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SYSTEMATIC REVIEW AND META-ANALYSIS

Relation of Different Fruit and Vegetable Sources With Incident Cardiovascular Outcomes: A Systematic Review and Meta-Analysis of Prospective Cohort Studies

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BACKGROUND: Public health policies reflect concerns that certain fruit sources may not have the intended benefits and that vegetables should be preferred to fruit. We assessed the relation of fruit and vegetable sources with cardiovascular outcomes using a systematic review and meta-analysis of prospective cohort studies.

METHODS AND RESULTS: MEDLINE, EMBASE, and Cochrane were searched through June 3, 2019. Two independent reviewers extracted data and assessed study quality (Newcastle-Ottawa Scale). Data were pooled (fixed effects), and heterogeneity (Cochrane-Q and I^2) and certainty of the evidence (Grading of Recommendations Assessment, Development, and Evaluation) were assessed. Eighty-one cohorts involving 4 031 896 individuals and 125 112 cardiovascular events were included. Total fruit and vegetables, fruit, and vegetables were associated with decreased cardiovascular disease (risk ratio, 0.93 [95% CI, 0.89–0.96]; 0.91 [0.88–0.95]; and 0.94 [0.90–0.97], respectively), coronary heart disease (0.88 [0.83–0.92]; 0.88 [0.84–0.92]; and 0.92 [0.87–0.96], respectively), and stroke (0.82 [0.77–0.88], 0.82 [0.79–0.85]; and 0.88 [0.83–0.93], respectively) incidence. Total fruit and vegetables, fruit, and vegetables were associated with decreased cardiovascular disease (0.89 [0.85–0.93]; 0.88 [0.86–0.91]; and 0.87 [0.85–0.90], respectively), coronary heart disease (0.81 [0.72–0.92]; 0.86 [0.82–0.90]; and 0.86 [0.83–0.89], respectively), and stroke (0.73 [0.65–0.81]; 0.87 [0.84–0.91]; and 0.94 [0.90–0.99], respectively) mortality. There were greater benefits for citrus, 100% fruit juice, and pommes among fruit sources and allium, carrots, cruciferous, and green leafy among vegetable sources. No sources showed an adverse association. The certainty of the evidence was "very low" to "moderate," with the highest for total fruit and/or vegetables, pommes fruit, and green leafy vegetables.

CONCLUSIONS: Fruits and vegetables are associated with cardiovascular benefit, with some sources associated with greater benefit and none showing an adverse association.

REGISTRATION: URL: https://www.clinicaltrials.gov; Unique identifier: NCT03394339.

Key Words: cardiovascular outcomes ■ cohort ■ fruit ■ nutrition ■ vegetables

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Increased fruit and vegetable consumption is the cornerstone of dietary guidance for cardiovascular disease (CVD) prevention. Their benefit as part of heart healthy diets is balanced against an increasing concern of their contribution to an excess intake of sugars.[1,2] Some influential commentators have even questioned the value of the proverbial "apple a day."[3] Public health outlets are emphasizing vegetables before fruit intake and discouraging the intake of certain sources of fruit, such as fruit

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CLINICAL PERSPECTIVE

What Is New?

- Public health policies discourage the consumption of certain fruit sources (eg, 100% fruit juice, dried fruit, and tropical fruit) because of their sugar content and emphasize vegetable consumption before fruit.
- We examined the relation of fruit and vegetable sources with cardiovascular disease outcomes.

What Are the Clinical Implications?

- In this systematic review and meta-analysis of 81 unique cohorts, we identified that fruits and vegetables are associated with cardiovascular benefit and no fruit or vegetable sources are associated with cardiovascular harm.
- Certain fruit and vegetable sources showed greater associations with cardiovascular benefit, including citrus, 100% fruit juice, and pommes fruit and allium, carrots, and cruciferous and green leafy vegetables.

Nonstandard Abbreviations and Acronyms

GRADE	Grading of Recommendations Assessment, Development, and Evaluation
NOS	Newcastle-Ottawa Scale

juice and dried, tropical, and canned fruit, some of which have been reflected in health policies.[4-8]

Given the longstanding perceived value of fruit and vegetables in reducing global CVD morbidity and mortality[9] and in light of developing efforts to limit dietary sugars, there is a need to reassess the role of different fruit and vegetable sources in CVD prevention. Whether different fruit and vegetable sources show comparable CVD risk reduction is unclear. Systematic reviews and meta-analyses of prospective cohort studies have shown evidence of a cardiovascular benefit of broad categories of fruits and vegetables,[10-16] but the relative contributions of specific fruit and vegetable sources and the certainty of the estimates for these sources are underexplored. We, therefore, conducted a systematic review and meta-analysis of prospective cohort studies using Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach to assess the role of different fruit and vegetable sources in CVD risk reduction and to quantify the certainty of the evidence to inform public health policy.

METHODS

All supporting data are available within the article and its online supplementary files. We followed the *Cochrane Handbook for Systematic Reviews and Interventions*[17] and reported results in accordance with Meta-Analysis of Observational Studies in Epidemiology[16] and Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines.[18] The protocol was registered at Clinicaltrials.gov (identifier, NCT03394339).

Search Strategy

We searched MEDLINE, EMBASE, and the Cochrane Library databases through June 3, 2019, using the search strategy presented in Table S1 and restrictions for prospective cohorts. We supplemented the search with manual searches of the references of included studies.

Study Selection

Prospective cohort studies that reported the association of fruit and/or vegetable intake with CVD, coronary heart disease (CHD), or stroke incidence and mortality with a minimum follow-up time of 1 year in individuals free of disease at baseline were included. Cohorts that presented data on exposures to fruits and vegetables within the context of a dietary index were not included unless fruits and/or vegetables were presented separately from the other components of the diet index.

Data Extraction

Two reviewers (A.Z., F.A.) independently extracted relevant information, including study design, sample size, subject characteristics, exposure, outcomes, assessment method, dose for each quantile, number of events, population, person-years of follow-up, duration of follow-up, covariates adjustments, and risk ratios (RRs; or odds ratios or hazard ratios) with 95% CIs for each quantile of exposure. We contacted authors for missing data. Data on CVD outcomes were extracted for exposures to total fruits and vegetables, fruits, vegetables, and their sources. Potatoes were not included in the present analysis as they are nutritionally classified as a starchy food and are largely omitted in quantifications of exposure to vegetables.

Outcomes

Outcomes were CVD, CHD, and stroke incidence and mortality.

Risk of Bias

Included studies were assessed for risk of bias with the Newcastle-Ottawa Scale (NOS),[19] which awards

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up to 9 points based on cohort selection (up to 4 points), outcome ascertainment (up to 3 points), and degree of covariate adjustments (up to 2 points with adjustment for age as the primary confounding variable awarded 1 point and adjustment for ≥7/9 secondary confounding variables, including sex, family history, smoking, markers of adiposity, energy intake, physical activity, presence of diabetes mellitus, hypertension [or related medications], and dyslipidemia [or related medications]). Studies achieving ≥7 points were considered high quality. Disagreements in NOS score between the 2 reviewers were resolved by a third reviewer (J.L.S.).

Statistical Analysis

Review Manager version 5.3 (The Nordic Cochrane Centre, Denmark) and STATA version 13.0 (StataCorp, TX) were used to conduct all analyses. We prespecified in our analysis plan the use of the generic inverse variance method with DerSimonian and Laird random effects models to pool the natural log-transformed RRs of extreme quantiles, comparing the highest versus the lowest (reference) exposures.[20] On the basis of a deviation from our prespecified analysis plan requested by the statistical reviewer, we present the generic inverse variance with fixed effects models as the primary analysis and the DerSimonian and Laird random effects models as a secondary analysis in the Supplemental Material. Hazard ratios and odds ratios (as cumulative incidence <10%) were considered equivalent to RR.[21] Studies that provided RR on a continuous scale (ie, per dose increment) were scaled to the highest quantile reported for the exposure in the respective cohort as necessary. Test for differences between fruit and vegetable categories were conducted in RevMan, with a test for subgroup differences, with $P<0.05$ indicating a significant difference between fruit categories or vegetable categories on a given outcome. We also conducted a dose-response analysis. A random-effects linear dose-response was modeled using a generalized least square trend (*glst*) for estimation of summarized dose-response data, as per Greenland and Longnecker[22] and Orsini.[23] A 2-stage multivariate random-effects method was used to model a nonlinear association using restricted cubic splines with 3 knots.[23] A Wald test was used to evaluate linear and nonlinear dose-response trends. The median dose of each quantile was used, and when not provided we chose the midpoint of the upper and lower boundaries for each quantile as the assigned dose. For open-ended lower and upper quantiles, we defined lowest and highest boundary as the same as the adjacent category cutoff. Servings per day were calculated, with one serving defined as 80 g of fruits and/or vegetables and their categories, with the exception of citrus fruit (122 g), fruit juice (125 g), and green leafy vegetables (88 g), or unless otherwise specified.[24]

Heterogeneity was assessed by the Cochran Q statistic and quantified by the I^2 statistic. An $I^2 \geq 50\%$ and $P_Q<0.1$ was considered evidence of substantial heterogeneity.[25,26] Sensitivity analyses and a priori subgroup analyses were used to explore sources of heterogeneity. We performed sensitivity analyses by systematically removing each study with recalculation of the summary estimates. A priori subgroup analyses were conducted for all comparisons with ≥10 observations. Subgroup analyses included age (less than median versus median or greater), sex (males, females, and mixed), follow-up years (less than median versus median or greater), number of covariates in extracted model (<8 versus ≥8 covariates), exposure assessment tool (validated Food Frequency Questionnaire [FFQ], unvalidated FFQ, and food record), risk of bias score (<6 versus ≥6), and country of data collection. Wald test in metaregression was used to assess differences within each subgroup. Because of the exploratory intent of our subgroup analyses, we did not prespecify adjustment for the false discovery rate in our prespecified analysis plan. On the basis of a deviation from our prespecified analysis plan requested by the statistical reviewer, we adjust for the false discovery rate in our subgroup analyses using the Holm-Bonferroni procedure. If ≥10 cohort comparisons were available, then publication bias was assessed by visual inspection of funnel plots for asymmetry and formal testing with the Begg and Egger tests. If publication bias was suspected ($P<0.10$), the Duval and Tweedie trim and fill method imputed missing study data in attempt to adjust for funnel plot asymmetry.[27]

Grading the Evidence

The GRADE method was used to assess the certainty of the evidence for each comparison on a 4-point scale, ranging from "very low" to "high."[28-40] Because of their inherent limitations, observational studies start at a "low" certainty of evidence that can be downgraded or upgraded based on established criteria. Criteria to downgrade included risk of bias (weight of studies shows high risk of bias by NOS), inconsistency (substantial unexplained heterogeneity, $I^2>50\%$, and $P_Q<0.10$), indirectness (presence of factors that limit generalizability based on populations, exposures, and outcomes), imprecision (95% CIs cross minimally important difference of 5% [RR, 0.95–1.05]), and publication bias (significant evidence of small study effects). Criteria to upgrade included a large risk estimate (RR <0.5 or >2 in the absence of plausible confounders), a dose-response gradient, and attenuation by plausible confounders.

RESULTS

Flow of the Literature

Figure 1 illustrates a flow of the literature. Of 4271 reports, we included a total of 117 publications[41-156] of 81

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unique prospective cohort studies of 4 031 896 individuals and 125 112 cardiovascular events.

Study Characteristics

The Table shows the characteristics of the included studies.[41-156] Participants were from 69 countries with cohorts distributed worldwide (36 from Europe, 23 from North America, 1 from South America, 17 from Asia, 4 from Australia, and 1 large global cohort including 18 countries worldwide). The median participant age at baseline was 55 (range, 7–90) years with a median follow-up of 11 (range, 2–37) years. Median (range) intakes in servings per day in the highest quantiles were 7.4 (2.6–10.4) fruits and vegetables, 2.6 (0.29–11.0) fruits, 2.85 (0.74–11.0) vegetables, 0.4 (0.3–0.5) bananas, 0.27 (0.13–0.7) berries, 0.71 (0.22–2.2) citrus fruit, 0.82 (0.4–2.28) fruit juice, 0.95 (0.29–2.0) pommes, 2.37 (2.1–2.65) watermelon, 0.54 (0.07–2) allium vegetables, 9.5 (5–14) carrots, 0.43 (0.1–3.0) cruciferous vegetables, 0.71 (0.25–1.5) green leafy vegetables, and 0.63 (0.29–2.0) tomatoes. Doses were not available for apricots and celery. Dietary intake was assessed by self-administered validated food frequency questionnaire (54%), interview administered validated FFQ (10%), unvalidated FFQ (19%), or 24-hour recalls/food records (17%).

Table S2 lists the variables that were statistically adjusted in the included studies. Age, the prespecified primary confounding variable, was adjusted for in 95% of included studies, of which 55% also adjusted for all 9 of the prespecified secondary confounding variables.

Study Quality

Table S3 summarizes the NOS assessment of included studies. There was a high risk of bias in associations

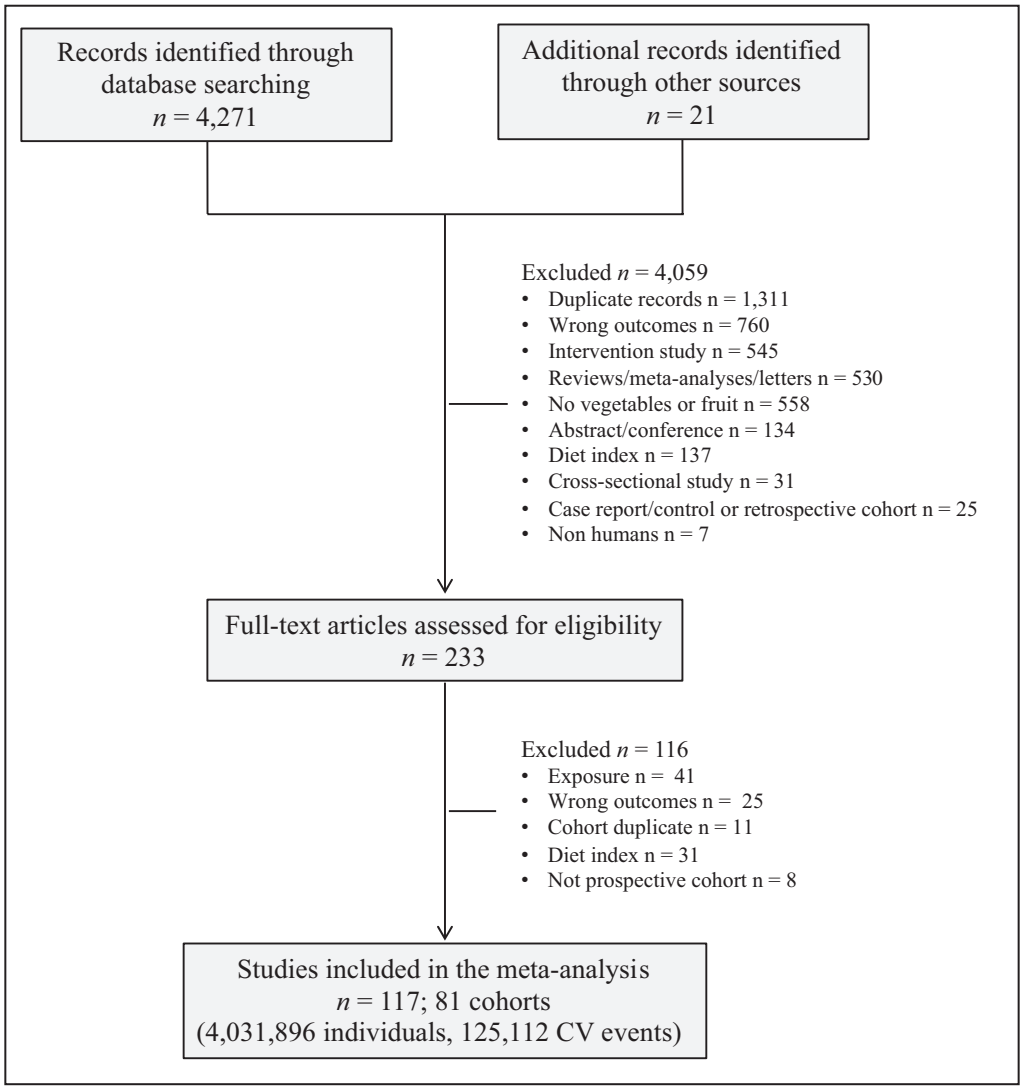


Figure 1. Summary of evidence search and selection.
CV indicates cardiovascular.

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Table. **Table of Study Characteristics**

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Adriouch, 2018[41]	NutriNet-Sante	France	84 158 (17 931:66 227)	44.1±14.5	4.9±1.6	24-h recall	Fruit category	3	CVD incidence CHD incidence Stroke incidence	602 309 293
Appleby, 2002[42]	Health Food Shoppers	United Kingdom	10 741 (4325:6416)	16–89	18–24	Unvalidated FFQ	Fruit	2	CVD mortality CHD mortality Stroke mortality	1202 (591:611) 605 (347:258) 356 (142:214)
Atkins, 2014[43]	British Regional Heart	England	3328 (3328:0)	60–79	11.3	Validated FFQ	Fruit and/or vegetable	2	CVD incidence CVD mortality CHD incidence	582 327 307
Bahadoran, 2017[44]	Theran Lipid and Glucose	Iran	2369 (1047:1322)	≥19	6	Validated FFQ	Vegetable categories	3	CVD risk	79
Bazzano, 2002[45]	National Health and Nutrition Examination Survey Epidemiologic Follow-up Study	United States	9608	25–74	19	Unvalidated FFQ	Fruit and vegetable	4	CVD mortality CVD incidence IHD mortality IHD incidence Stroke mortality Stroke incidence	1145 N/A 639 1786 218 888
Belin, 2011[46]	WHI-OS (Women's Health Initiative Observational Study)	United States	93 676 (0:93 676)	50–79	10	Self-administered validated FFQ	Fruit, vegetable	2	CVD incidence	6006
Bendinelli, 2011[47]	EPIC	Italy	29 689 (0:29 689)	50.0±7.9	7.85	Validated FFQ	Fruit, vegetable, categories	4	CHD incidence	144
Berard, 2017[48]	MONICA	France	1311	35–64	16–18	Food recall	Fruit, vegetable	5	CVD mortality	41
Bhupathiraju, 2013[49]	NHS (Nurses' Health Study) and HPFS (Health Professionals Follow-Up Study)	United States	113 276 (42 135:71 141)	40–75 (men) 30–55 (women)	22 (men) 24 (women)	Validated FFQ	Fruit and/or vegetable, categories	5	CHD incidence	6189 (3607:2582)
Bingham, 2008[50]	EPIC	United Kingdom	11 134	45–75	4	Validated FFQ	Fruit and vegetable	5	IHD risk	678
Blekkenhorst, 2017[51]	PLSAW (Perth Longitudinal Study of Aging Women)	Australia	1226 (0:1226)	75.1±2.7	15	Validated FFQ	Vegetable, categories	Per 5–75 g/d	CVD mortality IHD mortality Stroke mortality	238 128 92
Bos, 2014[52]	Rotterdam Study	The Netherlands	3570 (1405:2165)	69.4±6.3	12.9	Unvalidated FFQ	Fruit and vegetable	3	Stroke risk	545
Buijsse, 2008[53]	Zutphen Elderly Study	The Netherlands	559 (559:0)	65–84	15	Unvalidated FFQ	Vegetable category	Per 1-SD increase	CVD mortality	197

(*Continued*)

Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Buil-Cosiales, 2016[55]	PREDIMED (Prevención con Dleta Mediterránea)	Spain	7216	55–80	6	Validated FFQ	Fruit, vegetable, categories	5	CVD composite score CVD mortality MI incidence Stroke incidence	342 104 118 169
Buil-Cosiales, 2017[54]	SUN (Seguimiento University of Navarra)	Spain	17 007 (6633:10 374)	38	10.3	Validated FFQ	Fruit, vegetable, categories	5	CVD incidence	112
Cassidy, 2012[56]	NHS	United States	69 622 (0:69 622)	30–55	14	Validated FFQ	Fruit, vegetable	5	Stroke incidence	1803
Collin, 2019[57]	REGARDS (Reasons for Geographic and Racial Differences in Stroke)	United States	13 440 (7972:5469)	≥45	6±1.8	Validated FFQ	Fruit category	12 oz/d	CHD mortality	168
Conrad, 2018[58]	NHANES	United States	29 133 (13 926:15 207)	46.3 (95% CI, 45.8–46.7)	6.5	24-h recall	Vegetable	3	CVD mortality CHD mortality	726 556
Dauchet, 2004[59]	PRIME	France, North Ireland	8087 (8087:0)	50–59	5	Interview	Fruit category	3	CHD event	133
Dauchet, 2010[60]	PRIME	France, North Ireland	8060 (8060:0)	50–59	10	Interview-validated FFQ	Fruit and/or vegetable	3	CVD risk Acute coronary syndrome	612 367
Du, 2016[61]	China Kadoorie Biobank	China	451 665 (186 086:265 579)	50.5±10.4	N/A, ≈7.14 y	Interview unvalidated FFQ	Fruit	5	Acute coronary event Hemorrhagic stroke event Other CeVD events Ischemic stroke	2551 14 579 11 054 3523
Du, 2017[62]	China Kadoorie Biobank	China	462 342 (189 560:272 782)	51±10.5	≈7	Interview unvalidated FFQ	Fruit	4	CVD mortality IHD mortality Ischemic stroke mortality Hemorrhagic stroke mortality	6166 2038 585 2351
Elwood, 2013[63]	Carphilly Cohort Study	United Kingdom	2235 (2235:0)	45–59	30	Unvalidated FFQ	Fruit and vegetable	2	CVD incidence	N/A
Eriksen, 2015[64]	SABRE (Southhall and Brent Revised)	United Kingdom	2096	40–69	21	Validated FFQ	Fruit, vegetable	2	CVD incidence CHD incidence	571 520
Fitzgerald, 2012[65]	Women's Health Study	United States	34 827 (0:34 827)	55 (46–68) (mean [95% CI])	14.6	Validated FFQ	Fruit, vegetable	5	CVD risk	1094

