Int J Clin Exp Med 2017;10(7):9891-9900
www.ijcem.com /ISSN:1940-5901/IJCEM0055258

Review Article

Fish consumption and prostate cancer risk: a meta-analysis of 37 studies

Yuanqing Dai, Yao Bai, Xiaobo Zhang

Department of Geriatric Surgery, Xiangya Hospital, Central South University, Changsha, China

Received April 12, 2017; Accepted June 7, 2017; Epub July 15, 2017; Published July 30, 2017

Abstract: Background: Previous reports have suggested a potential association of fish consumption and the risk of prostate cancer. Since the associations between different studies were controversial, we therefore conducted a meta-analysis to re-assess the relationship between fish consumption and prostate cancer risk. Methods: Pertinent studies were identified by a search of PubMed, Embase, Web of Science and Medline until 31th of January in 2017. A random effect model was used to combine the relative risk (RR) with 95% confidence intervals (CI). Sensitivity analysis and publication bias were conducted. Results: Our meta-analysis was based on 37 studies (18 cohort studies and 19 case-control studies) involving 55401 cases. The total RR of prostate cancer risk for the highest *vs*. the lowest fish consumption was 0.956 (95% CI = 0.881-1.036), with its significant heterogeneity among studies (I^2 = 65.6%, P = 0.000). All of the included studies suggested a high quality, with the average NOS score of 7.35. Subgroup analyses by study design and ethnicity also showed nonsignificant associations between fish consumption and the risk of prostate cancer. And there was no publication bias of the meta-analysis about fish consumption and prostate cancer risk. Conclusion: We found that the highest fish consumption had no significant association on the risk of prostate cancer.

Keywords: Fish consumption, prostate cancer, meta-analysis

Introduction

Prostate cancer is a common cancer in men, accounting for approximately 25% of all cancers, and has the second highest incidence of cancer in men worldwide [1]. More than one million new prostate cancer patients were diagnosed in the year of 2015, presenting a tremendous burden for public health [2]. Although much effort has been directed toward prostate cancer prevention, many aspects of its etiology are still unknown. To address this serious challenge, it is necessary to explore strategies that might reduce the incidence of prostate cancer.

Fish consumption has been explored for the risk of prostate cancer, and also to assess the influence on progression to more clinically advanced disease and thereby prostate cancer mortality. The mechanistic rationale for a preventive effect of fish on prostate cancer is partially ascribed to the content of longchain marine omega-3 polyunsaturated fatty acids [3]. These compounds may play a role in the development and progression of prostate cancer to a more clinically advanced disease through, for example, antiinflammatory effects [3]. Two previous meta-analyses [4, 5] which included the studies until 2010 had been conducted to explore the association between fish consumption and prostate cancer risk. The results from them consistently suggested that there is no evidence of a protective association of fish consumption with prostate cancer incidence. However, many cohort studies [6-10] with large cases and participants were conducted in the recent years. Considering the results were not consistent, we then conducted an update meta-analysis to re-assess the relationship for prostate cancer risk with high fish consumption.

Materials and methods

Literature search

An electronic search of PubMed, Embase, Web of Science and Medline was performed until 31th of January in 2017. The keywords imputed

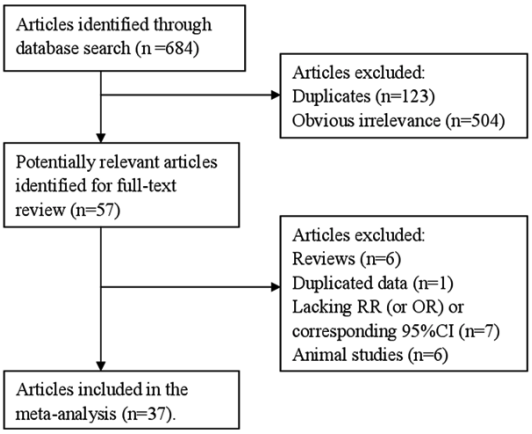

Figure 1. Study selection process for this meta-analysis.

are 'fish' OR 'diet' OR 'lifestyle' combined with 'prostate cancer' OR 'prostate carcinoma' with language in English or Chinese. The full texts of relevant citations from all the results identified have been inspected and analyzed. Relative references in the main outcomes have also been searched and reviewed. The study selection process was performed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [11].

Selection criteria

Evaluating all the studies above that presenting quantitative estimates regarding the linkage between fish consumption and the risk of prostate cancer and those studies meet the requirement was embraced in our research and then included for this study. We made the strict criteria for our studies as following: (1) The study design are cohort, case-control or cross-sectional studies; (2) Human population studies; (3) The outcome of interest was prostate cancer; (4) The independent variable of interest was fish consumption; (5) The risk estimates, such as Relative Risk (RR) and Odds Ratio (OR) with 95% Confidence Intervals (CI) were reported (or the numbers of case and control and the total numbers could calculate them). The studies could not satisfy such criteria were ruled out immediately.

Data extraction and quality assessment

The data extracted from the included studies in use were referring such aspects: author name, year of publication, country, design of study, fish type, age, number of case and participants, value of RR or OR with 95% CI and relative adjustments. A third reviewer was sought to make a common consensus on the abstracted data. The methodological quality of each study was assessed separately using the Newcastle-Ottawa-Scale (NOS) [12], which can either be used as a checklist or scale.

