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REVIEW

Potato consumption and risk of all cause, cancer and cardiovascular mortality: a systematic review and dose-response meta-analysis of prospective cohort studies

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ABSTRACT

A systematic review and meta-analysis of prospective cohort studies was conducted to examine the association of potato consumption and risk of all-cause, cancer and cardiovascular mortality in adults. We searched PubMed, Scopus databases up to September 2018 for all relevant published papers. All analyses were performed on HRs or RRs and 95% CIs. In twenty prospective studies, 25,208 cases were reported for all-cause mortality, 4877 for cancer mortality and 2366 for CVD mortality. No significant association was found between potato consumption and risk of all-cause (0.90; 95% CI: 0.8, 1.02, p = 0.096) and cancer (1.09; 95% CI: 0.96, 1.24, P = 0.204) mortality. In addition, no significant linear association was found between each 100 g/d increments in potato consumption and risk of all-cause (P = 0.7) and cancer (P = 0.09) mortality. Moreover, nonlinear association between potato consumption and risk of cancer mortality was non-significant (P-nonlinearity = 0.99). In addition, two of three studies which examined the association of potato consumption with CVD mortality did not find any significant relationship. There was no evidence for publication bias in this study. We failed to find significant association between potato consumption and risk of mortality. Further studies are required to confirm this issue.

KEYWORDS
Potato; cardiovascular disease; cancer; mortality; meta-analysis

Introduction

Cardiovascular disease (CVD) and cancers are the major causes of mortality, accounting for 33% and 15% of annual deaths worldwide (Hayat et al. 2010; Pagidipati and Gaziano 2013). Along with high rate of mortality, these chronic diseases cause serious morbidities and impute a huge economic burden on countries (Bloom et al. 2012).

Several modifiable factors including dietary intakes have been related to risk of CVD and cancer (Pagidipati and Gaziano 2013). For instance, consumption of a high carbohydrate diet has been associated with adverse effects on lipid and glucose metabolism, and subsequently to the risk of CVD and cancer (Ando et al. 2018; Ludwig et al. 2018; Mirrahimi et al. 2014).

Potato is known as one of the most popular foods all over the world. This starchy vegetable is a fundamental part of the most nation's food supplies because of its fruitfulness properties and favorable taste (Burlingame, Mouillé, and Charrondiere 2009; Zaheer and Akhtar 2016). Traditionally, potatoes have been identified as a vegetable; however, they are best classified as a refined starch because of a high amount of starch and unfavorable impact on the risk of disease (Chiuve and Willett 2007).

Potato is a rich source of vitamin C which could play a role in cardiovascular health (Camire, Kubow, and Donnelly 2009a; Hodgson et al. 2014). Potatoes have higher amount of potassium and lower amount of sodium than other fruits and vegetables (USDA 2005). The US Food and Drug Administration (FDA) has showed a favorable impact of foods that are rich in potassium and low in sodium, such as potatoes, and lowered risk of hypertension and stroke (Food and Administration 2000). It also contains other minerals including phosphorus, magnesium as well as dietary fiber (King and Slavin 2013). Due to the considerable amounts of water, potato is considered as a low energy density food item (Anderson, Soeandy, and Smith 2013). In addition, consumption of potato promote satiety in humans, therefore, it has a permanent place in different weight loss programs (Geliebter et al. 2013). However, high glycemic index and glycemic load of potato have made some concerns about its growing consumption (Zurbau et al. 2018). Potatoes and potato components have been shown to have beneficial effects on several cardio metabolic risk factors including decreasing blood pressure, improving lipid profiles, and lowering inflammatory biomarkers (McGill, Kurilich, and Davignon 2013a).

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Few studies showed a significant association between potato consumption and risk of CVD and cancers development and mortality (Pietinen et al. 1996; Skuladottir et al. 2006). However, other studies could not find any significant association between potato intake and mortality (Larsson and Wolk 2016; Osella et al. 2018; Veronese et al. 2017).

A recent systematic and meta-analysis examined the association of consumption of potato and risk of all-cause mortality (Schwingshackl et al. 2018). However, low studies were included in that meta-analysis. Moreover, potato was consumed in combination with other tubers in one of the included studies. In addition, to the best of our knowledge there was no systematic review for the association of potato intake, cancer and CVD mortality. Therefore, we conducted this systematic review and meta-analysis to summarize available findings for the association of potato consumption with risk of all cause, cancer and cardiovascular mortality in adults among prospective cohort studies.

Methods

Current systematic review and meta-analysis has been conducted according to the Preferred Reporting Items of Systematic Reviews and Meta-Analysis (PRISMA) statement guideline (Moher et al. 2009). The protocol was uploaded to the PROSPERO International prospective register of systematic reviews (www.crd.york.ac.uk/PROSPERO; CRD42018104183).

Search strategy

Relevant studies were searched in PubMed and Scopus databases up to September 2018, using MESH and non-MESH key words (Supplemental Methods in supplementary materials). We limited our searches to studies published in English language articles. Moreover, reference lists of eligible articles, including related reviews were also hand-searched. Two reviewers (MDM and AM) conducted assessment of publications based on titles and abstracts and then full text according to inclusion and exclusion criteria. If there was disagreement in making decision between 2 reviewers, it was discussed with the third one (LA). Unpublished data and gray literature such as conference papers, editorials, theses, and patents were not included.

Inclusion criteria

Studies were included in the current meta-analysis if they: (1) had full texts written in English language (2) were prospective cohort studies (3) were conducted in adults (age $\geq$18 y) (4) considered white unfried potatoes including boiled, baked, mashed potatoes and potato salad and fried potatoes (potato chips, French fries,.) or potatoes (not specified) as the exposure (5) examined the association for all-cause, CVD, or cancer mortality (6) reported hazard ratios (HRs), relative risks (RRs) or odds ratios (ORs) with corresponding 95% CIs for the association of potato consumption with all-cause, CVD, or cancer mortality.

Exclusion criteria

Studies were excluded if they (1) were ecologic, cross-sectional, case-control, or randomized clinical trials (RCTs) studies (2) were carried out on children, pregnant women or animals (3) did not report HRs, RRs or ORs with corresponding 95% CI (4) examined other cause of mortality (5) analyzed the association for potato consumption along with other food items (6) reported HRs, RRs or ORs and 95% CI for dietary patterns rather than food items. In addition, we excluded studies that assessed consumption of specific types of potatoes rather than white or yellow potatoes due to the different composition between them. If more than one study used the same data set, only study with the largest number of incident cases was included.

Data extraction

Two independent researchers (MDM, AM) extracted necessary data from the included studies. Any disagreements were discussed and resolved by a third investigator (LA). Following data were obtained from included studies: first author's name; year of publication; study location; duration of follow-up; age range and gender of participants; study design; health status of study population; study sample size and number of deaths; types of potato (boiled/fried/mashed/baked); methods used for exposure assessment; outcomes; outcome assessment methods; categories of potato intake; risk estimate [most adjusted measures; HRs, RRs, or ORs with their corresponding 95% CIs] variables, and potential confounders adjusted for. If a study provided data at different time points, only the latest one was recruited. We sent several E-mails for several times to the corresponding author of the studies which did not reported sufficient data.

Quality assessment

Newcastle-Ottawa Scale (NOS) scale was used to assess the quality of included studies (Margulis et al. 2014). This scale consists of three sections for the assessment of subject selection, comparability of subject, and exposure or outcome. A score of 0–4, 0–2, and 0–3 will be given for each section, respectively. Overall score will be between 0 and 9. In our study, studies with a score of 6 or above were considered as high quality.