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Fraser, 1992[66]	Adventis Health Study	United States	26 473 (10 003:16 740)	Men: 51.3±16.0 Women: 53.2±16.6 (mean±SD)	6	Validated FFQ	Fruit	3	CHD mortality CHD event	463 134
Gardener, 2011[67]	NOMAS (Northern Manhattan Study)	United States	2568 (924:1644)	69±10 (Mean±SD)	9	Interview validated FFQ	Fruit, vegetable	Continuous	CVD mortality CVD incidence MI incidence Ischemic stroke incidence	314 518 133 171
Gaziano, 1995[68]	Massachusetts Health Care Panel Study	United States	1299 (494:805)	≥66	4.75	Unvalidated FFQ	Fruit and vegetable categories	2	CVD mortality	161
Genkinger, 2004[69]	Odyssey	United States	6151 (2276:3875)	30–93	13	Validated FFQ	Fruit, vegetable categories	5	CVD mortality	378
Gillman, 1995[70]	Framingham Study	United States	832 (832:0)	45–65	18–22	24-h recall	Fruit and vegetable	5	Stroke mortality Stroke incidence	14 97
Goetz, 2016[71]	REGARDS	United States	16 678	≥45	6.0±1.9	Validated FFQ	Fruit categories	5	CHD events	589
Goetz, 2016[72]	REGARDS	United States	20 024 (9011:11 013)	≥45	6.5	Validated FFQ	Fruit, vegetable	5	Stroke incidence	524
Gunge, 2017[73]	Danish Diet, Cancer and Health Cohort	Denmark	57 053 (25 759:28 809)	50–64	13.6	Validated FFQ	Fruit and vegetable categories	2	MI incidence	2322 (1669:653)
Gunnell, 2013[74]	Health and Wellbeing Surveillance System	Australia	14 890 (6114:8776)	45–97	6	Validated FFQ	Fruit and vegetable	2	IHD hospitalization	538
Hansen, 2010[76]	Danish Diet, Cancer and Health	Denmark	53 383 (25 065:28 318)	50–64	7.7	Validated FFQ	Fruit, vegetable categories	4	Acute coronary syndrome	1075 (820:255)
Hansen, 2017[75]	Danish Diet, Cancer and Health	Denmark	55 338	50–64	13.5	Validated FFQ	Fruit and vegetable categories	2	Stroke incidence	2283
Harriss, 2007[77]	Melbourne Collaborative	Australia	40 653 16 673:23 980	40–69	10.4	Validated FFQ	Fruit, vegetable	4	CVD mortality IHD mortality	697 407
Hertog, 1997[78]	Caerphilly Prospective Study	South Wales	1900 (1900:0)	45–59	14.6	Validated FFQ	Vegetable categories	4	IHD mortality	131
Hirvonen, 2000[80]	Finnish Male Smokers in the ATBC Study	Finland	26 497 (26 497:0)	50–69	6.1	Validated FFQ	Fruit category	4	Cerebral infarction Subarachnoid hemorrhage Intracerebral hemorrhage	736 83 95

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Hirvonen, 2001[79]	Finnish Male Smokers in the ATBC Study	Finland	25, 373 (25, 373:0)	50–69	6.1	Validated FFQ	Fruit, vegetable, categories	5	CHD mortality MI event	815 1122
Hjartaker, 2015[81]	Migrant Study	Norway	9766 (9766:0)	42–73	20.3	Unvalidated FFQ	Fruit and/ or vegetable, categories	4	CVD mortality CHD mortality Stroke mortality	4595 2386 1034
Hodgson, 2016[82]	Australian Women aged 70–85 y	Australia	1456 0:1456	>70	15	Validated FFQ	Fruit category	3	CVD mortality	235
Holmberg, 2009[83]	Swedish National Farm Register	Sweden	1738 (1738:0)	50±6.0	12	Unvalidated FFQ	Fruit and vegetable	2	CHD incidence	138
Iso, 2007[84]	Japan Collaborative Cohort	Japan	N/A	40–79	N/A	Validated FFQ	Fruit or vegetable categories	3	IHD mortality CeVD mortality	N/A N/A
Jacques, 2015[85]	Framingham Offspring	United States	2880 (1302:1578)	28–62 (mean=54)	14.9	Validated FFQ	Fruit categories	3	CVD incidence CHD incidence	518 261
Johnsen, 2003[86]	Danish Diet, Cancer and Health	Denmark	54 506	50–64	3.09	Validated FFQ	Fruit and/or vegetable	5	Stroke incidence	266
Joshipura, 1999[87]	NHS and HPFS cohorts	United States	114 279 (38 683:75 596)	30–55 (men) 40–75 (women)	8 (men) 14 (women)	Validated FFQ	Fruit and/ or vegetable, categories	5	Ischemic stroke incidence	570 (366:204)
Joshipura, 2009[88]	NHS and HPFS cohorts	United States	109 788 (38 918:70 870)	30–55 (men) 40–75 (women)	14–16	Validated FFQ	Fruit and/ or vegetable, categories	5	CVD incidence	3892
Keli, 1996[89]	Zutphen Elderly	The Netherlands	552 (552:0)	50–69	15	Interview	Fruit, vegetable categories	3	Stroke risk	42
Kim, 2013[90]	British Women's Heart and Health Study	United Kingdom	3080 (0:3080)	60–79	7	Unvalidated FFQ	Fruit	2	CVD incidence	329
Knekt, 1994[93]	Finnish Mobile Clinic Health	Finland	5133 (2748:2385)	30–69	14	Interview unvalidated FFQ	Fruit, vegetable	3	CHD mortality	244 (186:58)
Knekt, 1996[92]	Finnish Mobile Clinic Health	Finland	5133 (2748:2385)	30–69	26	Interview unvalidated FFQ	Fruit or vegetable categories	4	CHD death	473 (324:149)
Knekt, 2000[91]	Finnish Mobile Clinic Health	Finland	9208	≥15	28	Interview unvalidated FFQ	Fruit or vegetable categories	5	CeVD incidence	824
Kobylecki, 2015[94]	Copenhagen City Heart	Denmark	78 527	20–100	10	Self-reported unvalidated FFQ	Fruit and vegetable	3	IHD incidence	2823
Kondo, 2019[95]	NIPPON DATA80	Japan	9115 (4002:5113)	30–79	29	3-d food record	Fruit or vegetable	3	CVD mortality	1070
Kvaavik, 2010[96]	Health and Lifestyle Survey	United Kingdom	4866 (2509:2377)	43.7±16.3	20	Interview Unvalidated FFQ	Fruit and vegetable	2	CVD mortality	431

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Lai, 2015[97]	UK Women's Cohort	United Kingdom	30 458 (0:30 458)	35–69	16.7	Validated FFQ	Fruit, categories	5–6	CVD mortality CHD mortality Stroke mortality	286 138 148
Larsson, 2009[98]	Finnish Male Smokers in the ATBC Study	Finland	26 556 (26 556:0)	50–69	13.6	Validated FFQ	Fruit, vegetable	5	Stroke incidence	2702
Larsson, 2013[99]	Swedish Mammography and Swedish Men Cohorts	Sweden	74 961 40 291:34 670	45–83	10.2	Validated FFQ	Fruit and/or vegetable, categories	5	Stroke incidence	4089
Leenders, 2013[101]	EPIC	Europe (10 countries)*	451 151 129 882:321 269	25–70	12.8 (median)	Validated FFQ and 7-d food record	Fruit and/or vegetable	4	CVD mortality	5125
Leenders, 2014[100]	EPIC	Europe (10 countries)*	451 151 (129 882:321 269)	25–70	13	Validated FFQ and 7-d food record	Fruit and/or vegetable	4	CHD mortality Stroke mortality	2139 1291
Lin, 2007[102]	NHS	United States	66 360 (0:66 360)	30–55	12	Validated FFQ	Fruit, vegetable, categories	5	CHD mortality MI event	324 938
Lin, 2017[103]	Survey of Health & Living Status of the Elderly	Taiwan	4176	≥50	11	Interview FFQ	Fruit and vegetable	2	CVD mortality	N/A
Liu, 2000[105]	Women's Health Study	United States	39 127 (0:39 127)	45–89	5	Validated FFQ	Fruit and/or vegetable	5	CVD incidence MI event	418 126
Liu, 2001[104]	The Physician's Health Study	United States	15 520 (15 520:0)	40–84	6	Validated FFQ	Vegetable	5	CHD incidence	1148
Mann, 1997[106]	The Oxford Vegetarian Study	United Kingdom	10 802 (4102:6700)	16–79	13.3	Validated FFQ	Fruit, vegetable, categories	3	IHD mortality	64
Manuel, 2015[107]	Canadian Community Health Survey	Canada	82 259 (37 483:44 746)	20–83 (men: 48.2; women: 49.4)	8.6	Interview FFQ	Fruit and vegetable	3	Stroke incidence	1551
Miller, 2017[108]	PURE (Prospective Urban and Rural Epidemiology)	18 Countries†	135 335	35–70	7.4 (Median)	Validated FFQ	Fruit and/or vegetable	4	CVD events MI Stroke Cardiovascular mortality	4784 N/A N/A N/A
Mink, 2007[109]	Iowa Women's Health	United States	34 492 (0:34 492)	55–69	16	Validated FFQ	Fruit or vegetable categories	3	CVD mortality CHD mortality Stroke mortality	2316 1329 469

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Mizrahi, 2009[110]	Finnish Mobile Clinic Health Examination Survey	Finland	3932	40–74	24	Interview	Fruit, vegetable, categories	4	Stroke risk	625
Mori, 2018[111]	Japan Public Health Center Based Prospective Study	Japan	88 184 (40 622:47 562)	45–74	16.9	Validated FFQ	Vegetable categories	5	CHD mortality Stroke mortality	1968 (1192:776) 1470 (856:614)
Mytton, 2018[112]	EPIC-Norfolk	England	22 992 (10 002:12 990)	40–79	16.4	7-d food record	Fruit and vegetable	5	CVD incidence	4965
Nagura, 2009[113]	Japan Collaborative	Japan	59 485 (25 206:34 279)	40–79	12.7	Validated FFQ	Fruit, vegetable	4	CVD mortality CHD mortality Stroke mortality	2243 (1207:1036) 452 (258:194) 1053 (559:494)
Nakamura, 2008[114]	Takayama	Japan	29 079 (13 355:15 724)	≥35 (men: 54.0; women: 55.1)	7.33	Validated FFQ	Fruit, vegetable	4	CVD mortality	384 (200:184)
Nechuta, 2010[115]	Shanghai Women's Health	China	71 243 (0:71 243)	40–70	9	Interview Validated FFQ	Fruit and vegetable	Daily	CVD mortality	775
Neelakantan, 2018[116]	Singapore Chinese Health Study	China	57 078	45–74	17	Validated FFQ	Fruit or vegetable	1 Serving/d	CVD mortality	4871
Ness, 2005[117]	Boyd Orr Cohort	United Kingdom (England and Scotland)	4028 (1995:2033)	3.5–11.2	37	Household survey	Fruit, vegetable	4	CHD mortality Stroke mortality	298 83
Nothlings, 2008[118]	EPIC	Europe (10 countries)†	10 262	35–70	9	Validated FFQ	Fruit or vegetable	80 g/d	CVD mortality	517
Okuda, 2015[119]	NIPPON DATA80	Japan	9112 (4000:5112)	30–79	24	Household survey	Fruit and/or vegetable	4	CVD mortality CHD mortality Stroke mortality	823 165 385
Oude Griep, 2010[120]	MORGEN	The Netherlands	19 819	20–59	10.5	Validated FFQ	Fruit, vegetable	4	CHD incidences	245
Oude Griep, 2011[122]	MORGEN	The Netherlands	20 069 (8988:11 081)	20–68	10.3	Validated FFQ	Fruit and vegetable	4	Stroke incidence	233
Oude Griep, 2011[121]	MORGEN	The Netherlands	20 069 (8989:11 081)	42±11	10.5	Validated FFQ	Fruit or vegetable categories	3–4	CHD incidence	245
Oyebode, 2014[123]	HSE (Health Survey for England)	England	65 226 (28 960:36 266)	56.6±14.3	7.7	24-h recall	Fruit and/or vegetable	4	CVD mortality	1554
Pham, 2007[124]	Miyako Study	Japan	9651 (4254:5397)	Men: 56.5±10.63; women: 57.4±10.89 (mean±SD)	13.8	Questionnaire	Fruit, vegetable	2	Stroke mortality	226
Rebello, 2014[125]	Singapore Chinese Health Study	China	53 469 (23 501:29 968)	45–7	15	Interview validated FFQ	Fruit and vegetable	5	IHD mortality	1660 (1022:638)

(*Continued*)

Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Rissanen, 2003[126]	Kuopio Ischaemic Heart Disease Risk Factor	Finland	1950 (1950:0)	42–60	12.8	4-d food record	Fruit and vegetable	5	CVD mortality	115
Saglimbene, 2017[127]	DIET-HD	Europe and South America	9757	N/A	1.5	Validated FFQ	Fruit, categories	2	CVD mortality	N/A
Sahyoun, 1996[128]	Nutrition Status Study	United States	680	60–101	9–12	3-d food record	Fruit, vegetable, categories	3	CHD mortality	101
Sauvaget, 2003[129]	Life Span Study	Japan	39 337 (14 966:23 471)	34–103	16	Validated FFQ	Fruit, vegetable, categories	3	Stroke mortality	1926 (692:1234)
Scheffers, 2019[130]	EPIC Netherlands and MORGEN	The Netherlands	34 560 (25 574:8986)	20–69	14.6	Validated FFQ	Fruit and categories	5	CVD incidence CHD incidence Stroke incidence	3801 2135 1135
Sesso, 2003[131]	WHS (Women's Health Study)	United States	38 445 (0:38 445)	45–89	6.9	Validated FFQ	Fruit or vegetable categories	4	CVD incidence	729
Sesso, 2003[133]	WHS	United States	38 445 (0:38 445)	≥45	7.2	Validated FFQ	Vegetable categories	5	CVD incidence MI incidence Stroke incidence	729 201 247
Sesso, 2007[132]	WHS	United States	38 176 (0:38 176)	54.5	10.1	Validated FFQ	Fruit category	4	CVD mortality CVD incidence MI incidence Stroke incidence	204 1004 289 339
Shah, 2018[134]	Cooper Center Longitudinal Study	United States	11 376 (8577:2799)	47	18	3-d food record	Fruit or vegetable	Continuous	CVD mortality	249
Sharma, 2013[135]	Multi Ethnic Cohort	United States	174 028 (78 410:95 618)	45–75	7.5	Validated FFQ	Fruit, vegetable	5	Stroke mortality	860 (434:426)
Sharma, 2014[136]	Multi Ethnic Cohort	United States	164 617 (72 866:91 751)	45–75	5–8	Validated FFQ	Fruit, vegetable	5	IHD mortality	1951 (1140:811)
Simila, 2013[137]	ATBC	Finland	21 955 (21 955:0)	50–69	19	Validated FFQ	Fruit, fruit juices	Daily	CHD risk	4379
Sonestedt, 2015[138]	Malmo Diet and Cancer	Sweden	26 445 (10 048:16 397)	44–74	14	Validated FFQ	Fruit, vegetable	5	CVD incidence CHD incidence Stroke incidence	2921 N/A N/A
Sotomayor, 2018[139]	Renal Transplant Recipients	The Netherlands	400 (217:183)	52±12	7.2	Unvalidated FFQ	Fruit	3	CVD mortality	49
Steffen, 2003[140]	ARIC (Atherosclerosis Risk in Communities)	United States	11 940 (5271:6669)	45–64 (men: 54.4±5.7; women: 54.1±5.7)	11	Interview validated FFQ	Fruit and vegetable	5	CHD incidence Ischemic stroke incidence	535 214

(*Continued*)

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Stefler, 2016[141]	HAPIEE (Health, Alcohol and Psychosocial Factors in Eastern Europe)	Poland, Russia, Czech Republic	19 263	57	7.1	Validated FFQ	Fruit and/or vegetable	4	CVD mortality CHD mortality Stroke mortality	438 226 109
Strandhagen, 2000[142]	Men Born in 1913	Sweden	730 (730:0)	54	26	Interview unvalidated FFQ	Fruit, vegetable	5	CVD mortality CVD incidence	226 209
Takachi, 2008[143]	Japan Public Health Center Based Prospective Study	Japan	77 891 (35 909:41 982)	45–74	5.9	Validated FFQ	Fruit and/or vegetable, categories	4	CVD incidence	1386 (830:556)
Tanaka, 2013[144]	Japan Diabetes Complications Study	Japan	1414	40–70	8.1 (Median)	Validated FFQ	Fruit and vegetable	4	CHD incidence Stroke incidence	96 68
Tognon, 2014[145]	MONICA	Denmark	1849 (901:948)	30–59	11	Food record	Fruit, vegetable	2	CVD mortality CVD incidence MI mortality MI incidence Stroke mortality Stroke incidence	223 755 64 161 40 167
Tucker, 2005[146]	Baltimore Longitudinal Study of Aging	United States	501 (501:0)	34–80	18	7-d food record	Fruit and/or vegetable	2	CHD mortality	71
Von Ruesten, 2013[147]	EPIC	Germany	23 531 (9098:14 433)	35–65	8	Validated FFQ	Fruit, vegetable, categories	Daily	CVD incidence	363
Vormund, 2015[148]	MONICA	Switzerland	17 861 (8663:9198)	16–92	21.4 (Mean)	24-h recall	Fruit, vegetable	Daily	CVD mortality	1385
Wang, 2016[149]	Linxian Nutrition Intervention Trials	China	2455 (1105:1340)	40–69	19–26	Unvalidated FFQ	Fruit and/or vegetable, categories	2	CHD mortality Stroke mortality	355 (men) 452 (women)
Watkins, 2000[150]	CPS-11 (Cancer Prevention Study 11)	United States	1 063 023 (453 962:609 061)	≥30	7	Unvalidated FFQ	Vegetable		CHD mortality	13 761 (9156:4605)
Whiteman, 1999[151]	OXCHECK	United Kingdom	10 522 (4929:5593)	35–64	9	Unvalidated FFQ	Fruit, green vegetables	2–3	IHD mortality	144
Yamada, 2011[152]	Jidni Medical School Cohort	Japan	10 623 (4147:6476)	N/A	10.7	Validated FFQ	Fruit category	5	CVD event MI event Stroke event	758 (270:488) 565 (383:182) 99 (76:23)
Yokoyama, 2000[153]	Shibata Study	Japan	2121 (880:1241)	≥40	20	Unvalidated FFQ	Fruit, vegetable	3	Stroke incidence	196 (91:105)

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Table. Continued

Study	Cohort	Country	Participants (Men:Women)	Age, y	Follow-Up, y	Dietary Assessment	Exposure	Quantiles	Outcomes	Incidence (Men:Women)
Yoshizaki, 2019[154]	Japan Public Health Centre Based Prospective Study	Japan	16 498 (7726:8772)	45–74	14	Validated FFQ	Fruit and/or vegetable	3	CHD incidence Stroke incidence	839 197
Yu, 2014[155]	Shanghai Men and Women's Health Study	China	122 635 (55 424:67 211)	40–74	5.4–9.8	Interview validated FFQ	Fruit and/or vegetable, categories	4	CHD incidence	365 (217:148)
Zhang, 2011[156]	Shanghai Men and Women's Health Study	China	134 796 (61 436:73 360)	40–74 40–70	4.5 10.2	Interview validated FFQ	Fruit, vegetable, and categories	5	CVD mortality	5393 (1951:3442)
Zhang, 2011[157]	MONICA	Finland	36 686 (17 287:19 399)	25–74	13.7	24-h recall	Fruit, veg	4	Stroke incidence	1478

ATBC, alpha-tocopherol, beta-carotene cancer prevention; CeVD indicates cerebrovascular disease; CHD, coronary heart disease; CVD, cardiovascular disease; DIET-HD, dietary intake, death and hospitalization in adult with early stage kidney disease treated with hemodialysis; EPIC, European Prospective Investigation Into Cancer and Nutrition; FFQ, Food Frequency Questionnaire; IHD, ischemic heart disease; MI, myocardial infarction; MONICA, monitoring of trends and determinants in cardiovascular disease; MORGEN, monitoring project on risk factors for chronic diseases; N/A, Not Available; NHANES, National Health and Nutrition Examination Survey; NIPPON DATA80, National Integrated Project for Prospective Observation of non-communicable disease and its trends in aged; and OXCHECK, Oxford and Collaborators health check.

*The EPIC cohort represented the following countries: France, Germany, Greece, Italy, the Netherlands, Spain, United Kingdom, Sweden, Denmark, and Norway.

†The PURE cohort represented the following countries: high income: Canada, Sweden, United Arab Emirates; middle income: Argentina, Brazil, Chile, China, Colombia, Iran, Malaysia, Poland, South Africa, Turkey, Occupied Palestinian Territory; low income: Bangladesh, India, Pakistan, Zimbabwe.

between fruit juice, cruciferous, green leafy, and tomato vegetables, and CHD and stroke mortality and citrus and stroke mortality as >35% of the pooled risk estimate was derived from Iso et al,[84] which was scored 5 on the NOS. The association between apricots and CVD mortality was derived from one study, Saglimbene et al,[158] which was scored 1 on the NOS. Although most studies had scores reduced because of self-administered ascertainment of exposure, 88% of studies received a total score ≥6, which was considered high quality.

Cardiovascular Disease

CVD Incidence

Figure 2 and Figures S1 through S11 show the relation of total and specific fruit and vegetables with CVD incidence. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.93 [95% CI, 0.89–0.96], no significant heterogeneity), fruits (RR, 0.91 [95% CI, 0.88–0.95], no significant heterogeneity), and vegetables (RR, 0.94 [95% CI, 0.90–0.97], no significant heterogeneity). Figures S12 and S13 summarize the relation of sources of fruit or vegetables with CVD incidence. A significant interaction by fruit source was observed (P<0.001), with significant associations with lower risk limited to citrus (RR, 0.88 [95% CI, 0.80–0.86], no significant heterogeneity) and pommes (RR, 0.76 [95% CI, 0.66–0.88], no significant heterogeneity). We found no significant associations from the highest versus lowest intakes of berries (RR, 1.27 [95% CI, 0.95–1.71], heterogeneity not applicable) and juice (RR, 1.00 [95% CI, 0.93–1.07], no significant heterogeneity) fruit. No interaction by vegetable source was observed (P=0.227).