Statistical analysis

The pooled measure was calculated as the inverse variance-weighted mean of the logarithm of RR with 95% CI. A random-effects model was used to combine study-specific RR (95% CI), which considers both within-study and between-study variation [13]. Statistical heterogeneity was analyzed using Cochran I^2, which depicts the percentage of variation across studies due to heterogeneity rather than chance [14]. The I^2 was used to assess heterogeneity, and I^2 values of 0, 25, 50 and 75% represent no, low, moderate and high heterogeneity, respectively [15]. Meta-regression with restricted maximum likelihood estimation [16] and subgroup analysis according to study design and ethnicity were performed to assess the potentially important covariates that might exert substantial impact on between-study heterogeneity. Publication bias was analyzed by using Egger's test and funnel plot [17]. Sensitivity analysis [18] was conducted to describe how robust the pooled estimator was when removing an individual studies at a time. STATA version 10.0 (StataCorp LP, College Station, Texas, USA) was used for the whole meta-analysis. Statistical significance was set at $P < 0.05$.

Results

Study selection

A flow diagram of the study selection process was showed in **Figure 1**. Database search led to retrieval of 684 records from PubMed, Embase, Web of Science and Medline. There were 123 duplicated records and 504 studies obvious irrelevance when reviewing the abstract and titles that did not meet our demands, which were eliminated from further analyses. After carefully review of the full-text versions of each record, we finally ruled out 20 articles. As a result, 37 studies [6-10, 19-50] involving 55401 cases were chosen for our meta-analysis. The characteristics of the included studies were presented in **Table 1**.

Table 1. Characteristics of the included studies

Study, year	Study design	Country	Age (years)	Participants Cases	Fish Type	RR (95% CI)	Quality scores	Adjustment for covariates
Allen et al. 2004	Cohort	Japan	51-89	18115 (196)	Total	1.77 (1.01-3.11)	8	Age, calendar period, city of residence, educational level, and radiation dose.
Allen et al. 2008	Cohort	Europe	44-95	142520 (2727)	Total	1.05 (0.91-1.20)	8	Center, educational level, height, marital status, total energy intake, and weight.
Amin et al. 2008	Case-control	Canada	64.5±8.3	917 (386)	Total	0.54 (0.32-0.89)	7	Age, alcohol use, cystitis, educational level, ethnicity, family history of prostate cancer, prostatitis, sexually transmitted infections, and smoking status.
Andersson et al. 1995	Case-control	Sweden	70.0±6.1	508 (256)	Total	1.8 (1.0-3.5)	7	Age.
Augustsson et al. 2003	Cohort	United States	40-75	47822 (2482)	Total	0.93 (0.80-1.08)	8	Age, caloric intake, fatty acids, lycopene, physical activity, retinol, and vitamin D.
Bosire et al. 2013	Cohort	United States	50-71	293464 (23453)	Total	0.79 (0.65-0.96)	7	Age, ethnicity, educational level, body mass index, smoking, physical activity, family history of prostate cancer, diabetes, energy, history prostate-specific antigen screening, and all other components in the specific index.
Chavarro et al. 2008	Cohort	United States	40-84	20167 (2161)	Total	1.11 (0.95-1.30)	7	Age, alcohol intake, BMI, dairy-product intake, meat intake, multivitamin use, physical activity, race, random assignment to aspirin or b-carotene, smoking intake, tomato-product intake, and vitamin E supplement use
Chen et al. 2005	Case-control	China	≥50	718 (237)	Total	1.15 (0.79-1.66)	6	Age and BMI.
Deneo-Pellegrini et a. 1999	Case-control	Uruguay	40-89	408 (175)	Total	0.9 (0.5-1.8)	7	Age, residence, urban/rural status, education, family history of prostate cancer in a first-degree relative and body mass index.
Fernandez et al. 1999	Case-control	Italy	40-75	8117 (127)	Total	0.7 (0.4-1.1)	7	Age, alcohol use, area of residence, BMI, educational level, and smoking status
Fradet et al. 2009	Case-control	United States	65.5±8.1	1012 (506)	Dark	0.43 (0.29-0.63)	8	Age, ethnicity, and institution, total fat intake, body mass index, smoking, PSA screening, and family history of prostate cancer did not materially alter our results.
Hu et al. 2008	Case-control	Canada	20-76	6838 (1799)	Total	0.8 (0.7-1.0)	7	Age, alcohol use, BMI, cigarette pack-years, educational level, residence province, total fruit and vegetable intake, and total energy intake.
Jain et al. 1999	Case-control	Canada	69.8	1253 (617)	Total	0.66 (0.50-0.89)	7	Age, BMI, educational level, log-conjugated and total linoleic acid, dietary fiber, folic acid, fruit intake, grain intake, retinol, total carotenoids, total energy intake, total fat, total plants, vegetable intake, vitamin C, vitamin E, marital status, multivitamin supplements used in past year, smoking, study area, and vasectomy.
Jian et al. 2004	Case-control	China	72.7±7.1	404 (130)	Salted	2.12 (1.17-3.86)	7	Age, BMI, educational level, caloric intake, family history of prostate cancer, fresh vegetable and fruit intake, income, marital status, physical activity, residence (rural or urban), and tea drinking.
Joshi et al. 2012	Case-control	United States	65-79	1813 (717)	Dark	0.9 (0.7-1.3)	8	Age, BMI, total calorie intake, family history of PCA, total fat intake, dietary vitamin D intake, alcohol consumption, total dairy intake, cigarette smoking, total fruit consumption, total vegetable consumption, red meat consumption, white meat consumption, processed meat consumption.
Key et al. 1997	Case-control	United Kingdom	68.1	656 (328)	Fatty	0.78 (0.47-1.29	6	Log total energy intake.
Key et al. 2014	Cohort	United Kingdom	40-89	150000 (3000)	White	1.03 (0.90-1.18)	8	NA.
Le Marchand et al. 1994	Cohort	United States	≥45	20316 (198)	Total	1.2 (0.8-1.8)	8	Age, ethnicity, and income.
Mills et al. 1989	Cohort	United States	≥25	14000 (180)	Total	1.47 (0.84-2.60)	7	Age.
Mina et al. 2008	Case-control	Canada	66±6	3141 (1534)	Salted	0.79 (0.61-1.02)	8	Age, cigarette pack-years, and race.
Outzen et al. 2016	Cohort	Denmark	50-64	27178 (1690)	Total	1.12 (0.97-1.29)	8	BMI, education, smoking status, smoking duration, smoking amount, participation in sport, red and processed meat intake, dairy products intake, alcohol intake, indicator for alcohol abstinence, indicator variable for fish intake.
Park et al. 2007	Cohort	United States	≥45	82483 (4404)	Total	1.04 (0.93-1.15)	7	BMI, educational level, ethnicity, family history of prostate cancer, smoking status, length of follow-up and total energy intake.

