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Nutrition and prostate cancer: review of the evidence

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ABSTRACT

Objectives: Of the possible causes of cancer, nutritional factors are supposed to play a major role in preventable cancers. Regarding prostate cancer, nutritional data remain contradictory. This article aims to review current evidence on the relation between nutrition and prostate cancer.

Material and methods: A systematic literature search for meta-analyses, systematic reviews, and pooled analyses was conducted in the PubMed database from its inception to September 2019. Eligible studies had to assess the association between nutrition and risk of prostate cancer.

Results: Generally, no evidence was found for an association between most food items or groups, including fruit, vegetables, meat, tea, coffee, and risk of prostate cancer. There was an inconsistent and weak positive association between milk and dairy foods and prostate cancer. Carbohydrates, vitamins, and minerals were not associated with prostate cancer. Furthermore, no association was found with dietary patterns such as vegetarian or pesco-vegetarian, but increased adherence to a Mediterranean diet seemed to have a protective effect. In general, large heterogeneity between studies was observed. Studies included in meta-analyses were mostly observational, and therefore prone to several inherent biases.

Conclusions: The evidence on any potential association between diet and prostate cancer is weak. The reductionist approach considering individual nutritional factors is not suitable, and conducting more observational studies or small randomised trials evaluating the impact of individual nutritional factors on prostate cancer will not bring further answers. Large, well-designed, randomised, controlled trials are mandatory in order to clarify the relationship between nutrition and prostate cancer.

KEY WORDS: diet, prostate cancer, review, nutrition, dietary patterns, dietary factors, dietary supplements.

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INTRODUCTION

In the 1980s, nutrition was estimated to contribute to the development of more than one third of cancers (35%) in Western countries [1], which made nutrition the second most important preventable cause of cancer, after smoking.

According to the latest burden of disease study, almost 530,000 deaths in 2016 in the US were attributed to dietary factors [2], which corresponds to approximately 19.3% of the total number of deaths in the United States of America (USA).

Prostate cancer is the second most commonly diagnosed cancer in men: almost 1.3 million men in the world were projected to be diagnosed with prostate cancer in 2018 [3]. Moreover, with an estimated number of associated deaths of 359,000 in 2018, prostate cancer is the fifth leading cause of death from cancer in men [3].

While prostate cancer aetiology remains mostly unknown, an association with nutritional factors is plausible; since prostate cancer presents a long latency, diet and nutrition might have an impact on its pro-

gression at several stages of the life cycle. Nonetheless, existing evidence supporting this association is mainly inconsistent [4-6].

Therefore, the aim of this article was to review the current evidence of the impact of several dietary factors on the risk of prostate cancer.

METHODS

A systematic literature search was conducted to identify meta-analyses or systematic reviews assessing the association between nutrition and prostate cancer. The literature search was restricted to articles published in English in the PubMed database from its inception to September 2019. A combination of key words and MeSH index terms was used including "prostate cancer" or "prostate neoplasm", "diet" or "dietary" or "dietary intake" and "meta-analysis" or "pooled analysis". Complementary searches were also conducted; more details are reported in the supplementary material (Appendix 1).

Eligible articles had to 1) present a meta-analysis or a pooled analysis of retrospective or prospective studies, and 2) report a summary estimate on risk of prostate cancer associated with either a measurement of food intake (dietary intake or supplementation) or with a measurement of the adherence to a dietary pattern.

Titles and abstracts were screened for eligibility. Full copies of eligible articles were retrieved and fully read.

For each article, the following information was extracted: the number and the design (cohort, case-control, or randomised controlled trial) of the included studies, total number of prostate cancer cases, sample size of the study, mean follow-up period, and the summary estimate with its corresponding confidence interval (CI). Moreover, information on the publication bias (type of assessment and results with *p*-value) as well as on the between-study heterogeneity (I^2 or *p*-value) were also extracted.

RESULTS

The literature search yielded 68 published meta-analyses investigating the association between nutrition and prostate cancer risk. The nutritional topics that were investigated were: vitamins and minerals (11 articles); fruit, vegetables, and carotenoids (11 articles); fat and fatty acids (10 articles); meat (11 articles); milk and dairy (seven articles); carbohydrates (four articles) and other dietary items (seven articles). Also, seven articles reported on the association between dietary patterns and risk of prostate cancer. Results in terms of strength and direction of the association are summarised in Table 1.

VITAMINS AND MINERALS

Eleven meta-analyses assessed the relationship between vitamins and/or minerals dietary intake or supplementation and risk of prostate cancer [7-17] (Appendix 2).

Overall, there was no statistically significant association between vitamin intake or supplementation and risk of prostate cancer, regardless of the type of vitamin and the study design. Only one meta-analysis of 18 observational studies found a significant decrease of 11% in prostate cancer risk when comparing highest and lowest dietary intake levels of vitamin C, with low to moderate heterogeneity ($I^2 = 39.4\%$) and no evidence of publication bias [7]. An earlier published meta-analysis [9], including two randomised, controlled trials (RCTs), compared vitamin C supplementation with placebo and found no association (summary relative risk [SRR] = 0.98 [95% CI: 0.91, 1.06]).

Five meta-analyses investigated the association between folic acid intake and risk of prostate cancer. Meta-analyses of observational studies did not suggest an association when comparing highest and lowest intakes of folate, with reported SRRs ranging from 0.83

TABLE 1. Summary of found evidence

	Decreases risk	Increases risk
Strong evidence	–	–
Probable evidence	**Nutrients:** lycopene **Dietary patterns:** adherence to WCRF/AICR dietary recommendations	**Food items/food groups:** milk and dairy
Limited-suggestive evidence	**Nutrients:** alpha-carotene, calcium **Food items/food groups:** • tofu, soy food • tomato • whole milk **Dietary patterns:** Mediterranean diet	**Nutrients:** • flavonoids • folic acid **Food items/food groups:** fried food
No evidence for an association	**Nutrients:** carbohydrates, vitamins (C, D, E, multivitamins), minerals, beta-carotene **Food items/food groups:** fruit and vegetables, meat and alternatives to meat (fish, seafood), eggs, tea, coffee, dietary acrylamide **Dietary patterns:** vegetarian, semi-vegetarian, and pesco-vegetarian diets	

(95% CI: 0.57, 1.20) to 1.02 (95% CI: 0.95, 1.09). Low to moderate amounts of heterogeneity were observed, with higher heterogeneity in case-control (I^2 = 57.7%) compared to cohort studies (I^2 = 0%). Summary relative risks were higher in RCTs compared to observational studies, but in two out of three meta-analyses, the association between intake of folate and risk of prostate cancer was not statistically significant. On the other hand, a meta-analysis of five RCTs found a significantly increased risk of 24% in patients randomised to folic acid compared to those randomised to placebo.

There was no association between zinc intake and risk of prostate cancer in two meta-analyses [10, 12], but the results remained heterogeneous (I^2 of 23.8 and 90%, respectively), and were based on a limited number of studies included in the analyses.

The results of studies investigating selenium intake and prostate cancer risk were inconsistent. A recently published Cochrane review of 21 observational studies found a statistically significant risk reduction of 16% when comparing highest and lowest selenium intake levels [14], with low heterogeneity across studies and no evidence of publication bias. However, the same review reported no association between selenium intake and prostate cancer risk in an analysis based on four RCTs. Similarly, two other meta-analyses [9, 12] found no association between selenium supplementation and risk of prostate cancer, but included a limited number of studies that were very heterogeneous (I^2 of 84% and 96%, respectively).

