REVIEW

Potato Consumption and Risk of Site-Specific Cancers in Adults: A Systematic Review and Dose-Response Meta-Analysis of Observational Studies

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ABSTRACT

The etiology of cancer type may vary significantly due to anatomy, embryology, and physiology of the cancer site. Although the association between potato consumption and colorectal cancer (CRC) was summarized in a 2018 meta-analysis of 5 cohort studies, to the best of our knowledge, no meta-analysis has evaluated potato consumption in relation to multiple cancer sites in adults. Medline/PubMed, ISI Web of Knowledge, Scopus, and the Cochrane Database of Systematic Reviews were searched for relevant publications through August 2020. We selected cohort or case-control studies conducted in adults that reported risk estimates (relative risk [RRs], HRs, and ORs) of potato intake for any cancer type. Random effects meta-analyses compared high and low intake categories. Twenty prospective cohort studies (total $n = 785{,}348$) including 19,882 incident cases, and 36 case-control studies (21,822 cases; 66,502 controls) were included. Among cohort studies, we did not find an association between high versus low intake of total potato (white and yellow) consumption and overall cancers: 1.04 (95% CI: 0.96, 1.11; $tau^2 = 0.005$, $n = 18$). We found no relation between total potato consumption (high compared with low intake) and risk of CRC, pancreatic cancer, colon, gastric, breast, prostate, kidney, lung, or bladder cancer in cohort or case-control studies. We did not find an association between high versus low consumption of potato preparations (boiled/fried/mashed/roasted/baked) and risk of gastrointestinal-, sex-hormone-, or urinary-related cancers in cohort or case-control studies. Certainty of the evidence was low for total cancer, CRC, colon, rectal, renal, pancreatic, breast, prostate, and lung cancer and very low for gastric and bladder cancer. In conclusion, potato intake or potato preparations were not associated with multiple cancer sites when comparing high and low intake categories. This finding was consistent with the findings from the 2018 meta-analysis regarding potato intake and risk of CRC. *Adv Nutr* 2021;12:1705–1722.

Keywords: potato, cancer, systematic review, meta-analysis, dose-response

Introduction

In recent years, the prevalence of cancer has dramatically increased in both developed and developing countries (1). In 2016, cancer was reported to be the second leading cause of death, responsible for one-sixth of the global mortality rate (2). Among important modifiable factors, much interest has been placed on diet in relation to cancer risk (3). Starchy foods and highly refined carbohydrates have been demonstrated to increase cancer risk in most studies (4).

As a non-cereal staple food consumed worldwide, an understanding of how potato consumption is associated with cancer is important (5). Potatoes are rich sources of fiber (resistant starch), essential nutrients (magnesium,

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Abbreviations used: CRC, colorectal cancer; GI, glycemic index; GL, glycemic load; GRADE, Grades of Recommendation, Assessment, Development and Evaluation; IGF-1, insulin-like growth factor-1; PC, pancreatic cancer; RCT, randomized clinical trial; ROBINS-E, Risk Of Bias In Nonrandomized Studies; RR, relative risk.

potassium, vitamin C, and vitamin B-6), and phytochemicals (lutein and zeaxanthin) (6), which are negatively associated with carcinogenesis (7–11). However, the benefits of potato consumption have also been questioned due to its high content of starch and high glycemic index (GI) (12). A review study showed that high dietary GI is associated with increased risk of colorectal cancer (CRC) and prostate cancer, and a high dietary glycemic load (GL) is related to an increased risk of breast and endometrial cancers (13–15).

The biological effects and nutrient content of potatoes are affected by preparation and cooking methods. For example, boiled potatoes have a higher GI than other kinds of potato preparations. Long-term consumption of high GI or GL diets may lead to chronic hyperinsulinemia (13). Insulin is itself a mitogen and also increases the bioactivity of insulin-like growth factors (IGF-1) which can promote cancer by inhibiting apoptosis and stimulating cell proliferation (13). Fried potatoes are associated with an increased risk of cancer due to a higher content of *trans* fatty acids, salts, and acrylamide (16–18). Acrylamide exerts a mutagenic effect because of the capacity of glycidamide, its epoxide metabolite, to form DNA adducts (19). Some evidence indicates that *trans* fatty acids and salt intake induce chronic inflammation, which may be related to carcinogenesis (20, 21). Preparation methods are of particular interest given the global transition from the consumption of fresh potatoes to potato products like French fries, potato chips, boiled, mashed, and baked potatoes (22).

Findings regarding the association between potato consumption and cancer risk have been contradictory. Several studies have shown a significant association between total potato (white and yellow) intake or any kind of potato preparations and risk of multiple cancer sites (23–26). Although other research has found no significant associations between total potato (white and yellow) intake or any specific kind of potato intake and risk of cancer (27–32).

Several studies have indicated that high starch and carbohydrate intake may be associated with a variety of cancers including CRC, prostate, lung, breast, and endometrial (13–15). Therefore, it is necessary to investigate the overall relation between potato consumption and different types of cancers. In addition, the consumption of fried potatoes has been associated with different types of cancer (26, 33). To the best of our knowledge, the present study is the first to systematically study the association between potato consumption and risk of cancer at multiple sites. One meta-analysis investigated the association between potato consumption and risk of CRC (34), however, cancer itself was not a focus of this study (it only included 1 type of cancer, i.e. CRC) among a host of other disease outcomes. Further, the cohort studies pooled in that meta-analysis (34) included 1 study on sweet potatoes (35). We identified an additional 2 cohort studies (36, 37) that were not included in that meta-analysis. We know of no meta-analysis that has investigated associations between potato intake and risk of cancer at multiple sites. Therefore, we conducted a systematic review and meta-analysis to examine this relation using observational studies.

Methods

The current systematic review and meta-analysis was conducted using the Preferred Reporting Items of Systematic Reviews and Meta-Analysis (PRISMA) statement guidelines (38).

Search strategy

Medline/PubMed, ISI Web of Knowledge, Scopus, and the Cochrane Database of Systematic Reviews were searched for studies on the association between potato consumption and risk of cancer published prior to August 2020. The query syntax was set using Medical Subject Headings (MeSH) and thesaurus search terms including: ("Potato∗" OR "French fries") AND (Cancer∗ OR Malignanc∗ OR Neoplas∗ OR Tumor∗ OR Carcinoma∗). References retrieved from the studies as well as relevant reports were also hand-searched to reduce the likelihood of missing any publications. These steps were performed by 3 independent investigators (MDM, HM, MRA). Any disagreements were resolved through discussion or, if necessary, by a fourth investigator (LA). The study protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO)(CRD42020150160).

Inclusion criteria

Articles were included in the systematic review and meta-analysis if they: *1*) had full texts written in English; *2*) had cohort or case-control or pooled study designs; *3*) were conducted with adults (aged ≥18 y); *4*) defined exposure as total potato consumption, boiled, baked, roasted, mashed, or fried (potato chips or French fries); *5*) reported either HRs, relative risks (RRs), or ORs with corresponding 95% CIs for the association between potato consumption and risk of cancer.

Exclusion criteria

Studies were eliminated if they were: *1*) from unpublished data or gray literature, such as conference articles, editorials, theses, and patents; *2*) animal, ecologic, cross-sectional studies, or randomized clinical trials (RCTs); *3*) carried out among pregnant women or children; *4*) did not report HRs, RRs, or ORs with corresponding 95% CIs; *5*) examined nonrelevant outcomes; *6*) analyzed potato consumption along with other food items; *7*) did not provide the full text. In addition, studies on specific types of potatoes other than white or yellow potatoes (such as sweet potatoes) were excluded because of their different nutritional composition (39). In the case of multiple articles using the same dataset, the study with the largest sample size was included.

Data extraction

The following data were obtained from each study: first author's name, year of publication, study origin, cohort name, duration of follow-up, age range and gender of participants, study design, sample size and number of cases, type of potato

preparation (boiled/fried/mashed/roasted/baked), methods applied for exposure assessment, outcomes, outcome evaluation methods, categories of potato intake, risk estimates and 95% CIs comparing the outcomes of interest in the highest category of potato consumption to the lowest category (maximally adjusted measures, if available), and potential confounders that were controlled in the study. We attempted to contact the corresponding author of the articles when they did not provide sufficient data (for risk estimates and/or 95% CIs) (40–47). However, we were unable to retrieve additional data through this method. If the study provided gender-specific associations, we pooled both risk estimates using fixed-effect models before entering them into the overall meta-analysis. These steps were carried out by 2 independent reviewers (MDM, MA). In the case of lack of consensus regarding study selection or data extraction, the principal investigator (LA) resolved the issue.

Risk of bias

Using the Risk Of Bias In Nonrandomized Studies (ROBINS-E) assessment tool (48), critical appraisal of the studies was done using 7 main domains (potential confounding, selection of participants, classification of exposure, departures from intended exposures, missing data, measurement of outcomes, and reporting bias). Studies were classified as having low, moderate, serious, or critical risk of bias (**Supplementary Table 1**). Quality *assessment* was performed by 2 authors (MA & AJ) independently, and a third party (LA) resolved any disagreements.

Certainty of evidence

Study quality was assessed using the Grades of Recommendation, Assessment, Development and Evaluation (GRADE) tool (49). Evaluation of the certainty of the evidence involves consideration of within-study risk of bias, the directness of the evidence, heterogeneity, precision of the effect or association estimates, and risk of publication bias in order to reach an overall certainty of the evidence rating of very low, low, moderate, or high for each outcome.