Richman et al. 2010	Cohort	United States	66.0±8.2	1294 (127)	Total	1.13 (0.70-1.84)	8	Age at diagnosis, energy intake, time from diagnosis to questionnaire, primary treatment, BMI, nonvigorous activity, Gleason sum at diagnosis, and prostate-specific antigen at diagnosis.
Rohrmann et al. 2007	Cohort	United States	≥35	2892 (199)	Total	0.86 (0.44-1.67)	8	Age, BMI at age 21 y, saturated fat intake, tomato-product intake, and total energy intake.
Schuurman et al. 1999	Cohort	Holland	55-69	58279 (642)	Total	1.03 (0.80-1.34)	7	Age, family history of prostate cancer, and socioeconomic status.
Severson et al. 1989	Cohort	United States	≥45	7999 (174)	Total	1.22 (0.74-2.01)	7	Age.
Sonoda et al. 2004	Case-control	Japan	59-73	280 (140)	Total	0.45 (0.20-1.02)	8	Energy intake and smoking.
Stott-Miller et al. 2013	Case-control	United States	35-74	3041 (1549)	Fried	1.41 (1.04-1.92)	8	Age, race, family history of prostate cancer, body mass index, PSA/DRE tests in previous 5 years, and education.
Sung et al. 1999	Case-control	China	≥50	270 (90)	Total	1.09 (0.61-1.96)	7	NA.
Talamini et al. 1992	Case-control	Italy	45-79	956 (271)	Total	0.79 (0.53-1.17)	7	Age, area of residence, BMI, and educational level.
Terry et al. 2001	Cohort	Sweden	43-82	6272 (466)	Total	0.43 (0.30-0.70)	7	Age, alcohol use, BMI, fruit and vegetable intake, milk intake, physical activity, processed meat intake, red meat intake, and smoking status
Torfadottir et al. 2013	Cohort	Iceland	46.8±6.9	2268 (345)	Total	1.05 (0.71-1.57)	8	Age at study entry in midlife, education, family history of prostate disease, going to a physician regularly, height in midlife, BMI in midlife, type 2 diabetes in midlife and concurrent salted or smoked fish-, fish oil-, milk-, rye bread-, and meat intake.
Tyagi et al. 2010	Case-control	India	69.7	909 (303)	Total	1.45 (1.01-2.09)	7	NA.
Ukoli et al. 2009	Case-control	Nigeria	56.09±12.1	324 (56)	Total	1.16 (0.50-2.68)	7	NA.
Villeneuve et al. 1999	Case-control	Canada	50-74	3246 (1623)	Total	1.0 (0.7-1.3)	6	Age, alcohol use, BMI, cigarette pack-years, coffee use, family history of cancer, fruit and fruit-juice intake, grain and cereal intake, income, meat intake, residence province, race, rice and pasta intake, tofu intake, and years since quitting smoking.
Wilson et al. 2016	Cohort	United States	61	1155 (184)	Total	0.83 (0.50-1.38)	8	Age at diagnosis and total energy intake, race, family history of prostate cancer, BMI, smoking, vigorous physical activity, total calcium intake, cooked tomato products intake, coffee intake, and clinical stage.
Wright et al. 2012	Cohort	Finland	50-69	27111 (1929)	Total	0.90 (0.79-1.02)	8	Age, energy intake, smoking dose and duration, trial intervention assignment, education level, and dietary fat intake

Abbreviations: NA: not available; CI: confidence interval; RR: relative risk.