FRUIT, VEGETABLES, AND CAROTENOIDS

Eleven meta-analyses reported on the association between fruit, vegetable, and carotenoid intake or supplementation and prostate cancer risk [9, 12, 18-26] (Appendix 3).

Two meta-analyses investigated concomitantly the relationship between fruit and vegetable intake and prostate cancer [22, 23], both finding no association between fruit intake and prostate cancer, with no significant heterogeneity and no evidence of publication bias. Moreover, a more recent meta-analysis found no association with prostate cancer when comparing highest and lowest quartiles of apple consumption. No association was found between vegetable intake and risk of prostate cancer.

Three meta-analyses reported on the association between raw and cooked tomato intake and risk of prostate cancer [18, 20, 24]. The most recent and largest one, published by Rowles *et al.* [24], reported a significantly reduced risk of prostate cancer when comparing highest and lowest intakes of total tomato, tomato foods, and cooked tomato and sauces, with corresponding SRRs of 0.81 (95% CI: 0.71, 0.92), 0.84 (95% CI: 0.72, 0.98), and 0.84 (95% CI: 0.73, 0.99), respectively. However, heterogeneity between studies remained high (I^2 of 73.1%, 76.7%, and 57.4%, respectively) and statistical tests suggested publication bias.

On the other hand, five meta-analyses of observational studies examined the association between lycopene intake – a carotenoid found in particularly large amounts in tomatoes and associated products – and risk of prostate cancer [18-20, 25, 26]. An overall reduced risk of prostate cancer was observed with increasing consumption of lycopene, with risk reductions ranging from 3 to 12% across these studies.

In the most recent meta-analysis, based on 25 observational studies, the decreased risk was statistically significant (SRR = 0.88 [95% CI: 0.78, 0.98]), when comparing highest and lowest categories of lycopene intake. However, there was significant between-study heterogeneity (I^2 = 56.7%, p = 0.001) and Begg's test suggested potential publication bias. When stratified by study design (e.g. cohort vs case-control studies), the association remained significant only for cohort studies, with a higher pooled estimate (SRR = 0.93 [95% CI: 0.79, 0.99]), when compared to case-control studies (SRR = 0.83 [95% CI: 0.67, 1.02]). Between-study heterogeneity was higher in case-control studies (65.5%) when compared to cohort studies (11%).

Fewer meta-analyses reported on the association between other carotenoids and prostate cancer risk. When comparing highest and lowest intake levels of α-carotene from 12 observational studies, we found a borderline significant reduction of 13% in prostate cancer risk. On the other hand, no association was found between β-carotene dietary intake and/or supplementation and risk of prostate cancer in observational studies and RCTs [9, 12, 26], with SRRs ranging from 0.90 to 1.18.

FAT AND FATTY ACIDS

Ten meta-analyses reported on the association between fat and/or fatty acids intake and risk of prostate cancer [27-36] (Appendix 4).

Two meta-analyses examined the relationship between total, saturated, and unsaturated fat intake and prostate cancer risk [32, 35]. Both found no association between saturated or unsaturated fat and risk of prostate cancer. However, results were divergent for total fat, with one meta-analysis finding a significantly increased risk (SRR = 1.17 [95% CI: 1.10, 1.25]), and the other finding no association (SRR = 1.00 [95% CI: 0.99, 1.01]).

Concerning fatty acids, seven meta-analyses reported on the association between intake of alpha-linolenic acid (ALA) and prostate cancer. Only two meta-analyses found an increased risk of prostate cancer associated with ALA intake [28, 32]. In the other five meta-analyses, there was no association between ALA intake and risk of prostate cancer, with SRRs varying from 0.95 to 1.30,

which were higher in case-control studies compared to cohort studies. Overall, there was no evidence of publication bias, and heterogeneity ranged from 0 to 90%.

Regarding intake of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), current evidence suggests there is no association with prostate cancer risk, with SRRs ranging from 0.99 to 1.11. There were low to large amounts of heterogeneity (I^2 ranging from 0 to 61%) and most studies lacked reporting of publication bias.

There seemed to be no evidence of an association between linoleic acid intake in two meta-analyses reporting relevant data, with SRRs ranging from 0.83 to 1.27, which were larger in analyses based on case-control studies compared to analyses based on cohort studies.

Of note, in most analyses, the reporting on publication bias and heterogeneity was incomplete, and the number of included studies was limited.

MEAT, FISH, SEAFOOD, ALTERNATIVES TO MEAT, AND EGGS

Eleven meta-analyses reported on the association between meat, alternatives to meat, fish, seafood, and/or egg intakes and risk of prostate cancer [37-47] (Appendix 5).

Three meta-analyses suggested there was no association between red meat intake and risk of prostate cancer, regardless of the type of red meat (processed or unprocessed), with SRRs ranging from 0.99 to 1.06. In most analyses, funnel plots and publication bias tests suggested potential publication bias, and between-study heterogeneity ranged from 0 to 61%. Also, based on pooled results from 15 cohort studies, there was no evidence of an association between poultry or seafood and prostate cancer risk [44].

Szymanski *et al.* [43] investigated the relationship between fish consumption and prostate cancer separately in 12 case-control and 12 cohort studies and found divergent results, with SRRs of 0.85 (95% CI: 0.72, 1.00) and 1.01 (95% CI: 0.90, 1.14), respectively.

Based on four meta-analyses of cohort and case-control studies, higher intakes of tofu and/or soy food were generally associated with a significantly lower risk of prostate cancer, in comparison with lower intakes, with corresponding SRRs ranging from 0.65 to 0.75. The most recent meta-analysis published by Applegate *et al.* [38] reported risk reductions of 19% and 17% when comparing highest and lowest consumption of total soy food and tofu, respectively.

No association was found between isoflavones (generally found in soy) and risk of prostate cancer [38, 47].

Results from three meta-analyses suggested no association between intake of eggs and risk of prostate cancer, regardless of the study design (cohort vs case-control) and the quantification of eggs intake. There was no evidence of publication bias, and heterogeneity was low to moderate (I^2 ranging from 0 to 52.2%). However, based on four studies, Keum *et al.* [41] found a significantly increased risk of fatal prostate cancer associated with egg intake (SRR = 1.47 [95% CI: 1.01, 2.14]) with moderate heterogeneity (I^2 = 40%).

More generally, there was no association between protein intake and risk of prostate cancer [42].

MILK, DAIRY PRODUCTS, AND SOURCES OF CALCIUM

Seven meta-analyses investigated the association between milk, dairy, and/or calcium dietary intakes and supplementation and risk of prostate cancer [48-54] (Appendix 6).

In the most complete meta-analysis of cohort studies, Aune *et al.* [48] suggested that high intakes of total dairy, milk, low-fat milk, cheese, total calcium, dietary calcium, and dairy calcium were associated with increased risk of prostate cancer, when compared to lower intakes. Corresponding SRRs varied from 1.07 to 1.18, with low to moderate between-study heterogeneity (I^2 ranging from 0 to 53%). Conversely, high intakes of whole milk were associated with an 8% decrease in prostate cancer risk, when compared to lower intakes. There was no significant association between other types of dairy products such as yoghurt, non-dairy calcium, and supplemental calcium intakes and prostate cancer risk. Overall, results from other meta-analyses of observational studies were consistent with those of Aune *et al.* [48].