Informative statements to communicate the findings of the systematic review were provided using recommendations of the GRADE working group (50). According to the GRADE recommendations, communicating the findings of reviews are based on effect size and the certainty of the evidence. Informative statements are provided using key words or phrases including: will, probably, may, and we are uncertain.

Statistical analysis

Effect estimates were pooled using a random-effects model employing the metan command in STATA (51). To examine the weight of each study, the SE for the log RR/HR/OR of each study was considered as the estimated variance of the log RR, using inverse variance methods (52). Risk estimates with the largest number of adjustment potential confounders were entered into the meta-analysis. Between-study heterogeneity was explored using tau^2 as an absolute measure (with the metan command in STATA) (53). In addition, a subgroup analysis (including formal statistical tests to see if the differences between subgroups were significant) was conducted based on gender (male, female, both), case number, exposure assessment tool (FFQ, non-FFQ instrument), energy adjustment (yes, no), BMI adjustment (yes, no), country, study design (cohort, case-control), and CRC (colon, rectal) (using the metan command) (51).

In addition, we conducted dose-response analyses using the methods proposed by Greenland and Longnecker (54) and Orsini et al. (55). A 2-stage random-effects dose-response meta-analysis was conducted to examine likely nonlinear associations between potato intake and cancer (56). The number of patients with cancer, sample size, and risk estimates were extracted from studies with $\geq$3 quantitative exposure categories. If a study did not report the sample size in each category, we considered it to be similar across categories. In addition, the median or mean potato intake for each category was also extracted. In studies reporting the frequency of potato consumption, weekly grams of potato intake were calculated based on a serving size of 100 g (57). Nonlinear associations were examined by modeling exposure levels with the use of restricted cubic splines with 3 knots at the 10th, 50th, and 95th percentiles of the distribution (using the glst command in STATA) (58, 59). The null hypothesis was that the coefficient of the second spline was equal to zero.

Furthermore, a linear dose-response association between potato consumption and cancer risk was examined for each 100-g/wk increment in consumption using a 2-stage generalized least-squares trend estimation method (with glst and metan commands in STATA) (56, 54, 60). First, study-specific slope lines were obtained (using the glst command) and afterwards, these lines were combined to get an overall average slope (56). Study-specific slope lines were combined through a random-effects model (with the metan command) (51). In addition, potential publication bias was assessed by visual inspection of funnel plots (acquired from the metafunnel command) and also through using Egger's regression test (with the metabias command) (61, 62). Besides pooled analyses, we carried out sensitivity analyses (using the metaninf command in STATA to determine whether the overall estimates were affected by the effect size of a single study) (63). All statistical analyses were performed with STATA software, version 11.0 (STATA Corp.). All P values were 2-tailed and P values <0.05 were considered statistically significant.

Results

The systematic literature search resulted in 8912 articles (**Figure 1**). After reviewing the article titles and abstracts, 6667 publications were excluded. Among the 260 remaining publications, 188 were excluded because: the population was not relevant; it was an ecologic, cross-sectional, RCT study, or a review article; potato consumption was analyzed along with other food items or as part of a dietary pattern; >1 study was conducted on the same population; sweet potatoes were examined; or insufficient information was given (e.g. they

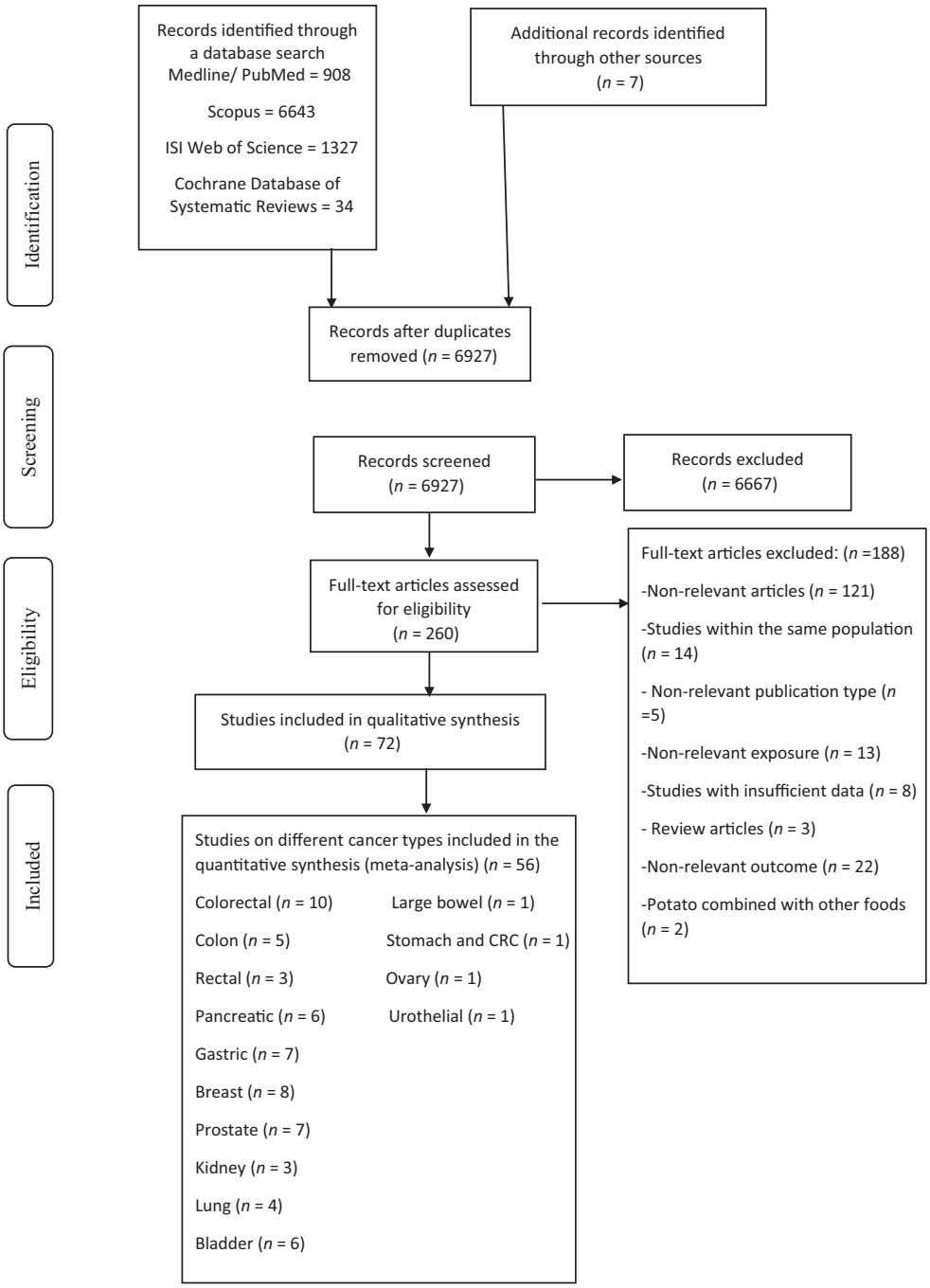

FIGURE 1 Flow diagram of study selection. CRC, colorectal cancer.

did not report the risk estimates or 95% CIs) or nonrelevant outcomes were presented. Finally, 20 cohort studies, 48 case-control studies, and 4 pooled analyses were included in the systematic review. Out of those, 20 prospective cohort studies and 36 case-control studies were included in the meta-analysis.

Study characteristics

We included a total of 73 studies published between 1988 and 2019. Twenty studies, with a total of 785,348 participants and 19,882 incident cases, were prospective cohort studies (24, 27–29, 36, 37, 64–73, 74–76). Forty-eight studies (23, 25, 26, 30–33, 66, 77–116) with 25,005 cases and 73,069 controls had case-control designs. Five studies (117–121) with 3,947,660 subjects, of which 35,760 had cancer were pooled analyses.

In most studies ($n = 50$), risk estimates were reported for both genders together, with only 2 studies providing gender-specific associations (72, 112). However, some studies were conducted with only females ($n = 17$) (24, 27, 28, 37, 66–68, 73, 76, 88, 95, 101, 104, 106, 113, 117, 121) or males

($n = 8$) (26, 29, 64, 69–71, 74, 110). The follow-up period in the cohort studies ranged from 0.75 to 22 y. Participant age ranged between 18 and 107 y. More detailed characteristics of the studies are presented in **Tables 1** and **2**.

In most studies, potato consumption was assessed using an FFQ. Fifty-eight studies reported the total quantity of potato consumption. Moreover, 14 studies reported the consumption of fried potatoes (31, 33, 66, 67, 77, 80, 90, 93, 104, 111, 113–116), 8 reported French fry consumption (26, 28, 31, 66, 67, 72, 93, 106), and 4 boiled/baked potato intake (67, 77, 110, 113).

Included in the systematic review, 10 studies reported results on CRC (24, 27, 36, 37, 73, 78, 89, 97, 105, 114), 8 studies were on breast cancer (28, 67, 68, 88, 101, 104, 113, 121), and 6 studies on pancreatic cancer (PC) (64, 74, 86, 102, 108, 119). We also included 7 studies on the analyses on prostate cancer (26, 29, 69–71, 84, 110), 7 on gastric cancer (23, 30, 81, 92, 95, 99, 116), 4 on kidney cancer (31, 76, 90, 120), 4 on lung cancer (25, 32, 72, 93), and 6 on bladder cancer (31, 33, 79, 94, 98, 122). Furthermore, 7 studies on colon cancer (24, 65, 66, 76, 96, 112, 118), 3 on rectal cancer (24, 65, 66), 3 on ovarian cancer (104, 106, 117), 2 on esophageal squamous cell cancer (82, 87), 2 on large bowel cancer (31, 104), 2 on gallbladder cancer (103, 109), 2 on nasopharyngeal cancer (107, 115), 2 on urothelial cancer (77, 111), and 6 on other cancer sites were evaluated in relation to potato consumption (75, 80, 83, 85, 91, 100). Thus, study-specific results are shown in Tables 1 and 2.