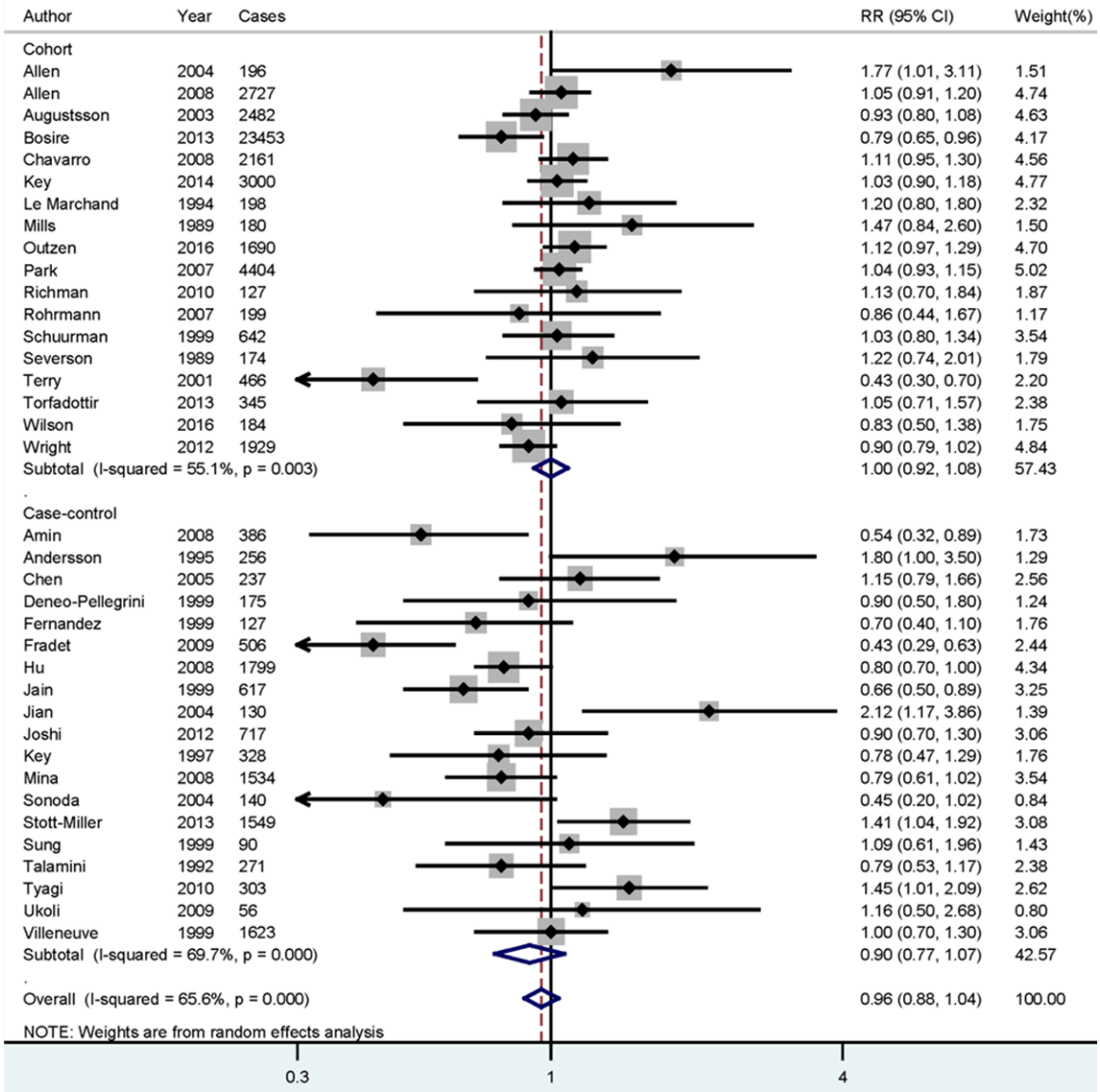

Figure 2. Forest plot for assessment of association between fish consumption and the risk of prostate cancer.

Among them, eighteen were prospective cohort studies and nineteen were case-control studies. Study populations were from four continents: Europe, Asia, America and Africa. The results of quality assessment of selected studies were showed in **Table 1**. Among the 37 studies included, all of the studies were in relative high quality (over 6 stars), with the average NOS score was 7.35.

Association between fish consumption and the risk of prostate cancer

The multivariate-adjusted RR of each study with the highest *vs*. the lowest fish consumption was available in **Figure 2**. The total RR of prostate cancer for the highest *vs*. the lowest fish consumption was 0.956 (95% CI = 0.881-1.036), with its significant heterogeneity among studies (I^2 = 65.6%, P = 0.020).

In order to explore the significant between-study heterogeneity founded in the overall analysis, univariate meta-regression with the covariates of publication year, location where the study was conducted, fish type and study design (case-control or prospective) was performed. No significant differences were found in the above-mentioned analysis.