However, in a meta-analysis of seven RCTs, Bristow *et al.* [49] found a significant 46% reduction in prostate cancer risk when comparing calcium supplementation and placebo.

CARBOHYDRATES, DIETARY FIBRE, GLYCAEMIC LOAD, AND GLYCAEMIC INDEX

Four meta-analyses reported on the association between carbohydrates, dietary fibre, and whole grain intake and risk of prostate cancer [55-58] (Appendix 7). One meta-analysis also investigated the relationship between glycaemic index (GI), glycaemic load (GL), and prostate cancer [57].

Based on 17 studies, Sheng *et al.* [56] found a decrease in the risk of prostate cancer when comparing highest and lowest dietary fibre intakes, with significant heterogeneity (I^2 = 53.6%, p = 0.005). No evidence of publication bias was observed. When stratifying the analysis by study design, the decreased risk was statistically significant in case-control studies, with no heterogeneity, but not significant in cohort studies, with very large heterogeneity.

Wang *et al.* [57] investigated the potential association between dietary fibre intake, whole grains, carbohydrates, glycaemic index (GI), and glycaemic load (GL) and risk of prostate cancer in a systematic review

of 27 studies. The authors found no clear evidence of a relationship with prostate cancer risk. Funnel plot inspection and Egger's and Begg's tests suggested no evidence of publication bias. Heterogeneity was generally moderate, ranging from 39.5 to 69.5% for dietary fibre intake and GI, respectively.

Based on five cohorts and 16 case-control studies, Fan *et al.* [55] found no association between carbohydrate intake and prostate cancer risk. Funnel plot inspection and statistical tests indicated no evidence of publication bias. Significant heterogeneity among included studies was observed (I^2 = 62.7%). Results were not altered when the analysis was stratified by study design (data not shown); however, larger heterogeneity was observed in case-control studies when compared to cohorts (68.5% vs 7.8%). Results published in a previous meta-analysis are also consistent [58].

OTHER DIETARY ITEMS

Seven meta-analyses reported on the association between intake of other dietary items or food groups and risk of prostate cancer [59-65]. Dietary items/groups assessed were various and included tea, and coffee, flavonoids, fried food, and dietary acrylamide (Appendix 8).

Based on seven cohort studies, there was no association between tea consumption and risk of prostate cancer. Results regarding coffee intake were inconsistent: when comparing highest and lowest intakes, two meta-analyses of 13 and five prospective studies reported significant protective effects of coffee, with risk reductions of 10% [59] and 21% [64], respectively. Conversely, another meta-analysis of 12 observational studies found a significant harmful effect of coffee [62].

Increased intake of flavonoids was also associated with significant increases in risk of prostate cancer. However, results seemed to be driven by one large study; after its removal from the analysis, the pooled estimate lost statistical significance.

Also, fried food seemed to increase the risk of prostate cancer, but the limited number as well as the case-control design of the included studies imply caution when interpreting these results

Finally, no association was found between intake of dietary acrylamide and prostate cancer.

DIETARY PATTERNS

Seven meta-analyses assessed the relationship between different dietary patterns and risk of prostate cancer [66-72] (Appendix 9).

When comparing highest and lowest adherence to healthy dietary patterns, two meta-analyses found no association with prostate cancer [67, 69]. On the other hand, Fabiani *et al.* [67] reported a significantly increased risk of prostate cancer associated with highest adherence to western and carbohydrate dietary patterns (considered unhealthy), with corresponding increases in risk of 34% and 64%, respectively. Based on seven case-control studies, Grosso *et al.* [69] reported a similar estimate, with a 44% increase in risk associated with higher adherence to unhealthy dietary patterns. Nonetheless, this association was not observed in cohort studies.

No association with risk of prostate cancer was found when comparing vegetarian, pesco-vegetarian, and semi-vegetarian diets to a non-vegetarian diet, based on results pooled from four, four, and two cohort studies, respectively [69].

The most recent meta-analysis investigating the Mediterranean diet – high in vegetables, olive oil, complex carbohydrates, lean meats, and antioxidants – was found to decrease the risk of prostate cancer by 5%, with no heterogeneity. However, this decrease was not statistically significant, and the authors suggested potential publication bias. Results from two other previously published meta-analyses were also consistent [71, 72].

Furthermore, a meta-analysis of seven cohort studies suggested that adherence to the World Cancer Research Fund and American Institute for Cancer Research (CRF/AICR) dietary recommendations was associated with a significantly lower risk of prostate cancer. These dietary recommendations included: limiting the consumption of energy-dense foods and avoiding sugar-sweetened beverages, eating mostly foods of plant origin, reducing consumption of red meat and avoiding that of processed meat, and limiting alcoholic beverages [70].

DISCUSSION

The relationships between nutrition and prostate cancer risk have been the subject of a myriad systematic reviews, and this article provides a comprehensive overview of these publications.

Current evidence does not support an association between carbohydrates, fat and fatty acids, fruit and vegetables, meat and alternatives for meat, vitamins, minerals, and tea and risk of prostate cancer.

Several meta-analyses of observational studies suggested a positive association between milk and dairy and prostate cancer risk. Regarding calcium, results were inconsistent between observational studies and randomised trials, the latter suggesting a protective effect of calcium when compared to placebo. These contradictory results among types of dairy products and sources of calcium suggest that other elements instead of fat and calcium might be responsible for the increase in risk of prostate cancer.

Conversely, a potentially beneficial role of lycopene was detected in observational studies. Lycopene is found in high concentration in the prostate and is the most potent antioxidant among the carotenoids [73, 74]. It is thought that through its antioxidant powers, lycopene could reduce DNA damage in the prostate

[75]. However, further research is needed in order to better understand the mechanisms of absorption and degradation of lycopene in the prostate as well as other factors modulating these mechanisms, which remain mostly unknown [73, 76].

However, a "reductionist" approach considering intake of single foods and prostate cancer risk may not be the most suitable approach. The literature is overwhelmed with such studies: there are peer-reviewed publications associating cancer with almost every existent single aliment [77]. Considering dietary patterns instead of single nutrients or foods might be more appropriate because they are generally not consumed separately and the health-related effects could be interdependent [78].

Regarding dietary patterns, adhering to a Mediterranean diet seemed to have a slightly protective effect on the risk of prostate cancer, but the association was not statistically significant. Adherence to WCRF/AICR dietary recommendations was also found to be associated with a decreased risk of prostate cancer. On the other hand, dietary patterns such as vegetarian or pesco-vegetarian did not seem to be associated with prostate cancer.

Our study attempted to present a comprehensive overview of the epidemiology of prostate cancer. However, some limitations should be addressed. Firstly, generally large amounts of between-study heterogeneity were observed. This heterogeneity could be explained by differences across studies in the definition of the categories of intake. For some foods or food groups, either their myriad of definitions (e.g. fibre, carbohydrate) or their heterogeneous reporting and measurement (e.g. fat intake) could account for large parts of the observed between-study heterogeneity.

Secondly, studies included in meta-analyses were generally observational (cohort and case-control studies) and therefore prone to several inherent biases such as selection bias or information bias, particularly recall bias.