The results of the quality assessment are presented in Supplemental Table 1. The ROBINS-E tool indicated an overall low to moderate risk of bias and serious risk in some studies. In most studies, bias originated from exposure misclassification and from possible confounding.

Total potato (white and yellow) and risk of overall cancer in cohort studies

In cohort studies, participants with the highest total potato consumption (white and yellow) did not show significantly elevated risk of total cancers compared with participants in the low category (Summary Effect Estimate: 1.04 [95% CI: 0.96, 1.11; $tau^2 = 0.005$, $n = 18$]) (**Figure 2**). There was no evidence of publication bias with Egger's regression test ($P = 0.489$) or when using a funnel plot (**Supplemental Figure 1**). Furthermore, sensitivity analyses showed that no individual study had a significant effect on the overall risk estimate (**Supplemental Figure 2**). Subgroup analysis showed total potato (white and yellow) intake significantly increase overall cancer in studies conducted in Europe (**Table 3**). A linear trend estimation indicated that a 100-g increment in total potato (white and yellow) intake was not associated with a higher risk of total cancer (pooled risk estimate: 1.00 [95% CI: 0.99, 1.00; $tau^2 < 0.001$, $n = 12$]). However, some evidence of a nonlinear dose-response association was observed between total potato (white and yellow) consumption and risk of total cancer (P nonlinearity $= 0.006$, $n = 12$ studies) (**Supplemental Figure 3**).

Total potato (white and yellow) and risk of site-specific cancer

In the highest versus lowest analysis that combined colon, rectal, and CRC as an outcome (total CRC), total potato (white and yellow) consumption increased the risk of total CRC in cohort studies. Comparing the highest with lowest total potato (white and yellow) consumption categories, no significant association was found between total potato consumption and separate risk of CRC, colon, PC, breast, prostate, gastric, bladder, kidney, and lung cancer in cohort or case-control studies. The findings are reported in **Table 4**. Subgroup analyses revealed that the association between total potato (white and yellow) consumption and risk of CRC was significant in studies conducted in European countries ($P = 0.003$), and in studies in which BMI was not adjusted for ($P = 0.003$) (Table 3). Subgroup analyses indicated that the association between total potato (white and yellow) intake and PC was significant in studies conducted in Europe ($P = 0.04$), in those with a higher number of cases ($P = 0.005$), in studies that adjusted for BMI ($P = 0.004$) as covariates, and that included either gender ($P = 0.032$) (Table 3). Between-subgroup heterogeneity was observed for BMI adjustment and continent for CRC. For PC, we observed between-subgroup heterogeneity for BMI adjustment and number of cases. Regarding breast cancer, between-subgroup heterogeneity was observed for the number of cases (Table 3).

Estimation of a linear trend indicated that a 100-g/wk increment in total potato (white and yellow) intake was not associated with a higher risk of CRC (Summary Effect Estimate: 1.00 [95% CI: 0.99, 1.01; $tau^2 = 0.0001$, $n = 3$]), PC (Summary Effect Estimate: 1.00 [95% CI: 0.99, 1.02; $tau^2 = 0.0001$, $n = 2$] [1 cohort and 1 case-control study]), breast cancer (Summary Effect Estimate: 0.99 [95% CI: 0.97, 1.00; $tau^2 < 0.001$, $n = 2$]). However, evidence of a nonlinear dose-response association was observed between potato consumption (white and yellow) and risk of CRC (P nonlinearity $= 0.019$, $n = 3$ cohort studies) (**Supplemental Figure 4**).

No indication of publication bias was found with Egger's regression test or with a funnel plot (see **Supplemental Figures 5–8**) for CRC ($P = 0.69$), PC ($P = 0.25$), breast cancer ($P = 0.07$), or prostate cancer ($P = 0.09$).

In the meta-analysis comparing the high versus low intake categories, no individual study significantly affected the overall risk estimates for CRC, PC, breast, and prostate cancer (**Supplemental Figures 9–12**).

Potato preparations (fried versus boiled, mashed, baked, roasted) and risk of cancer

In the meta-analysis comparing high with low intake categories, no association was found between fried potato consumption and risk of gastrointestinal cancers, sex-hormone-related cancers, urinary-system-related cancers, and lung cancer in cohort or case-control studies. Participants in the highest boiled/mashed/baked/roasted potato intake categories did not show a significantly elevated risk of sex-hormone-related cancers compared with participants in the

TABLE 1 Characteristics of cohort studies on the association of potato consumption on site-specific cancers in adults in the systematic review[1]