Whether the result of the research has publication bias or not were showed in **Figure 3**. The

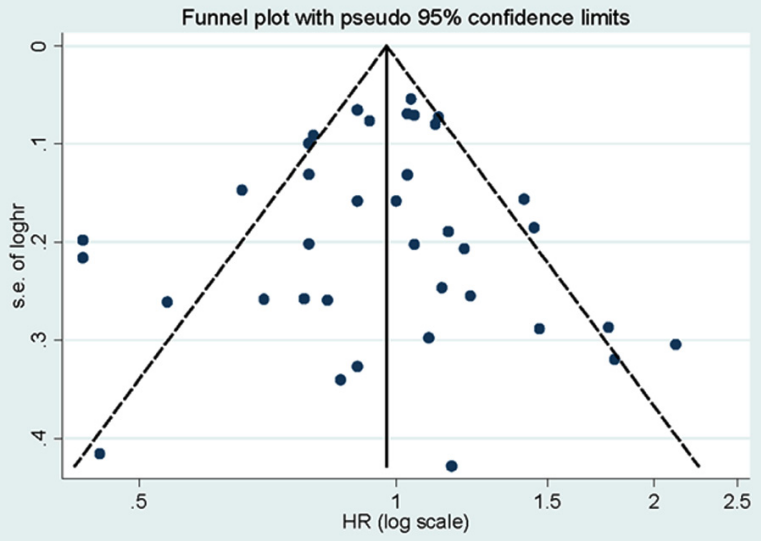

Figure 3. Funnel plot for assessment of publication bias.

Egger's test (P = 0.606) also showed that there was no publication bias of the meta-analysis about fish consumption and the risk of prostate cancer.

Sensitivity analysis (**Figure 4**) showed that no individual study had excessive influence on the association on fish consumption and prostate cancer risk when removed a study at a time.

Subgroup analyses

We classified the included studies into several subgroups for analysis, of which the result was shown in **Table 2**. Considering the study design, the studies conducted in cohort studies (RR = 1.000, 95% CI = 0.922-1.084) and case-control studies (RR = 0.904, 95% CI = 0.766-1.066) showed that no significant associations were found between them. When stratified by ethnicity, the populations from America (RR = 0.908, 95% CI = 0.812-1.016), Europe (RR = 0.947, 95% CI = 0.836-1.073) and Asia (RR = 1.279, 95% CI = 0.931-1.756) had nonsignificant association on prostate cancer risk with a high fish consumption.

Discussion

The findings from this meta-analysis of epidemiologic studies indicated that highest fish consumption had no significant association for the risk of prostate cancer. The associations were also not significant either in cohort studies or in the case-control studies with higher fish consumption. The result of subgroup analysis by geographic locations was consistent with the overall pooled result. Higher fish consumption had no significant association among American population, European population or Asian population. The total RR of prostate cancer for the highest vs. the lowest fish consumption was 0.956 (0.881-1.036), with its significant heterogeneity among studies (I^2 = 65.6%, P = 0.020).

However, evidence of significant between-study heterogeneity was found in the whole result and some subgroups analyses. As we all know, between-study heterogeneity is common in a meta-analysis, and exploring the heterogeneity is necessary in the report [51]. Therefore, we used univariate meta-regression with the covariates of publication year, location where the study was conducted, fish type and study design (case-control or prospective) to explore the between-study heterogeneity. No significant findings were found in the above-mentioned analysis. We then conducted subgroup analyses by study design and geographic locations to further explore the source of heterogeneity. However, the between-study heterogeneity was evidence in some subgroup analyses.

Although we did not obtain the positive result for prostate cancer with higher fish consumption, much cases and participants were included; this may achieve a much more comprehensive result. Most of the included studies had adjusted the covariates which may influence the result. Furthermore, we did not find any publication bias by the Egger's test and funnel plot. What's more, no individual study had excessive influence on the association of fish consumption and prostate cancer risk when removed a study at a time.

However, our meta-analysis still had several restrictions. Firstly, due to the unconformity of categories of fish consumption of each study, we did not do the dose-response analysis for fish consumption and prostate cancer risk.