The observed associations may be due to confounding factors. For instance, food intake can be the reflection of a more general behaviour, e.g. people who eat well have other healthy behaviours, such as exercising, not smoking, are less obese, etc. Other socioeconomic factors such as social class can also be a source of confounding. Thus, the observed associations could be more the result of confounding by the aforementioned lifestyle factors than a causal nutrition-prostate cancer risk relationship. These aspects also need to be taken into consideration when interpreting these results. Further studies, and in particular well-designed randomised controlled trials, are mandatory to estimate the levels of evidence and attempt to better clarify the associations between nutrition and prostate cancer.

Overall, evidence for associations was at best probable, but in most of the cases it remained suggestive/limited; however, this is part of a much bigger picture: generally, studies investigating associations between malignancies and nutritional ingredients are based on weak evidence [77]. Also, one problematic aspect of observational studies on nutrition is that they measure dietary intake only once (e.g. at baseline); however, dietary intake during follow-up are not necessarily reflected by the baseline measured consumption [79]. Measuring food intake or adherence to dietary patterns more than once could be more appropriate.

The lack of association and the inconclusiveness of results might also be a result of weakness of evidence, small effect sizes, and nutritional studies' flawed assessment of dietary intake.

Based on existent evidence, it remains difficult to draw conclusions regarding the relationship between diet and prostate cancer. It is disappointing to see that despite the great number of published studies, very few associations stand out. Another myriad of observational studies or small randomised trials will not bring further answers [80]. More specifically, large, well-designed, randomised, controlled trials, are required in order to obtain stronger levels of evidence and attempt to better clarify the associations between dietary factors and prostate cancer risk. Orienting future research towards other nutrition-related topics (e.g. food security, climate change as a consequence of food production, differential food access due to social inequalities, etc.) would potentially help better comprehend ways in which nutrition influences cancer, and more specifically, prostate cancer.

DISCLOSURE

The authors report no conflict of interest.

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AUTHORS' CONTRIBUTIONS

MD, PB prepared research concept and design. MD collected data, analysed them and wrote the article. PM critically revised the article. All authors contributed to preparing the final publication.

Appendix 1. Detailed literature search queries

MAIN LITERATURE SEARCH

("prostate cancer" OR ("prostate" and "cancer") OR "Prostate Neoplasms"[MeSH] OR "prostate carcinoma" OR "prostatic cancer" OR ("prostatic" AND "cancer") OR ("prostatic" AND "neoplasms"))
AND
("diet" OR Diet[MeSH] or "dietary" or "dietary intake" or "nutrition")
AND
("meta-analysis"[Publication Type] OR "meta-analysis as topic"[MeSH] OR "meta-analysis" OR "pooled analysis")

OTHER COMPLEMENTARY LITERATURE SEARCHES

VITAMIN D

("prostate cancer" OR ("prostate" and "cancer") OR "Prostate Neoplasms"[MeSH] OR "prostate carcinoma" OR "prostatic cancer" OR ("prostatic" AND "cancer") OR ("prostatic" AND "neoplasms"))
AND
("Vitamin D"[Mesh] OR "vitamin D" OR "Ergocalciferols"[Mesh] OR "ergocalciferol" OR "Cholecalciferol"[Mesh] OR "Cholecalciferol")
AND
("meta-analysis"[Publication Type] OR "meta-analysis as topic"[MeSH] OR "meta-analysis" OR "pooled analysis")

RED MEAT

("prostate cancer" OR ("prostate" and "cancer") OR "Prostate Neoplasms"[MeSH] OR "prostate carcinoma" OR "prostatic cancer" OR ("prostatic" AND "cancer") OR ("prostatic" AND "neoplasms"))
AND
("red meat" or "meat")
AND
("meta-analysis"[Publication Type] OR "meta-analysis as topic"[MeSH] OR "meta-analysis" OR "pooled analysis")

Appendix 2. Characteristics of meta-analyses investigating the association between vitamins and minerals and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH, RCT)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Vitamins									
Bai, 2015	Vitamin C	Highest vs lowest	18 (6 CH, 12 CC)	15,926	103,658	NR	0.89 (0.83, 0.94)	pEgger = 0.295, pBegg = 0.173, funnel plot inspection did not suggest PB	39.4%, 0.045
Jiang, 2010	Vitamin C supplementation	Vs placebo	2 RCT	2,346	44,002	6.1	0.98 (0.91, 1.06)	NR	0%, 0.47
	Vitamin E supplementation	Vs placebo	5 RCT	3,580	85,549	6.2	0.96 (0.85, 1.08)	NR	64%, 0.03
Gilbert, 2011	Vitamin D	Per 1000 IU increase	4 CH, 2 NCC	5,997	116,061	1.1-10	1.14 (0.98, 1.31)	Funnel plot inspection suggested PB	0%, 0.572
	Vitamin D	Per 1000 IU increase	5 CC	2,725	5,726	NA	0.83 (0.28, 2.43)	Funnel plot inspection suggested PB	49.2%, 0.097
Stratton, 2011	Vitamin E supplementation	Vs no vitamin E supplementation	10 (5 CH, 2 CC, 3 RCT)	21,608	501,292	5.7*	1.02 (0.89, 1.16)	NR	93%, 0.00001
	Multivitamins supplementation	Vs no multivitamin supplementation	5 (2 CH, 2 CC, 1 RCT)	3,837	305,851	10.6*	1.11 (0.95, 1.29)	NR	65%, 0.03
Folic acid									
Tio, 2014	Folate	Highest vs lowest	11 (5 CH, 6 CC)	15,336	146,782	NR	0.97 (0.89, 1.06)	pEgger = 0.22	41.9%, 0.07
		Highest vs lowest	5 CH	12,898	140,428	NR	1.00 (0.96, 1.05)	pEgger = 0.67	0%, 0.88
		Highest vs lowest	6 CC	2,438	6,354	NA	0.83 (0.57, 1.20)	pEgger = 0.95	57.7%, 0.04
Wang, 2014	Folate	Highest vs lowest	5 CH	NR	192,702	14.1	1.02 (0.95, 1.09)	pEgger = 0.694, pBegg = 0.806	0%, 0.959
Qin, 2013	Folic acid	Vs control	5 RCT	508	27,065	NR	1.17 (0.84, 1.62)	NR	54.3%, 0.07
Vollset, 2013	Folic acid	Vs placebo	12 RCT	656	44,177	5.5**	1.15 (0.94, 1.41)	NR	NR
Wien, 2012	Folic acid	> 0.4 g/day vs control	5 RCT	632	25,738	5.1	1.24 (1.03, 1.49)	NR	17%, 0.31
Minerals									
Vinceti, 2018	Selenium	Highest vs lowest	4 RCT	1,020	18,942	NR	1.01 (0.90, 1.14)	NR	0%
	Selenium	Highest vs lowest	21 observational studies	576,667	14,950	NR	0.84 (0.75, 0.95)	Funnel plot inspection did not suggest PB	35%
Mahmoud, 2016	Zinc	Highest vs lowest	17 (3 CH, 2 NCC, 11 CC, 1 RCT)	11,689	111,199	NR	1.07 (0.98, 1.16)	pEgger = 0.679, funnel plot inspection did not suggest PB	23.8%, 0.125
Stratton, 2011	Zinc supplementation	Vs no zinc supplementation	3 (1 CH, 2 CC)	2,930	40,082	NR	1.18 (0.71, 0.96)	NR	90%, 0.0001
	Selenium supplementation	Vs no selenium Supplementation	2 (1 CC, 1 RCT)	2,249	20,923	5.5*	1.57 (0.68, 3.61)	NR	96%, 0.00001
Jiang, 2010	Selenium supplementation	Vs placebo	2 RCT	912	18,700	6.5	0.78 (0.41, 1.48)	NR	84%, 0.01

*CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, * mean FU computed with FU values of cohort studies and/or RCTs, ** mean FU for all included studies including several cancer sites was abstracted, § I^2 represents the percentage of heterogeneity between studies not explained by chance*

Appendix 3. Characteristics of meta-analyses investigating the association between fruit, vegetables, and carotenoids and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH, RCT)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Fruit and vegetables									
Rowles, 2018	Total tomato intake	Highest vs lowest	26 (5 CH, 2 NCC, 19 CC)	22,555	266,037	NR	0.81 (0.71, 0.92)	pEgger = 0.011, pBegg = 0.003	73.1%, 0.000
	Raw tomato intake	Highest vs lowest	10 (3 CH,1 NCC, 6 CC)	9808	170,085	NR	0.95 (0.84, 1.09)	pEgger = 0.568, pBegg = 0.592	55.6%, 0.016
	Tomato foods intake	Highest vs lowest	18 (4 CH, 1 NCC, 13 CC)	14,215	211,841		0.84 (0.72, 0.98)	pEgger = 0.037, pBegg = 0.053	76.7%, 0.000
	Cooked tomato and sauces intake	Highest vs lowest	10 (3 CH, 1 NCC, 6 CC)	13,925	166,535	NR	0.84 (0.73, 0.99)	pEgger = 0.019, pBegg = 0.020	57.4%, 0.012
Petimar, 2017	Fruit intake	Highest vs lowest	15 CH	52,680	842,149	9-22	1.01 (0.98, 1.04)	NR	0.69
	Vegetables intake	Highest vs lowest	15 CH	52,680	842,149	9-22	0.99 (0.96, 1.02)	NR	0.55
Fabiani, 2016	Apple intake	Highest vs lowest	2 CC	1,344	8,073	NA	0.93 (0.79, 1.09)	NR	0%, 0.664
Meng, 2014	Fruit intake	Highest vs lowest	14 CH	26,297	1,078,471	11.1 (3-33)**	1.02 (0.98, 1.07)	pEgger = 0.092, pBegg = 0.511, funnel plot inspection did not suggest PB	0%, 0.929
	Vegetables intake	Highest vs lowest	12 CH	27,223	869,758	11.1 (3-33)**	0.97 (0.93, 1.01)	pEgger = 0.549, funnel plot inspection did not suggest PB	0%, 0.505
Chen, 2013	Raw tomato intake	Highest vs lowest	3 CH	2,292	84,525	5	0.81 (0.38, 0.95)	NR	74%, 0.02
	Cooked tomato intake	Highest vs lowest	2 CH	3,819	70,996	8	0.85 (0.69, 1.06)	NR	64%, 0.09
Etminan, 2004	Raw tomato intake	Highest vs lowest	9 (2 CH, 7 CC)	6,459	60,333	NR	0.89 (0.80, 1.00)	Funnel plot inspection did not suggest PB	0.05
	Cooked tomato intake	Highest vs lowest	6 (1 CH, 5 CC)	5,747	53,905	NR	0.81 (0.71, 0.92)	NR	0.90
Carotenoids									
Rowles, 2017	Lycopene intake	Highest vs lowest	25 (8 CH, 1 NCC, 16 CC)	36,336	443,815	10*	0.88 (0.78, 0.98)	pEgger = 0.130, pBegg = 0.032	56.7%, 0.001
Chen, 2015	Lycopene intake	Highest vs lowest	13 (2 CH, 3 NCC, 8 CC)	13,180	171,468	10*	0.91 (0.82, 1.01)	NR	45.5%, 0.037
	Lycopene intake	Per 5 mg/day	13 (2 CH, 3 NCC, 8 CC)	13,180	171,468	10*	0.97 (0.93, 1.01)	pEgger = 0.22, pBegg=0.20	50.2%, 0.020
Wang, 2015	Lycopene intake	Highest vs lowest	13 (4 CH, 9 CC)	7,327	178,643	6.8*	0.88 (0.76, 1.02)	NR	23.6%, 0.02
	Alpha-carotene	Highest vs lowest	12 (4 CH, 8 CC)	6,492	159,026	10.1*	0.87 (0.76, 0.99)	NR	15.5%, 0.16
	Beta-carotene	Highest vs lowest	19 (8 CH, 11 CC)	10,164	209,075	12*	0.90 (0.81, 1.01)	NR	26%, 0.10
Chen, 2013	Lycopene intake	Highest vs lowest	5 (3 CH, 2 NCC)	8,350	141,359	8	0.93 (0.86, 1.01)	Funnel plot inspection did not suggest PB	18%, 0.30
Stratton, 2011	Beta-carotene supplementation	Vs no beta-carotene supplementation	3 (1 CH, 1 CC, 1 RCT)	2,886	43,060	5.4*	1.18 (0.61, 2.30)	NR	95%, < 0.00001
Jiang, 2010	Beta-carotene supplementation	Vs placebo	3 RCT	2,332	61,656	7.4	0.97 (0.90, 1.05)	NR	0%, 0.37
Etminan, 2004	Lycopene intake	Highest vs lowest	10 (3 CH, 7 CC)	5,241	111,912	NR	0.89 (0.81, 0.98)	Funnel plot inspection did not suggest PB	0.23

*CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, * mean FU computed with FU values of cohort studies, ** mean FU for all studies (fruit and vegetables), § I^2 represents the percentage of heterogeneity between studies not explained by chance*