Statistical analysis

We pooled effect sizes from included studies using fixed effect meta-analysis. Risk estimates with the greatest degree of adjustment for potential confounders were used in this meta-analysis. Between-study heterogeneity was explored using Q Cochrane test and I^2 statistics (Higgins and Thompson 2002) . I^2 score of 50% or higher were considered as the presence of between study-heterogeneity. To reduce the heterogeneity we reanalyzed our data with the random-effect model. In addition, subgroup analysis with

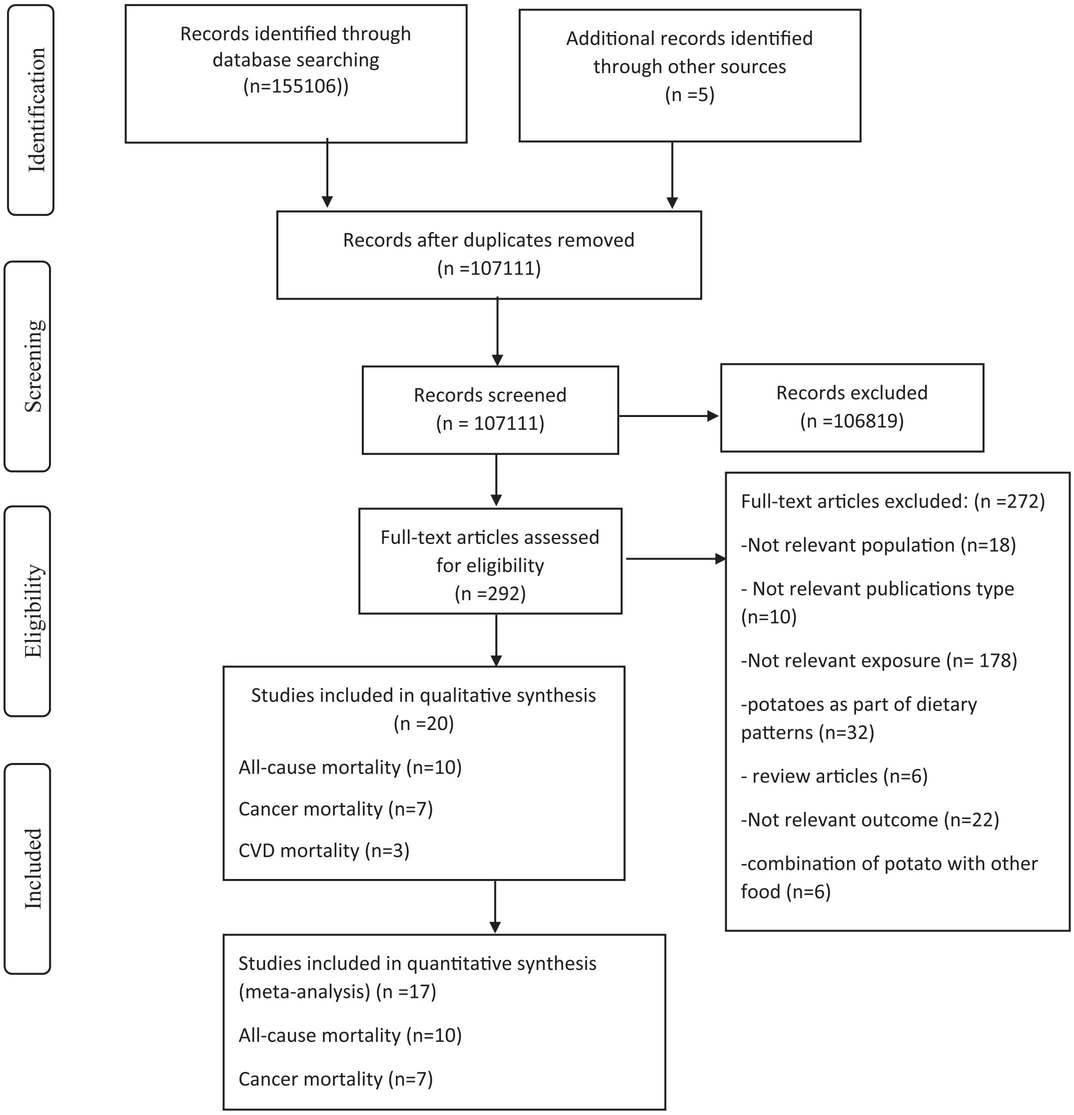

Figure 1. Flow diagram of study selection.

the fixed-effect model was conducted based on gender (male, female, both), sample size, case number, follow up years (>10 y, ≤10 y), exposure assessment tool (FFQ, non FFQ), energy adjustment (yes, no), BMI adjustment (yes, no).

In addition, we conducted dose-response analysis using the method proposed by Greenland and Longnecker et al., (Greenland and Longnecker 1992) and Orsini et al., (Orsini, Bellocco, and Greenland 2006). The number of cases, person-years and risk estimates were extracted from studies with at least 3 quantitative exposure categories. If a study did not provide the number of cases or person-years in each category, we considered it to be the same in each category. In addition, median or mean intake of potato for each category was also extracted. In studies reported the frequency of potato consumption, daily grams of potato intake were calculated considering a serving size to be 100 g (2008). The non-linear association was examined by modeling exposure levels with the use of restricted cubic splines with 3 kn at percentiles 10%, 50%, and 95% of the distribution (Orsini et al. 2012; Wang et al. 2014). The null hypothesis was that the coefficient of the second spline is equal to zero.

Furthermore, a linear dose-response association of potato consumption with mortality risk was examined for each 100-g/d increment in its consumption. In addition, potential publication bias was investigated using both Egger regression and Begg correlation tests (Begg and Mazumdar 1994; Egger et al. 1997). Moreover, sensitivity analysis was done by omitting one study at each stage to find if an individual study or a group of studies had considerable influence on our results. All statistical analyses were performed by the use of STATA software, version 11.0 (STATA Corp.). P values lower than 0.05 were considered as statistically significant.

Results

Systematic literature search resulted in 155111 records of them 107111 records remained after removing of duplications (Figure 1). After investigation of articles by the title and abstract, 106819 publications were excluded. Among 292 remained publications, 272 articles were excluded for the following reasons: not relevant population, ecologic, cross sectional, case–control or RCT studies, review articles, and studies analyzed consumption of potato along with other food items or in a frame of a dietary pattern. Finally, a total of 20 prospective cohort studies selected for the meta-analysis; from that, 10 studies investigated all-cause mortality, 7 studies cancer mortality, and other studies CVD mortality.

Study characteristics

We used a total of 26 reports from 20 studies in our meta-analysis. The studies were published between 1984 and 2018. 5 studies were conducted in the Japan (Huang et al. 2000; Khan et al. 2004; Kurozawa et al. 2004; Sakauchi et al. 2007; Tokui et al. 2005), 2 in United States (Veronese et al. 2017; Wilson et al. 2012), 2 in 10 European countries (Iestra et al. 2006; Sluik et al. 2014), 2 in Italy (Osella et al. 2018; Prinelli et al. 2015), 2 in Spain (Gonzalez et al. 2008; Guallar-Castillon et al. 2012) and the remained studies were conducted in Sweden (Larsson and Wolk 2016), France (Bongard et al. 2016), 9 European countries (Trichopoulou et al. 2007), Greece (Dilis et al. 2012), Denmark (Skuladottir et al. 2006), Finland (Pietinen et al. 1996) and California (Kahn et al. 1984). The number of participants were 417688 for all-cause mortality, 115172 for CVD mortality, and 268555 for cancer mortality. In most studies (n = 12) (Dilis et al. 2012; Gonzalez et al. 2008; Guallar-Castillon et al. 2012; Huang et al. 2000; Kahn et al. 1984; Osella et al. 2018; Prinelli et al. 2015; Sakauchi et al. 2007; Skuladottir et al. 2006; Sluik et al. 2014; Trichopoulou et al. 2007; Veronese et al. 2017), the effect size for both genders in combination was reported and in 5 studies reported separately (Iestra et al. 2006; Khan et al. 2004; Kurozawa et al. 2004; Larsson and Wolk 2016; Tokui et al. 2005). However, some studies were conducted only on men (n = 3) (Bongard et al. 2016; Pietinen et al. 1996; Wilson et al. 2012). We identified 25,208 cases of all-cause mortality, 2366 cases of cancer mortality, and 4878 cases of CVD mortality. Characteristics of these studies are presented in Table 1. Follow-up periods ranged from 6 to 21 y, and the age range was from 20 to 86 y. Potato consumption was assessed by food record questionnaire (n = 1) (Bongard et al. 2016) and dietary history questionnaire (n = 3) (Guallar-Castillon et al. 2012; Huang et al. 2000; Iestra et al. 2006) and the remained studies used food frequency questionnaires (Dilis et al. 2012; Gonzalez et al. 2008; Kahn et al. 1984; Khan et al. 2004; Kurozawa et al. 2004; Larsson and Wolk 2016; Osella et al. 2018; Pietinen et al. 1996; Prinelli et al. 2015; Sakauchi et al. 2007; Skuladottir et al. 2006; Sluik et al. 2013; Tokui et al. 2005; Trichopoulou et al. 2007; Veronese et al. 2017; Wilson et al. 2012).