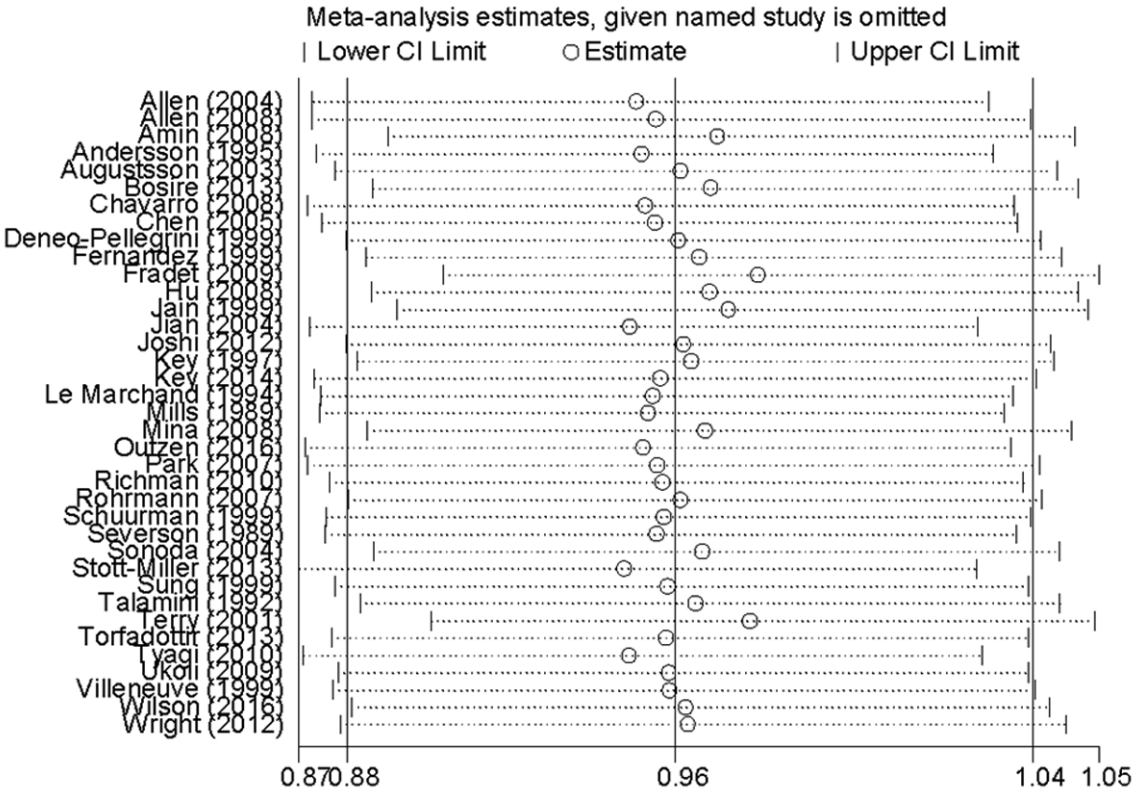

Figure 4. Sensitivity analyses for assessment of association between fish consumption and the risk of prostate cancer.

Table 2. Summary risk estimates of the overall and subgroup analyses on fish consumption and the prostate cancer risk

Subgroups	No. (cases)	No. studies	Risk estimate (95% CI)	Heterogeneity test	
				I^2 (%)	*P*-value
All studies	55401	37	0.956 (0.881-1.036)	65.6	0.000
Study design					
Prospective	44557	18	1.000 (0.922-1.084)	55.1	0.003
Case-control	10844	19	0.904 (0.766-1.066)	69.7	0.000
Ethnicity					
American	42468	19	0.908 (0.812-1.016)	66.9	0.000
European	11781	11	0.947 (0.836-1.073)	64.9	0.001
Asian	1096	6	1.279 (0.931-1.756)	56.0	0.045

Therefore, further studies with detailed category of fish consumption are wanted to assess the dose-response analysis. Secondly, thirty of the 37 studies were reported the total fish, while little study was reported the fatty, salted, dark, fried or white fish. We therefore pooled the result for prostate cancer risk with the high total fish consumption. The RR of prostate cancer for the highest vs. the lowest total fish consumption was 0.962 (95% CI = 0.885-1.046), consistent with the whole result and the subgroup analyses. Thirdly, half of the studies followed a case-control design that may lead to inherent recall and selection bias to retrospective studies. However, the association was not significant either in case-control studies or in the cohort studies. Finally, Study populations were from four continents: Europe, Asia, America and Africa. To our attention, only one study was come from Africa. Therefore, more studies conducted in Africa are wanted in the future studies.

In summary, this study suggested that highest fish consumption had no significant association on the risk of prostate cancer, either in cohort studies or in case-control studies. During some limitation existed in our study, further studies with large cases and participants are wanted to confirm this result.

Disclosure of conflict of interest

None.

Address correspondence to: Xiaobo Zhang, Department of Geriatric Surgery, Xiangya Hospital, Central South University, 87 Xiangya Road, Changsha 410008, Hunan Province, China. Tel: +86 731 8975-3053; Fax: +86 731 89753053; E-mail: xbzhang-123@126.com

References

[1] Jemal A, Bray F, Center MM, Ferlay J, Ward E and Forman D. Global cancer statistics. CA Cancer J Clin 2011; 61: 69-90.

[2] Hurst R, Hooper L, Norat T, Lau R, Aune D, Greenwood DC, Vieira R, Collings R, Harvey LJ, Sterne JA, Beynon R, Savovic J and Fairweather-Tait SJ. Selenium and prostate cancer: systematic review and meta-analysis. Am J Clin Nutr 2012; 96: 111-22.

[3] Chan JM, Gann PH and Giovannucci EL. Role of diet in prostate cancer development and progression. J Clin Oncol 2005; 23: 8152-60.

[4] Lovegrove C, Ahmed K, Challacombe B, Khan MS, Popert R and Dasgupta P. Systematic review of prostate cancer risk and association with consumption of fish and fish-oils: analysis of 495,321 participants. Int J Clin Pract 2015; 69: 87-105.

[5] Szymanski KM, Wheeler DC and Mucci LA. Fish consumption and prostate cancer risk: a review and meta-analysis. Am J Clin Nutr 2010; 92: 1223-33.

[6] Bosire C, Stampfer MJ, Subar AF, Park Y, Kirkpatrick SI, Chiuve SE, Hollenbeck AR and Reedy J. Index-based dietary patterns and the risk of prostate cancer in the NIH-AARP diet and health study. Am J Epidemiol 2013; 177: 504-13.