Appendix 4. Characteristics of meta-analyses investigating the association between fat and fatty acids and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Fat									
Xu, 2015	Total fat	Per 28.35 g (1 ounce)	13 CH	36,144	692,771	9.5 (2-17.4)	1.00 (0.99, 1.01)	pEgger = 0.93	5.0%, 0.34
	Saturated fat	Per 28.35 g (1 ounce)	9 CH	33,983	625,789	9.6 (2-17.4)	1.00 (1.00, 1.00)	pEgger = 0.01	14.3%, 0.32
	Unsaturated fat	Per 28.35 g (1 ounce)	10 CH	34,644	648,764	9.2 (2-17.4)	0.99 (0.96, 1.02)	pEgger = 0.16 and 0.92 for mono and poly-unsaturated fat	4.4%, 0.40
Dennis, 2004	Total fat	Per 45 g/day	15	NR	NR	NR	1.17 (1.10, 1.25)	NR	56%, 0.003
	Saturated fat	Per 25 g/day	14	NR	NR	NR	1.09 (0.99, 1.20)	NR	65%, < 0.001
	Monosaturated fat	Per 20 g/day	9	NR	NR	NR	1.04 (0.94, 1.15)	NR	30%, 0.17
	Polyunsaturated fat	Per 20 g/day	8	NR	NR	NR	1.06 (0.88, 1.27)	NR	13%, 0.32
Fatty acids									
Alexander, 2015	PUFA	Highest vs lowest intake	12 (9 CH, 3 NCC)	NR	255,643	9.8 (1.9-20)	1.00 (0.93, 1.09)	Funnel plot, Egger test, and Duval and Tweedie method did not indicate PB	50.4%, 0.019
Fu, 2015	ALA	Per 0.5 g/day	5 (3 CH, 2 NCC)	7781	183,495	8	0.99 (0.98, 1.00)	pBegg = 0.81	0%, 0.670
	EPA	Per 0.5 g/day	5 (3 CH, 2 NCC)	6525	153,903	8	1.02 (0.99, 1.05)	NR	36.1%, 0.181
Carleton, 2013	ALA	Highest vs lowest intake	12 (3 CH, 2 NCC, 7 CC)	14,795	227,309	8	1.08 (0.90, 1.29)	pEgger > 0.527,pBegg > 0.165	85%, < 0.00001
	ALA	Highest vs lowest intake	5 (3 CH, 2 NCC)	10,748	218,500	8	0.95 (0.84, 1.09)	NR	69%, 0.01
	ALA	Highest vs lowest intake	7 CC	4,047	8,809	NA	1.30 (0.81, 2.07)	NR	90%, < 0.00001
Chua, 2012	ALA	Highest vs lowest intake	5 CH	NR	228,668	NR	0.96 (0.86, 1.07)	pEgger = 0.34, pBegg = 0.30, funnel plot suggesting no PB	63%, 0.028
	EPA	Highest vs lowest intake	4 CH	NR	196,192	NR	1.00 (0.92, 1.08)	pEgger = 0.65, pBegg = 0.60, funnel plot suggesting no PB	61%, 0.055
	DHA	Highest vs lowest intake	4 CH	NR	196,192	NR	0.99 (0.92, 1.07)	pEgger = 0.54, pBegg = 1.0, funnel plot suggesting no PB	58%, 0.070
Carayol, 2010	ALA	Highest vs lowest intake	5 CH	10,748	NR	9	0.97 (0.86, 1.10)	NR	60.9%, 0.04
	ALA	> 1.5 g/day vs < 1.5 g/day	5 CH	10,169	NR	9	0.95 (0.91, 0.99)	NR	0%, 0.74
Simon, 2009	ALA	Highest vs lowest intake	11 (3 CH, 2 NCC, 6 CC)	NR	170,886	NR	1.09 (0.91, 1.32)	NR	< 0.01
Brouwer, 2004	ALA	Highest vs lowest intake*	9 (2CH, 2 NCC, 5 CC)	NR	NR	NR	1.70 (1.12, 2.58)	NR	NR
Dennis, 2004	Linoleic acid	Per 10 g/day	5	NR	NR	NR	0.96 (0.85, 1.09)	NR	21%, 0.39
	ALA	Per 1.5 g/day	5	NR	NR	NR	1.26 (1.10, 1.45)	NR	88%, < 0.001
	EPA	Per 0.5 g/day	2	NR	NR	NR	1.11 (1.00, 1.24)	NR	0%, 0.55
	DHA	Per 0.5 g/day	2	NR	NR	NR	1.05 (0.99, 1.11)	NR	0%, 0.76
Zock, 1998	Linoleic acid	Highest vs lowest intake	3 CC	654	1578	NR	1.27 (0.97, 1.66)	NR	NR
	Linoleic acid	Highest vs lowest intake	2 CH	399	62,771	NR	0.83 (0.56, 1.24)	NR	NR

CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, PUFA – poly-unsaturated fatty acids, ALA – alpha *linoleic acid, EPA – eicosapentaenoic acid, DHA – docosahexaenoic acid, *combined estimates for highest vs lowest ALA intakes and highest vs lowest ALA blood levels, also included estimates for comparing highest vs lowest ALA blood levels in this meta-analysis § I^2 represents the percentage of heterogeneity between studies not explained by chance*

Appendix 5. Characteristics of meta-analyses investigating the association between meat, fish, seafood, alternatives to meat, eggs, and proteins and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Meat									
Wu, 2016	Total red meat	> 120 g/day vs 20-40 g/day	15 CH	52,683	824,149	9-22	0.99 (0.94, 1.03)	NR	24%, 0.23
	Unprocessed red meat	>100 g/day vs < 20 g/day	15 CH	52,683	824,149	9-22	1.02 (0.98, 1.06)	NR	2%, 0.43
	Processed red meat	> 40 g/day vs < 5 g/day	15 CH	52,683	824,149	9-22	1.04 (1.01, 1.08)	NR	0%, 0.61
	Poultry	> 45 g/day vs < 5 g/day	15 CH	52,683	824,149	9-22	1.05 (1.00, 1.09)	NR	0%, 0.55
Bylsma, 2015	Total red meat	Highest vs lowest	10 CH	23,170	411,729	11.8	1.02 (0.92, 1.12)	pEgger = 0.963, funnel plot suggested slight PB	61%, 0.006
	Fresh red meat	Highest vs lowest	9 CH	13,007	243,396	11.8	1.06 (0.97, 1.16)	pEgger = 0.001, funnel plot suggested slight PB	38.3%, 0.113
	Processed red meat	Highest vs lowest	11 CH	27,705	568,147	12.8	1.05 (1.01, 1.10)	pEgger = 0.211	3.38%, 0.406
Alexander, 2010	Red meat	Highest vs lowest	15 CH	NR	NR	NR	1.00 (0.96, 1.05)	Funnel plot suggested slight PB	0.264
	Processed red meat	Highest vs lowest	11 CH	NR	NR	NR	1.05 (0.99, 1.12)	pEgger = 0.013, funnel plot suggested PB	0.088
Alternatives to meat									
Applegate, 2018	Total soy food	Highest vs lowest	16 (7 CH, 11 CC)	11,266	209,151	NR	0.71 (0.58, 0.85)	pEgger = 0.052, pBegg = 0.300	68.9%
	Non-fermented soy food	Highest vs lowest	11 (5 CH, 6 CC)	5,788	81,435	NR	0.65 (0.56, 0.83)	pEgger = 0.117, pBegg = 0.161	60.3%
	Tofu	Highest vs lowest	5 (3 CH, 2 CC)	865	32,618	NR	0.73 (0.57, 0.94)	pEgger = 0.093, pBegg = 0.221	4.5%
Hwang, 2009	Tofu	Highest vs lowest	5 (3 CH, 2 CC)	947	32,618	28*	0.73 (0.57, 0.92)	pBegg = 0.079	0.428
	Total soy food	Highest vs lowest	5 (2 CH, 3 CC)	2,395	65,539	21*	0.69 (0.57, 0.84)	pBegg = 0.295	0.544
	Non-fermented soy food	Highest vs lowest	8 (4 CH, 4 CC)	2,885	48,529	25*	0.75 (0.62, 0.89)	pEgger = 0.047, funnel plot suggested PB	0.413
Yan, 2009	Soy food	Highest vs lowest	14 (5 CH, 9 CC)	9,732	171,487	NR	0.74 (0.63, 0.89)	pEgger = 0.05, pBegg = 0.16	NR
Yan, 2005	Non-fermented soy food	Highest vs lowest (and none vs some)	8 (2 CH, 6 CC)	4217	25,742	NR	0.70 (0.59, 0.83)	Authors claim no evidence of PB	NR

Appendix 5. Cont.