	First author (ref)	Cohort name	Country	Year	Age range (y)	Gender	Sample size (*n*)	Cases (*n*)	Follow-up (y)	Exposure	Exposure assessment	Outcome	Outcome assessment	Categorical or continuous	OR, RR, or HR (95%CI)	Adjustment[2]
1	Agnoli et al. (36)	European Prospective Investigation into Cancer and Nutrition (EPIC)	Italy	2012		M/F	45,275	435	11.28	Potatoes	FFQ (valid)	CRC	Databases of the regional cancer registries	T3 vs. T1	HR: 1.12 (0.87–1.44)	—
2	Asli et al. (24)	Norwegian Women and Cancer (NOWAC)	Norway	2017	41–70	F	79788-79788-75474	275-637-100	12	Potatoes	FFQ (valid)	Rectal cancer, colon cancer, CRC	Registry linkage	T3 vs. T1	HR: 1.18(0.94, 1.48), HR: 1.63(1.15, 2.31), HR: 2.0 (1.07, 3.72)	1,2,5,6,8,16
3	Asli et al. (64)	HELGA	Norway	2018	40–55	M	38, 766	121	12	Potatoes	FFQ (valid)	PC	Registry linkage	T3 vs. T1	HR: 1.01(0.56, 1.84)	1,2,5,6,8,16
4	Flood et al. (27)	Breast Cancer Detection Demonstration Project (BCDDP)	USA	2002		F	45, 490	485	8.6	Potatoes	FFQ (valid)	CRC	National Death Index	Q5 vs. Q1	RR: 0.82 (0.65, 1.15)	3,5,6,7,8,16,17, 18,22,24,29,30
5	Michels et al. (65)	Nurses' Health Study (NHS) and the Health Professionals' Follow-up Study (HPFS)	USA	2000	30–75	M/F	136, 089	937-244	4.7	Potatoes	FFQ (valid)	Colon, colon, rectal, rectal	Medical records	Q4 vs. Q1 per 100-g/wk, Q4 vs. Q1 per 100-g/wk	RR: 1.11 (0.86–1.43), RR: 0.93 (0.76–1.15), RR: 1.18 (0.69–2.00), RR: 0.94 (0.63–1.41)	1,5,6,7,8,11,16,17, 18,30,34,36,37
6	Mucci et al. (66)	Swedish Mammography Cohort	Sweden	2005		F	61, 467	504-237	13.4	Pan-fried potatoes, potato chips, and French fries	FFQ (valid)	Colon, rectal, colon, rectal cancer	Swedish Cancer Register	T3 vs. T1, T3 vs. T2, T3 vs. T3, T3 vs. T4	RR: 1.1 (0.9–1.4), RR: 1.1 (0.8–1.6), RR: 0.9 (0.5–1.6), RR: 0.9 (0.4–2.0)	1,3,6,15,16,20,21
7	Wilson et al. (69)	Health Professionals Follow-up Study (HPFS)	USA	2008	40–75	M	47, 896	4174-5025-5025	10	Potatoes, French fries, chips	FFQ (valid)	Prostate cancer	Medical records	Q5 vs. Q1, Q5 vs. Q1	RR: 1.07 (0.96–1.19), RR: 0.88 (0.80–0.98), RR: 1.07 (0.98–1.17)	1,2,5,6,7,8,11, 12,16,17,18, 22,28,30,38
8	Wilson et al. (28)	Nurses' Health Study II	USA	2009	25–44	F	90, 628	1179	14	Potatoes, baked-roasted-mashed, chips	FFQ (valid)	Breast cancer	Biennial follow-up questionnaires	Q5 vs. Q1	RR: 0.98 (0.80, 1.19), RR: 0.97 (0.80, 1.17), RR: 0.98 (0.80, 1.19)	5,6,7,8,14,16, 17,21,31,32, 39,44,46
9	Verhoeven et al. (68)	Netherlands Cohort Study	Netherlands	1997	55–69	F	62573	650	4.3	Potatoes	FFQ	Breast cancer	Pathologically	Q5 vs. Q1	RR: 1.14 (0.81, 1.62)	1,11,16,17,31,32,35
10	Sonestedt et al. (67)	Malmoä Diet and Cancer	Sweden	2008	46–81	F	15, 773	544	10.3	Boiled potatoes, French and deep-fried potatoes	FFQ	Breast cancer	Histologically	Q5 vs. Q1, Q5 vs. Q1	HR: 0.91 (0.69–1.20), HR: 1.10 (0.88–1.40)	1,3,5,7,8,16,17,32,35
11	Drake et al. (71)	Malmoä Diet and Cancer cohort	Sweden	2012	45–73	M	8128	817	15	Potatoes	FFQ	Prostate cancer	Histologically	Q5 vs. Q1	HR: 0.87 (0.69, 1.09)	3,5,7,8,9,16,17,22
12	Lee et al. (76)	Nurses' Health Study (NHS) and the Health Professionals' Follow-up Study (HPFS)	USA	2006	30–75	M/F	34, 146	113	20	Potatoes	FFQ (valid)	Renal cancer	Medical records	≥5/wk vs. <2/wk	RR: 1.05 (0.77–1.43)	6,8,12,13,15,16,30
13	Feskanich et al. (72)	Nurses' Health Study (NHS) and the Health Professionals' Follow-up Study (HPFS)	USA	2000	30–55, 40–75	F-M	77283-47778	519-274	12 10	French fries	FFQ (valid)	Lung cancer	Participant report	Q5 vs. Q1	RR: 1.09 (0.79–1.50), RR: 1.05 (0.67–1.64)	1,8,16,17,30
14	Lemarchand et al. (29)	AGRIculture and CANcer (AGRICAN)	France	2016	50–75	M	81, 959	1672	0.75	Potatoes	FFQ	Prostate cancer	ICD-O-3		HR: 1.06 (0.94, 1.20)	3,6,8,9
15	Diallo et al. (70)	Supplémentation en Vitamines et Minéraux Antioxydants (SU.VI.MAX)	France	2016		M	3313	139	12.6	Potatoes	24-h dietary records	Prostate cancer	Biopsy	T3 vs. T1	HR: 0.93 (0.56, 1.45)	1,3,5,6,7,8,11,16,17,22
16	Kato et al. (73)	New York University Women's Health Study	USA	1997	34–65	F	14, 727	100	12.22	Potatoes	FFQ	CRC	Histologically	Q4 vs. Q1	RR:1.05(0.62–1.79)	1,3,6,9,16,20,22
17	Lin et al. (37)	Women's Health Study (WHS)	USA	2004	>45	F	39, 876	223	10	Potatoes	FFQ (valid)	CRC	Pathology reports	Q5 vs. Q1	RR: 1.21 (0.76–1.92)	1,6,7,8,11,16,17, 18,30,34,36,37
18	Stolzenberg-Solomo et al. (74)	α-Tocopherol, β-carotene Cancer Prevention	Finland	2002	50–69	M	27, 111	163	12	Potatoes	Dietary history questionnaire	PC	Finnish Cancer Registry		RR:1.02(0.63–1.64)	1,8,16
19	Steinmetz et al. (76)	Iowa Women's Health Study	USA	1994	55–69	F	41,837	212	5	Potatoes	FFQ (valid)	Colon cancer	State Health Registry	Q4 vs. Q1	RR:1.24(0.76–2.04)	1,16
20	Pols et al. (75)	Nambour Skin Cancer Study	Australia	2011	501	M/F	1056	103	6	Potatoes	FFQ	Basal cell cancer	Histologically	T3 vs. T1	RR: 0.7 (0.4 –1.0)	1,2,16,41,41,42,43
21	Koushik et al. (117)	Pooling Project of Prospective Studies of Diet and Cancer	Multinational countries	2005	27–93	F	560, 441	2068	7–22	Potatoes	FFQ (valid)	Ovarian cancer	Histologically	Per 1 serving/d	RR: 0.97(0.81–1.15)	6,7,8,16,31,32,34,36
22	Koushik et al. (118)	Pooling Project of Prospective Studies of Diet and Cancer	Multinational countries	2007	25–93	M/F	756, 217	5504	6–20	Potatoes	FFQ (valid)	Colon cancer	Medical records	1 serving/wk to $<\frac{1}{2}$ serving/d	RR: 0.87 (0.75–1.02), RR: 0.89 (0.77–1.04), RR: 1.02 (0.86–1.21)	3,5,6,8,11,16,17, 18,29,30,34,39
23	Koushik et al. (119)	Pooling Project of Prospective Studies of Diet and Cancer	Multinational countries	2012	22–104	M/F	862, 584	2212	7–20	Potatoes	FFQ (valid)	PC	Medical records	per 3 servings/wk	RR: 1.03(0.95–1.11)	6,8,16,12,17
24	Jung et al. (121)	Pooling Project of Prospective Studies of Diet and Cancer	Multinational countries	2012	18–104	F	993, 466	4749-19, 749	11–20	Potatoes	FFQ (valid)	Breast cancer (er+), breast cancer (er−)	Medical records	1 serving/d	RR: 1.12(0.99–1.26), RR: 1.04(0.96–1.13)	3,5,6,7,8,11,16, 17,31,32,36,39
25	Lee et al. (120)	Pooling Project of Prospective Studies of Diet and Cancer	Multinational countries	2007	22–107	M/F	774, 952	1478	7–20	Potatoes	FFQ (valid)	Renal cancer	Histologically	1 serving/d	RR: 0.98 (0.75–1.27)	1,6,8,13,16,17,32,44

[1] CRC, colorectal cancer; F, females; ICD-O-3, International Classification of Diseases for Oncology, 3rd revision; M, males; PC, pancreatic cancer; RR, relative risk; T, tertile; Q4, Quartile; Q5, Quintile.

[2] Adjustments: age (1), sex (2), education (3), race (4), height (4), BMI (5), physical activity (6), smoking (7), study center (8), year of interview (9), family history of cancer (10) and diabetes mellitus (11), hypertension (12), family income (13), number of meals per day (14), dietary intake of energy (15), alcohol (16), red meat (17), fruits and vegetables (18), fiber (19), fat (20), calcium (21), zinc (22), vitamin D (23), social class (24), employment in risky occupation (25), socioeconomic status (26), α-linolenic acid (27), nonsteroid anti-inflammatory drug (28), multivitamin supplements (29), age at menarche (30), parity (31), age at first pregnancy (32), hormone therapy (33), age at menopause (34), menopausal status (35), aspirin (36), prostate-specific antigen testing in previous period (37), oral contraceptive use (38), skin color (39), sunburn (40), elastosis (41), treatment allocation (42), age at first birth (43), ethnicity (44), glycemic load (45).

TABLE 2 Characteristics of case-control studies on the association of potato consumption on site-specific cancers in adults in the systematic review[1]