Two studies reported amounts of French fry consumption (Larsson and Wolk 2016; Wilson et al. 2012), one study reported boiled potato intake (Larsson and Wolk 2016) while, two other studies reported consumption of fried potato (Guallar-Castillon et al. 2012; Veronese et al. 2017). However, other studies combined amounts of different forms of potato consumption.

Among included studies, eight studies did not show a significant association between consumption of potatoes (regardless to its type) and risk of all-cause mortality (Bongard et al. 2016; Gonzalez et al. 2008; Iestra et al. 2006; Osella et al. 2018; Prinelli et al. 2015; Sluik et al. 2014; Trichopoulou et al. 2007; Veronese et al. 2017). However, an inverse association was found between total potato consumption and all-cause mortality in Khan et al. study (Kahn et al. 1984). With regards to fried potatoes, a prospective large cohort showed a direct association of its consumption with all-cause mortality risk (Veronese et al. 2017). However, another prospective study could not find a linear dose-response association between 100 gr increment in fried potato consumption and all-cause mortality (Guallar-Castillon et al. 2012).

In addition, a significant association was found between consumption of total potatoes and risk of cancer mortality in a Skuladottir et al., Study (Skuladottir et al. 2006). However, other studies failed to find such an association (Huang et al. 2000; Khan et al. 2004; Kurozawa et al. 2004; Sakauchi et al. 2007; Tokui et al. 2005). In addition, no association of French fry consumption with cancer mortality was found in the unique study in this field (Wilson et al. 2012).

Furthermore, several studies could not find a significant association between total potatoes consumption and risk of CVD mortality (Dilis et al. 2012; Larsson and Wolk 2016). Also Larsson et al. (Larsson and Wolk 2016) study on the association of fried potato with CVD mortality failed to find a significant relationship. However, Pietinen et al., found a significant inverse association between high potatoes consumption and chronic heart disease (CHD) mortality risk (Pietinen et al. 1996).

The methodological quality of all included studies was high (score $\geq$ 6) (Supplementary Table 1 in supplementary materials).

Potato intake and total mortality

Ten studies (Bongard et al. 2016; Gonzalez et al. 2008; Guallar-Castillon et al. 2012; Iestra et al. 2006; Kahn et al. 1984; Osella et al. 2018; Prinelli et al. 2015; Sluik et al. 2014; Trichopoulou et al. 2007; Veronese et al. 2017) with 12 effect sizes were included in the meta-analysis of potatoes intake and total mortality. Pooling effect sizes from these studies, no significant association was found between potato consumption and risk of total mortality (0.9; 95% CI: 0.8, 1.02, $p = 0.096$), with low heterogeneity ($P = 0.326$, $I2 = 13.5\%$) (Figure 2). In addition, visual inspection of funnel plot (supplementary Figure 1) and Egger test (P-Egger = 0.26) found no evidence of publication bias. Furthermore, sensitivity analysis showed that no individual study had significant effect on the overall effect size (supplementary Figure 2). Khan et al., showed that higher consumption of potato was associated with the reduced risk of all-cause

Table 1. Characteristics of these studies.

Authors (year)	Country	Age range (y)	Gender	Characteristics of subjects	Sample size	Cases	Follow-up (y)	Exposure	Exposure assessment	Outcome	Outcome assessment	Categorical or continuous	OR, RR or HR (95%CI)	Adjustment[a]
Osella et al (2018) (19)	Italy	≥50	M/F	General Practitioners	2442	396	11	Potatoes (not specified)	FFQ Self-reported	All-cause mortality	Death certificate	Q5 vs. Q1	HR :0.75 (0.53–1.07)	1,2,3,6,9,10, 13,18, 22,60
Veronese et al. (2017) (18)	USA	45–79	M/F	Free of CVD and diabetes	4400	236	8	Potatoes (boiled, mashed, baked, potato salad, fried, French, hash brown) Fried potatoes (fried, French, hash brown)	FFQ: Self-reported	All-cause mortality	Autopsy report, coroner's report, death certificate, medical records, National Death Index, obituary, or Social Security Death Index	Q5 vs. Q1	HR: 1.11 (0.65, 1.91) HR: 2.26 (1.15, 4.47)	1,2,3,4,6,7,8,9, 10,11,12,13,14
Larsson and Wolk (2016) (20)	Sweden	45–79	M	Patients at high risk of knee osteoarthritis	36,508	1713	13	Potatoes (boiled , French, fried)	FFQ: Self-reported	CVD mortality	Swedish National Patient and Cause of Death Registers	Q4 vs. Q1	HR: 0.94 (0.79, 1.12)	1,3,6,7,8,9,10, 15,16,17,18,19
		49–83	F	Patients at high risk of knee osteoarthritis	32,805	2290	13	Potatoes (boiled , French, fried)	FFQ: Self-reported	CVD mortality	Swedish National Patient and Cause of Death Registers	Q4 vs. Q1	HR: 0.88 (0.75, 1.02)	1,3,6,7,8,9,10, 15,16,17,18,19
		45–83	M/F	Patients at high risk of knee osteoarthritis	69313	4003	13	French fries fried potato boiled potato	FFQ: Self-reported	CVD mortality	Swedish National Patient and Cause of Death Registers	Q5 vs. Q1	HR: 1.19 (0.86, 1.66) HR: 1.10 (0.94, 1.29) HR: 0.94 (0.85, 1.05)	1,3,6,7,8,9,10, 15,16,17,18,19
Bongard et al. (2016) (45)	France	45–64	M	Healthy subjects	960	150	14.8	Potatoes (not specified)	3-day food record Interview	All-cause mortality	French national database	Q4 vs. Q1	HR: 1.12 (0.69–1.83)	1,6,7,8,20,21,22,23
Prinelli et al. (2015) (42)	Italy	40–74	M/F	Healthy subjects	974	193	20	Potatoes (not specified)	FFQ: Interview	All-cause mortality	Regional Registry of mortality	M2 vs. M1	HR: 0.99 (0.73–1.36)	1,2,3,6,7,8,9,24
Sluik et al. (2014) (40)	10 European countries	35–70	M/F	Persons with diabetes	6,384	830	9.9	Potatoes (not specified)	FFQ: Interview	all-cause mortality	death registries	Per 100 g increase	HR: 1.10 (0.91, 1.32)	2,3,7,8,10,25,26,27
				Persons without diabetes	258911	12135	9.9	Potatoes (not specified)	FFQ, food record and food recall: Interview	All-cause mortality	Death registries	Per 100 g increase	HR: 0.96 (0.90, 1.01)	2,3,7,8,10,25,26,27
Dilis et al. (2012) (47)	Greece	20–86	M/F	Persons without CVD and cancer at baseline	23929	240	10	Potatoes (not specified)	FFQ: Interview	CHD death	Hospital discharge data, medical records or death certificates	Per 1 SD increase	HR: 1.15 (0.82–1.61)	1,3,5,6,7,8,9,10, 28,29,30,31,32, 33,34,35,36,37, 38,39,40,41,42
Wilson et al. (2012) (39)	USA	40–75	M	Healthy men	47,896	642	20	French fries	FFQ: Self-reported	Prostate Cancer death	Family members and searches of the National Death Index	Q4 vs. Q1	RR: 1.03(0.79, 1.33)	1,4,5,6,7,8,9,10,43, 44,45,29,46,47, 48,49,50
Guallar-Castillon et al. (2012) (43)	Spain	29–69	M/F	Healthy adults	40,757	1135	11	Fried potato	dietary history questionnaire Interview	All-cause mortality	Regional mortality registries and the national mortality database	Per 100 g increase	HR: 0.90 (0.70, 1.15)	1,2,3,4,6,7,8,10,29, 30,32,33,35,43, 51,52,53,54,55
Gonzalez et al. (2008) (44)	Spain	60–85	M/F	Elderly people	288	88	6	Potatoes (not specified)	FFQ: Interview	All-cause mortality	Contact with each center	Per 1 SD increase	RR: 1.31(1.03–1.68)	1,2,3,4,6,7,8,9, 19,43,56,57
Sakauchi et al. (2007) (34)	Japan.	40–79	M/F	Volunteers from the general population	110792	71	13	Potatoes (not specified)	FFQ: Self-reprted	Ovarian cancer death	Death certificates	T3 vs. T1	HR: 1.54(0.66, 3.61)	1,3,6,7,53,58,59
Skuladottir et al. (2006) (17)	Denmark	50–64	M/F	Persons without cancer	57,053	295	11	Potatoes (not specified)	FFQ: Self-reprted	Lung cancer death	linkage to the Danish Civil Registration System	T3 vs. T1	HR:1.58(1.12, 2.23)	1,2,8,20,22