CVD Mortality

Figure 3 and Figures S14 through S30 show the relation of total and specific fruit and vegetables with CVD mortality. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.89 [95% CI, 0.85–0.93], substantial heterogeneity [I^2=68%, P<0.001]), fruits (RR, 0.88 [95% CI, 0.86–0.91], substantial heterogeneity [I^2=79%, P<0.001]), and vegetables (RR, 0.87 [95% CI, 0.85–0.90], substantial heterogeneity [I^2=59%, P<0.001]). Figures S31 and S32 summarize the association of sources of fruits or vegetables with CVD mortality. A significant interaction by fruit (P=0.001) and vegetable sources (P<0.001) was observed with significant associations with lower risk limited to pommes fruit (RR, 0.86 [95% CI, 0.80–0.92], no significant heterogeneity) and to allium (RR, 0.33 [95% CI, 0.22–0.49], heterogeneity not applicable), cruciferous (RR, 0.85 [95% CI, 0.82–0.89], no significant heterogeneity), and green leafy (RR, 0.87 [95% CI, 0.81–0.94], substantial heterogeneity [I^2=88%, P<0.001]) vegetables. There was

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Comparison	Cohorts	N	Events	Pooled Effect Estimates RR [95% CIs]	RR [95% Cis]	Heterogeneity I^2	*p* -value	*p*-value	GRADE
CVD INCIDENCE									
Fruit and Vegetables	**12**	**501,744**	**24,310**	**0.93 [0.89, 0.96]**		**26%**	**0.16**	**<0.001**	●●○○ **LOW**
Fruit	**17**	**577,323**	**27,205**	**0.91 [0.88, 0.95]**		**40%**	**0.03**	**<0.001**	●●●○ **MODERATE**
Apricots	-	-	-	-		-	-	-	- -
Bananas	-	-	-	-		-	-	-	- -
Berries	1	38,176	1,004	1.27 [0.95, 1.71]		-	-	0.110	●○○○ VERY LOW
Citrus	6	222,525	6,220	0.88 [0.80, 0.96]		33%	0.16	0.003	●●○○ LOW
Dried	-	-	-	-		-	-	-	- -
Grapes	-	-	-	-		-	-	-	- -
Juice	5	167,879	8,056	1.00 [0.93, 1.07]		0%	0.42	0.950	●○○○ VERY LOW
Pommes	5	149,437	2,578	0.76 [0.66, 0.88]		0%	0.94	<0.001	●●○○ LOW
Watermelon	-	-	-	-		-	-	-	- -
Vegetables	**14**	**539,683**	**22,810**	**0.94 [0.90, 0.97]**		**34%**	**0.07**	**<0.001**	●○○○ **VERY LOW**
Allium	2	40,814	808	0.79 [0.57, 1.10]		85%	0.01	0.160	●○○○ VERY LOW
Carrots	-	-	-	-		-	-	-	- -
Celery	-	-	-	-		-	-	-	- -
Cruciferous	7	273,878	6,824	0.99 [0.90, 1.08]		52%	0.04	0.780	●○○○ VERY LOW
Green Leafy	5	211,902	5,732	0.87 [0.76, 0.99]		42%	0.12	0.003	●●○○ LOW
Tomatoes	2	55,452	841	0.97 [0.78, 1.20]		9%	0.35	0.770	●○○○ VERY LOW
CHD INCIDENCE									
Fruit and Vegetables	**19**	**619,182**	**17,987**	**0.88 [0.83, 0.92]**		**17%**	**0.24**	**<0.001**	●●●○ **MODERATE**
Fruit	**18**	**1,170,021**	**23,856**	**0.88 [0.84, 0.92]**		**12%**	**0.30**	**<0.001**	●●●○ **MODERATE**
Apricots	-	-	-	-		-	-	-	- -
Bananas	1	122,635	365	0.76 [0.56, 1.02]		29%	0.24	0.070	●○○○ VERY LOW
Berries	4	100,296	2,233	0.94 [0.82, 1.09]		74%	<0.01	0.420	●○○○ VERY LOW
Citrus	10	364,978	8,333	0.91 [0.85, 0.98]		0%	0.70	0.009	●●○○ LOW
Dried	-	-	-	-		-	-	-	- -
Grapes	1	66,360	938	1.13 [0.78, 1.64]		-	-	0.530	●○○○ VERY LOW
Juice	4	109,898	7,589	0.99 [0.92, 1.07]		0%	0.61	0.770	●○○○ VERY LOW
Pommes	8	371,684	4,866	0.90 [0.84, 0.97]		25%	0.22	0.005	●○○○ VERY LOW
Watermelon	1	122,635	365	0.87 [0.64, 1.18]		0%	0.36	0.370	●○○○ VERY LOW
Vegetables	**18**	**696,330**	**17,172**	**0.92 [0.87, 0.96]**		**53%**	**<0.01**	**<0.001**	●●○○ **LOW**
Allium	5	210,964	1,734	0.93 [0.80, 1.09]		20%	0.29	0.390	●○○○ VERY LOW
Carrots	-	-	-	-		-	-	-	- -
Celery	-	-	-	-		-	-	-	- -
Cruciferous	8	347,453	9,383	1.01 [0.95, 1.07]		0%	0.48	0.710	●●○○ LOW
Green Leafy	5	170,250	6,696	0.82 [0.76, 0.89]		40%	0.14	<0.001	●●●○ MODERATE
Tomatoes	3	134,494	1,283	0.80 [0.57, 1.13]		0%	0.45	0.200	●○○○ VERY LOW
STROKE INCIDENCE									
Fruit and Vegetables	**14**	**532,667**	**11,091**	**0.82 [0.77, 0.88]**		**37%**	**0.07**	**<0.001**	●●●○ **MODERATE**
Fruit	**18**	**987,993**	**43,702**	**0.82 [0.79, 0.85]**		**34%**	**0.06**	**<0.001**	●●●○ **MODERATE**
Apricots	-	-	-	-		-	-	-	- -
Bananas	-	-	-	-		-	-	-	- -
Berries	4	143,662	5,967	1.03 [0.94, 1.13]		50%	0.08	0.470	●○○○ VERY LOW
Citrus	8	225,613	7,142	0.88 [0.82, 0.94]		51%	0.04	<0.001	●●○○ LOW
Dried	-	-	-	-		-	-	-	- -
Grapes	-	-	-	-		-	-	-	- -
Juice	4	148,839	1,705	0.82 [0.68, 0.99]		73%	0.02	0.040	●○○○ VERY LOW
Pommes	5	230,881	7,657	0.89 [0.84, 0.95]		0%	0.51	<0.001	●●●○ MODERATE
Watermelon	-	-	-	-		-	-	-	- -
Vegetables	**16**	**564,531**	**13,607**	**0.88 [0.83, 0.93]**		**50%**	**<0.01**	**<0.001**	●●●○ **MODERATE**
Allium	2	84,169	4,912	0.89 [0.80, 0.99]		0%	0.70	0.030	●○○○ VERY LOW
Carrots	-	-	-	-		-	-	-	- -
Celery	-	-	-	-		-	-	-	- -
Cruciferous	6	255,726	7,706	0.98 [0.91, 1.05]		62%	0.02	0.490	●○○○ VERY LOW
Green Leafy	4	196,456	4,798	0.88 [0.79, 0.98]		0%	0.42	0.020	●●○○ LOW
Tomatoes	1	38,445	247	0.20 [0.05, 0.82]		-	-	0.030	●●○○ LOW

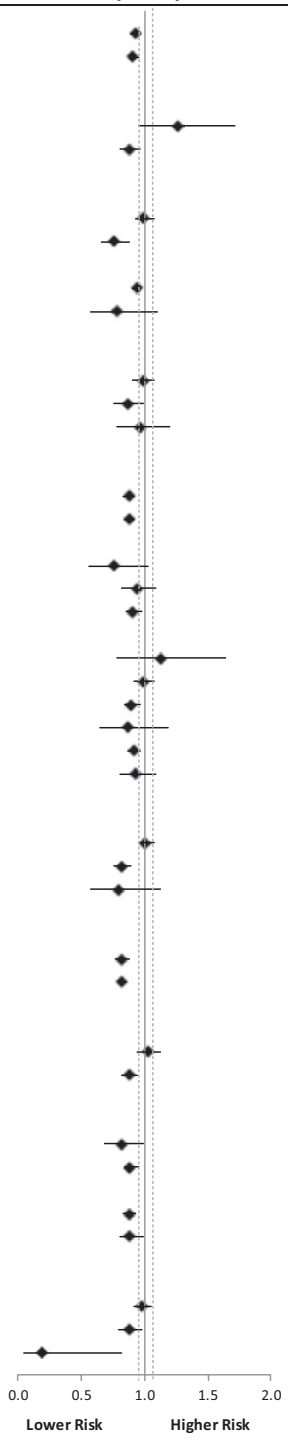


Figure 2. Relation between intake of fruits and vegetables and total incident cardiovascular disease (CVD) (highest vs lowest level of intake).

Pooled risk estimates are represented by the black diamond, with principal exposures highlighted in bold. Principal exposures (fruits and vegetables, fruits, and vegetables) represent the pooled data of the risk estimates reported for these exposures and were not tabulated by pooling fruit and vegetable varieties. Values of $I^2 \geq 50\%$ indicate substantial heterogeneity, with significance at $P>0.10$. The mean important difference of 5% change in relative risk, indicating a clinically relevant association with lower or higher risk, is indicated by the dashed gray lines. CHD indicates coronary heart disease; GRADE, Grading of Recommendations Assessment, Development, and Evaluation; and RR, risk ratio.

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Comparison	Cohorts	N	Events	Pooled Effect Estimates RR [95% CIs]	Heterogeneity I^2	Heterogeneity *p*-value	*p*-value	GRADE
CVD MORTALITY								
Fruit and Vegetables	**14**	**798,391**	**17,439**	**0.89 [0.85, 0.93]**	**68%**	**<0.01**	**<0.001**	●●○○ LOW
Fruit	**25**	**1,581,506**	**39,623**	**0.88 [0.86, 0.91]**	**79%**	**<0.01**	**<0.001**	●●○○ LOW
Apricots	1	9,757	515	1.84 [1.27, 2.67]	-	-	0.001	●○○○ VERY LOW
Bananas	1	9,766	4,595	1.06 [0.87, 1.29]	-	-	0.550	●○○○ VERY LOW
Berries	4	112,892	7,401	0.97 [0.92, 1.03]	15%	0.32	0.300	●○○○ VERY LOW
Citrus	3	74,716	7,197	0.95 [0.90, 1.02]	62%	0.05	0.150	●○○○ VERY LOW
Dried	2	31,757	447	0.93 [0.63, 1.37]	0%	0.51	0.720	●○○○ VERY LOW
Grapes	3	74,716	7,197	0.90 [0.81, 1.01]	61%	0.08	0.080	●○○○ VERY LOW
Juice	1	30,458	286	0.81 [0.58, 1.13]	-	-	0.220	●○○○ VERY LOW
Pommes	5	85,929	7,947	0.86 [0.80, 0.92]	25%	0.26	<0.001	●○○○ VERY LOW
Watermelon	-	-	-	-	-	-	-	- -
Vegetables	**24**	**1,101,435**	**33,516**	**0.87 [0.85, 0.90]**	**59%**	**<0.01**	**<0.001**	**●●○○ LOW**
Allium	1	1,226	238	0.33 [0.22, 0.49]	-	-	<0.001	●○○○ VERY LOW
Carrots	2	10,325	4,792	0.92 [0.85, 1.01]	36%	0.21	0.080	●○○○ VERY LOW
Celery	1	34,492	2,316	0.91 [0.83, 1.01]	-	-	0.070	●○○○ VERY LOW
Cruciferous	7	187,730	13,081	0.85 [0.82, 0.89]	86%	<0.01	<0.001	●○○○ VERY LOW
Green Leafy	5	40,893	6,661	0.87 [0.81, 0.94]	88%	<0.01	<0.001	●○○○ LOW
Tomatoes	3	45,557	7,072	0.98 [0.93, 1.04]	23%	0.27	0.510	●○○○ VERY LOW
CHD MORTALITY								
Fruit and Vegetables	**5**	**489,635**	**3,240**	**0.81 [0.72, 0.92]**	**4%**	**0.39**	**0.001**	**●●●○ MODERATE**
Fruit	**21**	**1,398,863**	**14,786**	**0.86 [0.82, 0.90]**	**62%**	**<0.01**	**<0.001**	**●●○○ LOW**
Apricots	-	-	-	-	-	-	-	- -
Bananas	1	9,964	2,384	1.04 [0.81, 1.34]	-	-	0.760	●○○○ VERY LOW
Berries	5	105,420	5,141	0.98 [0.91, 1.05]	49%	0.07	0.560	●○○○ VERY LOW
Citrus	6	180,574	5,309	0.91 [0.85, 0.96]	0%	0.71	0.00	●○○○ VERY LOW
Dried	1	30,458	138	0.79 [0.47, 1.31]	-	-	0.360	●○○○ VERY LOW
Grapes	3	106,782	2,846	0.97 [0.77, 1.21]	41%	0.18	0.770	●○○○ VERY LOW
Juice	3	141,710	1,249	0.87 [0.75, 1.01]	71%	0.02	0.070	●○○○ VERY LOW
Pommes	5	146,407	4,650	0.84 [0.76, 0.92]	0%	0.52	<0.001	●○○○ VERY LOW
Watermelon	-	-	-	-	-	-	-	- -
Vegetables	**19**	**1,968,325**	**26,007**	**0.86 [0.83, 0.89]**	**21%**	**0.18**	**<0.001**	**●●●○ MODERATE**
Allium	4	75,434	1,280	0.67 [0.57, 0.79]	88%	<0.01	<0.001	●○○○ VERY LOW
Carrots	1	10,802	64	0.76 [0.37, 1.58]	-	-	0.470	●○○○ VERY LOW
Celery	1	34,492	1,329	0.92 [0.80, 1.06]	-	-	0.250	●○○○ VERY LOW
Cruciferous	6	296,772	7,420	0.91 [0.85, 0.98]	88%	<0.01	0.010	●○○○ VERY LOW
Green Leafy	5	148,133	4,591	0.86 [0.78, 0.94]	0%	0.61	0.001	●○○○ VERY LOW
Tomatoes	3	175,088	3,657	0.92 [0.82, 1.04]	0%	0.63	0.180	●○○○ VERY LOW
STROKE MORTALITY								
Fruit and Vegetables	**6**	**499,732**	**3,051**	**0.73 [0.65, 0.81]**	**0%**	**0.57**	**<0.001**	**●●●○ MODERATE**
Fruit	**14**	**1,282,756**	**10,899**	**0.87 [0.84, 0.91]**	**75%**	**<0.01**	**<0.001**	**●●○○ LOW**
Apricots	-	-	-	-	-	-	-	- -
Bananas	1	9,766	1,034	1.04 [0.70, 1.54]	-	-	0.840	●○○○ VERY LOW
Berries	2	40,224	1,182	0.97 [0.82, 1.15]	0%	0.66	0.750	●○○○ VERY LOW
Citrus	4	145,204	3,869	0.90 [0.86, 0.95]	82%	<0.01	<0.001	●●○○ LOW
Dried	1	30,458	152	0.95 [0.80, 1.13]	-	-	0.580	●○○○ VERY LOW
Grapes	2	40,224	1,182	0.74 [0.53, 1.02]	39%	0.2	0.070	●○○○ VERY LOW
Juice	2	128,270	2,232	0.67 [0.60, 0.76]	0%	0.93	<0.001	●●○○ LOW
Pommes	3	74,716	1,651	0.91 [0.77, 1.09]	0%	0.59	0.310	●○○○ VERY LOW
Watermelon	-	-	-	-	-	-	-	- -
Vegetables	**12**	**780,441**	**7,551**	**0.94 [0.90, 0.99]**	**62%**	**<0.01**	**0.010**	**●●○○ LOW**
Allium	2	3,671	544	0.99 [0.78, 1.24]	96%	<0.01	0.940	●○○○ VERY LOW
Carrots	1	9,766	1,034	0.54 [0.48, 0.61]	-	-	<0.001	●●○○ LOW
Celery	-	-	-	-	-	-	-	- -
Cruciferous	5	195,452	5,065	0.92 [0.85, 1.01]	18%	0.29	0.080	●○○○ VERY LOW
Green Leafy	4	126,971	4,103	0.90 [0.83, 0.97]	50%	0.09	0.005	●●○○ LOW
Tomatoes	2	108,260	3,107	1.03 [0.94, 1.12]	0%	0.83	0.540	●○○○ VERY LOW

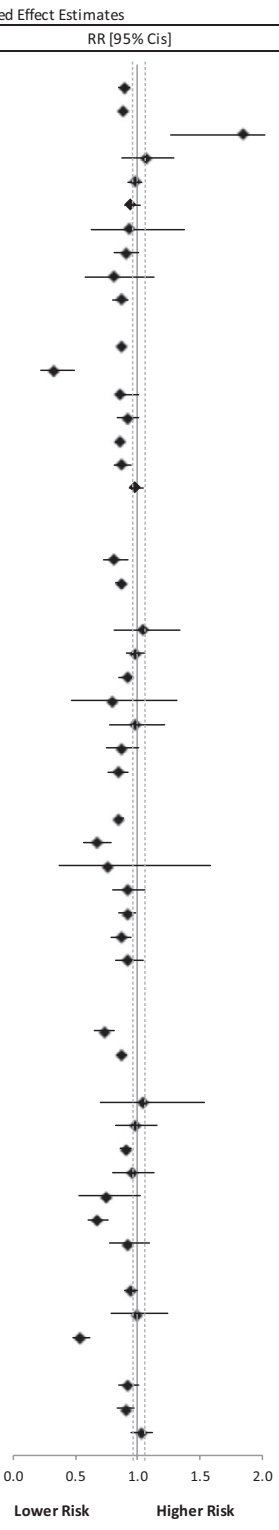


Figure 3. Relation between intake of fruits and vegetables and cardiovascular mortality (highest vs lowest level of intake). Pooled risk estimates are represented by the black diamond, with principal exposures highlighted in bold. Principal exposures (fruits and vegetables, fruits, and vegetables) represent the pooled data of the risk estimates reported for these exposures and were not tabulated by pooling fruit and vegetable varieties. Values of $I^2 \geq 50\%$ indicate substantial heterogeneity, with significance at $P>0.10$. The mean important difference of 5% change in relative risk, indicating a clinically relevant association with lower or higher risk, is indicated by the dashed gray lines. CHD indicates coronary heart disease; CVD, cardiovascular disease; GRADE, Grading of Recommendations Assessment, Development, and Evaluation; and RR, risk ratio.

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a significant increased risk with CVD mortality from the highest versus lowest intake of apricots (RR, 1.84 [95% CI, 1.27–2.67], heterogeneity not applicable). We found no significant associations from the highest versus lowest intakes of bananas (RR, 1.06 [95% CI, 0.87–1.29], heterogeneity not applicable), berries (RR, 0.97 [95% CI, 0.92–1.03], no significant heterogeneity), citrus (RR, 0.95 [95% CI, 0.90–1.02], substantial heterogeneity [I^2=62%, P=0.049]), juice (RR, 0.81 [95% CI, 0.58–1.13], heterogeneity not applicable), and grapes (RR, 0.90 [95% CI, 0.81–1.01], substantial heterogeneity [I^2=61%, P=0.077) fruit and carrots (RR, 0.92 [95% CI, 0.85–1.01], no significant heterogeneity), celery (RR, 0.91 [95% CI, 0.83–1.01], heterogeneity not applicable), and tomato (RR, 0.98 [95% CI, 0.93–1.04], no significant heterogeneity) vegetables.

Figures S33 through S55 show the dose-response analyses for total and specific fruit and vegetables and CVD incidence and mortality. A nonlinear model best fit the data for citrus fruit and incident CVD (P=0.033), with a plateau at 0.5 servings/day, total fruits and vegetables with CVD mortality (P<0.001), with a plateau at 4 daily servings, and fruits and CVD mortality (P=0.003), with a plateau in risk reduction after 2 daily servings. An inverse dose-response gradient was found for the following associations: total fruits and vegetables (RR, 0.97 [95% CI, 0.96–0.99] per serving/day), fruits (RR, 0.97 [95% CI, 0.95–0.99] per serving/day), pommes (RR, 0.87 [95% CI, 0.75–0.99] per serving/day), and green leafy vegetables (RR, 0.72 [95% CI, 0.56–0.93]) with CVD incidence and total fruits and vegetables (RR, 0.72 [95% CI, 0.56–0.93] per serving/day), fruits (RR, 0.92 [95% CI, 0.89–0.96] per serving/day), and vegetables (RR, 0.94 [95% CI, 0.92–0.97] per serving/day) with CVD mortality.

Coronary Heart Disease

CHD Incidence

Figure 2 and Figures S56 through S69 show the relation of total and specific fruit and vegetables with CHD incidence. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.88 [95% CI, 0.83–0.92], no significant heterogeneity), fruits (RR, 0.88 [95% CI, 0.84–0.92], no significant heterogeneity), and vegetables (RR, 0.92 [95% CI, 0.87–0.96], substantial heterogeneity [I^2=53%, P=0.002]). Figures S70 and S71 summarize the relation of sources of fruits or vegetables with CHD incidence. No interaction by fruit source was observed (P=0.375). A significant interaction by vegetable sources was seen (P<0.001) with significant associations with lower risk limited to green leafy vegetables (RR, 0.82 [95% CI, 0.76–0.89], no significant heterogeneity). We found no significant associations from the highest versus lowest intakes of allium (RR, 0.93 [95% CI, 0.80–1.09], no significant heterogeneity), cruciferous (RR, 1.01 [95% CI, 0.95–1.07], no significant heterogeneity), and tomato (RR, 0.80 [95% CI, 0.57–1.13], no significant heterogeneity) vegetables.