	First author (ref)	Country	Year	Gender	Cases (*n*)	Controls (*n*)	Age - mean or range (cases)	Age - mean or range (controls)	Exposure	Exposure assessment	Outcome	Outcome assessment	Categorical or continuous	OR, RR, or HR (95%CI)	Adjustment[2]
1	Annema et al (78)	Australia	2011	M/F	834	939	40–79	40–79	Potatoes	FFQ (valid)	CRC	Australian Cancer Registry	Q4 vs. Q1	OR: 1.14 (0.87,1.5)	1,2,6,7,8,16,17,30,27
2	Kampman et al. (96)	Netherland	1995	M/F	232	259	>75	75>	Potatoes	Dietary history	Colon cancer	Cancer registries	Q4 vs. Q1	OR: 0.67 (0.37–1.22)	1,2,11,16,17
3	Polesel et al. (107)	Italy	2013	M/F	198	594	18–76	18–76	Potatoes	FFQ (valid)	Nasopharyngeal	Histologically	T3 vs. T1	OR: 1.53 (0.84–2.76)	1,2,3,8,9,10,17
4	Rai et al. (109)	India	2006	M/F	153	153	53.10 ± 12.27	50.49 ± 12.48	Potatoes	FFQ	Gallbladder	Histologically	T3 vs. T1	OR: 0.74 (0.25–2.19)	
5	Tayyem et al. (114)	Jordan	2014	M/F	220	281	>18	>18	Mashed potatoes, fried potatoes	FFQ (valid)	CRC		Q4 vs. Q1, Q4 vs. Q1	OR: 1.12 (0.06–21.16), OR: 1.85 (0.54–6.35)	3,7,8,16,44,19,32
6	Torkuz et al. (115)	Turkey	2011	M/F	183	183	18–75	18–75	Fried potatoes	FFQ	Nasopharyngeal cancer	Histologically	T3 vs. T1	OR: 1.44 (0.91–2.28)	
7	Boeing te al. (81)	Germany	1991	M/F	143	579	32–80	32–80	Potatoes	FFQ	Stomach cancer	Histologically	T3 vs. T1	RR: 0.81 (0.50, 1.32)	1,2,9
8	Bostetti et al. (82)	Italy	2000	M/F	304	743	39–77	39–77	Potatoes	FFQ	Esophageal cancer	Histologically	Q5 vs. Q1	OR: 0.88 (0.53, 1.48)	1,2,3,8,16,17
9	Bravi et al. (85)	Italy	2006	M/F	768	2078	22–79	22–79	Potatoes	FFQ (valid)	Oral and pharyngeal cancer	Histologically	Q5 vs. Q1	OR: 1.85 (1.19, 2.86),	1,2,3,6,8,10,17
10	Chan et al. (86)	USA	2005	M/F	532	1701	21–85	21–85	Potatoes	FFQ (valid)	PC	Histologically	Q4 vs. Q1	OR: 1.4 (1.0–1.9)	1,2,16
11	Cornee et al. (30)	France	1994	M/F	92	128	66.6 ± 10.4	66.6 ± 10.4	Potatoes	Dietary history	Gastric cancer	Histologically	T3 vs. T1	OR: 1.47 (0.72, 2.98)	1,2,16,26
12	Stefani et al. (87)	Uruguay	2005	M/F	160-200	320-400	30–89 ,40–89	30–89, 40–89	Potatoes	FFQ	Gastric cancer, esophagus cancer	Histologically, microscopically	Per 25 g/d	OR: 1.05 (0.93.1.18), OR: 1.05 (0.93.1.18)	1,3,8,16,17,18,23
13	Hoang et al. (92)	South Korea	2016	M/F	415	830	53	53	Potatoes	FFQ	Gastric cancer	Histologically	T3 vs. T1	OR: 0.79 (0.57–1.09)	3,7,8,11,14
14	Deneo-Pellegrini et al (89)	Uruguay	1996	M/F	160	287	30–84	30–84	Potatoes	FFQ	CRC	National Cancer Registry	T3 vs. T1	RR: 0.74 (0.48–1.17)	1,2,3,6,11,16,17
15	Heck et al. (91)	France	2008	M/F	513	713			Potatoes	FFQ	Hypopharyngeal cancer	Pathological	Q4 vs. Q1	OR: 0.48 (0.06, 3.86)	1,2,3,4,17
16	Ito et al. (95)	Japan	2002	F	508	36, 490	>30	>30	Potatoes	FFQ	Gastric cancer	Histologically	Q4 vs. Q1	OR: 0.78 (0.53, 1.13)	1,8,10,11
17	Levi et al (97)	Switzerland	1999	M/F	223	491	<75	<75	Potatoes	FFQ (valid)	CRC	Histologically	T3 vs. T1	OR: 1.41 (0.85, 2.33)	1,2,3,4,6,7,16,17
18	Bich NN et al. (80)	Japan	2009	M/F	670	672			Fried potatoes	FFQ	Stomach and CRC	Interview	T3 vs. T1	OR: 0.88 (0.42–1.69)	1,2
19	Ping et al (105)	Sahara	1998	M/F	100	265	40–84	40–84	Potatoes	FFQ	CRC	Medical records		OR: 1.46 (0.90–2.37)	
20	Steinmetz et al. (112)	Australia	1993	M/F	121-99	241-197	63	63	Mashed potatoes	FFQ	Colon cancer	Histologically	Q4 vs. Q1	OR: 1.42 (0.72–2.80), OR: 1.37 (0.63–2.99)	2,11,16,17,19,26,33
21	Radosavljevic et al. (33)	Serbia	2005	M/F	130	130	64.91	64.91	Fried	FFQ	Bladder cancer	Clinical signs pathohistological finding	T3 vs. T1	OR: 6.31 (2.91–13.70)	8
22	Pandey et al. (103)	India	2002	M/F	64	101	45/95	45/95	Potatoes	30-d recall method	Gallbladder cancer			OR: 1.25 (0.19–10.3)	
23	Mucci et al. (31)	Sweden	2003	M/F	23-14-124-173	3-54-202-42	51–77	51–77	French Fries, potato crisps, pan-fried potatoes, potatoes au gratin, total potatoes	FFQ	Bladder cancer	Population-based cancer registry	Q4 vs. Q1	OR: 0.7 (0.4–1.1), OR: 0.9 (0.4–1.8), OR: 1.3 (0.7–2.4), OR: 0.5 (0.2–1.0), OR: 1.6 (0.7–3.5)	1,2,6,8,16,17,18,19,21
		Sweden	2003	M/F	36-16-49-15	33-54-202-42	51–77	51–77	French fries-potato crisps-pan-fried potato-potato au gratin- total potato	FFQ	Kidney cancer	Population -based cancer registry	Q4 vs. Q1	OR: 0.7 (0.3–1.6), OR: 0.7 (0.3–1.9), OR: 0.7 (0.4–1.4), OR: 1.1 (0.4–2.6), OR: 0.8 (0.3–2.2)	1,2,6,8,16,17,18,19,21
		Sweden	2003	M/F	58-48-226-59	33-54-202-42	51–77	51–77	French fries-potato crisps-pan-fried potato-potato au gratin- total potato	FFQ	Large bowel cancer	Population-based cancer registry	Q4 vs. Q1	OR: 0.8 (0.5–1.4), OR: 1.3 (0.8–2.1), OR: 0.8 (0.5–1.2) OR: 1.5 (0.8–2.7), OR: 1.2 (0.6–2.2)	1,2,6,8,16,17,18,19,21
24	Isa et al. (94)	China	2013	M/F	487	469	60–79	60–79	Potatoes	FFQ	Bladder cancer	Hospital	Q4 vs. Q1	OR: 0.4 (0.2–0.9)	1,2,8
25	Balbi et al. (79)	Uruguay	2001	M/F	144	576	40–89	40–89	Potatoes	FFQ	Bladder cancer	Microscopically	T3 vs. T1	OR: 0.38 (0.23, 0.64)	1,2,3,6,8,16,17,25
26	Stejneck et al. (111)	Sweden	1990	M/F	418	511	79		Fried potatoes	Questionnaire	Urothelial cancer	Histologically, cytologically regional cancer registry	Weekly vs. more seldom, T3 vs. T1	RR: 1.6 (1.1, 2.6), RR: 1.8 (1.2–2.7)	1,2,8
27	Demetriou et al. (88)	Cyprus	2012	F	935	817	40–70		Potatoes	FFQ	Breast cancer	Histologically	Per 100-g/wk	OR: 0.95 (0.87, 1.03)	1,5,6,7,11,31,34
28	Mourouti et al. (101)	Greece	2015	F	250	250	44–68	44–68	Potatoes	FFQ (valid)	Breast cancer	Biopsy		OR: 1.23 (0.88, 1.68)	1,6,7,8,11,31,34,35
29	Tajaddini et al. (113)	Iran	2015	F	306	309	25–65	25–65	Baked/boiled potato, fried potatoes	FFQ (valid)	Breast cancer	Histologically	T3 vs. T1	OR: 0.45 (0.28–0.71), OR: 0.64 (0.39–1.05)	1,6,16,32,36
30	Pelucchi et al. (104)	Italy	2003	F	2569-1031-1953	2588-2411-4154	<79	<79	Fried/baked	FFQ (valid)	Breast cancer-ovarian cancer-large bowel cancer	Interview	T3 vs. T1	OR: 0.9 (0.8–1.1), OR: 1.1 (0.9–1.3), OR: 0.8 (0.7–1.0)	1,2,3,6,8,9,16,17
31	Stott-Milleret et al. (26)	USA	2013	M	1549	1492	35–74	35–74	French fries-snack chips	FFQ	Prostate cancer	Population-based tumor registry	T3 vs. T1	OR: 1.37 (1.11–1.69), OR: 1.08 (0.89–1.32)	1,3,4,6,11,42

(*Continued*)

TABLE 2 (*Continued*)

	First author (ref)	Country	Year	Gender	Cases (*n*)	Controls (*n*)	Age - mean or range (cases)	Age - mean or range (controls)	Exposure	Exposure assessment	Outcome	Outcome assessment	Categorical or continuous	OR, RR, or HR (95%CI)	Adjustment[2]
32	Russnes et al. (110)	Norway	2016	M	1499	1112	67/25	67/25	Boiled potatoes	FFQ	Prostate cancer	Histologically, cytologically	T3 vs. T1	OR: 1.12(0.87–1.42)	1,3,6,8,9,16,22,23
33	Plagens-Rotman et al. (106)	Poland	2018	F	167	683	21–84	21–84	French fries and chips	Questionnaire	Ovarian cancer	Histologically	Per 100-g/wk	OR: 2.06 (0.53–7.99)	7
34	Grieb et al. (90)	Georgia	2006	M/F	333	333	64	64	Fried potatoes	FFQ (valid)	Renal cancer	Interview	Q4 vs. Q1	OR: 2.05 (1.19, 3.53)	1,2,4,6,8,14
35	Andreatta et al. (77)	Argentina	2010	M/F	168	334	55	55	Boiled Fried potatoes	FFQ (valid)	Urinary tract cancer	Histopathologically	T3 vs. T1	OR: 0.47 (0.13–1.63)	1,2,6,7,8,25,26
36	Hue et al. (41)	China	1997	M/F	227	227	53.2	53.2	Potatoes	FFQ	Lung cancer	Histologically	Q4 vs. Q1	OR 0.8 (0.5–1.5)	8,14
37	Hue et al. (34)	Canada	2002	M/F	161	483	>20	>20	French fries/fried potatoes	FFQ	Lung cancer	Pathologically	Q4 vs. Q1	OR 1.7 (1.0–3.0)	1,3,9,16,25
38	Dosil-Diaz et al. (25)	Spain	2008	M/F	295	322	>35	>35	Potatoes	FFQ(valid)	Lung cancer	Histologically	T3 vs. T1	OR: 0.08 (0.03–0.22)	1,2,8,26
39	Hu et al. (27)	China	1988	M/F	241	241	25–80	25–80	Potatoes	Interview	Stomach cancer	Histologically	-	OR: 1.54 (1.03–2.33)	8,17
40	Polesel et al. (108)	Italy	2009	M/F	326	652	63	63	Potatoes	FFQ (valid)	PC	Histologically	Q4 vs. Q1	OR: 1.79 (1.12–2.86)	1,2,3,6,8,9,10,12,16,17
41	Lucenteforte et al. (99)	Italy	2008	M/F	230	547	22–80	22–80	Potatoes	FFQ	Stomach cancer	Histologically	Q5 vs. Q1	OR: 2.04 (1.05–3.98)	1,2,3,6,8,10,11,16
42	Bosetti et al. (83)	Italy	2002	M/F	527	1297	61	61	Potatoes	FFQ (valid)	Laryngeal cancer	Histologically		OR: 1.86 (1.29–2.68)	1,2,3,9,16,17
43	Lin et al. (98)	USA	2009	M/F	844	888	64.4	64.9	White potatoes	FFQ (valid)	Bladder cancer	Histologically	Q4 vs. Q1	OR: 1.02 (0.75–1.39)	1,2,8,16,17,45
44	Ohba et al. (102)	Japan	1996	M/F	141	282	64.4	64.4	Potatoes	FFQ	PC	Pathologically	per 3 servings/wk	RR:1.06 (0.84–1.35)	1,2,9
45	Bravi et al. (84)	Italy	2006	M	1369	1451	46–74	-	Potatoes	FFQ (valid)	Prostate	Biopsy	Q4 vs. Q1	OR: 0.9 (0.7–1.16)	1,3,6,7,8,9,16,17
46	Malagoli et al. (100)	Italy	2019	M/F	380	719	55		Potatoes	FFQ	Melanoma	Biopsy	T3 vs. T1	OR: 0.83 (0.6–1.16)	3,6,16
47	Maso et al. (122)	Italy	2019	M/F	690	665	25–84	27–84	Potatoes	FFQ	Bladder	Histologically	Q4 vs. Q1	OR: 0.79 (0.54–1.13)	1,2,3,8,9,12,16
48	Shah et al. (116)	Malaysia	2014	M/F	58	134	57.9 ± 12.79	57.6 ± 11.80	Sauced or cooked potatoes Fried potato	Dietary history	Gastric	Histologically		OR: 0.19 (0.05–0.68) OR: 0.56 (0.29–1.07)	- -