(*continued*)

Table 1. Continued.

Authors (year)	Country	Age range (y)	Gender	Characteristics of subjects	Sample size	Cases	Follow-up (y)	Exposure	Exposure assessment	Outcome	Outcome assessment	Categorical or continuous	OR, RR or HR (95%CI)	Adjustment[a]
Iestra et al. (2006) (41)	10 European countries	≥70	M	Post-myocardial infarction (MI) patients	284	188	10	Potatoes (not specified)	dietary history method: Interview	All-cause mortality	Medical records	–	HR:1.15 (0.85–1.55)	1,2,3,6,7,8, 10,26,43
			F	Post-myocardial infarction (MI) patients	142	56	10	Potatoes (not specified)	dietary history method: Interview	All-cause mortality	Medical records	–	HR:0.75 (0.42–1.32)	1,2,3,6,7,8, 10,26,43
Trichopoulou et al. (2007) (46)	Nine European countries	≥60	M/F	Persons without coronary heart disease, stroke, or cancer	74 607	4047	7.4	Potatoes (not specified)	FFQ, food record, 24 h dietary recall Interview	All-cause mortality	Mortality registries	Per 100 g increase	HR: 1.01(0.98, 1.03)	1,2,4,6,7,8,9,10,43,-61
Tokui et al. (2005) (35)	Japan	40–79	M	Aged persons	46465	547	13	Potatoes (not specified)	FFQ: Interview	Stomach cancer death	Death certificates	Q4 vs. Q1	HR: 1.01(0.79,1.26)	1
			F	Aged persons	64327	285	13	Potatoes (not specified)	FFQ: Interview	Stomach cancer death	Death certificates	Q4 vs. Q1	HR: 0.86(0.57, 1.29)	1
Kurozawa et al. (2004) (36)	Japan	40–59	M	Persons without cancer	18058	21	9	Potatoes (not specified)	FFQ: Interview or self-reported	Hepatocellular Carcinoma death	Death certificates	T3 vs. T1	HR: 1.05(0.54, 2.01)	–
		60–79	F	Persons without cancer	18221	20	9	Potatoes (not specified)	FFQ: Interview or self-reported	Hepatocellular Carcinoma death	Death certificates	T3 vs. T1	HR: 0.53(0.17, 1.65)	–
Khan et al. (2004) (37)	Japan	40–97	M	Patients without cancer	1524	155	13.8	Potatoes (not specified)	FFQ self-reported	Lung, colorectal, gastric, Pancreatic cancer	Death registries	–	RR:1.2(0.8,1.9)	1,8
			F	Patients without cancer	1634	89	13.8	Potatoes (not specified)	FFQ self-reported	Lung, colorectal, gastric, Pancreatic cancer	Death registries	–	RR:1.3(0.7, 2.5)	1,8
Huang et al. (2000) (38)	Japan	40–79	M/F	Gastric cancer patients	877	241	6	Potatoes (not specified)	dietary questions: Interview	gastric cancer death	Kaplan-Meier method	–	HR: 0.85 (0.58–1.24)	1,2,60
Pietinen et al. (1996) (16)	Finland	50–69	M	Free of CVD and diabetes	21 930	635	6.1	Potatoes (not specified)	FFQ	CHD mortality	Central Population Register	T5 vs. T1	RR: 0.74 (0.57, 0.97)	1,3,6,7,8,9,10,18,39
Kahn et al. (1984) (48)	California	≥30	M/F	Healthy adults	27539	5754	21	Potatoes (not specified)	FFQ	All-cause mortality	Matching computer tapes	Q4 vs. Q1	OR: 0.81 (0.67, 0.97)	1

[a]Adjustment: age (1), sex (2), education (3), race (4), height (5), BMI (6), physical activity (7), smoking (8)), dietary intake of energy (9), alcohol (10), income (11), depression (12), adherence to Mediterranean diet (13), Charlson comorbidity index (14), family history of MI before 60 y of age (15), aspirin use (16), walking or bicycling (17), history of hypertension (18), history of hypercholesterolemia (19), center (20), payment of income tax (21), presence of a serious chronic condition (22), diet quality score (23), time spent watching TV (24), prevalence of heart disease (25), prevalence of cancer or stroke (26), diabetes medication use (27), arterial blood pressure (28), meat (29), vegetables (30), Legumes (31), Fruits and nuts (32), Dairy foods (33), Cereals (34), fish (35), eggs (36), Sugar and confectioneries (sweets) (37), Non-alcoholic beverages (38), Saturated lipids (39), Monounsaturated lipids (40), Polyunsaturated lipids (41), Monounsaturated: saturated lipid ratio (42), previous endoscopy (42), diabetes (43), family history of prostate cancer (44), multivitamin use (45), tomato sauce (46), calcium intake (47), alpha linolenic acid (48), supplemental vitamin E (49), PSA testing in previous period (50), hyperlipidemia (51), cancer (52), oral contraceptives (53), menopause (54), hormone replacement therapy (55), chewing ability (56), self-perceived health (57), number of full pregnancies (58), hormone therapy (59), job (60), waist to hip ratio (61).

CHD, coronary heart disease; CVD, cardiovascular disease; FFQ, food frequency questionnaire; HR, hazard ratio; OR, odds ratio; M^2, median; SD, standard deviation; Q^4, Quartile; Q5, quintile; T^3, tertile; RR, relative risk; USA, United States.