CHD Mortality

Figure 3 and Figures S72 through S87 show the relation of total and specific fruit and vegetables with CHD mortality. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.81 [95% CI, 0.72–0.92], no significant heterogeneity), fruits (RR, 0.86 [95% CI, 0.82–0.90], substantial heterogeneity [I^2=62%, P<0.001]), and vegetables (RR, 0.86 [95% CI, 0.83–0.89], no significant heterogeneity). Figures S88 and S89 summarize the relation of sources of fruits or vegetables with CHD mortality. No significant interaction was found by fruit sources (P=0.144). A significant interaction by vegetable source was seen (P=0.023), with significant associations with lower risk limited to allium (RR, 0.67 [95% CI, 0.57–0.79], substantial heterogeneity [I^2=88%, P<0.001]), cruciferous (RR, 0.91 [95% CI, 0.85–0.98], substantial heterogeneity [I^2=88%, P<0.001]), and green leafy (RR, 0.86 [95% CI, 0.78–0.94], no significant heterogeneity) vegetables. We found no significant associations from the highest versus lowest intakes of carrots (RR, 0.76 [95% CI, 0.37–1.58], heterogeneity not applicable), celery (RR, 0.92 [95% CI, 0.80–1.06], heterogeneity not applicable), and tomato (RR, 0.92 [95% CI, 0.82–1.04], no significant heterogeneity) vegetables.

Figures S90 through S116 show the dose-response analyses for fruit and vegetables and CHD incidence and mortality. A nonlinear model best fit the data for citrus fruit (P=0.005) and green leafy vegetables (P=0.004) and incident CHD and total fruits and vegetables and CHD mortality (P=0.044), with plateaus in risk reductions following 0.5, 0.5, and 3 daily servings, respectively. An inverse dose-response was found in the associations between total fruits and vegetables (RR, 0.97 [95% CI, 0.96–0.98] per serving/day), fruits (RR, 0.96 [95% CI, 0.93–0.99] per serving/day), vegetables (RR, 0.98 [95% CI, 0.95–0.99] per serving/day), and green leafy vegetables (RR, 0.85 [95% CI, 0.76–0.94] per serving/day) with CHD incidence and fruits (RR, 0.94 [95% CI, 0.90–0.97] per serving/day) and vegetables (RR, 0.89 [95% CI, 0.83–0.96] per serving/day) with CHD mortality.

Stroke

Stroke Incidence

Figure 2 and Figures S117 through S127 show the relation of total and specific fruit and vegetables with

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stroke incidence. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.82 [95% CI, 0.77–0.88], no significant heterogeneity), fruits (RR, 0.82 [95% CI, 0.79–0.85], no significant heterogeneity), and vegetables (RR, 0.88 [95% CI, 0.83–0.93], substantial heterogeneity [I^2=50%, *P*=0.006]). Figures S128 and S129 summarize the relation of sources of fruits or vegetables with stroke incidence. A significant interaction by fruit (*P*=0.017) and vegetable sources (*P*=0.044) was observed with significant associations with lower risk limited to citrus (RR, 0.88 [95% CI, 0.82–0.94], substantial heterogeneity [I^2=51%, *P*=0.04]), juice (RR, 0.82 [95% CI, 0.68–0.99], substantial heterogeneity [I^2=73%, *P*=0.02]), and pommes (RR, 0.89 [95% CI, 0.84–0.95], no significant heterogeneity) fruit and to allium (RR, 0.89 [95% CI, 0.80–0.99], no significant heterogeneity), green leafy (RR, 0.88 [95% CI, 0.79–0.98], no significant heterogeneity), and tomato (RR, 0.20 [95% CI, 0.05–0.82], heterogeneity not applicable) vegetables. We found no significant associations from the highest versus lowest intakes of berries (RR, 1.03 [95% CI, 0.94–1.13], substantial heterogeneity [I^2=50%, *P*=0.078]) fruit and cruciferous (RR, 0.98 [95% CI, 0.91–1.05], substantial heterogeneity [I^2=62%, *P*=0.022]) vegetables.

Stroke Mortality

Figure 3 and Figures S130 through S144 show the relation of total and specific fruits and vegetables with stroke mortality. We found a lower risk associated with the highest versus the lowest intakes of fruits and vegetables (RR, 0.73 [95% CI, 0.65–0.81], no significant heterogeneity), fruits (RR, 0.87 [95% CI, 0.84–0.91], substantial heterogeneity [I^2=75%, *P*<0.001]), and vegetables (RR, 0.94 [95% CI, 0.90–0.99], substantial heterogeneity [I^2=62%, *P*=0.001]). Figures S145 and S146 summarize the relation of sources of fruit or vegetables with stroke mortality. A significant interaction by fruit (*P*<0.001) and vegetable sources (*P*<0.001) was observed with significant associations, with lower risk limited to citrus (RR, 0.90 [95% CI, 0.86–0.95], substantial heterogeneity [I^2=82%, *P*<0.001]) and juice (RR, 0.67 [95% CI, 0.60–0.76], no significant heterogeneity) fruit and carrots (RR, 0.54 [95% CI, 0.48–0.61], heterogeneity not applicable) and green leafy (RR, 0.90 [95% CI, 0.83–0.97], substantial heterogeneity [I^2=50%, *P*=0.09]) vegetables. We found no significant associations from the highest versus lowest intakes of bananas (RR, 1.04 [95% CI, 0.70–1.54], heterogeneity not applicable), berries (RR, 0.97 [95% CI, 0.82–1.15], no significant heterogeneity), grapes (RR, 0.74 [95% CI, 0.53–1.02], no significant heterogeneity), and pommes (RR, 0.91 [95% CI, 0.77–1.09], no significant heterogeneity) fruit and allium (RR, 0.99 [95% CI, 0.79–1.24], substantial heterogeneity [I^2=96%, *P*<0.001]), cruciferous (RR, 0.92 [95% CI, 0.85–1.01], no significant heterogeneity), and tomato (RR, 1.03 [95% CI, 0.94–1.12], no significant heterogeneity) vegetables.

Figures S147 through S171 show the dose-response analyses for fruit and vegetables and stroke mortality and incidence. A nonlinear model best fit the data for citrus fruit (*P*=0.039) and vegetables (*P*=0.012) and stroke incidence and fruit (*P*<0.001) and green leafy (*P*=0.043) vegetables and stroke mortality, with plateaus in risk reductions following 0.5, 1, 2, and >0.7 daily servings, respectively. An inverse dose-response gradient was found in the associations between total fruits and vegetables (RR, 0.95 [95% CI, 0.92–0.98] per serving/day), fruits (RR, 0.92 [95% CI, 0.88–0.96] per serving/day), citrus fruit (RR, 0.83 [95% CI, 0.69–0.98] per serving/day), pommes (RR, 0.87 [95% CI, 0.79–0.96] per serving/day), green leafy vegetables (RR, 0.88 [95% CI, 0.79–0.97] per serving/day), and tomatoes (RR, 0.67 [95% CI, 0.52–0.87] per serving/day) with stroke incidence and fruits and vegetables (RR, 0.93 [95% CI, 0.88–0.98] per serving/day), fruits (RR, 0.85 [95% CI, 0.78–0.92] per serving/day), vegetables (RR, 0.93 [95% CI, 0.87–0.99] per serving/day), citrus fruit (RR, 0.67 [95% CI, 0.57–0.80] per serving/day), fruit juice (RR, 0.54 [95% CI, 0.36–0.89] per serving/day), carrots (RR, 0.44 [95% CI, 0.28–0.69] per serving/day), and green leafy vegetables (RR, 0.85 [95% CI, 0.73–0.98] per serving/day) with stroke mortality.

Sensitivity Analyses

The systematic removal of each study did not modify the direction or significance of the association estimates or the evidence for heterogeneity (data not shown).

Subgroup Analyses

Figures S172 through S188 illustrate a priori categorical subgroup analyses. There were no statistically significant subgroup differences. Inverse associations were predominately limited to studies with statistical adjustments of ≥8 potential confounders. Confining analyses to studies using validated exposure assessment techniques did not alter the associations. No effect modification was seen by sex, age, follow-up duration, NOS, or study location.

Publication Bias

Figures S189 through S205 illustrate publication bias analyses for comparisons with at least 10 observations. Visual inspection and formal analysis with the Begg and Egger test did not show evidence of

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publication bias in any comparison, except for vegetable intake with CVD (P_{Begg}=0.015, P_{Egger}=0.004), CHD (P_{Begg}=0.018, P_{Egger}=0.004), and stroke (P_{Begg}=0.545, P_{Egger}=0.018) mortality and fruit intake with stroke mortality (P_{Begg}=0.820, P_{Egger}=0.031), which were subsequently unsupported by the trim and fill test.

GRADING OF RECOMMENDATIONS ASSESSMENT, DEVELOPMENT, AND EVALUATION

Figures 2 and 3 and Tables S4 through S9 summarize the GRADE assessments. The certainty of the evidence was rated as "moderate" for 11, "low" for 21, and "very low" for 52 of the exposure-outcome relationships. Our certainty in the evidence was strongest for the associations of total fruits and vegetables with lower risks of CHD incidence and CHD and stroke mortality; fruits with lower risks of CVD, CHD, and stroke incidence; vegetables with lower risks of CHD mortality and stroke incidence; pommes fruit with lower risks of stroke incidence; and green leafy vegetables with lower risks of CHD incidence. The evidence was rated as "moderate" in each case, because of an upgrade for dose-response gradient in the absence of any downgrades. The associations for specific types of fruits and vegetables were rated largely as "very low," because of downgrades for imprecision, risk of bias, indirectness, and/or inconsistency. The fixed effects model improved our certainty in the evidence for fruit and CVD incidence by improving precision of the pooled risk estimate. There were no other marked differences between the random effects and fixed effects models.

DISCUSSION

We conducted a systematic review and meta-analysis of 81 unique prospective cohorts involving 4 031 896 individuals and 125 112 cardiovascular events to assess the relation of total and specific fruit and vegetable consumption on CVD incidence and mortality outcomes. Pooled analyses of highest versus lowest consumption illustrate a lower risk in CVD, CHD, and stroke incidence or mortality by 7% to 27% from total fruit and vegetable intake, 9% to 18% from fruit intake, and 5% to 14% from vegetable intake. Of the specific fruit sources, highest versus lowest intakes of citrus and pommes fruit showed significant risk reductions in most CVD outcomes, from 9% to 12% and from 10% to 24%, respectively, and fruit juice showed a significant risk reduction in stroke incidence and mortality by 18% and 33%, respectively. Most notably of the vegetable categories, one daily serving of green leafy vegetables was associated with 12% to 18% risk reduction in CVD, CHD, and stroke incidence and CHD mortality. There was a consistent linear dose-response between fruits and vegetables and CHD, with a maximum daily intake of 7 fruit and 7 vegetable servings showing a risk reduction of ≈20% and ≈30% in CHD incidence and mortality, respectively.

Findings in the Context of Existing Literature

Our findings are consistent with those of previous systematic review and meta-analyses, which also detected inverse associations between fruits and/or vegetables and CVD mortality and incident outcomes.[10,14,159] Our analyses were in line with those reported most recently by Aune et al, who observed the lowest risk on CVD, CHD, and stroke from maximum intakes of total fruits and vegetables.[10] This is despite our division of CVD outcomes differing significantly, with the present study distinguishing between mortality and incidence data. Our findings on individual fruits and vegetables were also relatively consistent, highlighting a high versus low intake of citrus and pommes fruit, fruit juice, and green leafy vegetables as protective on CVD outcomes, suggesting they may independently play a valuable role in the diet. Nonetheless, the current study benefited from the inclusion of updated and novel large prospective cohorts, namely, the SUN (Seguimiento University of Navarra)[160] and PURE (Prospective Urban and Rural Epidemiology)[161] cohorts, which combined contributed an additional 152 342 individuals and 4896 events to our analyses.

Numerous mechanisms have been proposed to explain the benefits of fruit and vegetable consumption on the cardiovascular system. Perhaps the most supported hypothesis is through their essential contribution to total dietary fiber, an established modifier of CVD risk factors.[162,163]

Fruits with highlighted benefits in the present review tend to be of low glycemic index, a characteristic with demonstrated CVD risk factor reductions.[164] Their consumption has also been associated with improved weight management[165] and decreased prevalence of obesity,[166] a risk factor attributed to 7% to 44% of CVD incidence,[167] likely because of their low energy density and displacement of high calorie foods in the diet. The relationships between the extensive list of micronutrients offered by fruits and vegetables and CVD risk reduction has also been widely explored. They are a key source of antioxidants in the diet, necessary for eradicating free radicals, and may defend against damaging lipid oxidation.[168] Individual sources may offer distinct benefits, such as green leafy vegetables, which are dense in dietary nitrates, a compound linked to reductions in early prognostic

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markers of CVD.[169-171] Interestingly, however, we did not observe a benefit from high consumption of berries as the most concentrated fruit source of antioxidants. Several vasoactive minerals, such as potassium, magnesium, and calcium, are also obtained from fruits and vegetables in the diet.[172-174] Although each mechanism may be individually biologically plausible, the complexity of the nutrient combinations cannot be underestimated. A whole food approach is necessary to evaluate their efficacy in CVD risk reduction as it can account for additive and multiplicative mechanisms.

Strengths and Limitations

Our systematic review and meta-analysis has several strengths. It provides a comprehensive synthesis of the available knowledge on consumption of fruits, vegetables, and their varieties and CVD outcomes of importance to public health and clinical practice. We included a systematic search strategy to ensure all published prospective cohort data were identified and used a priori established approaches to explore the pooled risk estimates, including dose-response analyses. Finally, the certainty of the evidence was assessed using the GRADE approach with the evidence upgraded in several cases for the presence of a protective inverse dose-response gradient for the association of total fruits and vegetables, fruits, vegetables, and green leafy vegetables with CVD outcomes.

There are also several limitations of our systematic review and meta-analysis. Although ≈90% of the included prospective cohort studies were of high quality, residual confounding (measured and unmeasured) cannot be ruled out in observational studies. This issue is addressed in the GRADE assessment, which starts observational studies as "low" certainty. We downgraded the certainty of evidence because of imprecision in 55 of the 84 associations as the upper 95% CI crossed the minimal clinically important difference of a 5% reduction in relative risk, from which evidence of harm could not be excluded in 30 associations. Because of limited number of observations, indirectness was also present in several cases and the lack of reported exposures for different tropical fruit limited our exploration of this fruit category. Another source of uncertainty leading to downgrades in the evidence was the presence of high risk of bias in several of the studies that presented data on specific sources of fruits and vegetables. Last, the evidence was downgraded for inconsistency based on the presence of substantial unexplained heterogeneity in 19 of the 84 associations.

Balancing the strengths and limitations, the certainty of the evidence was rated as "very low" to "low" for most of the exposure-outcome relationships for the association of fruits and vegetables with cardiovascular outcomes. The highest ("moderate") rated evidence was for the cardiovascular benefit of total fruits and vegetables, fruits, vegetables, pommes fruit, and green leafy vegetables. The least certainty was for other specific fruit and vegetable sources.

Implications

Addressing the low prevalence of adequate fruit and vegetable consumption remains an important global health target.[175] With average intakes of 1 and 1.7 servings of fruit and vegetables per day, respectively, in developed countries, such as the United States,[150] there is an opportunity to increase intakes to meet the established minimum recommendations of 5 daily servings and realize the cardiovascular benefits.[176] We observed a linear dose relationship between fruits and vegetables and CHD and stroke risk, suggesting an increased cardiovascular benefit with additional servings and that targets beyond "5 a day" should also be considered. Successful strategies for increasing fruit and vegetable intake, nevertheless, are lacking and may benefit from emphasizing a larger variety of sources. Our synthesis highlighted that different sources of fruit, including 100% fruit juice, are associated with comparable CVD risk reduction as that of vegetables. Public health guidance to limit the intake of certain fruit sources because of concerns related to their contribution to sugars may have unintended harm in preventing people from meeting fruit and vegetable targets for CVD risk reduction.

CONCLUSIONS

Current evidence supports the role of a variety of fruits and vegetables for CVD prevention. Higher intakes of fruits and/or vegetables are associated with improvements in all CVD outcomes, with fruit associated with the largest risk reductions. Greater benefits may be seen for some fruits, including citrus, pommes, and 100% fruit juice, and vegetables, including allium, cruciferous, and green leafy vegetables, supporting recommendations for emphasizing specific fruit and vegetable sources in dietary guidelines. No fruit and vegetable sources were adversely associated with CVD, including fruit sources of concern, such as 100% fruit juice and dried fruit. Our certainty in the evidence ranges from "very low" to "moderate," with the least certainty for specific sources of fruits and vegetables and the highest certainty for broad categories. More research of specific food sources of fruits and vegetables is needed to improve our estimates.

ARTICLE INFORMATION

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Supplementary Materials

Tables S1–S9
Figures S1–S205

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Supplemental Material

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Table S1. Search Strategy.

Search #	Medline 1946 to June 03, 2019	Embase 1946 to June 03, 2019	Cochrane Through to June 03, 2019
1	Vegetables/	exp vegetable/	Vegetables/
2	vegetable*.tw,kf.	vegetable*.tw,kw.	vegetable*.ti,ab,hw.
3	Vegetable Products/	1 or 2	1 or 2
4	or/1-3	fruit.mp.	fruit.mp.
5	fruit.mp.	exp Fruit/	Fruit/
6	exp Fruit/	4 or 5	4 or 5
7	5 or 6	3 or 6	3 or 6
8	4 or 7	cardiovascular disease/	cardiovascular diseases/
9	cardiovascular disease/	cardiovascular.tw,kw.	exp myocardial ischemia/
10	exp myocardial ischemia/	exp heart muscle ischemia/	cardiovascular.ti,ab,hw.
11	cardiovascular.tw,kf.	isch?em*.tw,kw.	isch?em*.ti,ab,hw.
12	isch?em*.tw,kf.	coronary.tw,kw.	coronary.ti,ab,hw.
13	coronary.tw,kf.	myocard*.tw,kw.	myocard*.ti,ab,hw.
14	myocard*.tw,kf.	angina.tw,kw.	angina.ti,ab,hw.
15	angina.tw,kf.	exp cerebrovascular disease/	exp cerebrovascular disorders/
16	exp cerebrovascular disorders/	stroke.tw,kw.	stroke*.ti,ab,hw.
17	stroke*.tw,kf.	cerebral vascular.tw,kw.	cerebral vascular.ti,ab,hw.
18	cerebral vascular.tw,kf.	cerebrovascular.tw,kw.	cerebrovascular.ti,ab,hw.
19	cerebrovascular.tw,kf.	Or / 8-18	Or / 8-
20	Or / 9-19	exp cohort analysis/	
21	exp cohort studies/	exp longitudinal study/	
22	cohort*.tw.	exp prospective study/	
23	controlled clinical trial.pt.	exp follow up/	
24	Epidemiologic methods/	cohort$.tw.	
25	limit 24 to yr=1971-1988	Or / 20-24	
26	Or / 21- 25	7 and 19	
27	8 and 20	25 and 26	
28	26 and 27		

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Table S2. Confounding Variables Among 117 Studies of Fruit and Vegetables and Cardiovascular Disease Outcomes.

Study	Adriouch, 2018[42]	Appleby, 2002[43]	Atkins, 2014[44]	Bahadoran, 2017[45]	Bazzano, 2002[46]	Belin, 2011[47]	Bendinelli, 2011[48]	Berard, 2017[49]	Bhupathiraju, 2013[50]	Bingham, 2008[51]	Blekkenhorst, 2017[52]
No. of variables fully adjusted model	13	3	8	2	10	10	12	5	13	9	10
No. of multivariable models presented	1	1	2	2	2	1	2	1	2	1	8
Timing of measurement of confounding variables	BL	BL	BL	BL	BL, 1982-84, 86, 87, 92	BL	BL	BL	1984-86, q2y	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓	✓		✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables											
Sex		✓	✓		✓		✓	✓		✓	N/A
Smoking	✓	✓	✓		✓	✓	✓		✓	✓	✓
BMI	✓		✓			✓			✓		✓
Physical activity	✓		✓		✓	✓	✓		✓	✓	✓
Alcohol	✓				✓		✓		✓	✓	✓
Blood pressure	✓					✓	✓			✓	
Energy	✓		✓		✓	✓	✓	✓	✓	✓	
Diabetes					✓	✓	✓				✓
Cholesterol						✓	✓			✓	✓
Other Confounding variables											
Education	✓				✓	✓	✓	✓			
Socioeconomic status			✓								✓
Menopause and/or hormone Use	✓						✓		✓		
Region/location											
Randomization treatment											✓
Ethnicity/nationality	✓				✓	✓					
Marital status											
Study center								✓			
Survey season	✓										
Employment status											
Follow-up duration											
Dietary Intake											
Vitamin/supplement					✓				✓		
Fruit and/or vegetable	✓										
Saturated fat											
Whole grains											
Fish/shellfish									✓		
Meat							✓				
Red meat									✓		
Dietary pattern score			✓	✓							
Processed meat											
Coffee											
Fibre											
Folate											
Sodium											
Vitamin E											
Disease History											
MI or family history of MI									✓		
CHD or family history of CHD											
CVD or family history of CVD				✓							
Medications											
ASA											✓
Other confounding variables not listed:	Sleep, WC								Cereal fibre, Trans fat	Weight	GFR

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Study	Bos, 2014[53]	Buijsse, 2008[54]	BuilCosiales, 2016[56]	BuilCosiales, 2017[55]	Cassidy, 2012[57]	Collin, 2019[58]	Conrad, 2018[59]	Dauchet, 2004[60]	Dauchet, 2010[61]	Du, 2016[62]
No. of variables fully adjusted model	7	15	17	14	13	12	10	10	12	13
No. of multivariable models presented	1	4	1	3	1	4	1	1	1	2
Timing of measurement of confounding variables	BL	BL	BL	1999, q2y	1976, q2y	BL	BL	BL	BL	BL
Pre-specified primary confounding variables										
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables										
Sex	✓	✓	✓	✓	✓	✓	✓			✓
Smoking	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
BMI	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Physical activity		✓	✓		✓	✓		✓	✓	✓
Alcohol		✓	✓	✓	✓	✓				✓
Blood pressure	✓		✓	✓	✓			✓	✓	
Energy		✓	✓	✓	✓	✓				
Diabetes	✓		✓		✓			✓	✓	
Cholesterol					✓			✓	✓	
Other Confounding variables										
Education			✓	✓		✓	✓	✓	✓	✓
Socioeconomic status		✓				✓	✓			✓
Menopause and/or hormone Use					✓					
Region/location						✓				✓
Randomization treatment			✓							
Ethnicity/nationality							✓			
Marital status				✓						
Study center			✓					✓	✓	
Survey season										✓
Employment status								✓	✓	
Follow-up duration										
Dietary Intake										
Vitamin/supplement					✓				✓	
Fruit and/or vegetable			✓							
Saturated fat		✓				✓				
Whole grains			✓	✓						
Fish/shellfish										
Meat										✓
Red meat										
Dietary pattern score										
Processed meat										
Coffee										
Fibre		✓				✓				
Folate		✓								
Sodium										
Vitamin E										
Disease History										
MI or family history of MI										
CHD or family history of CHD	✓		✓	✓						
CVD or family history of CVD										
Medications										
ASA					✓					
Other confounding variables not listed:		Vitamin C, trans/PUFA, α-tocopherol	Olive oil, Statins	Dyslipidemia, Legumes, Olive oil			Cardiomet-abolic meds, added sugar, SFA:M/PUFA		Dyslipidemi	Dairy, Preserved vegetables

