientific information into practice aimed at preventing the risk of developing type 2 diabetes by optimized consumption of animal foods.

However, limitations are also present. First, we included studies from published meta-analyses and, therefore, we could have missed individual studies not identified by our predefined systematic search strategy. Second, we could not fully account for relevant factors that affect the risk of type 2 diabetes, such as gender, ethnicity, age, smoking status, socioeconomic factors or family history of diabetes, since not all of the studies provided the information needed to perform subgroup analyses. Third, most of the included meta-analyses were of low methodological quality. Finally, no data were reported in the primary studies on the socio-economic status which is, at the same time, a risk factor for diabetes and a determinant of food choices. Therefore, this may act as a confounder we cannot account for.

5. Conclusions

This systematic review of the available evidence shows that the habitual consumption of meat, especially red (i.e., 100 g/day) and processed types (i.e., 50 g/day), is associated with a substantially increased risk of type 2 diabetes. Conversely, we found that a moderate amount (i.e., 200 g/day) of total dairy products, particularly low-fat items, and yogurt (i.e., 100 g/day) is associated with a decreased risk of type 2 diabetes. This benefit is not shared by full-fat dairy and cheese consumption, which shows a neutral relationship with the risk of type 2 diabetes. Also a moderate intake of egg (i.e., 1 egg daily) has no association with type 2 diabetes risk. Finally, fish consumption on the overall is not associated with type 2 diabetes incidence. However, there is a suggestion that a moderate fish consumption (nearly 50 g/day) is associated with a lower risk of type 2 diabetes in Asian populations and with an increased risk among the European/American ones.

Based on these findings, we can conclude that in the perspective of the optimization of type 2 diabetes prevention, the consumption of red and processed meat should be substantially restricted; conversely, the habitual inclusion in the diet of moderate amounts of dairy products – especially low-fat ones and yogurt – should be encouraged. Fish and eggs can be part of the diet in moderate amounts.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements:

G.R. and O.V. are responsible for study design; A.G., I.C., G.R., O.V, M.V., are responsible for writing; G.R., O.V., and M.V. have primary responsibility for the final content. All authors have read and approved the final manuscript.

Funding

This research was partially funded by (1) a research grant from the "Barilla Center for Food and Nutrition Foundation (BCFN)" within the framework of a project aimed at an evidence-based reformulation of the Food Pyramid for the prevention of cardiovascular disease; (2) Ministero della Salute Progetto di Ricerca Finalizzata RF-2016-02364513. G.R. is member of the scientific advisory board of the Barilla Health and Well-being advisory board, of the Nutrition Foundation of Italy and of "Istituto Nutrizionale Carapelli". The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, or in the decision to publish the results.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2022.110071.

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