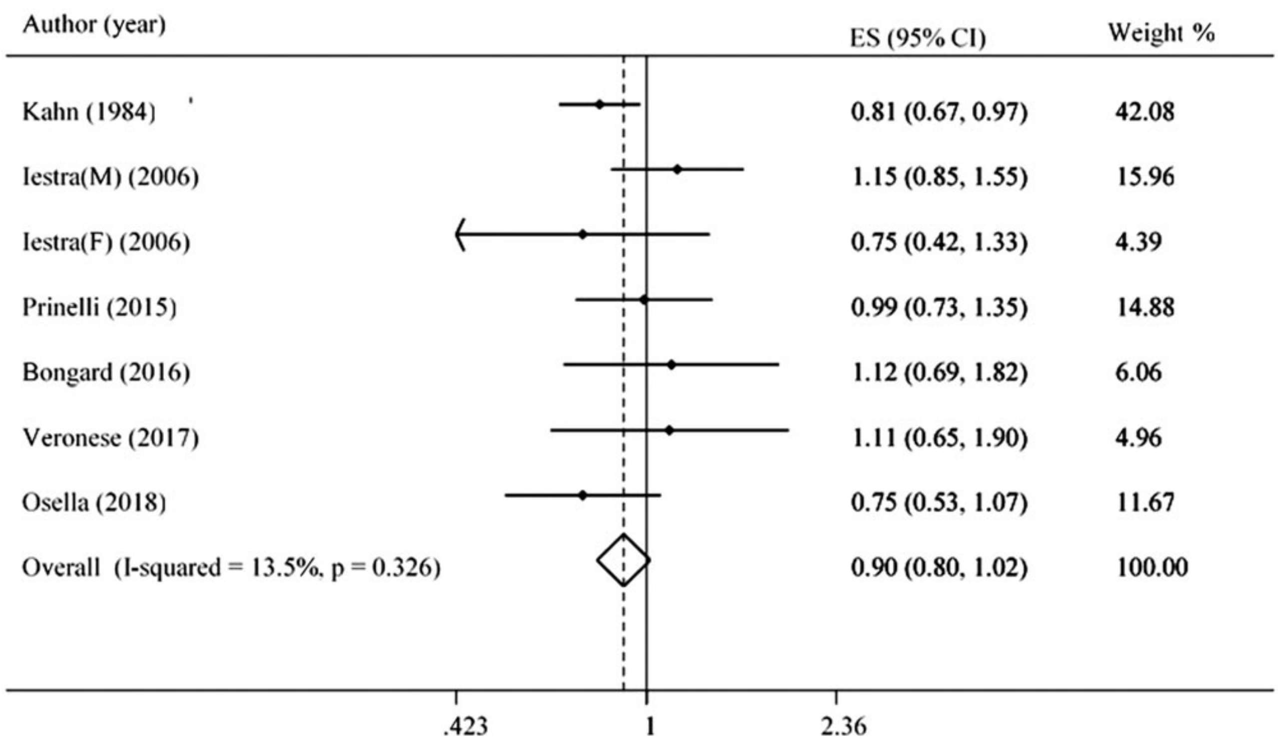

Figure 2. Forest plot showing the association of potato intake and all-cause mortality in adults using the fixed effects model. CI, confidence interval; HR, hazard ratio.

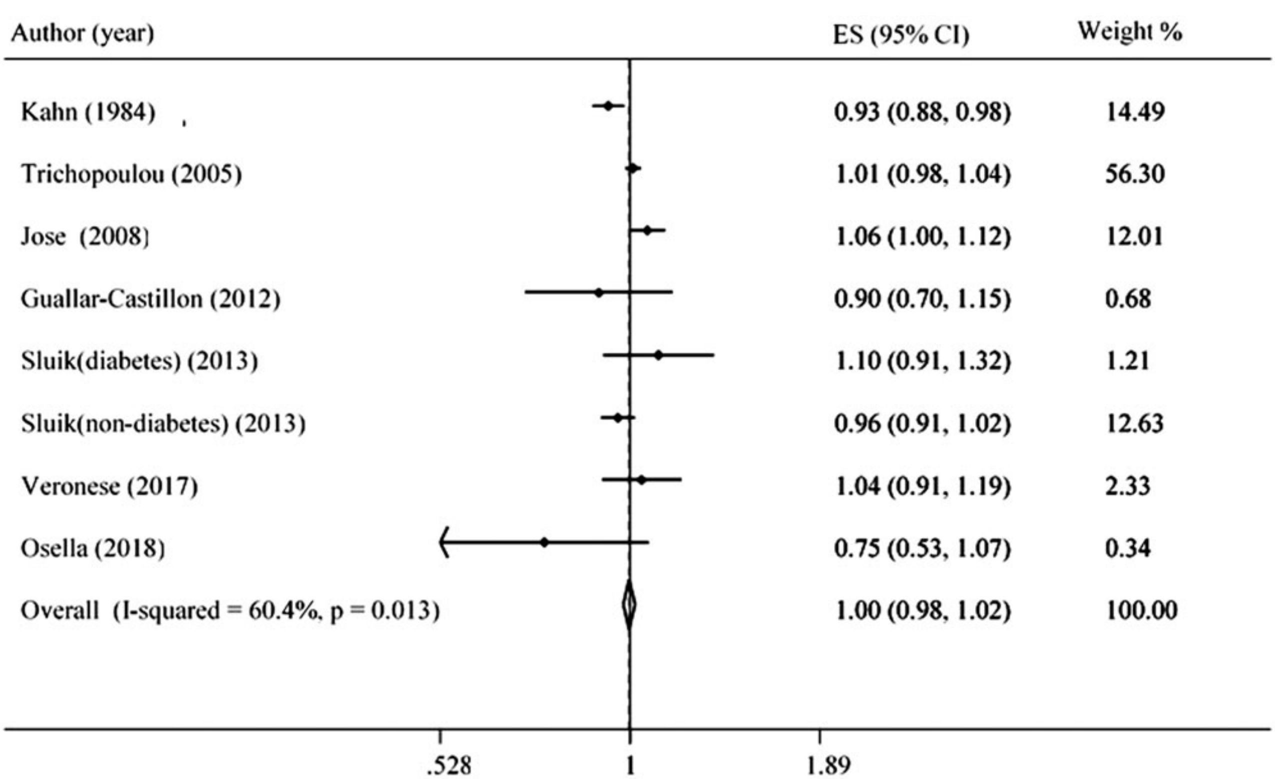

Figure 3. Forest plot showing association of potato intake and all-cause mortality in adults per 100 g/d increase using the random effects model. CI, confidence interval; HR, hazard ratio.

mortality. It should be noted that the study was old and examined association of potato consumption with mortality risk only at baseline. So, we removed it and performed analysis. No significant association was found between potato consumption and risk of total mortality (0.97; 95% CI: 0.83, 1.14, $p = 0.77$), with low heterogeneity ($P = 0.460$,

Table 2. Pooled estimates of association of potato with risk of all-cause, CVD and cancer mortality within different subgroups.