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Study	Du, 2017[63]	Elwood, 2013[64]	Eriksen, 2015[65]	Fitzgerald, 2012[66]	Fraser, 1992[67]	Gardener, 2011[68]	Gaziano, 1995[69]	Genkinger, 2004[70]	Gillman, 1995[71]	Goetz, 2016[72]	Goetz, 2016[73]
No. of variables fully adjusted model	12	3	9	10	6	7	6	6	7	12	10
No. of multivariable models presented	14	1	1	1	1	1	1	2	1	1	1
Timing of measurement of confounding variables	BL	1979, q5y	BL	BL	BL	qy.	1976, qy	BL	BL	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables											
Sex	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Smoking	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓
BMI	✓		✓					✓	✓		
Physical activity	✓			✓	✓	✓			✓	✓	✓
Alcohol	✓		✓	✓						✓	
Blood pressure			✓								
Energy				✓	✓	✓		✓		✓	✓
Diabetes				✓			✓		✓		
Cholesterol			✓				✓	✓	✓		
Other Confounding variables											
Education	✓			✓		✓				✓	✓
Socioeconomic status	✓	✓	✓							✓	✓
Menopause and/or hormone Use				✓							
Region/location	✓									✓	✓
Randomization treatment				✓							
Ethnicity/nationality						✓					✓
Marital status											
Study center											
Survey season	✓										
Employment status			✓								
Follow-up duration											
Dietary Intake											
Vitamin/supplement											
Fruit and/or vegetable											
Saturated fat											
Whole grains											
Fish/shellfish											
Meat	✓										
Red meat											
Dietary pattern score											✓
Processed meat											
Coffee											
Fibre											
Folate											
Sodium											
Vitamin E											
Disease History											
MI or family history of MI											
CHD or family history of CHD											
CVD or family history of CVD											
Medications											
ASA											
Other confounding variables not listed:	Preserved vegetables				Weight		Functional status			Trans FA MUFA:SFA, %E sweets	

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Study	Gunge, 2017[74]	Gunnell, 2013[75]	Hansen, 2010[77]	Hansen, 2017[76]	Harriss, 2007[78]	Hertog, 1997[79]	Hirvonen, 2000[81]	Hirvonen, 2001[80]	Hjartaker, 2015[82]	Hodgson, 2016[83]	Holmberg, 2009[84]
No. of variables fully adjusted model	18	10	11	13	15	13	10	11	9	15	0
No. of multivariable models presented	4	1	2	2	2	1	1	1	1	2	0
Timing of measurement of confounding variables	BL	BL	BL	BL	BL	BL, q5y	BL	BL	BL	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	
Pre-specified secondary confounding variables											
Sex	✓	✓	✓	✓	✓				✓	✓	
Smoking	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	
BMI	✓	✓	✓		✓	✓	✓	✓	✓	✓	
Physical activity	✓	✓	✓	✓	✓		✓	✓	✓	✓	
Alcohol	✓		✓	✓		✓			✓	✓	
Blood pressure	✓	✓	✓	✓	✓	✓	✓	✓		✓	
Energy	✓			✓	✓	✓				✓	
Diabetes				✓	✓		✓	✓		✓	
Cholesterol	✓	✓	✓	✓		✓	✓	✓		✓	
Other Confounding variables											
Education	✓		✓	✓	✓		✓	✓			
Socioeconomic status						✓			✓	✓	
Menopause and/or hormone Use	✓										
Region/location					✓						
Randomization treatment							✓	✓		✓	
Ethnicity/nationality					✓						
Marital status								✓			
Study center											
Survey season		✓		✓							
Employment status											
Follow-up duration	✓										
Dietary Intake											
Vitamin/supplement									✓		
Fruit and/or vegetable	✓				✓						
Saturated fat			✓								
Whole grains	✓		✓								
Fish/shellfish	✓										
Meat					✓						
Red meat	✓										
Dietary pattern score					✓						
Processed meat	✓										
Coffee									✓		
Fibre											
Folate											
Sodium											
Vitamin E						✓					
Disease History											
MI or family history of MI											
CHD or family history of CHD				✓		✓	✓	✓			
CVD or family history of CVD					✓					✓	
Medications											
ASA										✓	
Other confounding variables not listed:	WC	Charlson index, DM hospitalization		Weight		Vitamin C, B-carotene, Dietary fat				Cancer	

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Study	Iso, 2007[85]	Jacques, 2015[86]	Johnsen, 2003[87]	Joshipura, 1999[88]	Joshipura, 2009[89]	Keli, 1996[90]	Kim, 2013[91]	Knekt, 1994[94]	Knekt, 1996[93]	Knekt, 2000[92]	Kobylecki, 2015[95]
No. of variables fully adjusted model	3	5	13	12	14	7	0	5	6	17	12
No. of multivariable models presented	1	2	2	1	198-86, q2y	1	0	2	1	1	3
Timing of measurement of confounding variables	BL	1991, q3-4y	BL	1980-6, q2y	1980-6, q2y	1960-73, 77, 85	BL	BL	BL	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓	✓	✓	✓	✓		✓	✓	✓	✓
Pre-specified secondary confounding variables											
Sex	✓	✓	✓	✓					✓		✓
Smoking		✓	✓	✓	✓	✓		✓	✓	✓	✓
BMI		✓	✓	✓	✓				✓	✓	✓
Physical activity			✓	✓	✓						✓
Alcohol			✓	✓	✓	✓					✓
Blood pressure			✓	✓	✓	✓		✓	✓	✓	✓
Energy		✓	✓	✓	✓	✓		✓		✓	
Diabetes			✓		✓					✓	
Cholesterol			✓	✓	✓	✓		✓	✓	✓	✓
Other Confounding variables											
Education			✓								
Socioeconomic status											✓
Menopause and/or hormone Use				✓	✓						
Region/location	✓									✓	
Randomization treatment											
Ethnicity/nationality											
Marital status											
Study center											
Survey season											
Employment status											
Follow-up duration											
Dietary Intake											
Vitamin/supplement				✓	✓						✓
Fruit and/or vegetable											
Saturated fat										✓	
Whole grains					✓						
Fish/shellfish						✓					
Meat											
Red meat			✓								
Dietary pattern score											
Processed meat											
Coffee											
Fibre										✓	
Folate											
Sodium											
Vitamin E										✓	
Disease History											
MI or family history of MI				✓							
CHD or family history of CHD					✓						
CVD or family history of CVD											
Medications											
ASA					✓						
Other confounding variables not listed:			Ω-3-FA							Occupation, Vit C/E,Querc P/MUFA	Maximal oxygen intake, CRP

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Study	Kondo, 2019[96]	Kvaavik, 2010[97]	Lai, 2015[98]	Larsson, 2009[99]	Larsson, 2013[100]	Leenders, 2013[102]	Leenders, 2014[101]	Lin, 2007[103]	Lin, 2017[104]	Liu, 2000[106]
No. of variables fully adjusted model	7	8	8	14	16	11	11	13	6	8
No. of multivariable models presented	1	2	2	2	2	1	1	2	1	3
Timing of measurement of confounding variables	BL	BL	BL	BL	BL	BL	BL	1990, q2y	BL	BL
Pre-specified primary confounding variables										
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables										
Sex	✓	✓	✓	✓	✓	✓	✓		✓	
Smoking	✓		✓	✓	✓	✓	✓	✓		
BMI		✓	✓	✓	✓	✓	✓	✓	✓	
Physical activity			✓	✓	✓	✓	✓	✓		✓
Alcohol	✓		✓	✓	✓	✓	✓	✓		✓
Blood pressure		✓		✓	✓			✓	✓	✓
Energy	✓			✓	✓			✓		
Diabetes		✓		✓	✓	✓	✓	✓	✓	✓
Cholesterol				✓				✓		✓
Other Confounding variables										
Education					✓	✓	✓		✓	
Socioeconomic status		✓	✓							
Menopause and/or hormone Use								✓		
Region/location										
Randomization treatment				✓						✓
Ethnicity/nationality										
Marital status										
Study center						✓	✓			
Survey season										
Employment status										
Follow-up duration										
Dietary Intake										
Vitamin/supplement								✓		✓
Fruit and/or vegetable	✓		✓		✓	✓	✓			
Saturated fat										
Whole grains										
Fish/shellfish	✓									
Meat						✓	✓			
Red meat					✓					
Dietary pattern score										
Processed meat					✓					
Coffee					✓					
Fibre										
Folate				✓						
Sodium	✓									
Vitamin E								✓		
Disease History										
MI or family history of MI					✓					
CHD or family history of CHD		✓								
CVD or family history of CVD				✓					✓	
Medications										
ASA					✓			✓		
Other confounding variables not listed:		Respiratory diseases		Magnesium						

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Study	Liu, 2001[105]	Mann, 1997[107]	Manuel, 2015[108]	Miller, 2017[109]	Mink, 2007[110]	Mizrahi, 2009[111]	Mori, 2018[112]	Mytton, 2018[113]	Nagura, 2009[114]	Nakamura, 2008[115]
No. of variables fully adjusted model	11	5	1	17	11	8	16	16	16	15
No. of multivariable models presented	2	1	1	1	2	1	3	2	3	3
Timing of measurement of confounding variables	BL	BL	BL	BL	BL	BL	BL	BL	BL	BL
Pre-specified primary confounding variables										
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables										
Sex	✓	✓		✓		✓	✓	✓	✓	✓
Smoking	✓	✓		✓	✓	✓	✓	✓	✓	✓
BMI	✓	✓			✓	✓	✓		✓	✓
Physical activity	✓			✓	✓	✓	✓	✓	✓	✓
Alcohol	✓						✓	✓	✓	✓
Blood pressure	✓			✓	✓	✓	✓	✓	✓	✓
Energy				✓	✓	✓	✓	✓		✓
Diabetes	✓			✓	✓		✓	✓	✓	✓
Cholesterol	✓			✓		✓		✓		
Other Confounding variables										
Education				✓	✓			✓	✓	✓
Socioeconomic status		✓								
Menopause and/or hormone Use					✓					✓
Region/location				✓						
Randomization treatment	✓									
Ethnicity/nationality										
Marital status					✓					✓
Study center				✓			✓			
Survey season										
Employment status							✓			
Follow-up duration										
Dietary Intake										
Vitamin/supplement	✓						✓			
Fruit and/or vegetable				✓			✓		✓	
Saturated fat									✓	✓
Whole grains										
Fish/shellfish										
Meat										
Red meat				✓						
Dietary pattern score										
Processed meat										
Coffee							✓			
Fibre				✓						
Folate										
Sodium							✓		✓	✓
Vitamin E										
Disease History										
MI or family history of MI								✓		
CHD or family history of CHD										
CVD or family history of CVD										
Medications										
ASA										
Other confounding variables not listed:				Waist:hip, bread, white meat	Waist:hip		Green tea	Family hx of diabetes/ stroke	Sleep, stress, Ω-3 FA, diet cholesterol	Dietary protein

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Study	Nechuta, 2010[116]	Neelakantan, 2018[117]	Ness, 2005[118]	Nothlings, 2008[119]	Okuda, 2015[120]	Oude Griep, 2010[121]	Oude Griep, 2011[123]	Oude Griep, 2011[122]	Oyebode, 2014[124]	Pham, 2007[125]
No. of variables fully adjusted model	7	12	8	11	11	12	15	15	8	9
No. of multivariable models presented	2	1	2	2	3	3	3	3	2	1
Timing of measurement of confounding variables	BL	BL	BL	BL	BL	BL	BL	BL	2001, qy	BL
Pre-specified primary confounding variables										
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables										
Sex	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Smoking		✓		✓	✓	✓	✓	✓	✓	✓
BMI	✓	✓			✓		✓	✓	✓	✓
Physical activity	✓	✓							✓	
Alcohol				✓	✓	✓	✓	✓	✓	✓
Blood pressure		✓		✓						✓
Energy		✓	✓	✓	✓	✓	✓	✓		
Diabetes		✓		✓			✓	✓		✓
Cholesterol							✓	✓		
Other Confounding variables										
Education	✓	✓				✓	✓	✓	✓	
Socioeconomic status	✓		✓						✓	
Menopause and/or hormone Use						✓	✓	✓		
Region/location			✓							
Randomization treatment										
Ethnicity/nationality		✓								
Marital status	✓									
Study center										
Survey season			✓							
Employment status										
Follow-up duration										
Dietary Intake										
Vitamin/supplement						✓	✓	✓		
Fruit and/or vegetable		✓								✓
Saturated fat										
Whole grains		✓				✓	✓	✓		
Fish/shellfish		✓			✓	✓	✓	✓		
Meat					✓					
Red meat										
Dietary pattern score										
Processed meat						✓	✓	✓		
Coffee										
Fibre										
Folate										
Sodium					✓					
Vitamin E										
Disease History										
MI or family history of MI				✓		✓	✓	✓		
CHD or family history of CHD										
CVD or family history of CVD										
Medications										
ASA										
Other confounding variables not listed:		Sleep, nuts, legumes, dairy	Child food expenditure, Townsend	Cancer hx, insulin tx, Waist:Hip	Dairy, soy					Blood transfusion

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Study	Rebello, 2014[126]	Rissanen, 2003[127]	Saglimbene, 2017[128]	Sahyoun, 1996[129]	Sauvaget, 2003[130]	Scheffers, 2019[131]	Sesso, 2003[132]	Sesso, 2003[134]	Sesso, 2007[133]	Shah, 2018[135]	Sharma, 2013[136]
No. of variables fully adjusted model	20	10	N/A	4	13	12	16	16	18	10	7
No. of multivariable models presented	3	4	N/A	3	4	4	2	2	4	2	1
Timing of measurement of confounding variables	BL	BL	N/A	BL	BL	BL	BL	BL	BL	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓		✓	✓	✓	✓	✓	✓	✓	
Pre-specified secondary confounding variables											
Sex		✓		✓	✓	✓				✓	
Smoking	✓	✓			✓	✓	✓	✓	✓	✓	✓
BMI	✓	✓			✓	✓	✓	✓	✓	✓	
Physical activity	✓					✓	✓	✓	✓	✓	✓
Alcohol	✓				✓	✓	✓	✓	✓	✓	✓
Blood pressure	✓	✓			✓	✓	✓	✓	✓	✓	
Energy	✓							✓	✓		✓
Diabetes		✓			✓		✓	✓	✓	✓	
Cholesterol		✓				✓	✓	✓	✓	✓	
Other Confounding variables											
Education	✓				✓	✓					✓
Socioeconomic status											
Menopause and/or hormone Use	✓						✓	✓	✓		
Region/location					✓						
Randomization treatment							✓	✓	✓		
Ethnicity/nationality	✓										✓
Marital status											
Study center											
Survey season	✓										
Employment status											
Follow-up duration		✓									✓
Dietary Intake											
Vitamin/supplement		✓									
Fruit and/or vegetable							✓	✓	✓		
Saturated fat	✓						✓	✓			
Whole grains											
Fish/shellfish											
Meat											
Red meat	✓										
Dietary pattern score						✓					
Processed meat											
Coffee											
Fibre							✓	✓	✓		
Folate							✓	✓	✓		
Sodium											
Vitamin E							✓				
Disease History											
MI or family history of MI					✓		✓	✓	✓		
CHD or family history of CHD											
CVD or family history of CVD										✓	
Medications											
ASA											
Other confounding variables not listed:	Sleep, bread, legumes, soy egg, PUFA	Maximal oxygen		Functional status, Health	Birth cohort, animal prod, radiation				Vitamin C, flavonoid, potassium		

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Study	Sharma, 2014[137]	Simila, 2013[138]	Sonestedt, 2015[139]	Sotomayer, 2019[140]	Steffen, 2003[141]	Stefler, 2016[142]	Strandhagen, 2000[143]	Takachi, 2008[144]	Tanaka, 2013[145]	Tucker, 2005[147]	Tognon, 2014[146]
No. of variables fully adjusted model	5	2	14	16	12	12	5	11	21	10	6
No. of multivariable models presented	1	1	3	4	3	1	2	2	3	3	1
Timing of measurement of confounding variables	BL	BL	BL	BL	BL	BL	BL	BL	BL	1961, biennially	BL
Pre-specified primary confounding variables											
Age		✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables											
Sex			✓	✓	✓	✓	✓	✓	✓	✓	✓
Smoking	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓
BMI	✓		✓	✓	✓			✓	✓	✓	✓
Physical activity	✓		✓	✓	✓	✓		✓	✓	✓	✓
Alcohol	✓		✓	✓	✓	✓		✓	✓	✓	
Blood pressure				✓	✓		✓	✓	✓		
Energy			✓		✓	✓		✓	✓	✓	
Diabetes	✓			✓				✓	✓		
Cholesterol				✓	✓		✓		✓		
Other Confounding variables											
Education			✓	✓	✓	✓					✓
Socioeconomic status				✓							
Menopause and/or hormone Use											
Region/location											
Randomization treatment		✓									
Ethnicity/nationality					✓						
Marital status						✓					
Study center								✓			
Survey season			✓								
Employment status											
Follow-up duration				✓						✓	
Dietary Intake											
Vitamin/supplement						✓		✓		✓	
Fruit and/or vegetable			✓			✓					
Saturated fat									✓	✓	
Whole grains			✓								
Fish/shellfish											
Meat			✓								
Red meat											
Dietary pattern score					✓						
Processed meat											
Coffee			✓								
Fibre											
Folate											
Sodium									✓		
Vitamin E											
Disease History											
MI or family history of MI											
CHD or family history of CHD											
CVD or family history of CVD											
Medications											
ASA											
Other confounding variables not listed:			Fermented milk	eGFR, proteinuria, primary renal disease, hsCRP		Birth cohort, house score			†		

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Table S2. *Page 11/11*

Study	Von Ruesten, 2013[148]	Vormund, 2015[149]	Wang, 2016[150]	Watkins, 2000[151]	Whiteman, 1999[152]	Yamada, 2011[153]	Yokoyama, 2000[154]	Yoshizaki, 2019[155]	Yu, 2014[156]	Zhang, 2011[157]	Zhang, 2011[158]
No. of variables fully adjusted model	11	8	7	17	3	11	9	17	13	17	11
No. of multivariable models presented	2	3	1	1	1	2	1	3	2	1	1
Timing of measurement of confounding variables	BL, q2-3y	BL	BL	BL	BL	BL	BL	BL	BL	BL	BL
Pre-specified primary confounding variables											
Age	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Pre-specified secondary confounding variables											
Sex	✓	✓	✓	✓	✓	✓	✓	✓		✓	✓
Smoking	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
BMI	✓	✓	✓	✓		✓	✓	✓	✓	✓	✓
Physical activity	✓			✓		✓	✓	✓	✓	✓	✓
Alcohol	✓		✓	✓		✓	✓	✓		✓	✓
Blood pressure	✓			✓		✓	✓	✓	✓	✓	✓
Energy								✓	✓	✓	
Diabetes				✓				✓	✓	✓	✓
Cholesterol	✓					✓	✓	✓	✓	✓	✓
Other Confounding variables											
Education	✓			✓		✓			✓	✓	
Socioeconomic status									✓	✓	
Menopause and/or hormone Use										✓	
Region/location		✓	✓			✓					
Randomization treatment											
Ethnicity/nationality		✓		✓							
Marital status		✓		✓		✓					
Study center							✓				
Survey season		✓	✓								
Employment status				✓			✓				
Follow-up duration											
Dietary Intake											
Vitamin/supplement	✓								✓	✓	
Fruit and/or vegetable	✓							✓			✓
Saturated fat										✓	
Whole grains											
Fish/shellfish								✓	✓		
Meat								✓			
Red meat				✓					✓		
Dietary pattern score											
Processed meat											
Coffee				✓							
Fibre											
Folate											
Sodium								✓			
Vitamin E											
Disease History											
MI or family history of MI											
CHD or family history of CHD							✓			✓	
CVD or family history of CVD											
Medications											
ASA				✓							
Other confounding variables not listed:				Stroke, Diuretics				Mental stress		Occupation, stroke	Stroke

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ASA - acetylsalicylic acid; BL - baseline; CHD – coronary heart disease; CRP – C-reactive protein; CVD – cardiovascular disease; GFR – glomerular filtration rate; FA – fatty acid; MI – myocardial infarction; M/PUFA - mono/poly-unsaturated fatty acids; Querc – quercetin supplement; q*X*y - confounding variables measured once every *X* years; WC – waist circumference.

*Tanaka et al. (2013) adjusted for the following additional confounding variables: dyslipidemia, HbA1c, oral antihyperglycemic agents, insulin, retinopathy, dietary cholesterol, dietary fat and Ω-3 and Ω-6 FA.