[7] Torfadottir JE, Valdimarsdottir UA, Mucci LA, Kasperzyk JL, Fall K, Tryggvadottir L, Aspelund T, Olafsson O, Harris TB, Jonsson E, Tulinius H, Gudnason V, Adami HO, Stampfer M and Steingrimsdottir L. Consumption of fish products across the lifespan and prostate cancer risk. PLoS One 2013; 8: e59799.

[8] Key TJ. Nutrition, hormones and prostate cancer risk: results from the European prospective investigation into cancer and nutrition. Recent Results Cancer Res 2014; 202: 39-46.

[9] Wilson KM, Mucci LA, Drake BF, Preston MA, Stampfer MJ, Giovannucci E and Kibel AS. Meat, fish, poultry, and egg intake at diagnosis and risk of prostate cancer progression. Cancer Prev Res (Phila) 2016; 9: 933-41.

[10] Outzen M, Tjonneland A, Christensen J and Olsen A. Fish consumption and prostate cancer risk and mortality in a Danish cohort study. Eur J Cancer Prev 2016; [Epub ahead of print].

[11] Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann Intern Med 2009; 151: 264-9, W64.

[12] Stang A. Critical evaluation of the Newcastle-Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. Eur J Epidemiol 2010; 25: 603-5.

[13] DerSimonian R and Laird N. Meta-analysis in clinical trials. Control Clin Trials 1986; 7: 177-88.

[14] Higgins JP and Thompson SG. Quantifying heterogeneity in a meta-analysis. Stat Med 2002; 21: 1539-58.

[15] Higgins JP, Thompson SG, Deeks JJ and Altman DG. Measuring inconsistency in meta-analyses. BMJ 2003; 327: 557-60.

[16] Higgins JP and Thompson SG. Controlling the risk of spurious findings from meta-regression. Stat Med 2004; 23: 1663-82.

[17] Egger M, Davey Smith G, Schneider M and Minder C. Bias in meta-analysis detected by a simple, graphical test. BMJ 1997; 315: 629-34.

[18] Tobias A. Assessing the in fluence of a single study in the meta-analysis estimate. Stata Tech Bull 1999; 47: 15-7.

[19] Mills PK, Beeson WL, Phillips RL and Fraser GE. Cohort study of diet, lifestyle, and prostate cancer in Adventist men. Cancer 1989; 64: 598-604.

[20] Severson RK, Nomura AM, Grove JS and Stemmermann GN. A prospective study of demographics, diet, and prostate cancer among men of Japanese ancestry in Hawaii. Cancer Res 1989; 49: 1857-60.

[21] Talamini R, Franceschi S, La Vecchia C, Serraino D, Barra S and Negri E. Diet and prostatic cancer: a case-control study in northern Italy. Nutr Cancer 1992; 18: 277-86.

[22] Le Marchand L, Kolonel LN, Wilkens LR, Myers BC and Hirohata T. Animal fat consumption and prostate cancer: a prospective study in Hawaii. Epidemiology 1994; 5: 276-82.

[23] Andersson SO, Baron J, Wolk A, Lindgren C, Bergstrom R and Adami HO. Early life risk factors for prostate cancer: a population-based case-control study in Sweden. Cancer Epidemiol Biomarkers Prev 1995; 4: 187-92.

[24] Key TJ, Silcocks PB, Davey GK, Appleby PN and Bishop DT. A case-control study of diet and prostate cancer. Br J Cancer 1997; 76: 678-87.

[25] Deneo-Pellegrini H, De Stefani E, Ronco A and Mendilaharsu M. Foods, nutrients and prostate cancer: a case-control study in Uruguay. Br J Cancer 1999; 80: 591-7.