	No of studies				ES (95% CI)				P value				P-heterogeneity				I^2(%)			
Group	Cancer mortality	Cancer mortality (linear)	All-cause mortality	All-cause mortality (linear)	Cancer mortality	Cancer mortality (linear)	All-cause mortality	All-cause mortality (linear)	Cancer mortality	Cancer mortality (linear)	All-cause mortality	All-cause mortality (linear)	Cancer mortality	Cancer mortality (linear)	All-cause mortality	All-cause mortality (linear)	Cancer mortality	Cancer mortality (linear)	All-cause mortality	All-cause mortality (linear)
Total	10	6	6	7	1.08 (0.94, 1.25)	1.05 (0.99, 1.12)	0.9 (0.8, 1.01)	0.99 (0.95, 1.04)	0.248	0.09	0.09	0.7	0.329	0.052	0.326	0.013	12.3	54.5	13.5	60.4
Exposure assessment tool																				
FFQ	9	–	3	6	1.12 (0.97, 1.28)	–	0.90 (0.73, 1.12)	1.01 (0.98, 1.03)	0.09	–	0.384	0.375	0.391	–	0.374	0.09	5.3	–	0	46.2
Non FFQ	1	–	3	1	0.85 (0.58, 1.24)	–	1.06 (0.84, 1.34)	0.90 (0.70, 1.15)	0.402	–	0.599	0.405	–	–	0.420	–	–	–	0	–
Energy adjustment																				
Yes	1	–	3	4	1.03 (0.79, 1.33)	–	0.90 (0.73, 1.12)	1.01 (0.99, 1.04)	0.824	–	0.384	0.155	–	–	0.374	0.163	–	–	0	41.4
No	9	–	3	3	1.10 (0.95, 1.28)	–	1.06 (0.84, 1.34)	0.96 (0.91, 1.02)	0.183	–	0.599	0.236	0.261	–	0.420	0.329	20.4	–	0	10.1
BMI adjustment																				
Yes	2	1	5	5	1.06 (0.83, 1.36)	1.07 (0.92, 1.23)	0.96 (0.81, 1.13)	1.01 (0.99, 1.04)	0.614	0.357	0.646	0.182	0.375	–	0.365	0.195	0	–	7.3	33.9
No	8	5	1	2	1.09 (0.94, 1.27)	1.05 (1.02, 1.08)	1.12 (0.68, 1.82)	0.97 (0.91, 1.02)	0.239	<0.001	0.649	0.304	0.222	0.027	–	0.171	25.9	63.5	–	46.7
Case																				
Lower1[,2,3,4]	5	3	4	4	1.16 (0.87, 1.54)	1.02 (0.92, 1.14)	1.03 (0.86, 1.25)	1.05 (1.0, 1.10)	0.303	0.592	0.702	0.052	0.647	0.537	0.601	0.275	0	0	0	22.6
Higher1[,2,3,4]	5	3	2	3	1.06 (0.92, 1.23)	1.05 (1.02, 1.09)	0.84 (0.62, 1.13)	1.0(0.97, 1.02)	0.367	<0.001	0.255	0.975	0.111	0.009	0.232	0.209	46.8	7.9	29.9	36
Follow up (year)																				
≤10	3	2	3	5	0.86 (0.62, 1.18)	0.98 (0.84, 1.15)	1.06 (0.83, 1.34)	1.01 (0.98, 1.03)	0.35	0.833	0.63	0.322	0.59	0.425	0.425	0.163	0	0	0	38.7
>10	7	4	3	2	1.13 (0.98, 1.31)	1.05 (1.02, 1.09)	0.91 (0.74, 1.13)	0.84 (0.69, 1.03)	0.07	<0.001	0.42	0.108	0.35	0.023	0.344	0.406	10.4	68.5	6.4	0
Gender																				
Both	3	2	3	–	1.22 (0.95, 1.55)	1.13 (1.02, 1.25)	0.90 (0.73, 1.126)	–	0.111	0.017	0.384	–	0.052	0.271	0.374	–	66.3	17.4	0	
Male	4	2	2	–	1.07 (0.90, 1.27)	1.07 (1.03, 1.11)	1.14 (0.88, 1.47)	–	0.413	<0.001	0.310	–	0.949	0.544	0.928	–	0	0	0	–
Female	3	2	1	–	0.92 (0.66, 1.28)	0.98 (0.93, 1.04)	0.75 (0.42, 1.33)	–	0.629	0.627	0.325	–	0.343	0.470			6.7	0		
Sample size																				
Higher1[,2,3,4]	7	3	3	3	1.10 (0.95, 1.28)	1.02 (0.97, 1.07)	0.90 (0.73, 1.12)	1.0(0.97, 1.02)	0.188	0.042	0.384	0.975	0.373	<0.001	0.374	0.209	26	68.5	0	36
Lower1[,2,3,4]	3	4	3	4	1.03 (0.79, 1.34)	1.07 (1.03, 1.11)	1.06 (0.84, 1.34)	1.05 (1.0, 1.10)	0.8	0.379	0.599	0.052	0.230	0.402	0.420	0.275	0	0	0	22.6

[a]Sample size < 10000 vs ≥ 10000, case < 200 vs ≥ 200 for cancer mortality.
[b]Sample size <50000 vs ≥50000, case <200 vs ≥ 200 for cancer mortality (leaner).
[c]Sample size < 1000vs ≥1000, case < 200 vs ≥ 200 for all-cause mortality.
[d]Sample size < 10000vs ≥ 10000, case < 1000 vs ≥ 1000 for all-cause mortality.
[e]CVD, Cardiovascular disease; FFQ, Food frequency questionnaire.

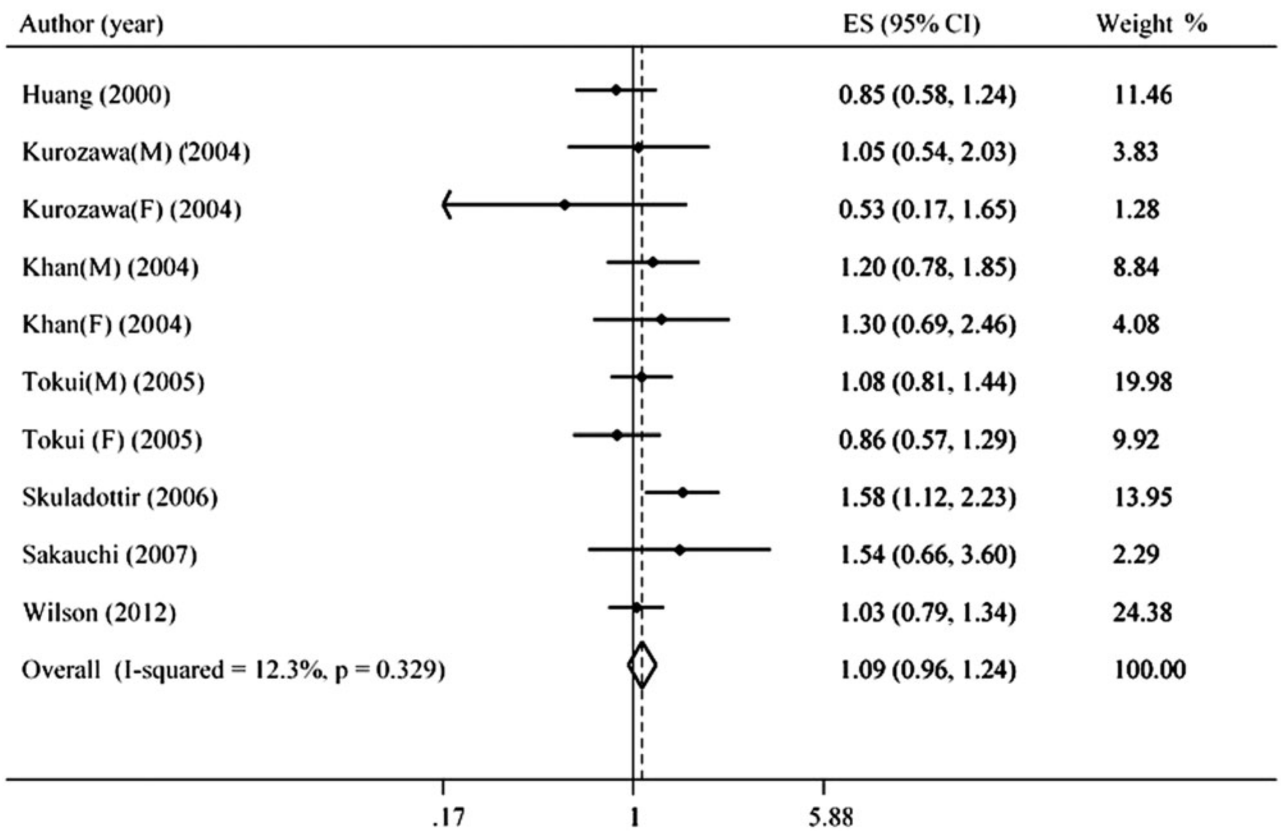

Figure 4. Forest plot showing the association of potato intake and cancer mortality in adults using the fixed effects model. CI, confidence interval; HR, hazard ratio.

I2 = 0%). We also conducted subgroup analysis without this study, however, findings remained unchanged.