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Table S3: Newcastle-Ottawa Scale (NOS) for Assessing the Quality of Cohort Studies

Study	Selection*	Outcome†	Comparability‡	Total§
Adriouch, 2018[42]	3	2	2	7
Appleby, 2002[43]	1	1	1	3
Atkins, 2014[44]	3	3	1	7
Bahadoran, 2017[45]	2	1	0	3
Bazzano, 2002[46]	2	3	1	6
Belin, 2011[47]	3	3	2	8
Bendinelli, 2011[48]	3	3	2	8
Berard, 2017[49]	3	3	1	7
Bhupathiraju, 2013[50]	2	2	1	5
Bingham, 2008[51]	2	0	2	4
Blekkenhorst, 2017[52]	2	3	2	7
Bos, 2014[53]	2	3	2	7
Buijsse, 2008[54]	3	3	1	7
Buil-Cosiales, 2016[56]	3	3	2	8
Buil-Cosiales, 2017[55]	3	1	2	6
Cassidy, 2012[57]	2	2	2	6
Collin, 2019[58]	3	3	1	7
Conrad, 2018[59]	3	3	1	7
Dauchet, 2004[60]	3	3	2	8
Dauchet, 2010[61]	4	3	2	9
Du, 2016[62]	4	3	1	8
Du, 2017[63]	4	3	1	8
Elwood, 2013[64]	3	3	1	7
Eriksen, 2015[65]	3	3	2	8
Fitzgerald, 2012[66]	2	2	1	5
Fraser, 1992[67]	2	3	2	7
Gardener, 2011[68]	4	2	1	7
Gaziano, 1995[69]	2	3	1	6
Genkinger, 2004[70]	3	3	1	7
Gillman, 1995[71]	3	3	2	8
Goetz, 2016[72]	3	2	1	6
Goetz, 2016[73]	3	3	1	7
Gunge, 2017[74]	3	3	2	8

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Study	Selection*	Outcome†	Comparability‡	Total§
Gunnell, 2013[75]	3	1	2	6
Hansen, 2010[77]	3	3	2	8
Hansen, 2017[76]	2	3	2	7
Harriss, 2007[78]	3	3	2	8
Hertog, 1997[79]	2	3	2	7
Hirvonen, 2000[81]	2	3	2	7
Hirvonen, 2001[80]	2	3	2	7
Hjartaker, 2015[82]	2	3	1	6
Hodgson, 2016[83]	2	3	2	7
Holmberg, 2009[84]	2	3	0	5
Iso, 2007[85]	2	2	1	5
Jacques, 2015[86]	3	3	1	7
Johnsen, 2003[87]	3	2	2	7
Joshipura, 1999[88]	2	2	2	6
Joshipura, 2009[89]	2	3	2	7
Keli, 1996[90]	4	3	1	8
Kim, 2013[91]	1	3	0	4
Knekt, 1994[94]	4	3	1	8
Knekt, 1996[93]	2	3	2	7
Knekt, 2000[92]	4	3	2	9
Kobylecki, 2015[95]	3	3	2	8
Kondo, 2019[96]	3	3	1	7
Kvaavik, 2010[97]	4	3	1	8
Lai, 2015[98]	3	3	1	7
Larsson, 2009[99]	2	3	2	7
Larsson, 2013[100]	3	3	2	8
Leenders, 2013[102]	3	3	2	8
Leenders, 2014[101]	3	3	2	8
Lin, 2007[103]	2	2	2	6
Lin, 2017[104]	3	3	1	7
Liu, 2000[106]	2	3	2	7
Liu, 2001[105]	2	3	2	7
Mann, 1997[107]	2	3	1	6
Manuel, 2015[108]	4	3	1	8
Miller, 2017[109]	3	3	2	8

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Study	Selection*	Outcome†	Comparability‡	Total§
Mink, 2007[110]	3	3	2	8
Mizrahi, 2009[111]	4	3	2	9
Mori, 2018[112]	3	3	2	8
Mytton, 2018[113]	3	3	2	8
Nagura, 2009[114]	3	3	2	8
Nakamura, 2008[115]	2	3	2	7
Nechuta, 2010[116]	3	3	1	7
Neelakantan, 2018[117]	3	3	2	8
Ness, 2005[118]	3	3	1	7
Nothlings, 2008[119]	2	3	1	6
Okuda, 2015[120]	3	3	1	7
Oude Griep, 2010[121]	3	3	1	7
Oude Griep, 2011[123]	2	3	2	7
Oude Griep, 2011[122]	2	3	2	7
Oyebode, 2014[124]	3	3	1	7
Pham, 2007[125]	3	3	2	8
Rebello, 2014[126]	3	3	1	7
Rissanen, 2003[127]	2	3	2	7
Saglimbene, 2017[128]	1	0	0	1
Sahyoun, 1996[129]	1	3	1	5
Sauvaget, 2003[130]	2	3	2	7
Scheffers, 2019[131]	3	3	2	8
Sesso, 2003[132]	2	3	2	7
Sesso, 2003[134]	2	3	2	7
Sesso, 2007[133]	3	2	2	7
Shah, 2018[135]	3	3	2	8
Sharma, 2013[136]	3	2	0	5
Sharma, 2014[137]	3	2	0	5
Simila, 2013[138]	2	3	1	6
Sonestedt, 2015[139]	4	3	2	9
Sotomayer, 2019[140]	1	3	2	6
Steffen, 2003[141]	4	3	2	9
Stefler, 2016[142]	2	3	1	6
Strandhagen, 2000[143]	2	3	1	6
Takachi, 2008[144]	3	3	2	8

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Study	Selection*	Outcome†	Comparability‡	Total§
Tanaka, 2013[145]	2	3	2	7
Tognon, 2014[146]	3	3	1	7
Tucker, 2005[147]	2	3	1	6
Von Ruesten, 2013[148]	3	2	2	7
Vormund, 2015[149]	3	3	1	7
Wang, 2016[150]	1	3	1	5
Watkins, 2000[151]	3	3	2	8
Whiteman, 1999[152]	3	3	1	7
Yamada, 2011[153]	2	3	2	7
Yokoyama, 2000[154]	2	3	2	7
Yoshizaki, 2019[155]	3	3	2	8
Yu, 2014[156]	3	3	2	8
Zhang, 2011[157]	3	3	2	8
Zhang, 2011[158]	3	2	2	7

*Maximum 4 points awarded for representativeness of exposed cohort, selection of non-exposed cohort, exposure assessment, and demonstration outcome not present at baseline.

†Maximum 3 points awarded for outcome assessment, follow-up length, and adequacy of follow-up.

‡Maximum 2 points awarded for adjusting for the pre-specified primary confounding variable (age) and 5 of the 7 pre-specified secondary confounding variables (sex, family history of CVD, smoking, body mass index, blood pressure (or hypertension/medications), cholesterol (or dyslipidemia/medications) and presence of diabetes mellitus.

§A maximum of 9 points could be awarded.

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Table S4. GRADE Assessment for Fruits and Vegetables and Cardiovascular Disease Incidence

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Cardiovascular Disease Incidence (follow-up median 10 years)										
12	observational	not serious	not serious	not serious	serious[1]	undetected	dose-response gradient[2]	24,310/501,744 (4.9%)	0.93 (0.89, 0.96)	⨁⨁◯◯ **LOW**
Fruit Consumption on Cardiovascular Disease Incidence (follow-up median 10 years)										
16	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[3]	27,204/577,323 (4.7%)	0.91 (0.88, 0.95)	⨁⨁◯◯ **LOW**
Vegetable Consumption on Cardiovascular Disease Incidence (follow-up median 11 years)										
14	observational	not serious	not serious	not serious	serious[4]	undetected	none	22,810/539,683 (4.2%)	0.94 (0.90, 0.97)	⨁◯◯◯ **VERY LOW**
Berries Consumption on Cardiovascular Disease Incidence (follow-up median 10 years)										
1	observational	not serious	not serious[5]	serious[6]	serious[7]	undetected[8]	none	1,004/38,176 (2.6%)	1.27 (0.95, 1.71)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Cardiovascular Disease Incidence (follow-up median 10 years)										
6	observational	not serious	not serious	not serious	serious[9]	undetected[8]	dose-response gradient[10]	6,220/222,525 (2.8%)	0.88 (0.80, 0.96)	⨁⨁◯◯ **LOW**
Fruit Juice Consumption on Cardiovascular Disease Incidence (follow-up median 15 years)										
5	observational	not serious	not serious	not serious	serious[11]	undetected[8]	none	8,056/167,879 (4.8%)	1.00 (0.93, 1.07)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Cardiovascular Disease Incidence (follow-up median 8 years)										
5	observational	not serious	not serious	serious[12]	not serious	undetected[8]	dose-response gradient[13]	2,578/149,437 (1.7%)	0.76 (0.66, 0.88)	⨁⨁◯◯ **LOW**
Allium Vegetables Consumption on Cardiovascular Disease Incidence (follow-up median 7 years)										
2	observational	not serious	serious[14]	serious[15]	serious[16]	undetected[8]	none	808/40,814 (2.0%)	0.79 (0.57, 1.10)	⨁◯◯◯ **VERY LOW**
Cruciferous Vegetables Consumption on Cardiovascular Disease Incidence (follow-up median 9 years)										
7	observational	not serious	serious[17]	not serious	serious[18]	undetected[8]	none	6,824/273,878 (2.5%)	0.99 (0.90, 1.08)	⨁◯◯◯ **VERY LOW**
Green Leafy Vegetables Consumption on Cardiovascular Disease Incidence (follow-up median 7 years)										
5	observational	not serious	not serious	not serious	serious[19]	undetected[8]	dose-response gradient[20]	5,732/211,902 (2.7%)	0.87 (0.76, 0.99)	⨁⨁◯◯ **LOW**
Tomatoes Consumption on Cardiovascular Disease Incidence (follow-up median 9 years)										
2	observational	not serious	not serious	serious[21]	serious[22]	undetected[8]	none	841/55,452 (1.5%)	0.97 (0.78, 1.20)	⨁◯◯◯ **VERY LOW**

[1] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.89) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.96) crosses the MID.

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[2] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between total fruit and vegetable intake and incident CVD (p<0.001).

[3] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and incident CVD (p=0.004).

[4] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.90) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.97) crosses the MID.

[5] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.

[6] Downgrade for serious indirectness as evidence is based on 1 cohort of female health-professionals residing in the USA and may not be generalizable to different populations.

[7] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.95 to 1.27) includes both clinically important benefit (RR≤0.95) and harm (RR≥1.05).

[8] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[9] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.80) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.96) crosses the MID.

[10] Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between citrus fruit intake and CVD incidence (p=0.033).

[11] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.93 to 1.07) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[12] Downgrade for serious indirectness as evidence is based on a predominately (>78%) female population and may not be generalizable to different populations.

[13] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between pommes intake and incident CVD (p=0.043).

[14] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=85%, p=0.01), which could not be explored through sensitivity due to only 2 observations available.

[15] Downgrade for serious indirectness as evidence is based on a predominately (97%) female populations of which most are health professionals, and may not be generalizable to different populations.

[16] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.57) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.10) crosses the MID.

[17] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=52%, p=0.04). Although the removal of Buil-Cosiales et al. 2016 during sensitivity analysis did partially explain the heterogeneity (I^2=27%, p=0.22), the presence of residual heterogeneity could not be excluded.

[18] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.90 to 1.08) includes both clinically important benefit (RR≤0.95) and harm (RR≥1.05).

[19] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.76) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.

[20] Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between green leafy vegetables intake and CVD mortality (p=0.01)

[21] Downgrade for serious indirectness as evidence is based on a predominately (88%) female population and may not be generalizable to different populations.

[22] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.78 to 1.20) includes both clinically important benefit (RR≤0.95) and harm (RR≥1.05).

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Table S5. GRADE Assessment for Fruits and Vegetables and Cardiovascular Disease Mortality

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Cardiovascular Disease Mortality (follow-up median 11 years)										
14	observational	not serious	serious[1]	not serious	not serious	undetected	dose-response gradient[2]	17,439/798,391 (2.2%)	0.89 (0.85, 0.93)	⨁⨁◯◯ **LOW**
Fruit Consumption on Cardiovascular Disease Mortality (follow-up median 11 years)										
27	observational	not serious	serious[3]	not serious	not serious	undetected	dose-response gradient[4]	39,623/1,581,506 (2.5%)	0.88 (0.86, 0.91)	⨁⨁◯◯ **LOW**
Vegetable Consumption on Cardiovascular Disease Mortality (follow-up median 10 years)										
21	observational	not serious	serious[5]	not serious	not serious	undetected	dose-response gradient[6]	33,516/1,101,435 (3.0%)	0.87 (0.85, 0.90)	⨁⨁◯◯ **LOW**
Apricot Consumption on Cardiovascular Disease Mortality (follow-up median 1.5 years)										
1	observational	serious[7]	not serious[8]	serious[9]	not serious	undetected[10]	none	515/9,757 (5.3%)	1.84 (1.27, 2.67)	⨁◯◯◯ **VERY LOW**
Bananas Consumption on Cardiovacular Disease Mortality 16(follow-up median 20.3 years)										
1	observational	not serious	not serious[8]	serious[12]	serious[13]	undetected[10]	none	4,595/9,766 (47.1%)	1.06 (0.87, 1.29)	⨁◯◯◯ **VERY LOW**
Berries Consumption on Cardiovascular Disease Mortality (follow-up median 16 years)										
4	observational	not serious	not serious	serious[14]	serious[15]	undetected[10]	none	7,401/112,892 (6.6%)	0.97 (0.92, 1.03)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Cardiovascular Disease Mortality (follow-up median 17 years)										
3	observational	not serious	not serious[16]	serious[17]	serious[18]	undetected[10]	none	7,197/74,716 (9.6%)	0.95 (0.90, 1.02)	⨁◯◯◯ **VERY LOW**
Dried Fruit Consumption on Cardiovascular Disease Mortality (follow-up median 17 years)										
2	observational	not serious	not serious	not serious	serious[19]	undetected[10]	none	447/31,757 (1.4%)	0.93 (0.63, 1.37)	⨁◯◯◯ **VERY LOW**
Fruit Juice Consumption on Cardiovascular Disease Mortality (follow-up median 17 years)										
1	observational	not serious	not serious[8]	serious[20]	serious[21]	undetected[10]	none	286/30,458 (0.9%)	0.81 (0.58, 1.13)	⨁◯◯◯ **VERY LOW**
Grapes Consumption on Cardiovascular Disease Mortality (follow-up median 16.7 years)										
3	observational	not serious	not serious[22]	serious[23]	serious[24]	undetected[10]	none	7,197/74,716 (9.6%)	0.90 (0.81, 1.01)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Cardiovascular Disease Mortality (follow-up median 16 years)										
5	observational	not serious	not serious	serious[25]	not serious	undetected[10]	none	7,947/85,929 (9.2%)	0.86 (0.80, 0.92)	⨁◯◯◯ **VERY LOW**
Allium Vegetables Consumption on Cardiovascular Disease Mortality (follow-up median 15 years)										
1	observational	not serious	not serious[8]	serious[26]	not serious	undetected[10]	none	238/1,226 (19.4%)	0.33 (0.22, 0.49)	⨁◯◯◯ **VERY LOW**
Carrots Consumption on Cardiovacular Disease Mortality (follow-up median 18 years)										

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2	observational	not serious	not serious	serious[27]	serious[28]	undetected[10]	none	4,792/10,325 (46.4%)	0.92 (0.85, 1.01)	⨁◯◯◯ **VERY LOW**
Celery Consumption on Cardiovascular Disease Mortality (follow-up median 16 years)										
1	observational	not serious	not serious[8]	serious[29]	serious[30]	undetected[10]	none	2,316/34,492 (6.7%)	0.91 (0.83, 1.01)	⨁◯◯◯ **VERY LOW**
Cruciferous Vegetables Consumption on Cardiovascular Disease Mortality (follow-up median 12 years)										
7	observational	not serious	serious[31]	not serious	not serious	undetected[10]	none	13,081/187,730 (7.0%)	0.85 (0.82, 0.89)	⨁◯◯◯ **VERY LOW**
Green Leafy Vegetables Consumption on Cardiovascular Disease Mortality (follow-up median 21 years)										
5	observational	not serious	serious[32]	not serious	not serious	undetected[10]	none	6,661/40,893 (16.3%)	0.87 (0.81, 0.94)	⨁⨁◯◯ **LOW**
Tomatoes Consumption on Cardiovascular Disease Mortality (follow-up median 16 years)										
3	observational	not serious	not serious	serious[33]	serious[34]	undetected[9]	none	7,072/45,557 (15.5%)	0.98 (0.93, 1.04)	⨁◯◯◯ **VERY LOW**

[1] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=68%, p<0.001) which could not be explained by sensitivity analyses.

[2] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit and vegetable intake and CVD mortality (p<0.011). The MKSPLINE procedure indicated a departure from linearity (p<0.001) at a threshold of 4 servings/day as observed by visual inspection.

[3] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=79%, p<0.001), which could not be explained by sensitivity analyses.

[4] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and CVD mortality (p=0.005).

[5] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=59%, p<0.001), which could not be explained by sensitivity analyses.

[6] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and CVD mortality (p<0.001).

[7] Downgrade for serious risk of bias as the effect estimate is based on Saglimbene et al. 2017, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9)

[8] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available

[9] Downgrade for serious indirectness as evidence is based on 1 cohort of patients receiving hemodialysis and may not be generalizable to different populations.

[10] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[11] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available

[12] Downgrade for serious indirectness as evidence is based on 1 male cohort and may not be generalizable to different populations

[13] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.87 to 1.29) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[14] Downgrade for serious indirectness as evidence is based on a predominately (91%) female population and may not be generalizable to different populations.

[15] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.92) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 1.03) crosses the MID.

[16] No downgrade for inconsistency as the presence of inter-study heterogeneity (I^2=62%, p=0.05) was explained by the removal of Lai et al. 2015 (I^2=0%, p=0.63) during sensitivity analysis.

[17] Downgrade for serious indirectness as the evidence is based on a predominately (87%) female population and may not be generalizable to different populations.

[18] Downgrade for serious imprecision, as upper bound of the 95% CIs (RR 1.02) crosses the MID (RR<0.95).

[19] Downgrade for serious imprecision, as upper bound of the 95% CIs (RR 1.37) crosses the MID (RR<0.95).

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[20] Downgrade for serious indirectness as evidence is based on 1 female cohort residing in the United Kingdom and may not be generalizable to different populations.
[21] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.58 to 1.13) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).
[22] No downgrade for inconsistency as the presence of inter-study heterogeneity (I^2=61%, p=0.08) was explained by the removal of Lai et al. 2015 (I^2=0%, p=0.93) during sensitivity analysis.
[23] Downgrade for serious indirectness as evidence is based on a predominately (87%) female population and may not be generalizable to different populations.
[24] Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 1.01) crosses the MID (RR<0.95).
[25] Downgrade for serious indirectness as evidence is based on a predominately (87%) female population and may not be generalizable to different populations.
[26] Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.
[27] Downgrade for serious indirectness as evidence is based on 2 male cohorts and may not be generalizable to different populations.
[28] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 1.01) crosses the MID.
[29] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available
[30] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.76) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.
[31] Downgrade for serious inconsistency as there was evidence for substantial inter-study heterogeneity (I^2=86%, p<0.00001), which could not be explained by sensitivity analyses.
[32] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=88%, p<0.00001), which could not be explained by sensitivity analyses.
[33] Downgrade for serious indirectness as evidence is based on only 3 isolated geographical regions (Norway and Massachusetts and Iowa, USA) and may not be generalizable to different populations.
[34] Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 1.04) includes crosses the MID (RR<0.95).

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Table S6. GRADE Assessment for Fruits and Vegetables and Coronary Heart Disease Incidence

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Coronary Heart Disease Incidence (follow-up median 10 years)										
19	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[1]	17,987/619,182 (2.9%)	0.88 (0.83, 0.92)	⨁⨁⨁◯ **MODERATE**
Fruit Consumption on Coronary Heart Disease Incidence (follow-up median 10 years)										
20	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[2]	23,856/1,170,021 (2.0%)	0.88 (0.84, 0.92)	⨁⨁⨁◯ **MODERATE**
Vegetable Consumption on Coronary Heart Disease Incidence (follow-up median 10 years)										
18	observational	not serious	not serious[3]	not serious	serious[4]	undetected	dose-response gradient[5]	17,172/696,330 (2.5%)	0.92 (0.87, 0.96)	⨁⨁◯◯ **LOW**
Bananas Consumption on Coronary Heart Disease Incidence (follow-up median 7.6 years)										
1	observational	not serious	not serious[6]	serious[7]	serious[8]	undetected[9]	none	365/122,635 (0.3%)	0.76 (0.56, 1.02)	⨁◯◯◯ **VERY LOW**
Berries Consumption on Coronary Heart Disease Incidence (follow-up median 8 years)										
4	observational	not serious	serious[10]	not serious	serious[11]	undetected[9]	none	2,233/100,296 (2.2%)	0.94 (0.82, 1.09)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Coronary Heart Disease Incidence (follow-up median 9 years)										
10	observational	not serious	not serious	not serious	serious[12]	undetected	dose-response gradient[12]	8,333/364,978 (2.3%)	0.91 (0.85, 0.98)	⨁⨁◯◯ **LOW**
Fruit Juice Consumption on Coronary Heart Disease Incidence (follow-up median 15 years)										
4	observational	not serious	not serious	not serious	serious[14]	undetected[9]	none	7,589/109,898 (6.9%)	0.99 (0.92, 1.07)	⨁◯◯◯ **VERY LOW**
Grapes Consumption on Coronary Heart Disease Incidence (follow-up median 12 years)										
1	observational	not serious	not serious[6]	serious[15]	serious[16]	undetected[9]	none	8,333/364,978 (2.3%)	0.91 (0.85, 0.98)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Coronary Heart Disease Incidence (follow-up median 8 years)										
8	observational	not serious	not serious	not serious	serious[17]	undetected[9]	none	4,886/371,684 (1.3%)	0.90 (0.84, 0.97)	⨁◯◯◯ **VERY LOW**
Watermelon Consumption on Coronary Heart Disease Incidence (follow-up median 7.6 years)										
1	observational	not serious	not serious	serious[16]	serious[19]	undetected[9]	none	365/122,635 (0.3%)	0.87 (0.64, 1.18)	⨁◯◯◯ **VERY LOW**
Allium Vegetables Consumption on Coronary Heart Disease Incidence(follow-up median 10 years)										
5	observational	not serious	not serious	not serious	serious[20]	undetected[9]	none	1,734/210,964 (0.8%)	0.93 (0.80, 1.09)	⨁◯◯◯ **VERY LOW**
Cruciferous Vegetables Consumption on Coronary Heart Disease Incidence(follow-up median 11 years)										
8	observational	not serious	not serious	not serious	not serious	undetected[9]	none	9,383/347,453 (2.7%)	1.01 (0.95, 1.07)	⨁⨁◯◯ **LOW**

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Green Leafy Vegetables Consumption on Coronary Heart Disease Incidence(follow-up median 16 years)										
5	observational	not serious	not serious	not serious	not serious	undetected[9]	dose-response gradient[21]	6,696/170,250 (3.9%)	0.82 (0.76, 0.89)	⨁⨁⨁◯ **MODERATE**
Tomatoes Consumption on Coronary Heart Disease Incidence(follow-up median 8 years)										
3	observational	not serious	not serious	serious[22]	serious[23]	undetected[9]	none	1,283/134,494 (1.0%)	0.80 (0.57, 1.13)	⨁◯◯◯ **VERY LOW**

[1] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit and vegetable intake and coronary heart disease incidence (CHD) ($p<0.001$).