[1] CRC, colorectal cancer; F, females; M, males; PC, pancreatic cancer; RR, relative risk; T, tertile; Q1, Quartile1/Quintile 1; Q4, Quartile; Q5, quintile.

[2] Adjustments: age (1), sex (2), education (3), race (4), height (4), BMI (5), physical activity (6), smoking (7), study center (8), year of interview (9), family history of cancer (10) and diabetes mellitus (11), hypertension (12), family income (13), number of meals per day (14), dietary intake of energy (15), alcohol (16), red meat (17), fruit and vegetable (18), fiber (19), fat (20), calcium (21), zinc (22), vitamin D (23), social class (24), employment in risky occupation (25), socioeconomic status (26), α-linolenic acid (27), nonsteroid anti-inflammatory drug (28), multivitamin supplements (29), age at menarche (30), parity (31), age at first pregnancy (32), hormone therapy (33), age at menopause (34), menopausal status (35), aspirin (36), prostate-specific antigen testing in previous period (37), oral contraceptive use (38), skin color (39), sunburn (40), elastosis (41), treatment allocation (42), age at first birth (43), ethnicity (44), glycemic load (45).

low category in cohort or case-control studies. The summary findings are reported in Table 4.

Certainty of evidence

The certainty of the evidence was assessed using GRADE. We found that studies on the associations of potato consumption with total cancer, CRC, colon, rectal, pancreatic, renal, lung, breast, and prostate cancers were of low quality, whereas studies on bladder and gastric cancers had very low quality. Furthermore, associations between fried potato consumption with gastrointestinal cancer, and sex-hormone-related cancer were low quality, whereas studies on urinary-related cancers and lung cancer had very low quality (**Table 5**).

The following informative statements were based on the GRADE tool assessment: total potato (white and yellow) intake may not increase the risk of total cancer, CRC, colon, pancreatic, breast, prostate, lung, or renal cancers. Fried potato intake may not increase the risk of gastrointestinal cancers, and sex-hormone-related cancers. Total potato (white and yellow) intake may not increase the risk of gastric and bladder cancers but the evidence is very uncertain. Fried potato intake may not increase the risk of and urinary-related cancers and lung cancer but the evidence is very uncertain (**Supplemental Table 2**).

Discussion

The present meta-analysis examined the association between potato consumption and risk of site-specific cancers with data from 20 cohort and 36 case-control studies. Our findings showed a significant association between total potato (white and yellow) consumption and risk of total CRC. In addition, total potato (white and yellow) intake was not associated with a higher risk of total or other site-specific cancers. Furthermore, no association was found between potato preparations (fried/boiled, mashed, baked, roasted) and risk of other site-specific cancers. However, we observed a positive nonlinear association between potato consumption and the risk of total cancer and CRC based on a nonlinear analysis.

We did not observe a significant positive association between total potato (white and yellow) consumption and total cancer risk in cohort studies. However, nonlinear dose-response associations have been observed in prior cohort studies. Findings from case-control studies are subject to several methodological limitations (123). For example, they are prone to recall bias and selection bias, which can make it difficult to draw firm conclusions. Therefore, we did not perform analyses of total cancers for case-control studies. A systematic review and dose response meta-analysis of 2 prospective studies did not suggest a significant association between potato consumption and total cancer risk (124).

A significant positive association has been reported between potato intake and the risk of total cancer in European populations, who tend to consume high quantities of refined carbohydrates, bread, potatoes, pasta, and rice

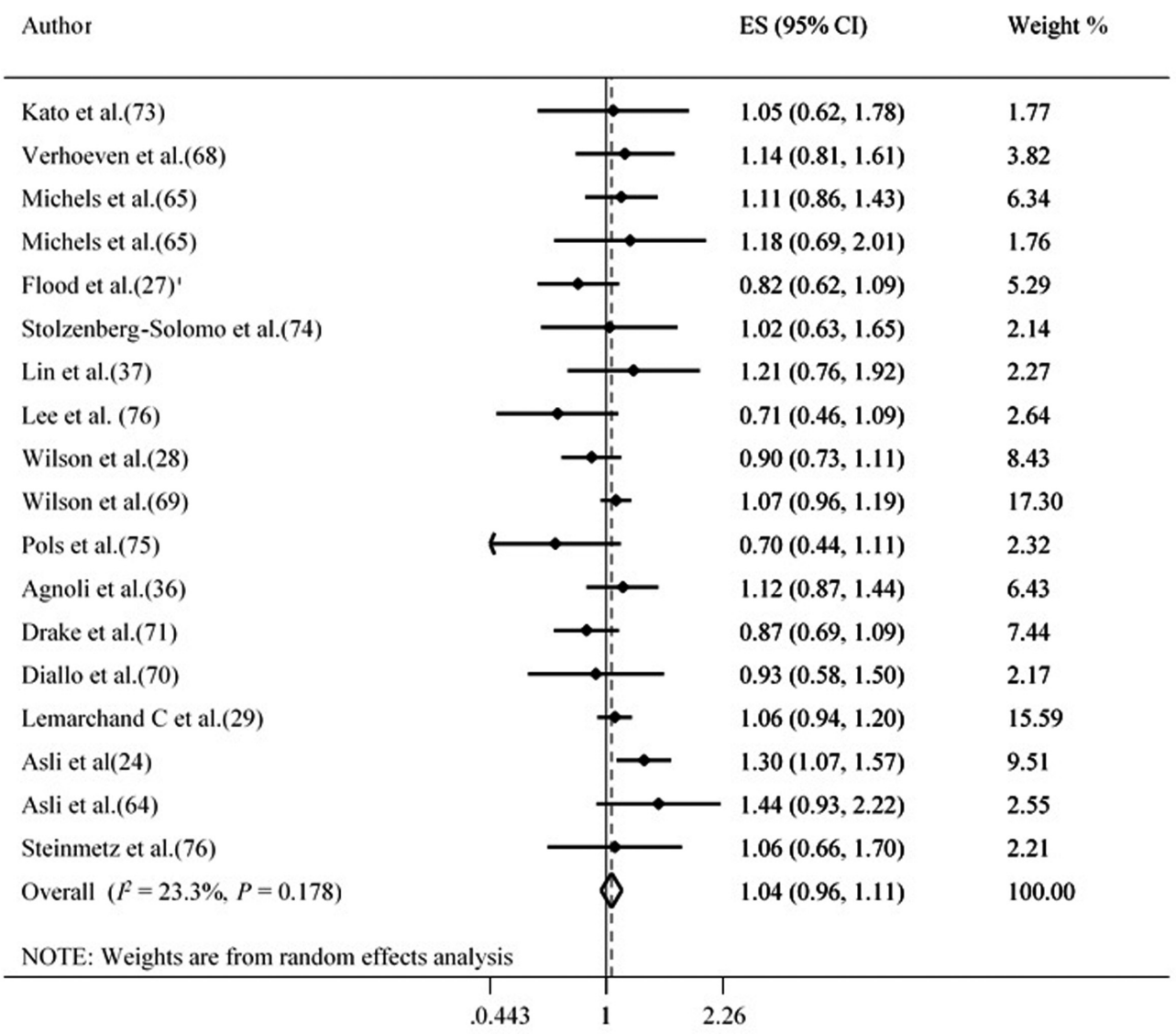

FIGURE 2 Forest plot derived from random-effects meta-analysis of studies investigating the association between high versus low total potato intake and total cancer in adults. ES, effect size.

(125). High carbohydrate intake may be an index of a highly endogenous insulin environment, and insulin and the modulation of IGF-1 have been indicated to act as cancer-promoting agents for several types of cancers (13). Also, we found a significant marginal association between potato and risk of total cancers in studies not adjusted for BMI. Lack of adjustment for BMI may be an explanation for these significant positive associations between potato intake and risk of total cancer. Obesity, a major determinant of insulin resistance and hyperinsulinemia, has been related to cancer (126).