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Fish and seafood									
Wu, 2016	Seafood	> 40 g/day vs < 5 g/day	15 CH	52,683	824,149	9-22	1.04 (0.98, 1.09)	NR	25%, 0.22
Szymanski, 2010	Fish	Highest vs lowest	12 CH	445,820	13,924	NR	1.01 (0.90, 1.14)	pEgger = 0.84, pBegg = 0.78, funnel plot inspection did not suggest PB	0.005
	Fish	Highest vs lowest	12 CC	15,582	5777	NA	0.85 (0.72, 1.00)	pEgger = 0.62, pBegg = 0.68, funnel plot inspection did not suggest PB	0.05
Eggs									
Wu, 2016	Eggs	>25 g/day vs <5 g/day	15 CH	52,683	824,149	9-22	0.99 (0.96, 1.02)	NR	0%, 0.97
Keum, 2015	Eggs	Per increase of 5 eggs/week	6	3,655	NR	NR	1.00 (0.88, 1.14)	pEgger = 0.72	0%, 0.69
Xie, 2012	Eggs	Highest vs lowest	6 CH	4,087	247,432	NR	0.97 (0.87, 1.07)	pEgger = 0.401, pBegg = 0.452	0%, 0.441
	Eggs	Highest vs lowest	11 CC	3,714	10,779	NR	1.09 (0.86, 1.31)	pEgger = 0.151, pBegg = 0.533, funnel plot inspection did not suggest PB	52.2%, 0.022
Isoflavones									
Applegate, 2018	Isoflavones	Highest vs lowest	6 (2 CH, 1 NCC, 3 CC)	11,812	133,061	NR	1.03 (0.97, 1.09)	pEgger = 0.802, pBegg = 0.707	44.9%
Yan, 2009	Isoflavones	Highest vs lowest	8 (2 CH , 6 CC)	8,353	133,270	NR	0.88 (0.76, 1.02)	Authors claim no evidence of PB	NR
Protein									
Mao, 2018	Protein	Highest vs lowest	12 (1 RCT, 8 CH, 3 CC)	13,483	286,245	9.5*	0.99 (0.93, 1.06)	pEgger = 0.296, funnel plot inspection did not suggest PB	0%, 0.656

*CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, * mean FU computed with FU values of cohort studies; § I^2 represents the percentage of heterogeneity between studies not explained by chance*

Appendix 6. Characteristics of meta-analyses investigating the association between milk, dairy, and calcium and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2\S}$ or *p*-value)
Milk and dairy									
Aune, 2015	Total dairy	Highest vs lowest	15 CH	38,107	848,395	10.7	1.09 (1.02, 1.17)	pEgger = 0.08, pBegg = 0.02	43%, 0.04
	Milk	Highest vs lowest	15 CH	11,392	566,146	9.7	1.11 (1.03, 1.21)	pEgger = 0.06, pBegg = 0.66	21%, 0.22
	Whole milk	Highest vs lowest	8 CH	19,664	448,719	11.6	0.92 (0.85, 0.99)	pEgger = 0.04, pBegg = 0.11	0%, 0.69
	Low fat milk	Highest vs lowest	6 CH	19,430	432,943	12.2	1.14 (1.05, 1.25)	NR	51%, 0.09
	Cheese	Highest vs lowest	11 CH	22,950	887,759	11.3	1.07 (1.01, 1.13)	pEgger = 0.57, pBegg = 0.44	0%, 0.56
	Yoghurt	Highest vs lowest	6 CH	18,351	623,112	7.4	1.12 (0.97, 1.29)	pEgger = 0.45, pBegg = 0.62	67%, 0.02
Huncharek, 2008	Dairy	Highest vs lowest	11 CH	10,952	300,172	12.2	1.11 (1.03, 1.19)	NR	0.33
	Milk	Highest vs lowest	11 CH	4,452	195,440	12.8	1.06 (0.91, 1.23)	NR	NR
	Cheese	Highest vs lowest	7 CH	7,213	211,702	12.5	1.11 (0.99, 1.25)	NR	NR
Qin, 2007	Milk or dairy	Highest vs lowest	13 CH	7,546	297,119	4-23	1.13 (1.02, 1.24)	Funnel plot inspection did not suggest PB, pEgger = 0.45	NR
Gao, 2005	Dairy	Highest vs lowest	10 CH	8,383	282,887	11.2	1.11 (1.00, 1.22)	Funnel plot inspection did not suggest PB	28%, > 0.2
Qin, 2004	Milk	Highest vs lowest	11 CC	2,929	6,949	NA	1.68 (1.34, 2.12)	NR	< 0.05
Calcium									
Rahmati, 2018	Total calcium	Highest vs lowest	12 (11 CH, 1 CC)	NR	905,046	9.9*	1.15 (1.04, 1.27)	pBegg = 0.02	59.7%, 0.006
Aune, 2015	Total calcium	Highest vs lowest	9 CH	33,127	750,275	9.6	1.10 (1.01, 1.21)	pEgger = 0.26, pBegg = 0.12	50%, 0.04
	Dietary calcium	Highest vs lowest	15 CH	35,493	800,879	8.9	1.18 (1.08, 1.30)	pEgger = 0.11, pBegg = 0.37	53%, 0.008
	Dairy calcium	Highest vs lowest	7 CH	10,493	479,666	10.5	1.13 (1.02, 1.24)	pEgger = 0.31, pBegg = 0.13	46%, 0.08
	Non-dairy calcium	Highest vs lowest	4 CH	13,067	442,796	7.5	0.91 (0.79, 1.05)	pEgger = 0.92, pBegg = 1.00	15%, 0.32
	Supplemental calcium	Highest vs lowest (and use vs no use)	9 (8 CH, 1 RCT)	30,232	498,516	8.5	1.00 (0.95, 1.05)	pEgger = 0.36, pBegg = 0.31	0%, 0.68
Bristow,2013	Calcium supplementation	Calcium vs placebo	7 RCT	48	7,693	3.9	0.54 (0.30, 0.96)	Funnel plot inspection did not suggest PB	0%
Huncharek, 2008	Dairy calcium	Highest vs lowest	4 CH	2,282	56,327	13.1	1.18 (1.06, 1.33)	NR	0.02
	Total/dietary calcium	Highest vs lowest	5 CH	8,327	199,993	10.2	1.15 (1.02, 1.30)	NR	NR
Gao, 2005	Calcium	Highest vs lowest	6 CH	7,154	222,940	7.5	1.39 (1.09, 1.77)	Funnel plot inspection did not suggest PB	45%, 0.107

*CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, * mean FU computed with FU values of cohort studies; § I^2 represents the percentage of heterogeneity between studies not explained by chance*

Appendix 7. Characteristics of meta-analyses investigating the association between carbohydrates, dietary fibre, and GI/GL and prostate cancer risk