[2] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and CHD ($p=0.005$).

[3] No downgrade for inconsistency as the presence of inter-study heterogeneity ($I^2=53\%$, $p=0.002$) was explained by the removal of Dauchet et al. 2010 ($I^2=0\%$, $p=0.5$)

[4] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.87) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.96) crosses the MID.

[5] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between vegetable intake and CHD ($p<0.001$).

[6] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available

[7] Downgrade for serious indirectness as evidence is based on only 1 geographical regions (China) and may not be generalizable to different populations.

[8] Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 1.02) crosses the MID (RR<0.95).

[9] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[10] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity ($I^2=74\%$, $p=0.008$), which could not be explained by sensitivity analyses.

[11] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.82 to 1.09) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[12] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.

[13] Upgrade for a dose-response gradient, as the MKSPLINE analysis indicated a significant non-linear inverse relationship between citrus intake and incident CHD ($p=0.005$).

[14] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.92 to 1.07) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[15] Downgrade for serious indirectness as evidence is based on 1 female cohort of health professionals and may not be generalizable to different populations.

[16] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.

[17] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.84) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.97) crosses the MID.

[18] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.64 to 1.18) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[19] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.80 to 1.09) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[20] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and CVD mortality ($p=0.002$). The MKSPLINE procedure indicated a departure from linearity ($p=0.004$) at threshold of 0.5 servings/day as observed by visual inspection.

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[21] Downgrade for serious indirectness as the evidence is based only on female populations, predominately (77.9%) of which reside in USA, and may not be generalizable to different populations.

[22] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.57 to 1.13) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05)

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Table S7. GRADE Assessment for Fruits and Vegetables and Coronary Heart Disease Mortality

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Coronary Heart Disease Mortality (follow-up median 18 years)										
5	observational	not serious	not serious	not serious	not serious	undetected[1]	dose-response gradient[2]	3,240/489,635 (0.7%)	0.81 (0.72, 0.92)	⨁⨁⨁◯ **MODERATE**
Fruit Consumption on Coronary Heart Disease Mortality (follow-up median 13 years)										
21	observational	not serious	serious[3]	not serious	not serious	undetected	dose-response gradient[4]	14,786/1,398,863 (1.1%)	0.86 (0.82, 0.90)	⨁⨁◯◯ **LOW**
Vegetable Consumption on Coronary Heart Disease Mortality (follow-up median 13 years)										
18	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[5]	26,007/1,968,325 (1.3%)	0.86 (0.83, 0.89)	⨁⨁⨁◯ **MODERATE**
Bananas Consumption on Coronary Heart Disease Mortality (follow-up median 20 years)										
1	observational	not serious	not serious[6]	serious[7]	serious[8]	undetected[1]	none	2,384/9,964 (4.9%)	1.04 (0.81, 1.34)	⨁◯◯◯ **VERY LOW**
Berries Consumption on Coronary Heart Disease Mortality (follow-up median 17 years)										
5	observational	not serious	not serious	not serious	serious[9]	undetected[1]	none	5,141/105,420 (4.9%)	0.98 (0.91, 1.05)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Coronary Heart Disease Mortality (follow-up median 16 years)										
6	observational	not serious	not serious	serious[10]	serious[11]	undetected[1]	none	5,309/180,574 (2.9%)	0.91 (0.85, 0.96)	⨁◯◯◯ **VERY LOW**
Dried Fruit Consumption on Coronary Heart Disease Mortality (follow-up median 17 years)										
1	observational	not serious	not serious[6]	serious[12]	serious[13]	undetected[1]	none	38/30,458 (0.1%)	0.79 (0.47, 1.31)	⨁◯◯◯ **VERY LOW**
Fruit Juice Consumption on Coronary Heart Disease Mortality (follow-up median 17 years)										
3	observational	serious[14]	not serious	not serious[15]	serious[16]	undetected[1]	none	1,249/141,170 (0.9%)	0.87 (0.75, 1.01)	⨁◯◯◯ **VERY LOW**
Grapes Consumption on Coronary Heart Disease Mortality (follow-up median 17 years)										
3	observational	not serious	not serious	serious[17]	serious[18]	undetected[1]	none	2,846/106, 782 (2.7%)	0.97 (0.77, 1.21)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Coronary Heart Disease Mortality (follow-up median 19 years)										
5	observational	not serious	not serious	serious[19]	not serious	undetected[1]	none	4,650/146,407 (3.2%)	0.84 (0.76, 0.92)	⨁◯◯◯ **VERY LOW**
Allium Vegetables Consumption on Coronary Heart Disease Mortality (follow-up median 15 years)										
4	observational	not serious	serious[20]	serious[21]	not serious	undetected[1]	none	1,280/75,434 (1.7%)	0.67 (0.57, 0.79)	⨁◯◯◯ **VERY LOW**
Carrots Consumption on Coronary Heart Disease Mortality (follow-up median 13years)										

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1	observational	not serious	not serious[6]	serious[22]	serious[23]	undetected[1]	none	64/10,802 (0.6%)	0.76 (0.37, 1.58)	⨁◯◯◯ **VERY LOW**
Celery Consumption on Coronary Heart Disease Mortality (follow-up median 16 years)										
1	observational	not serious	not serious[24]	serious[25]	serious[26]	undetected[1]	none	1,329/34,492 (3.9%)	0.92 (0.80, 1.06)	⨁◯◯◯ **VERY LOW**
Cruciferous Vegetables Consumption on Coronary Heart Disease Mortality (follow-up median 16 years)										
6	observational	serious[27]	serious[28]	not serious	serious[29]	undetected[1]	none	7,420/296,772 (2.5%)	0.91 (0.85, 0.98)	⨁◯◯◯ **VERY LOW**
Green Leafy Vegetables Consumption on Coronary Heart Disease Mortality (follow-up median 17 years)										
5	observational	serious[30]	not serious	not serious	not serious	undetected[1]	none	4,591/148,133 (3.1%)	0.86 (0.78. 0.94)	⨁◯◯◯ **VERY LOW**
Tomatoes Consumption on Coronary Heart Disease Mortality (follow-up median 16 years)										
3	observational	serious[31]	not serious	not serious	serious[32]	undetected[1]	none	3,657/175,088 (2.1%)	0.92 (0.82, 1.04)	⨁◯◯◯ **VERY LOW**

[1] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[2] Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between fruit and vegetable intake and CHD mortality (p=0.044)

[3] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=62%, p<0.0001). Although heterogeneity could be partially explained by the removal of Du et al. 2017 (I^2=44%, p=0.01) and Hjartaker et al. 2015 (I^2=46%, p=0.007) during sensitivity analyses, the presence of residual heterogeneity could not be excluded.

[4] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and CHD mortality (p<0.001).

[5] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between vegetable intake and CHD mortality (p=0.005).

[6] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.

[7] Downgrade for serious indirectness as evidence is based on 1 male cohort and may not be generalizable to different populations.

[8] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.81 to 1.34) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[9] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.91 to 1.05) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[10] Downgrade for serious indirectness as evidence is based on a predominately (≥69.6%) female populations and may not be generalizable to different populations.

[11] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.96) crosses the MID.

[12] Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.

[13] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.47 to 1.31) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[14] Downgrade for serious risk of bias as 56% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

[15] No downgrade for inconsistency as the presence of inter-study heterogeneity (I^2=71%, p=0.02) was explained by the removal of Collin et al. 2019 (I^2=0%, p=0.45).

[16] Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 1.01) crosses the MID (RR<0.95).

[17] Downgrade for serious indirectness as evidence is based on a predominately (91%) female population of which the majority are health professionals and may not be generalizable to different populations.

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[18] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.77 to 1.21) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[19] Downgrade for serious indirectness as evidence is based on a predominately (82.1%) female populations and may not be generalizable to different populations.

[20] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=88%, p<0.00001). Although heterogeneity could be partially explained by the removal of Blekkenhorst et al. 2017 (I^2=47%, p=0.13) during sensitivity analyses, the presence of residual heterogeneity could not be excluded.

[21] Downgrade for serious indirectness as evidence is based on a predominately (95.4%) female populations and may not be generalizable to different populations.

[22] Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.

[23] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.37 to 1.58) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[24] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.

[25] Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.

[26] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.80 to 1.06) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[27] Downgrade for serious risk of bias as 39.3% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9).

[28] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=88%, p<0.00001) which could not be explained by sensitivity analyses.

[29] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.

[30] Downgrade for serious risk of bias as 36.8% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9)

[31] Downgrade for serious risk of bias as 48.0% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9)

[32] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.82) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 1.04) crosses the MID.

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Table S8. GRADE Assessment for Fruits and Vegetables and Stroke Incidence

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Stroke Incidence (follow-up median 9 years)										
14	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[1]	11,091/532,667 (2.1%)	0.82 (0.77, 0.88)	⨁⨁⨁◯ **MODERATE**
Fruit Consumption on Stroke Incidence (follow-up median 14 years)										
17	observational	not serious	not serious	not serious	not serious	undetected	dose-response gradient[2]	43,702/987,983 (4.4%)	0.82 (0.79, 0.85)	⨁⨁⨁◯ **MODERATE**
Vegetable Consumption on Stroke Incidence (follow-up median 14 years)										
16	observational	not serious	serious[3]	not serious	not serious	undetected	dose-response gradient[4]	13,607/564,531 (2.4%)	0.82 (0.83, 0.93)	⨁⨁⨁◯ **MODERATE**
Berries Consumption on Stroke Incidence (follow-up median 10 years)										
4	observational	not serious	not serious[5]	not serious	serious[6]	undetected[7]	none	5,967/143,662 (4.2%)	1.03 (0.94, 1.13)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Stroke Incidence (follow-up median 11 years)										
8	observational	not serious	serious[8]	not serious	not serious	undetected[7]	dose-response gradient[9]	7,142/225,613 (3.2%)	0.88 (0.82, 0.94)	⨁⨁◯◯ **LOW**
Fruit Juice Consumption on Stroke Incidence (follow-up median 11 years)										
4	observational	not serious	not serious[10]	not serious	serious[11]	undetected[7]	none	1,705/148,839 (1.2%)	0.82 (0.68, 0.99)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Stroke Incidence (follow-up median 14 years)										
5	observational	not serious	not serious	not serious	not serious	undetected[7]	dose-response gradient[12]	7,364/146,723 (5.0%)	0.89 (0.84, 0.95)	⨁⨁⨁◯ **MODERATE**
Allium Vegetables Consumption on Stroke Incidence (follow-up median 28 years)										
2	Observational	not serious	not serious	serious[13]	serious[14]	undetected[7]	none	4,912/84,169 (5.8%)	0.89 (0.80, 0.99)	⨁◯◯◯ **VERY LOW**
Cruciferous Vegetables Consumption on Stroke Incidence (follow-up median 12 years)										
6	observational	not serious	serious[15]	not serious	serious[16]	undetected[7]	none	7,706/255,726 (3.0%)	0.98 (0.91, 1.05)	⨁◯◯◯ **VERY LOW**
Green Leafy Vegetables Consumption on Stroke Incidence (follow-up median 9 years)										
4	observational	not serious	not serious	not serious	serious[17]	undetected[7]	dose-response gradient[18]	4,798/196,456 (2.4%)	0.88 (0.79, 0.98)	⨁⨁◯◯ **LOW**
Tomatoes Consumption on Stroke Incidence (follow-up median 7 years)										
1	observational	not serious	not serious[19]	serious[20]	not serious	undetected[7]	dose-response gradient[21]	247/38,445 (0.6%)	0.20 (0.05, 0.82)	⨁⨁◯◯ **LOW**

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[1] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit and vegetable intake and stroke incidence (p=0.002).

[2] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and stroke incidence (p<0.001).

[3] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=50%, p=0.006) that could not be explained during sensitivity analysis.

[4] Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between vegetable intake and stroke incidence with a departure from linearity at 1.5 servings/day (p=0.012)

[5] No downgrade for inconsistency as the presence of inter-study heterogeneity (I^2=50%, p=0.08) was explained by the removal of Hirvonen et al. 2000 – cerebral infraction (I^2=0%, p=0.41)
during sensitivity analysis.

[6] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.94 to 1.13) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05)

[7] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[8] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=51%, p=0.04). Although the removal of Larsson et al. 2013 (I^2=37%, p=0.14) or Yamada et al. 2011 (I^2=39%, p=0.12) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

[9] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between citrus fruit intake and stroke incidence (p=0.033) and an MKSPLINE analysis revealed a significant non-linear inverse relationship between citrus fruit intake and stroke incidence (p=0.039).

[10] No downgrade for inconsistency as the presence of inter-study heterogeneity (I^2=73%, p=0.02) was explained by the removal of Scheffers et al. 2019 (I^2=0%, p=0.47)

[11] Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 0.99) crosses the MID (RR<0.95).

[12] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between pommes intake and stroke incidence (p=0.003). MKSPLINE analyses could not be conducted due to small sample size.

[13] Downgrade for serious indirectness as evidence is based on cohorts residing in Northern Europe and may not be generalizable to different populations.

[14] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.80) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.

[15] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=62%, p=0.02). Although the removal of Larsson et al. 2013 (during sensitivity analysis did partially explain the heterogeneity (I^2=40%, p=0.16), the presence of residual heterogeneity could not be excluded.

[16] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.91 to 1.05) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[16] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.79) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.

[17] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between green leafy vegetable intake and stroke incidence (p=0.008). MKSPLINE analyses could not be conducted due to small sample size.

[18] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.

[19] Downgrade for serious indirectness as evidence is based on only 1 cohort of females for USA and may not be generalizable to different populations.

[20] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between tomato intake and stroke incidence (p=0.002). MKSPLINE analyses could not be conducted due to small sample size.

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Table S9. GRADE Assessment for Fruits and Vegetables and Stroke Mortality

Quality Assessment								Study Event Rates (%)	Relative Risk (95% CI)	Certainty
No. of Cohorts	Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
Fruit and Vegetable Consumption on Stroke Mortality (follow-up median 19 years)										
6	observational	not serious	not serious	not serious	not serious	undetected[1]	dose-response gradient[2]	3,051/499,732 (0.6%)	0.73 (0.65, 0.81)	⨁⨁⨁◯ **MODERATE**
Fruit Consumption on Stroke Mortality (follow-up median 20 years)										
14	observational	not serious	serious[3]	not serious	not serious	undetected	dose-response gradient[4]	10,899/1,282,756 (0.8%)	0.87 (0.84, 0.91)	⨁⨁◯◯ **LOW**
Vegetable Consumption on Stroke Mortality (follow-up median 15 years)										
12	observational	not serious	serious[5]	not serious	serious[6]	undetected	dose-response gradient[7]	7,551/780,441 (1.0%)	0.94 (0.90, 0.99)	⨁⨁◯◯ **LOW**
Bananas Consumption on Stroke Mortality (follow-up median 20 years)										
1	observational	not serious	not serious[8]	serious[9]	serious[10]	undetected[1]	none	1,.34/9,766 (10.6%)	1.04 (0.70, 1.54)	⨁◯◯◯ **VERY LOW**
Berries Consumption on Stroke Mortality (follow-up median 19 years)										
2	observational	not serious	not serious	serious[11]	serious[12]	undetected[1]	none	1,182/40,224 (2.9%)	0.97 (0.82, 1.15)	⨁◯◯◯ **VERY LOW**
Citrus Fruit Consumption on Stroke Mortality (follow-up median 20 years)										
4	observational	serious[13]	serious[14]	not serious	not serious	undetected[1]	dose-response gradient[15]	3,869/145,204 (2.7%)	0.90 (0.86, 0.95)	⨁⨁◯◯ **LOW**
Dried Fruit Consumption on Stroke Mortality (follow-up median 17 years)										
1	observational	not serious	not serious	serious[16]	serious[17]	undetected[1]	none	152/30,458 (0.5%)	0.95 (0.80, 1.13)	⨁◯◯◯ **VERY LOW**
Fruit Juice Consumption on Stroke Mortality (follow-up median 17 years)										
2	observational	serious[18]	not serious	not serious	not serious	undetected[1]	dose-response gradient[19]	2,232/128,270 (1.7%)	0.67 (0.60, 0.76)	⨁⨁◯◯ **LOW**
Grapes Consumption on Stroke Mortality (follow-up median 19 years)										
2	observational	not serious	not serious	serious[20]	serious[21]	undetected[1]	none	1,182/40224 (2.9%)	0.74 (0.53, 1.02)	⨁◯◯◯ **VERY LOW**
Pommes Consumption on Stroke Mortality (follow-up median 17 years)										
3	observational	not serious	not serious	serious[22]	serious[23]	undetected[1]	none	1,651/74,716 (2.2%)	0.91 (0.77, 1.09)	⨁◯◯◯ **VERY LOW**
Allium Vegetable Consumption on Stroke Mortality (follow-up median 19 years)										
2	observational	not serious	serious[24]	not serious	serious[25]	undetected[1]	none	544/3,671 (14.8%)	0.99 (0.79, 1.24)	⨁◯◯◯ **VERY LOW**
Carrots Consumption on Stroke Mortality (follow-up median 20 years)										
1	observational	not serious	not serious[8]	serious[9]	not serious	undetected[1]	dose-response gradient[26]	1,034/9,766 (10.6%)	0.54 (0.48, 0.61)	⨁⨁◯◯ **LOW**

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Cruciferous Vegetables Consumption on Stroke Mortality (follow-up median 20 years)										
5	observational	serious[27]	not serious	not serious	serious[28]	undetected[1]	none	5,065/195,452 (2.6%)	0.92 (0.85, 1.01)	⨁◯◯◯ **VERY LOW**
Green Leafy Vegetables Consumption on Stroke Mortality (follow-up median 21 years)										
4	observational	serious[29]	serious[30]	not serious	serious[31]	undetected[1]	dose-response gradient[32]	4,103/126,971 (3.2%)	0.90 (0.83, 0.97)	⨁⨁◯◯ **LOW**
Tomatoes Consumption on Stroke Mortality (follow-up median 20 years)										
2	observational	serious[33]	not serious	not serious	serious[33]	undetected[1]	none[34]	3,107/108,260 (2.9%)	1.03 (0.94, 1.12)	⨁◯◯◯ **VERY LOW**

[1] No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).

[2] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit and vegetable intake and stroke mortality (p=0.005).

[3] Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I^2=75%, p<0.00001) which could not be explained by sensitivity analyses.

[4] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and stroke mortality (p<0.001) and an MKSPLINE analysis revealed a significant non-linear inverse relationship between fruit intake and stroke mortality (p<0.001)

[5] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=62%, p=0.0010). Although the removal of Wang et al. 2013 (I^2=43%, p=0.05) or Leeanders et al. 2014 (I^2=48%, p=0.02) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

[6] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.90) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.

[7] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between vegetable intake and stroke mortality (p=0.025).

[8] No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.

[9] Downgrade for serious indirectness as evidence is based on 1 male cohort and may not be generalizable to different populations

[10] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.70 to 1.54) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[11] Downgrade for serious indirectness as evidence is based on a predominately (76%) female population and may not be generalizable to different populations.

[12] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.82) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.15) crosses the MID.

[13] Downgrade for serious risk of bias as 75.3% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

[14] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=82%, p=0.0001). Although the removal of Wang et al. 2016 (I^2=40%, p=0.17) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

[15] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between citrus fruit intake and stroke mortality (p<0.001).

[16] Downgrade for serious indirectness as evidence is based on one female population and may not be generalizable to different populations.

[17] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.80 to 1.13) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[18]Downgrade for serious risk of bias as 62% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

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[19] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit juice intake and CHD mortality (p=0.002). MKSPLINE analyses could not be conducted due to small sample size.

[20] Downgrade for serious indirectness as evidence is based on a predominately (76%) female population and may not be generalizable to different populations.

[21] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.53 to 1.02) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[22] Downgrade for serious indirectness as evidence is based on a predominately (87%) female population and may not be generalizable to different populations.

[23] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.77) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.09) crosses the MID.

[24] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=96%, p<0.00001).

[25] Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.79 to 1.24) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).

[26] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between carrots intake and stroke mortality (p<0.001).

[27] Downgrade for serious risk of bias as 79.4% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

[28] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.01) crosses the MID.

[29] Downgrade for serious risk of bias as 50.0% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

[30] Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I^2=50%, p=0.09). Although the removal of Appleby et al. 2002 (I^2=36%, p=0.20) or Wang et al. 2016 (I^2=25%, p=0.05) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

[31] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.83) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.97) crosses the MID.

[32] Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between green leafy vegetable intake and CHD mortality (p=0.032). MKSPLINE analyses could not be conducted due to small sample size.

[33] Downgrade for serious risk of bias as 60.4% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

[34] Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.94) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.12) crosses the MID.

[35] Dose-response gradient could not be assessed due to insufficient dose ranges available to determine the presence of a linear/non-linear dose response.

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Figure S1. Relation between total fruit and vegetable intake and cardiovascular disease incidence (highest vs. lowest level of intake).