Dose-response analysis

Seven studies (Gonzalez et al. 2008; Guallar-Castillon et al. 2012; Kahn et al. 1984; Osella et al. 2018; Sluik et al. 2014; Trichopoulou et al. 2007; Veronese et al. 2017) were included in a dose response meta-analysis of the association between potato consumption and risk of total mortality. Among these studies, six studies (Gonzalez et al. 2008; Kahn et al. 1984; Osella et al. 2018; Sluik et al. 2014; Trichopoulou et al. 2007; Veronese et al. 2017) reported the association for total potato intake and the rest studies for fried potato (Guallar-Castillon et al. 2012). Linear dose-response meta-analysis showed no significant association between 100 g/d increment in potato consumption and risk of all-cause mortality (1.00; 95% CI: 0.98, 1.02, P = 0.79), with moderate heterogeneity (P = 0.013, I2 = 60.4%) (Figure 3). In addition, there was not any evidence of publication bias based on egger test (P-Egger = 0.95) and funnel plot (supplementary Figure 3). When we excluded the unique study (Guallar-Castillon et al. 2012) in which consumption of fried potato was assessed, findings remained unchanged (0.99; 95% CI: 0. 95, 1.04, P = 0.811), with moderate heterogeneity (P = 0.009, I2 = 64.8%). Findings remained unchanged after subgroup analysis based on pre-defined factors (Table 2). Due to scare studies with sufficient data for a nonlinear dose–response association meta-analysis, we could not perform such additional analysis.

Potato intake and cancer mortality

Seven studies (Huang et al. 2000; Khan et al. 2004; Kurozawa et al. 2004; Sakauchi et al. 2007; Skuladottir et al. 2006; Tokui et al. 2005; Wilson et al. 2012) were included in the analysis of potato (6 studies) or French fries (1 study) intake and cancer mortality. The summary HR was (1.09; 95% CI: 0.96, 1.24, P = 0.204), with low heterogeneity (I2 = 12.3%, P = 0.329) (Figure 4). Excluding 1 study (Wilson et al. 2012) that involved only French fries, the summary HR was (1.11; 95% CI: 0.95, 1.28, P = 0.18), with low heterogeneity (I2 = 20.4%). we conducted subgroup analyses based on pre-defined factors. However, these factors were not the cause of insignificancy. Funnel plot (supplementary Figure 4) and egger test (P-Egger = 0.86) did not find any evidence of publication bias. In addition, sensitivity analysis did not provided an evidence for the influence of an individual study on the overall effect size (supplementary Figure 5).

Dose-response analysis

4 studies (Kurozawa et al. 2004; Sakauchi et al. 2007; Skuladottir et al. 2006; Tokui et al. 2005) with 6 reports were included in the dose-response analysis. The summary RR for each 100-g/d increase in potato intake was (1.05; 95% CI: 0.99, 1.12, P = 0.09), with moderate heterogeneity (P = 0.052, I2 = 54.5%) (Figure 5A) and no publication bias (P-Egger = 0.97) (supplementary Figure 6). Subgroup analysis showed that sample size, case number, BMI adjustment, follow up duration and gender were source of heterogeneity. There was significant association of

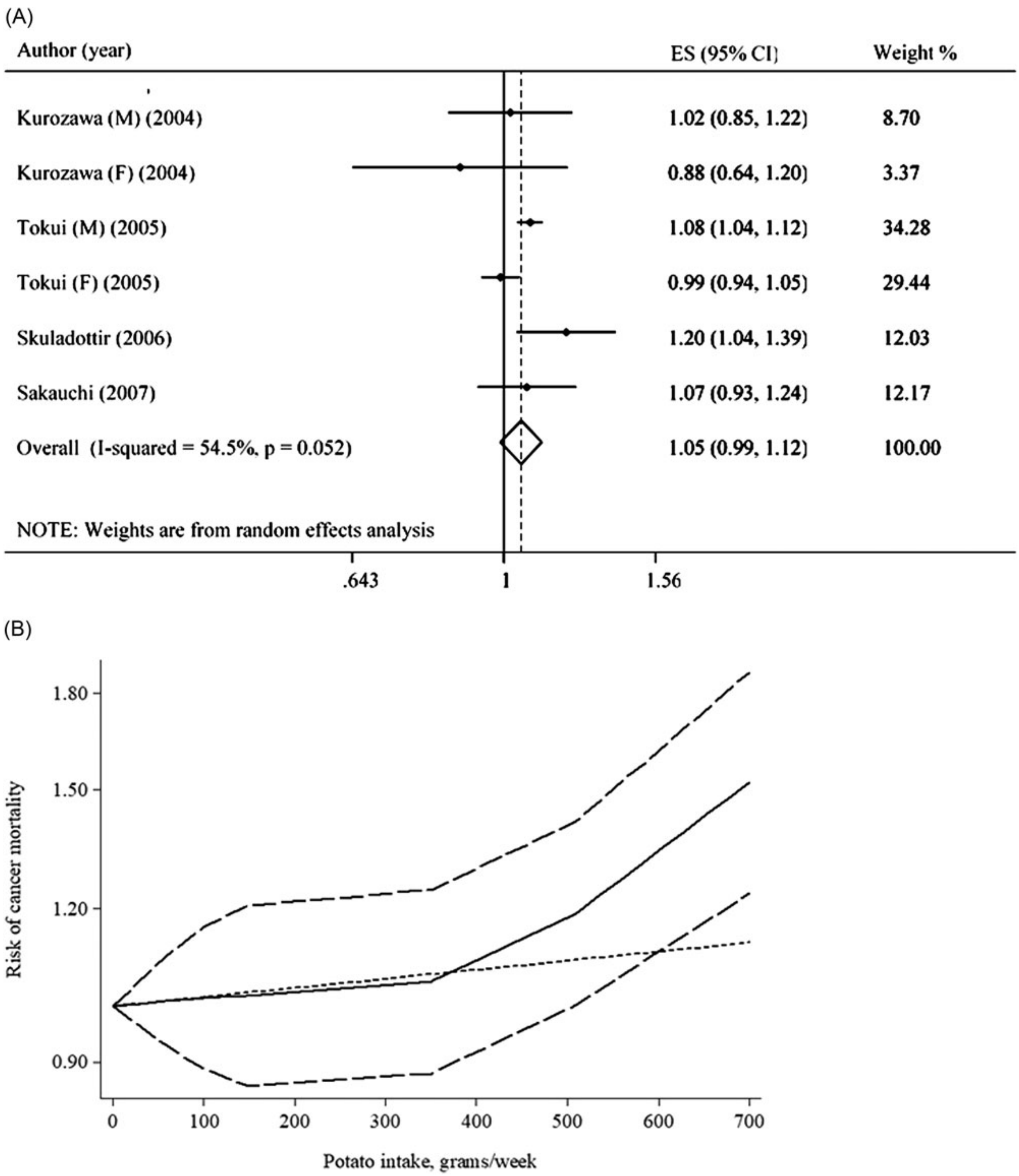

Figure 5. A Forest plot showing association of potato intake and cancer mortality in adults per 100 g/d increase using the random effects model. CI, confidence interval; HR, hazard ratio. B nonlinear association of potato intake and cancer mortality in adults.

potato intake with cancer mortality for each 100-g/d increase in potato intake in higher sample size (>50000) ($P = 0.042$), higher cases (≥200) ($P < 0.001$), higher duration of follow up (>10 y) ($P < 0.001$), both genders ($P = 0.017$) and males ($P < 0.001$), studies without adjustment for BMI ($P < 0.001$) (Table 2). There was no evidence of a nonlinear association (P-nonlinearity = 0.99) (Figure 5B).

Potato intake and CVD mortality

There were only three studies about the association of potato consumption and CVD mortality which were insufficient for performing meta-analysis. Therefore, we could not perform mete-analysis about association of potato intake and CVD mortality.

Discussion

We failed to find significant association between consumption of potatoes and risk of all-cause mortality as well as risk of mortality from cancers and CVDs. In addition, we did not find a nonlinear association between potato consumption and risk of cancer mortality. Moreover, there was no linear dose-response association between each 100-g/d increment in potato intake and risk of all-cause and cancer mortality.