We found a significant association between potato consumption and risk of total CRC in cohort studies. A recent meta-analysis by Schwingshackl et al. did not find any association between potato consumption and risk of CRC (34). One study included in the review was restricted to a specific kind of potato (sweet potato) (35). Some research suggests that a typical Western dietary pattern comprised of high amounts of red meat, processed meat, potatoes, and refined carbohydrates is associated with a higher risk of CRC (127, 128). One case-control study showed tendencies toward a higher risk of colon cancer in participants who consumed high quantities of potato (112). Another study with a case-control design showed an association between potato consumption and increased risk of rectal cancer among whites, but not among African Americans (129). The etiology of colon and rectum cancer may vary significantly due to the anatomy, embryology, and physiology of the colon and the rectum (130). Our meta-analysis showed a significant association between total potato (white and yellow) consumption and rectal cancer but no relation with colon cancer. However, only a few studies

TABLE 3 Results of subgroup analysis for potato consumption and risk of site-specific cancer in adults[1]

Group	Studies (*n*)	ES (95% CI)	*P* value	*P*-within subgroups heterogeneity	*P*-between subgroups heterogeneity	tau^2
Total cancer						
total	18	1.04 (0.96, 1.11)	0.345	0.178	—	0.005
Gender						
Female	7	1.05 (0.94, 1.18)	0.311	0.113	0.706	0.017
Male	6	1.02 (0.95, 1.10)	0.474	0.318		0.001
Both	5	1.09 (0.94, 1.27)	0.217	0.258		0.010
BMI adjustment						
Yes	11	0.99 (0.87, 1.11)	0.881	0.333	0.327	0.002
No	7	1.06 (0.99, 1.13)	0.069	0.129		0.021
Continent						
America	9	1.01 (0.93, 1.10)	0.678	0.394	0.111	0.001
Europe	8	1.08 (1.004, 1.18)	0.039	0.298		0.005
Australia	1	0.70 (0.44, 1.10)	0.127	—		0.127
Number of cases						
<500	11	0.99 (0.87, 1.11)	0.881	0.333	0.327	0.005
≥500	7	1.06 (0.99, 1.13)	0.069	0.129		0.005
Colorectal cancer						
Study design						
Cohort	5	1.09 (0.91, 1.31)	0.310	0.132	0.99	0.017
Case-control	4	1.13 (0.86, 1.49)	0.372	0.152		0.033
Gender						
Female	4	1.08 (0.83, 1.40)	0.133	0.07	0.97	0.037
Both	5	1.12 (0.93, 1.35)	0.102	0.259		0.010
Energy adjustment						
Yes	8	1.08 (0.94, 1.26)	0.255	0.130	0.28	0.015
No	1	1.46 (0.90, 2.36)	0.126	—		<0.001
BMI adjustment						
Yes	6	1.03 (0.87, 1.22)	0.703	0.187	0.04	0.014
No	3	1.29 (1.09, 1.52)	0.003	0.658		<0.001
Continent						
America	4	0.89 (0.73, 1.09)	0.269	0.386	0.04	0.0006
Europe	3	1.24 (1.07, 1.44)	0.003	0.575		<0.001
Australia	1	1.14 (0.86, 1.49)	0.346	—		<0.001
Africa	1	1.46 (0.90, 2.36)	0.126	—		<0.001
Number of cases						
<400	5	1.12 (0.87, 1.45)	0.345	0.245	0.98	0.021
≥400	4	1.09 (0.91, 1.32)	0.325	0.075		0.020
Pancreatic cancer						
Study design						
Cohort	2	1.23 (0.87, 1.72)	0.228	0.296	0.83	0.005
Case-control	3	1.23 (0.95, 1.60)	0.110	0.147		0.025
Gender						
Male	1	1.02 (0.63, 1.64)	0.935	—	0.49	<0.001
Both	4	1.26 (1.02, 1.55)	0.032	0.214		0.015
BMI adjustment						
Yes	2	1.59 (1.15, 2.19)	0.004	0.505	0.04	<0.001
No	3	1.09 (0.91, 1.30)	0.315	0.791		<0.001
Number of cases						
<300	2	1.05 (0.85, 1.30)	0.640	0.888	0.08	<0.001
≥300	3	1.38 (1.10, 1.73)	0.005	0.378		<0.001
Continent						
America	1	1.20 (0.87, 1.65)	0.266	—	0.33	<0.001
Europe	3	1.38 (1.01, 1.89)	0.040	0.253		0.020
Asia	1	1.06 (0.83, 1.34)	0.630	—		<0.001
Breast cancer						
Study design						
Cohort	2	0.95 (0.80, 1.14)	0.646	0.253	0.98	23
Case-control	2	0.95 (0.82, 1.10)	0.543	0.089		65
BMI adjustment						
Yes	3	0.93 (0.83, 1.05)	0.290	0.211	0.29	35.7
No	1	1.14 (0.80, 1.61)	0.459	—		—

(*Continued*)

TABLE 3 (*Continued*)

Group	Studies (*n*)	ES (95% CI)	*P* value	*P*-within subgroups heterogeneity	*P*-between subgroups heterogeneity	tau^2
Energy adjustment						
Yes	3	0.92 (0.82, 1.04)	0.209	0.455	0.10	0
No	1	1.23 (0.89, 1.69)	0.202	—		—
Number of cases						
<1000	2	1.18 (0.93, 1.50)	0.155	0.75	0.04	0
≥1000	2	0.90 (0.79, 1.02)	0.103	0.99		0
Prostate cancer						
Study design						
Cohort	4	1.03 (0.96, 1.12)	0.318	0.405	0.28	0
Case-control	1	0.90 (0.69, 1.15)	0.414	—		—
BMI adjustment						
Yes	4	1.04 (0.97, 1.12)	0.246	0.612	0.13	0
No	1	0.87 (0.69, 1.09)	0.233	—		—
Dietary assessment tool						
FFQ	4	1.02 (0.95, 1.10)	0.436	0.274	0.67	22
24-h dietary records	1	0.93 (0.57, 1.49)	0.765	—		—
Number of cases						
<1000	2	0.88 (0.71, 1.08)	0.228	0.804	0.12	0
≥1000	3	1.04 (0.97, 1.13)	0.221	0.456		0
Continent						
America	1	1.07 (0.96, 1.19)	0.217	—	0.31	—
Europe	4	0.99 (0.90, 1.09)	0.890	0.386		1.3

[1] ES, effect size.

have examined potato consumption in relation to colon or rectum cancer separately. Therefore, we may have had limited statistical power in this subgroup analysis. In addition, a pooled analysis in 2007 did not show a significant relation between potato consumption and risk of colon cancer (118).

We did not find a significant positive relation between total potato (white and yellow) consumption and risk of PC in cohort or case-control studies. This finding was consistent with a 2012 pooled analysis of 14 cohort studies on the association of total potato intake (per 606-g/wk increase in intake) with PC (119). Findings from that analysis revealed no significant linear association between each 606-g/wk increment in potato consumption and risk of PC (119). A meta-analysis of 10 cohort studies did not support an association between diets with a high GI, GL, total carbohydrates, or sucrose and PC risk (131).

We found no associations between total potato (white and yellow) intake and risk of other site-specific cancers such as breast, prostate, lung, gastric, kidney, and bladder cancer. With respect to specific kinds of potato preparation, we did not find an association between fried potatoes and risk of gastrointestinal-, sexual hormone-, and kidney-related cancers. We found no significant association between boiled/mashed/baked/roasted potato intake and sex-hormone-related cancers. In line with our findings, a systematic review and meta-analysis of 32 epidemiological studies concluded that dietary acrylamide was not related to the risk of oral and pharyngeal, esophageal, stomach, colorectal, pancreatic, laryngeal, lung, breast, endometrial, ovarian, prostate, bladder, or lymphoid malignancies (132).

Potato consumption might induce both beneficial and harmful effects on health. Potatoes are a rich source of essential nutrients including starch, fiber, trace minerals (such as potassium), vitamins (such as vitamin C), and phytochemicals (lutein and zeaxanthin), which are necessary for the body to stay healthy (133). Large amounts of catalase enzyme are found in potatoes, which converts hydrogen peroxide into oxygen and water, and can prevent cell injury. On the other hand, potatoes have toxic compounds, such as α-solanine and α-chaconine which are known to induce toxicity. It is also noteworthy that the biological effects and nutrient content of potatoes may be impressed by preparation and cooking ways. Fried potatoes are typically high in dietary fats, in particular, *trans* fatty acids, salts, and acrylamide, which have been related to increased risk of cancer in some studies (16–18). Foodborne toxins such as acrylamide are formed when starchy foods such as potatoes and potato products are cooked at temperatures above 121°C. The highest concentrations of acrylamide are found in potato chips and French fries that are cooked at high temperatures. However, deep frying at 170°C is known to effectively lower the concentration of toxic compounds, whereas microwaving is only somewhat effective and freeze-drying or dehydration has little effect (133). One study showed that boiling, baking, and microwave potato preparation methods can reduce vitamin C, thiamin, riboflavin, niacin, folic acid, and vitamin B-6 (134). Additionally, boiled potato has a

TABLE 4 Findings of meta-analyses for the consumption of total potato and potato preparation with site-specific cancers in adults[1]