First author, year	Dietary item assessed	No. of studies and design (CC, CH)	Total cases	Sample size	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Carbohydrates							
Wang, 2015	Carbohydrate	13 (3 CH, 10 CC)	7,757	76,049	0.96 (0.81, 1.14)	Funnel plot, pEgger = 0.598, pBegg = 0.428	51.2%, 0.017
Fan, 2018	Carbohydrate	21 (5 CH, 16 CC)	11,573	98,739	1.11 (0.98, 1.26)	Funnel plot inspection did not suggest PB, pEgger non-significant	62.7%, 0.000
Zhai, 2015	Carbohydrate	5 CH, 13 CC	8,046	84,687	1.06 (0.93, 1.20)	pEgger = 0.14, pBegg = 0.82	46.8%, 0.02
	Carbohydrate	5 CH	3,679	74,115	1.06 (0.88, 1.28)	NR	24%, 0.261
	Carbohydrate	13 CC	4,367	10,572	1.04 (0.87, 1.23)	NR	51.3%, 0.017
Dietary fibre							
Sheng, 2015	Dietary fibre	17 (5 CH, 12 CC)	13,484	255,026	0.89 (0.77, 1.01)	Funnel plot, pEgger = 0.946, pBegg = 0.753	53.6%, 0.005
		5 CH	9,640	247,310	0.94 (0.77, 1.11)	NR	74.3%, 0.004
		12 CC	3,844	7,716	0.82 (0.68, 0.96)	NR	17%, 0.277
Wang, 2015	Dietary fibre	16 (5 CH, 11 CC)	13,330	255,670	0.94 (0.85, 1.05)	Funnel plot, pEgger = 0.545, pBegg = 0.558	39.5%, 0.053
Whole grains							
Wang, 2015	Whole grains	8 (3 CH, 5 CC)	8,877	96,246	1.13 (0.98, 1.30)	Funnel plot, pEgger = 0.475, pBegg = 1.000	52.5%, 0.040
GI/GL							
Wang, 2015	Glycaemic index	6 (3 CH, 3 CC)	26,656	352,882	1.06 (0.96, 1.18)	Funnel plot, pEgger = 0.299, pBegg = 0.260	69.5%, 0.006
	Glycaemic load	5 (3 CH, 2 CC)	26,500	352,452	1.04 (0.91, 1.18)	Funnel plot, pEgger = 0.247, pBegg = 0.221	67%, 0.016

CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, GI – glycaemic index, GL – glycaemic load. Mean FU was not reported in any of the studies; all studies compared highest and lowest levels of exposure, § I^2 represents the percentage of heterogeneity between studies not explained by chance

Appendix 8. Characteristics of meta-analyses investigating the association between other dietary items and prostate cancer risk

First author, year	Dietary item assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Coffee and tea									
Grosso, 2017	Coffee	Highest vs lowest	13 CH	34,105	539,577	NR	0.90 (0.85, 0.95)	NR	17%
Zhang, 2014	Tea	Per 1 cup per day increase	7 CH	4,837	187,017	7.0-37.0	1.02 (0.98, 1.06)	NR	62.7%, 0.009
		Highest vs lowest	7 CH	4,837	187,017	7.0-37.0	1.05 (0.87, 1.27)	NR	59.1%, 0.017
Yu, 2011	Coffee	Highest vs no coffee/lowest	5 CH	NR	63,787	NR	0.79 (0.61, 0.98)	NR	57.1%, 0.053
Park, 2010	Coffee	Highest vs lowest	12 (4 CH, 8 CC)	4,775	59,328	NA	1.16 (1.01, 1.33)	pEgger < 0.001, funnel plot suggested PB	6.5%
		Highest vs lowest	4 CH	666	49,348	NR	1.06 (0.83, 1.35)	NR	0%
		Highest vs lowest	8 CC	4,109	9,980	NA	1.21 (1.03, 1.43)	NR	27.4%
Flavonoids									
Guo, 2016	Flavonoids	Highest vs lowest	4 (3 CH, 1 CC)	8,863	112,100	18*	1.12 (1.02, 1.23)	pEgger = 0.476, pBegg = 1.000	0%, 0.962
Fried food									
Lippi, 2015	Fried food	Highest vs lowest	4 CC	2,579	4,856	NA	1.35 (1.17, 1.57)	NR	43%
Dietary acrylamide									
Pelucchi, 2015	Dietary acrylamide	Per 10 µg/day increase	6 (4 CH, 2 CC)	13,559	NR	NR	1.00 (0.99, 1.02)	NR	0.74
		Highest vs lowest	6 (4 CH, 2 CC)	13,559	NR	NR	1.00 (0.93, 1.08)	NR	0.81

CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer,
** mean FU computed with FU values of cohort studies; § I^2 represents the percentage of heterogeneity between studies not explained by chancew*

Appendix 9. Characteristics of meta-analyses investigating the association between dietary patterns and prostate cancer risk

First author, year	Diet/dietary pattern assessed	Analysis conducted	No. of studies and design (CC, CH)	Total cases	Sample size	Mean FU (range)	SRR (95% CI)	Publication bias (type of assessment and results with *p*-value)	Heterogeneity ($I^{2§}$ or *p*-value)
Godos, 2017	Vegetarian diet	Vs non-vegetarian diet	4 CH	1,935	60,391	14.3	0.83 (0.63, 1.10)	Funnel plot inspection did not suggest PB	56%, 0.11
	Pesco-vegetarian diet	Vs non-vegetarian diet	4 CH	1,935	60,391	14.3	1.00 (0.75,1.34)	Funnel plot inspection did not suggest PB	53%, 0.12
	Semi-vegetarian diet	Ws non-vegetarian diet	2 CH	1,478	44,797	14.1	1.18 (0.95, 1.45)	Funnel plot inspection did not suggest PB	0%, 0.97
Jankovic, 2017	WCRF/AICR diet	Per 1 point increase in WCRF/AICR diet score	7 CH	4,975	361,616	10-15	0.94 (0.92, 0.97)	NR	0%
Grosso, 2017	Healthy dietary pattern	Highest vs lowest	7 CC	2,648	8,028	NA	0.99 (0.85, 1.15)	NR	18%
	Healthy dietary pattern	Highest vs lowest	3 CH	4,156	66,131	11.7	0.99 (0.90, 1.08)	NR	0%
	Unhealthy dietary pattern	Highest vs lowest	7 CC	2,648	8,028	NA	1.44 (1.21, 1.71)	NR	62%
	Unhealthy dietary pattern	Highest vs lowest	3 CH	4,156	66,131	11.7	0.87 (0.71, 1.07)	NR	59%
Fabiani, 2016	Healthy dietary pattern	Highest vs lowest	12 (3 CH, 9 CC)	7,410	75,718	12.1*	0.96 (0.88, 1.04)	pEgger = 0.538, pBegg = 0.583	0%, 0.724
	Western dietary pattern	Highest vs lowest	12 (3 CH, 9 CC)	7,410	75,718	12.1*	1.34 (1.08, 1.65)	pEgger = 0.045, pBegg = 0.583	74.6%, 0.0001
	Carbohydrate dietary pattern	Highest vs lowest	4 CC	1,888	4,118	NA	1.64 (1.35, 2.00)	pEgger = 0.799, pBegg = 1.000	0%, 0.393
Cheng, 2019	Mediterranean dietary pattern	Highest vs lowest	10 (5 CH, 5 CC)	33,451	403,320	13.4*	0.95 (0.90, 1.01)	pEgger = 0.770, pBegg = 0.049; asymmetric funnel plot	12.7%, 0.326
Schwingshackl, 2017	Mediterranean dietary pattern	Highest vs lowest	6 (3 CH, 3 CC)	29,806	350,814	13.7*	0.96 (0.92, 1.00)	NR	0%
Schwingshackl, 2014	Mediterranean dietary pattern	Highest vs lowest	5 (4 CH, 1 CC)	29,867	425,778	13.7*	0.96 (0.92, 0.99)	No suggestion of PB (data not shown)	0%

*CC – case-control, NCC – nested case-control, CH – cohort, RCT – randomised controlled trial, FU – follow-up, CI – confidence interval, NA – not applicable, NR – not reported, PB – publication bias, PCa – prostate cancer, * mean FU computed with FU values of cohort studies; § I^2 represents the percentage of heterogeneity between studies not explained by chance*