[26] Jain MG, Hislop GT, Howe GR and Ghadirian P. Plant foods, antioxidants, and prostate cancer risk: findings from case-control studies in Canada. Nutr Cancer 1999; 34: 173-84.
[27] Villeneuve PJ, Johnson KC, Kreiger N and Mao Y. Risk factors for prostate cancer: results from the Canadian National Enhanced Cancer Surveillance System. The Canadian Cancer Registries Epidemiology Research Group. Cancer Causes Control 1999; 10: 355-67.
[28] Sung JF, Lin RS, Pu YS, Chen YC, Chang HC and Lai MK. Risk factors for prostate carcinoma in Taiwan: a case-control study in a Chinese population. Cancer 1999; 86: 484-91.
[29] Fernandez E, Chatenoud L, La Vecchia C, Negri E and Franceschi S. Fish consumption and cancer risk. Am J Clin Nutr 1999; 70: 85-90.
[30] Schuurman AG, van den Brandt PA, Dorant E and Goldbohm RA. Animal products, calcium and protein and prostate cancer risk in The Netherlands Cohort Study. Br J Cancer 1999; 80: 1107-13.
[31] Terry P, Lichtenstein P, Feychting M, Ahlbom A and Wolk A. Fatty fish consumption and risk of prostate cancer. Lancet 2001; 357: 1764-6.
[32] Augustsson K, Michaud DS, Rimm EB, Leitzmann MF, Stampfer MJ, Willett WC and Giovannucci E. A prospective study of intake of fish and marine fatty acids and prostate cancer. Cancer Epidemiol Biomarkers Prev 2003; 12: 64-7.
[33] Jian L, Zhang DH, Lee AH and Binns CW. Do preserved foods increase prostate cancer risk? Br J Cancer 2004; 90: 1792-5.
[34] Sonoda T, Nagata Y, Mori M, Miyanaga N, Takashima N, Okumura K, Goto K, Naito S, Fujimoto K, Hirao Y, Takahashi A, Tsukamoto T, Fujioka T and Akaza H. A case-control study of diet and prostate cancer in Japan: possible protective effect of traditional Japanese diet. Cancer Sci 2004; 95: 238-42.
[35] Allen NE, Sauvaget C, Roddam AW, Appleby P, Nagano J, Suzuki G, Key TJ and Koyama K. A prospective study of diet and prostate cancer in Japanese men. Cancer Causes Control 2004; 15: 911-20.
[36] Chen YC, Chiang CI, Lin RS, Pu YS, Lai MK and Sung FC. Diet, vegetarian food and prostate carcinoma among men in Taiwan. Br J Cancer 2005; 93: 1057-61.
[37] Park SY, Murphy SP, Wilkens LR, Henderson BE and Kolonel LN. Fat and meat intake and prostate cancer risk: the multiethnic cohort study. Int J Cancer 2007; 121: 1339-45.
[38] Rohrmann S, Platz EA, Kavanaugh CJ, Thuita L, Hoffman SC and Helzlsouer KJ. Meat and dairy consumption and subsequent risk of prostate cancer in a US cohort study. Cancer Causes Control 2007; 18: 41-50.
[39] Amin M, Jeyaganth S, Fahmy N, Begin LR, Aronson S, Jacobson S, Tanguay S, Kassouf W and Aprikian A. Dietary habits and prostate cancer detection: a case-control study. Can Urol Assoc J 2008; 2: 510-5.
[40] Hu J, La Vecchia C, DesMeules M, Negri E, Mery L; Canadian Cancer Registries Epidemiology Research Group. Meat and fish consumption and cancer in Canada. Nutr Cancer 2008; 60: 313-24.
[41] Mina K, Fritschi L, Johnson KC; Canadian Cancer Registries Epidemiology Research G. An inverse association between preserved fish and prostate cancer: results from a population-based case-control study in Canada. Nutr Cancer 2008; 60: 222-6.
[42] Chavarro JE, Stampfer MJ, Hall MN, Sesso HD and Ma J. A 22-y prospective study of fish intake in relation to prostate cancer incidence and mortality. Am J Clin Nutr 2008; 88: 1297-303.
[43] Allen NE, Key TJ, Appleby PN, Travis RC, Roddam AW, Tjonneland A, Johnsen NF, Overvad K, Linseisen J, Rohrmann S, Boeing H, Pischon T, Bueno-de-Mesquita HB, Kiemeney L, Tagliabue G, Palli D, Vineis P, Tumino R, Trichopoulou A, Kassapa C, Trichopoulos D, Ardanaz E, Larranaga N, Tormo MJ, Gonzalez CA, Quiros JR, Sanchez MJ, Bingham S, Khaw KT, Manjer J, Berglund G, Stattin P, Hallmans G, Slimani N, Ferrari P, Rinaldi S and Riboli E. Animal foods, protein, calcium and prostate cancer risk: the European Prospective Investigation into Cancer and Nutrition. Br J Cancer 2008; 98: 1574-81.
[44] Ukoli FA, Taher K, Egbagbe E, Lomotey M, Oguike T, Akumabor P, Osime U and Beech D. Association of self-reported consumption of cooked meat, fish, seafood and eggs with prostate cancer risk among Nigerians. Infect Agent Cancer 2009; 4 Suppl 1: S6.
[45] Fradet V, Cheng I, Casey G and Witte JS. Dietary omega-3 fatty acids, cyclooxygenase-2 genetic variation, and aggressive prostate cancer risk. Clin Cancer Res 2009; 15: 2559-66.
[46] Richman EL, Stampfer MJ, Paciorek A, Broering JM, Carroll PR and Chan JM. Intakes of meat, fish, poultry, and eggs and risk of prostate cancer progression. Am J Clin Nutr 2010; 91: 712-21.
[47] Tyagi B, Manoharan N and Raina V. A case control study on prostate cancer in Delhi. Asian Pac J Cancer Prev 2010; 11: 397-401.
[48] Joshi AD, John EM, Koo J, Ingles SA and Stern MC. Fish intake, cooking practices, and risk of prostate cancer: results from a multi-ethnic case-control study. Cancer Causes Control 2012; 23: 405-20.

[49] Wright ME, Bowen P, Virtamo J, Albanes D and Gann PH. Estimated phytanic acid intake and prostate cancer risk: a prospective cohort study. Int J Cancer 2012; 131: 1396-406.
[50] Stott-Miller M, Neuhouser ML and Stanford JL. Consumption of deep-fried foods and risk of prostate cancer. Prostate 2013; 73: 960-9.
[51] Munafo MR and Flint J. Meta-analysis of genetic association studies. Trends Genet 2004; 20: 439-44.