Cancer type	Studies, *n*	Summary effect sizes (95% CI)	tau^2
Total potato intake			
Total CRC[2]			
Total	13	1.09 (0.98, 1.22)	0.008
Cohort	8	1.12 (1.004, 1.25)	0.0006
Case-control	5	1.05 (0.79, 1.39)	0.048
CRC[3]			
Total	9	1.11 (0.97, 1.28)	0.015
Cohort	5	1.10 (0.92, 1.32)	0.017
Case-control	4	1.13 (0.86, 1.49)	0.033
Colon cancer			
Total	4	1.09 (0.93, 1.28)	0.0005
Cohort	3	1.13 (0.97, 1.33)	<0.0001
Case-control	1	0.67 (0.36, 1.21)	<0.0001
Rectal cancer			
Cohort	2	1.48 (1.10, 1.98)	<0.0001
Pancreatic cancer			
Total	5	1.21 (1.01, 1.45)	0.008
Cohort	2	1.23 (0.87, 1.72)	0.005
Case-control	3	1.23 (0.95, 1.60)	0.025
Gastric cancer			
Case-control	7	1.005 (0.69, 1.46)	0.161
Breast cancer			
Total	4	0.98 (0.85, 1.12)	0.006
Cohort	2	0.97 (0.78, 1.21)	0.006
Case-control	2	1.02 (0.75, 1.37)	0.031
Prostate cancer			
Total	5	1.03 (0.95, 1.10)	<0.0001
Cohort	4	1.04 (0.96, 1.12)	<0.0001
Case-control	1	0.90 (0.70, 1.16)	<0.0001
Renal cancer			
Total	2	0.73 (0.48, 1.06)	<0.0001
Cohort	1	0.71 (0.46, 1.08)	<0.0001
Case-control	1	0.80 (0.29, 2.16)	<0.0001
Bladder cancer			
Case-control	5	0.72 (0.46, 1.14)	0.195
Lung cancer			
Case-control	2	0.80 (0.49, 1.29)	<0.0001
Fried potato intake			
Gastrointestinal cancer			
Total	9	1.03 (0.89, 1.19)	<0.0001
Cohort	4	1.07 (0.90, 1.27)	<0.0001
Case-control	5	0.95 (0.74, 1.23)	<0.0001
Sex-hormone-related cancer			
Total	6	1.01 (0.91, 1.11)	0.007
Cohort	3	0.97 (0.90, 1.05)	0.003
Case-control	3	1.15 (0.76, 1.75)	0.084
Urinary-related cancer			
Case-control	9	1.26 (0.82, 1.93)	0.321
Lung cancer			
Total	2	1.26 (0.81, 1.95)	0.058
Cohort	1	1.07 (0.82, 1.39)	<0.0001
Case-control	1	1.70 (0.98, 2.94)	<0.0001
Boiled/mashed/baked potato intake			
Total	4	0.87 (0.66, 1.14)	<0.0001
Cohort	2	0.95 (0.81, 1.11)	0.054
Case-control	2	0.73 (0.30, 1.77)	0.379

[1] CRC, colorectal cancer.
[2] Total CRC included CRC, colon, and rectal cancers.
[3] CRC included.

TABLE 5 GRADE evidence profile[1]

Outcome	Studies *n*	Study design	Risk of bias	Inconsistency	Indirectness	Imprecision	Other considerations	ES (95% CI)	Assumed risk	Absolute risk (95% CI)	Certainty
Total potato											
Total cancer	18	Cohort	Serious	Not serious	Not serious	Serious	None	1.04 (0.96, 1.11)	68 per 10000	2.72 per 10000 (−7.48 to 2.75)	⊕⊕◯◯ Low due to risk of bias and imprecision
CRC	9	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.11 (0.97, 1.28)	81 per 10000	8.9 per 10000 (−22.68 to 2.43)	⊕⊕◯◯ Low due to risk of bias and imprecision
Colon cancer	4	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.09 (0.93, 1.28)	68 per 10000	6.12 per 10000 (−19.04 to 4.27)	⊕⊕◯◯ Low due to risk of bias and imprecision
Rectal cancer	2	Cohort	Serious	Not serious	Not serious	Serious	None	1.47 (1.10, 1.98)	17 per 10000	7.99 per 10000 (−16.66 to −1.7)	⊕⊕◯◯ Low due to risk of bias and imprecision
Pancreatic cancer	5	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.21 (1.01, 1.45)	19 per 10000	3.99 per 10000 (−8.55 to −0.19)	⊕⊕◯◯ Low due to risk of bias and imprecision
Gastric cancer	7	Case-control	Serious	Serious	Not serious	Serious	None	1.005 (0.69, 1.46)	137 per 10000	0.68 per 10000 (−63.02 to 42.47)	⊕◯◯◯ Very low due to risk of bias, imprecision, and inconsistency
Breast cancer	4	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	0.98 (0.85, 1.12)	130 per 10000	2.6 per 10000 (−15.6 to 19.5)	⊕⊕◯◯ Low due to risk of bias and imprecision
Prostate cancer	5	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.03 (0.95, 1.10)	204 per 10000	6.12 per 10000 (−20.4 to 10.2)	⊕⊕◯◯ Low due to risk of bias and imprecision
Renal cancer	2	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	0.73 (0.48, 1.06)	24 per 10000	6.48 per 10000 (−1.44 to 12.48)	⊕⊕◯◯ Low due to risk of bias and imprecision
Lung cancer	2	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	0.80 (0.49, 1.29)	4781 per 10000	956 per 10000 (−1386 to 2438)	⊕⊕◯◯ Low due to risk of bias and imprecision
Bladder cancer	5	Case-control	Serious	Serious	Not serious	Serious	None	0.72 (0.46, 1.14)	5094 per 10000	956 per 10000 (−713 to 1426)	⊕◯◯◯ Very low due to risk of bias, imprecision, and inconsistency
Fried potato											
Gastrointestinal cancers	9	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.03 (0.89, 1.19)	81 per 10000	2.43 per 10000 (−15.39 to 8.91)	⊕⊕◯◯ Low due to risk of bias and imprecision

(*Continued*)

TABLE 5 (*Continued*)

Outcome	Studies *n*	Study design	Risk of bias	Inconsistency	Indirectness	Imprecision	Other considerations	ES (95% CI)	Assumed risk	Absolute risk (95% CI)	Certainty
Sex-hormone-related cancers	6	Cohort & case-control	Serious	Not serious	Not serious	Serious	None	1.01 (0.91, 1.11)	130 per 10000	1.3 per 10000 (−14.3 to 11.7)	⊕⊕◯◯ Low due to risk of bias and imprecision
Urinary-related cancers	9	Cohort & case-control	Serious	Serious	Not serious	Serious	None	1.26 (0.82, 1.93)	3283 per 10000	853 per 10000 (−3053 to 590)	⊕◯◯◯ Very low due to risk of bias, imprecision, and inconsistency
Lung cancer	2	Cohort & case-control	Serious	Serious	Not serious	Serious	None	1.26 (0.86, 1.95)	57 per 10000	14.82 per 10000 (−54.15 to 7.98)	⊕◯◯◯ Very low due to risk of bias, imprecision, and inconsistency

[1]Symbols indicate the following strength of evidence: ⊕⊕⊕⊕, high (further research is very unlikely to change our confidence in the estimate of association); ⊕⊕⊕◯, moderate (further research is likely to have an important impact on our confidence in the estimate of association and may change the estimate); ⊕⊕◯◯, low (further research is very likely to have an important impact on our confidence in the estimate of association and is likely to change the estimate); and ⊕◯◯◯, very low (any estimate of association is very uncertain). CRC, colorectal cancer; ES, effect size; GRADE, Grading of Recommendations, Assessment, Development and Evaluation.

high GI compared with other kinds of potatoes, which is mainly due to the conversion of native starch granules into rapidly digestible starch (RDS) (135). Therefore, the amount of essential nutrients decreases with the potato processing, whereas its GI increases.

To the best of our knowledge, this is the first systematic review and meta-analysis to investigate the association between potato consumption and risk of cancer in multiple sites. Strengths of this study include a large sample size including different geographic regions with different dietary patterns. Our findings were stable and robust in sensitivity analyses. Moreover, findings were adjusted for a great number of confounding factors in the studies that were included and we found no evidence of publication bias. We used the GRADE tool to assess the certainty of the evidence. The certainty of the evidence was rated as low for the association between total potato (white and yellow) consumption and total cancer, CRC, colon, rectal, renal, pancreatic, breast, prostate, and lung cancers and was rated as very low for gastric and bladder cancers. Furthermore, the certainty of the evidence was rated as low for the association between fried potato consumption and gastrointestinal cancers, and sex-hormone-related cancers and was rated as very low for urinary-related cancers and lung cancer.

Besides these strengths, several limitations should be kept in mind when interpreting our findings. First, most of the studies that were included had case-control designs. Case-control studies are subject to recall bias, selection bias, and reverse causation bias. Second, data on different types of potatoes as well as potato preparation and processing methods were not available in most of the studies, which prevented us from performing a more accurate detailed analysis. Third, use of a self-administered questionnaire to assess potato consumption could result in measurement errors. Use of an FFQ for capturing variation in dietary intake, especially potato intake, in different regions might also lead to bias due to the fact that different types of potatoes are more commonly found in different geographical areas (which is not taken into account in the FFQ). Fourth, most of the studies did not adjust other food items when investigating the association between potato consumption and risk of cancer. Because our meta-analysis was conducted on observational studies, we cannot conclude the possible specific effects of residual confounding on the results of both each separate study and also the pooled estimates.

Considering the importance of potatoes as a food source in many parts of world, these findings warrant further investigation. Prospective cohort studies with consistent, improved methods of estimating intake are warranted to investigate the associations between specific kinds of potato and cancer.

Conclusion

Overall, we did not find a significant association between potato consumption and risk of site-specific cancers when

comparing high and low categories or when using linear dose-response analyses. More cohort studies are needed to confirm the findings related to cancers with low or very low certainty evidence.

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