We could not find a significant association between potato consumption and risk of all-cause mortality. A recent meta-analysis did not find a significant association between consumption of potato and risk of all-cause mortality (Schwingshackl et al. 2018). However, only 6 studies were included in that meta-analysis. Moreover, potato consumption was analyzed along with consumption of other tubers in one of the included studies. A systematic review published in 2016, including 5 observational studies, could not provide considerable evidence for association between potato consumption and risk of obesity, type 2 diabetes, and CVD (Borch et al. 2016). However, higher consumption of potato in another observational study was associated with the reduced risk of all-cause mortality (Kahn et al. 1984). It should be noted that the study was old and examined association of potato consumption with mortality risk only at baseline. We did not have enough data to investigate the association for fried potato. Two previous studies in this area have reported controversial findings (Guallar-Castillon et al. 2012; Veronese et al. 2017). These inconsistence findings might be due to the type of oils used for frying (Guallar-Castillon et al. 2012).

We could not find a significant association between consumption of potatoes and risk of cancer mortality. In addition, we did not find a significant dose-response association between potato intake and cancer mortality. To the best of our knowledge, there is no systematic review available investigating the association of potato consumption with risk of cancer mortality. However, a pooled analysis of 20 cohort studies did not show a significant relationship between consumption of potatoes and risk of breast cancer (Jung et al. 2013). In addition, a large cohort study also could not find a significant association between consumption of potatoes and risk of prostate cancer (Diallo et al. 2016). Otherwise, another cohort study showed significantly higher risk of lung cancer mortality in smoker patients with the highest consumption of potatoes than those with the lowest consumption (Skuladottir et al. 2006). However, it should be noted that cigarette smoking reduces insulin-like growth factor binding protein (IGFBP)-3 concentrations in a dose-dependent manner (Kaklamani et al. 1999), which subsequently leads to higher circulating free insulin growth factor-1(IGF-1). Previous studies have shown a significant positive association between serum concentrations of IGF-1 and risk of cancer progression and mortality (Renehan et al. 2004).

We did not have enough data to investigate the association between consumption of each specific type of potato and risk of cancer mortality in this meta-analysis. A previous study conducted by Wilson et al., could not found a significant association between French fries consumption and risk of mortality from prostate cancer (Wilson et al. 2012). Therefore, further studies are needed to investigate association between consumption of these forms of potatoes and risk of cancer mortality.

As there were only three studies on the association of potato consumption with risk of CVD mortality, we could not perform meta-analysis. Among that three studies only one study showed a significant association between potato consumption and risk of CHD mortality (Pietinen et al. 1996). However, the follow up duration of that study was only 6.1 years. Another study which was conducted in 69,313 Swedish men and women, did not show a significant association between consumption of boiled potatoes, fried potatoes, or French fries and risk of CVD and mortality through 13-y follow-up (Larsson and Wolk 2016). In addition, a cohort also could not find a significant association between potato consumption and risk of CHD mortality, after 10 years follow-up (Dilis et al. 2012). Recent systematic review and meta-analysis showed boiled potato consumption is not related to risk for CHD, stroke and hypertension but could increase risk of T2D. Moreover, French-fries consumption could increase risk of T2D and hypertension (Schwingshackl et al. 2018). An Iranian cross-sectional study showed a positive relation between potato consumption, high fasting blood glucose level and diabetes mellitus (Khosravi-Boroujeni et al. 2012). Another cross-sectional study suggested potato consumption could increase obesity but not blood pressure in Iranian adolescents (Heidari-Beni et al. 2015). Previous study showed cardio metabolic factors were associated with CVD mortality (Zhang et al. 2018). These inconsistence findings might be suggested to be due to the different types of potatoes used in those studies. However, available evidences are insufficient to reach a firm conclusion.

Potatoes contain more potassium than other fruits and vegetables and are very low in sodium (USDA 2005) which associated with cardiovascular health (Appel et al. 2006). Potatoes are an important dietary source of vitamin C throughout the world and also provide fiber, magnesium, calcium, vitamin B6, niacin, iron and folate (McGill, Kurilich, and Davignon 2013b; Totland et al. 2012)), which their intakes are related to the reduced risk of several chronic diseases (Camire, Kubow, and Donnelly 2009b; Darooghegi Mofrad et al. 2018). However, dietary glycemic index (GI) and glycemic load (GL) of potato is also high (van Bakel et al. 2009; Wirfalt et al. 2002). Previous studies have associated higher dietary GI and GL to the increased risk of several chronic diseases (Fan et al. 2012; Mirrahimi et al. 2014; Sieri et al. 2015). Moreover, it has been found that dietary patterns rich in potatoes are commonly unhealthy (Veronese et al. 2017). The nutrient content of potatoes is influenced by preparation and cooking methods. Fried potatoes typically contain high amounts of dietary fats, in particular trans-fatty acids, and salts, in which their consumption is related to the increased risk of mortality (Siri-Tarino et al. 2010). Augustin et al. (1978) showed boiling, baking, and microwave method can reduce vitamin C, thiamin, riboflavin, niacin, folic acid, and vitamin B6 in potatoes (Augustin et al. 1978). One study showed that roasting or baking could enhance the availability of potatoes' minerals (Gahlawat and Sehgal 1998). Heating of potatoes converts native starch granules to rapidly digestible starch (RDS). So, boiled potato have high glycemic index than other kinds (García-Alonso and Goni 2000). Therefore, potato consumption might induce both beneficial and harmful influences on health. It seems that the effects of potato consumption on morbidity and mortality are greatly depended on subjects' health condition and type of consumed potato. Therefore, it is recommended to design further studies on the association between different types of potatoes and risk of mortality among patients with different health conditions.

To the best of our knowledge, this is the first systematic review and meta-analysis investigating the association of potato consumption with cardiovascular and cancer mortality. Included studies were longitudinal large scale prospective cohorts with high-quality methodology. In addition, we found low evidence for between study heterogeneity among studies about cancer and all-cause mortality. Moreover, findings were adjusted for a wide range of confounding factors in the included studies. Besides these strengths, several limitations should be kept at mind when interpreting findings. Potato consumption was frequently assessed through dietary patterns in those studies. In addition, information for preparation methods was not available in most cases. Furthermore, a self-administered questionnaire was used to assess dietary intakes of participants in most included studies, which may result in some measurement errors. Moreover, participants were only able to report their potato consumption frequency, but not its portion size. We did not have enough data for each form of potato preparation (e.g., boiled, fried, mashed, baked) and different types of potatoes to perform separate meta-analysis. In addition, the number of studies about the association of potato intake and CVD mortality was not so sufficient to conduct meta-analysis. Dietary intake of other food items was not considered as an important confounding factor in most included studies. The final limitation of the study centers on all-cause mortality as the primary outcome variable. There is no indication of the reason for the mortality. Therefore, the number of subjects considered in both all-cause and other causes may be inaccurate. In conclusion, this study did not find significant association between consumption of potatoes and risk of all-cause, CVD, and cancer mortality. Further randomized controlled clinical trial studies considering consumption of different types of potatoes are needed to confirm these findings.

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Conflict of interest

The authors have no conflict of interest.

Authors' contribution

MDM, LA, AS designed the research. MDA and AM independently done the literature search and screening; MDM analyzed data; MDM and AM wrote the manuscript and AM helped improve English writing; LA and AS revised the manuscript. All authors read and approved the final manuscript.

Abbreviations

BMI	body mass index
CHD	coronary heart disease
CI	confidence intervals
CVD	cardiovascular disease
FFQ	food frequency questionnaire
GI	glycemic index
GL	glycemic load
HR	hazard ratios
IGF-1	insulin growth factor-1
IGFBP-3	insulin-like growth factor binding protein-3
OR	odds ratios
MESH	medical subject headings
NOS	Newcastle-Ottawa scale
PRISMA	preferred reporting items for systematic reviews and meta-analyses
RCT	randomized clinical trials
RR	rate ratio
US	united states

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