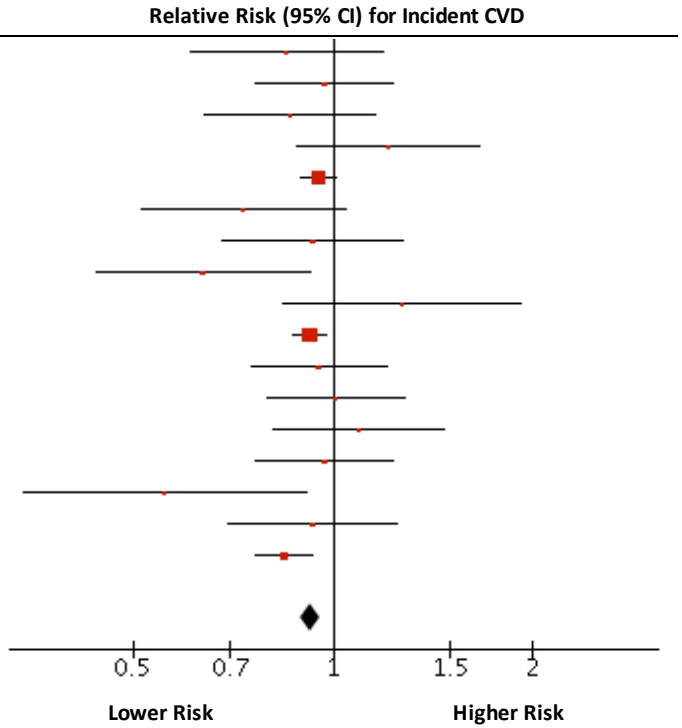
TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	418	1.1%	0.85 [0.61, 1.19]
Japan Public Health Center - Takachi 2008 - M	35,909	830	2.1%	0.97 [0.77, 1.23]
Japan Public Health Center - Takachi 2008 - F	41,982	556	1.4%	0.86 [0.64, 1.15]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	1.2%	1.21 [0.88, 1.65]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	34.3%	0.95 [0.90, 1.01]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	1.0%	0.73 [0.52, 1.04]
PRIME - Dauchet 2010 - former smokers	3,353	237	1.2%	0.93 [0.68, 1.28]
PRIME - Dauchet 2010 - current smokers	2,297	230	0.9%	0.64 [0.44, 0.93]
PRIME - Dauchet 2010 - never smokers	2,410	145	0.7%	1.27 [0.84, 1.92]
WHI-OS - Belin 2011	93,676	6,006	34.3%	0.92 [0.87, 0.98]
Carphilly Cohort - Elwood 2013	2,235	752	2.1%	0.95 [0.75, 1.20]
British Regional Heart - Atkins 2014	3,328	582	2.1%	1.01 [0.80, 1.28]
SABRE - Eriksen 2015 - European	1,090	225	1.4%	1.09 [0.82, 1.47]
SABRE - Eriksen 2015 - South Asian	1,006	346	2.1%	0.97 [0.77, 1.23]
PREDIMED- Buil-Cosiales 2016	7,216	342	0.5%	0.56 [0.34, 0.91]
PURE - Miller 2017	135,335	4,784	1.4%	0.93 [0.69, 1.25]
EPIC Norfolk - Mytton 2018	22,992	4,965	12.3%	0.84 [0.76, 0.93]
Total (95% CI)	**501,744**	**24,310**	**100.0%**	**0.93 [0.89, 0.96]**

Heterogeneity: Chi² = 21.52, df = 16 (P = 0.16); I² = 26%

Test for overall effect: Z = 4.42 (P < 0.00001)



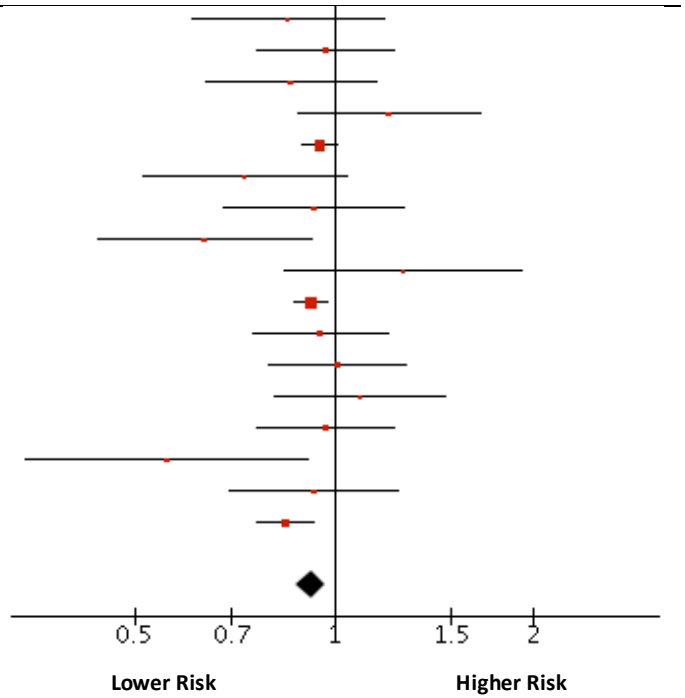
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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	418	2.3%	0.85 [0.61, 1.19]
Japan Public Health Center - Takachi 2008 - M	35,909	830	4.3%	0.97 [0.77, 1.23]
Japan Public Health Center - Takachi 2008 - F	41,982	556	2.9%	0.86 [0.64, 1.15]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	2.6%	1.21 [0.88, 1.65]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	22.6%	0.95 [0.90, 1.01]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	2.1%	0.73 [0.52, 1.04]
PRIME - Dauchet 2010 - former smokers	3,353	237	2.6%	0.93 [0.68, 1.28]
PRIME - Dauchet 2010 - current smokers	2,297	230	1.9%	0.64 [0.44, 0.93]
PRIME - Dauchet 2010 - never smokers	2,410	145	1.5%	1.27 [0.84, 1.92]
WHI-OS - Belin 2011	93,676	6,006	22.6%	0.92 [0.87, 0.98]
Carphilly Cohort - Elwood 2013	2,235	752	4.3%	0.95 [0.75, 1.20]
British Regional Heart - Atkins 2014	3,328	582	4.3%	1.01 [0.80, 1.28]
SABRE - Eriksen 2015 - European	1,090	225	2.9%	1.09 [0.82, 1.47]
SABRE - Eriksen 2015 - South Asian	1,006	346	4.3%	0.97 [0.77, 1.23]
PREDIMED- Buil-Cosiales 2016	7,216	342	1.1%	0.56 [0.34, 0.91]
PURE - Miller 2017	135,335	4,784	2.9%	0.93 [0.69, 1.25]
EPIC Norfolk - Mytton 2018	22,992	4,965	15.0%	0.84 [0.76, 0.93]
Total (95% CI) [Random Effects]	**501,744**	**24,310**	**100.00%**	**0.92 [0.88, 0.97]**

Heterogeneity: Tau² = 0.00; Chi² = 21.52, df = 16 (P = 0.16); I² = 26%

Test for overall effect: Z = 3.02 (P = 0.002)



All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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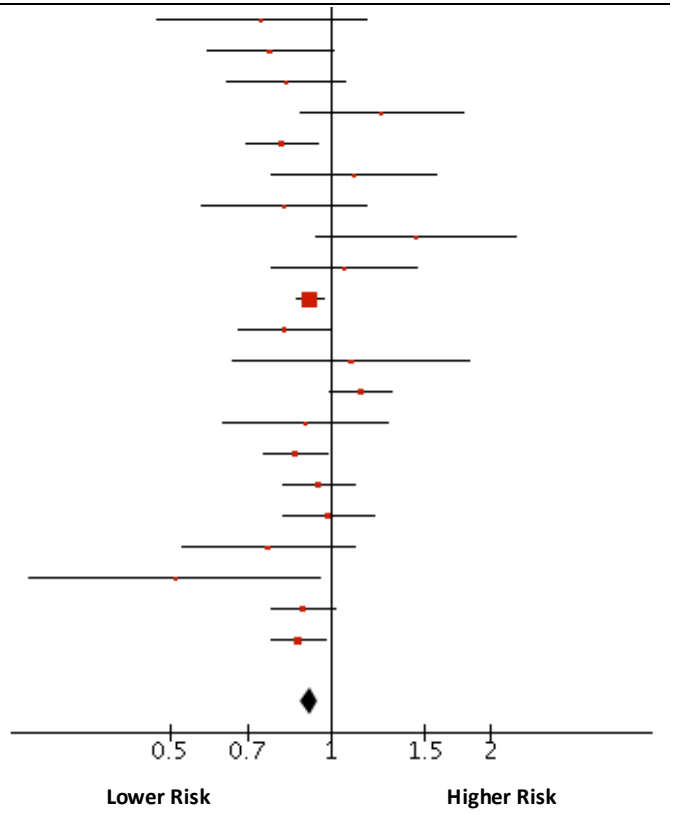
FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	0.7%	0.74 [0.47, 1.16]
Japan Public Health Center - Takachi 2008 - F	41,982	556	1.8%	0.77 [0.59, 1.01]
Japan Public Health Center - Takachi 2008 - M	35,909	830	2.1%	0.83 [0.64, 1.07]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	1.1%	1.25 [0.88, 1.77]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	5.4%	0.81 [0.69, 0.95]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	1.1%	1.11 [0.78, 1.57]
PRIME - Dauchet 2010 - current smokers	2,297	230	1.1%	0.82 [0.58, 1.17]
PRIME - Dauchet 2010 - never smokers	2,410	145	0.7%	1.45 [0.94, 2.23]
PRIME - Dauchet 2010 - former smokers	3,353	237	1.4%	1.06 [0.78, 1.45]
WHI-OS - Belin 2011	93,676	6,006	38.6%	0.91 [0.86, 0.97]
WHS - Fitzgerald 2012	34,827	1,094	3.5%	0.82 [0.67, 1.00]
British Women's Heart & Health - Kim 2013	3,080	329	0.5%	1.09 [0.66, 1.82]
EPIC Potsdam - Von Ruesten 2013	23,531	363	7.1%	1.14 [0.99, 1.31]
British Regional Heart - Atkins 2014	3,328	582	1.1%	0.90 [0.63, 1.27]
MONICA Danish - Tognon 2014	1,849	755	7.1%	0.86 [0.75, 0.99]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	1,694	5.4%	0.95 [0.81, 1.11]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	1,227	3.5%	0.99 [0.81, 1.20]
PREDIMED- Buil-Cosiales 2016	7,216	342	1.0%	0.76 [0.53, 1.11]
SUN - Buil-Cosiales - 2017	17,007	112	0.3%	0.51 [0.27, 0.96]
PURE - Miller 2017	135,335	4,784	7.1%	0.89 [0.77, 1.02]
EPIC NL and MORGEN - Scheffers 2019	34,560	3,801	9.7%	0.87 [0.77, 0.98]
Total (95% CI)	**577,323**	**27,205**	**100.0%**	**0.91 [0.88, 0.95]**

Heterogeneity: Chi² = 33.12, df = 20 (P = 0.03); I² = 40%

Test for overall effect: Z = 4.88 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	1.5%	0.74 [0.47, 1.16]
Japan Public Health Center - Takachi 2008 - F	41,982	556	3.5%	0.77 [0.59, 1.01]
Japan Public Health Center - Takachi 2008 - M	35,909	830	3.9%	0.83 [0.64, 1.07]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	2.3%	1.25 [0.88, 1.77]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	7.3%	0.81 [0.69, 0.95]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	2.3%	1.11 [0.78, 1.57]
PRIME - Dauchet 2010 - current smokers	2,297	230	2.3%	0.82 [0.58, 1.17]
PRIME - Dauchet 2010 - never smokers	2,410	145	1.6%	1.45 [0.94, 2.23]
PRIME - Dauchet 2010 - former smokers	3,353	237	2.8%	1.06 [0.78, 1.45]
WHI-OS - Belin 2011	93,676	6,006	13.4%	0.91 [0.86, 0.97]
WHS - Fitzgerald 2012	34,827	1,094	5.6%	0.82 [0.67, 1.00]
British Women's Heart & Health - Kim 2013	3,080	329	1.2%	1.09 [0.66, 1.82]
EPIC Potsdam - Von Ruesten 2013	23,531	363	8.3%	1.14 [0.99, 1.31]
British Regional Heart - Atkins 2014	3,328	582	2.3%	0.90 [0.63, 1.27]
MONICA Danish - Tognon 2014	1,849	755	8.3%	0.86 [0.75, 0.99]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	1,694	7.3%	0.95 [0.81, 1.11]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	1,227	5.6%	0.99 [0.81, 1.20]
PREDIMED- Buil-Cosiales 2016	7,216	342	2.1%	0.76 [0.53, 1.11]
SUN - Buil-Cosiales - 2017	17,007	112	0.8%	0.51 [0.27, 0.96]
PURE - Miller 2017	135,335	4,784	8.3%	0.89 [0.77, 1.02]
EPIC NL and MORGEN - Scheffers 2019	34,560	3,801	9.5%	0.87 [0.77, 0.98]
Total (95% CI) [Random Effects]	**577,323**	**27,205**	**100.0%**	**0.91 [0.86, 0.97]**

Heterogeneity: Tau² = 0.01; Chi² = 33.12, df = 20 (P = 0.03); I² = 40%

Test for overall effect: Z = 3.07 (P = 0.002)

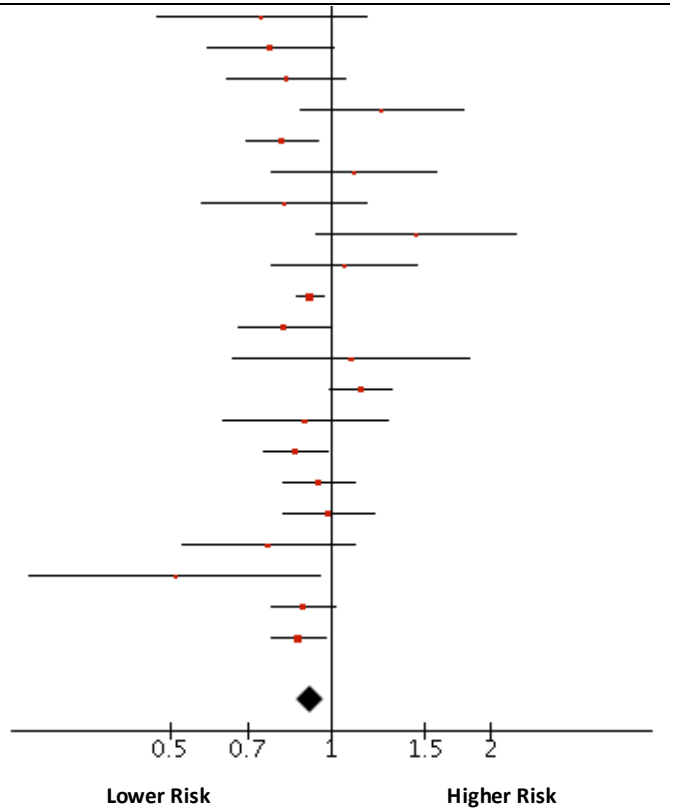


Figure S2. Relation between fruit intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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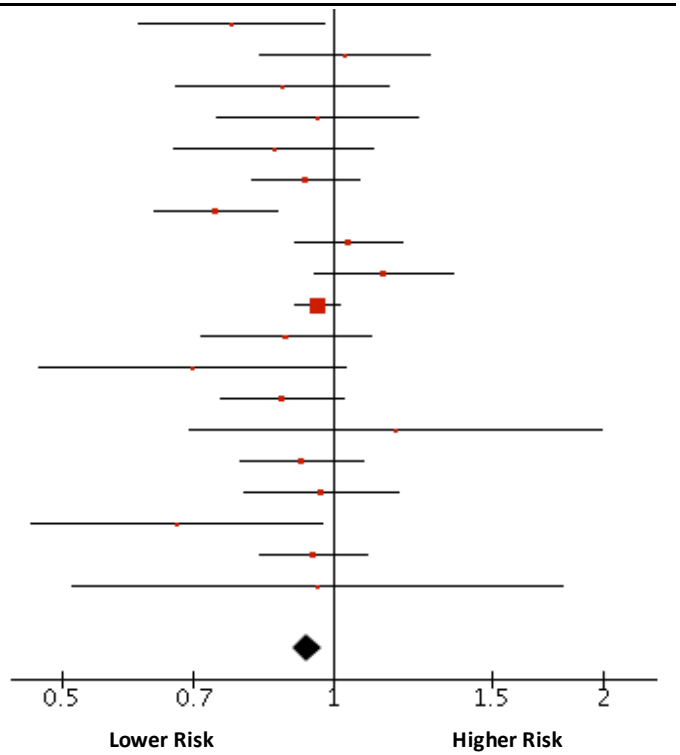
VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	209	2.4%	0.77 [0.61, 0.98]
Japan Public Health Center - Takachi 2008 - M	35,909	582	2.9%	1.03 [0.83, 1.28]
Japan Public Health Center - Takachi 2008 - F	41,982	556	1.8%	0.88 [0.67, 1.16]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	2.0%	0.96 [0.74, 1.24]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	2.0%	0.86 [0.67, 1.11]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	7.0%	0.93 [0.81, 1.07]
PRIME - Dauchet 2010 - current smokers	2,297	230	5.4%	0.74 [0.63, 0.87]
PRIME - Dauchet 2010 - former smokers	3,353	237	7.0%	1.04 [0.91, 1.19]
PRIME - Dauchet 2010 - never smokers	2,410	145	4.3%	1.14 [0.95, 1.36]
WHI-OS - Belin 2011	93,676	6,006	38.4%	0.96 [0.91, 1.02]
WHS - Fitzgerald 2012	34,827	1,094	2.9%	0.89 [0.71, 1.10]
EPIC Potsdam - Von Ruesten 2013	23,531	363	0.9%	0.70 [0.47, 1.03]
MONICA Danish - Tognon 2014	1,849	755	5.4%	0.88 [0.75, 1.03]
British Regional Heart - Atkins 2014	3,328	582	0.5%	1.17 [0.69, 1.99]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	1,694	5.4%	0.92 [0.79, 1.08]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	1,227	3.5%	0.97 [0.80, 1.18]
PREDIMED- Buil-Cosiales 2016	7,216	342	1.0%	0.67 [0.46, 0.97]
PURE - Miller 2017	135,335	4,784	7.0%	0.95 [0.83, 1.09]
SUN - Buil-Cosiales - 2017	17,007	112	0.3%	0.96 [0.51, 1.80]
Total (95% CI)	**539,683**	**22,810**	**100.0%**	**0.94 [0.90, 0.97]**

Heterogeneity: Chi² = 27.44, df = 18 (P = 0.07); I² = 34%

Test for overall effect: Z = 3.51 (P = 0.0004)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	209	4.0%	0.77 [0.61, 0.98]
Japan Public Health Center - Takachi 2008 - M	35,909	582	4.5%	1.03 [0.83, 1.28]
Japan Public Health Center - Takachi 2008 - F	41,982	556	3.1%	0.88 [0.67, 1.16]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	3.5%	0.96 [0.74, 1.24]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	8.2%	0.93 [0.81, 1.07]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	3.5%	0.86 [0.67, 1.11]
PRIME - Dauchet 2010 - former smokers	3,353	237	8.2%	1.04 [0.91, 1.19]
PRIME - Dauchet 2010 - never smokers	2,410	145	6.0%	1.14 [0.95, 1.36]
PRIME - Dauchet 2010 - current smokers	2,297	230	7.0%	0.74 [0.63, 0.87]
WHI-OS - Belin 2011	93,676	6,006	15.0%	0.96 [0.91, 1.02]
WHS - Fitzgerald 2012	34,827	1,094	4.5%	0.89 [0.71, 1.10]
EPIC Potsdam - Von Ruesten 2013	23,531	363	1.7%	0.70 [0.47, 1.03]
British Regional Heart - Atkins 2014	3,328	582	0.9%	1.17 [0.69, 1.99]
MONICA Danish - Tognon 2014	1,849	755	7.0%	0.88 [0.75, 1.03]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	1,227	5.2%	0.97 [0.80, 1.18]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	1,694	7.0%	0.92 [0.79, 1.08]
PREDIMED- Buil-Cosiales 2016	7,216	342	1.8%	0.67 [0.46, 0.97]
SUN - Buil-Cosiales - 2017	17,007	112	0.7%	0.96 [0.51, 1.80]
PURE - Miller 2017	135,335	4,784	8.2%	0.95 [0.83, 1.09]
Total (95% CI) [Random Effects]	**539,683**	**22,810**	**100.0%**	**0.92 [0.88, 0.97]**

Heterogeneity: Tau² = 0.00; Chi² = 27.44, df = 18 (P = 0.07); I² = 34%

Test for overall effect: Z = 2.93 (P = 0.003)

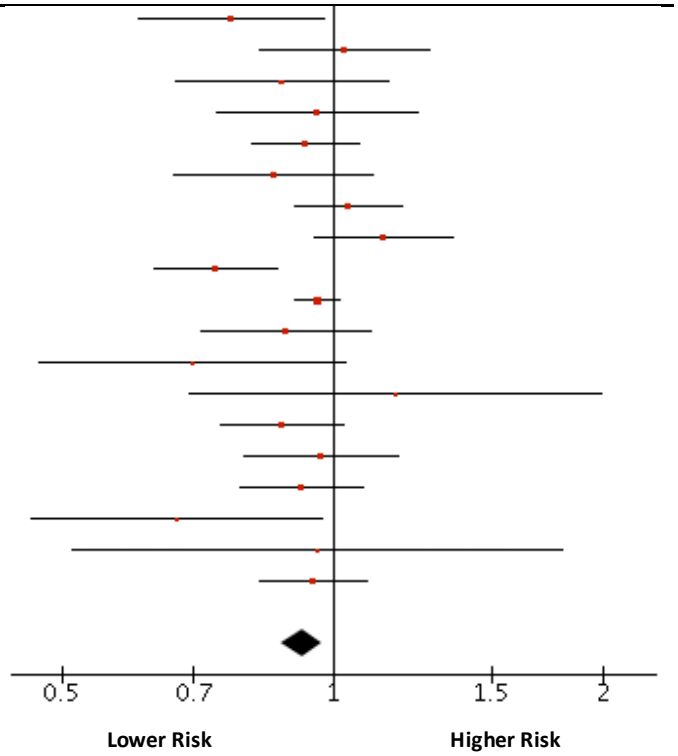


Figure S3. Relation between vegetable intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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BERRIES AND CARDIOVASCULAR DISEASE INCIDENCE

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2007	38,176	1,004	100.00%	1.27 [0.95, 1.71]
Total (95% CI)	**38,176**	**1,004**	**100.0%**	**1.27 [0.95, 1.71]**

Heterogeneity: Not applicable
Test for overall effect: Z = 1.60 (P = 0.11)

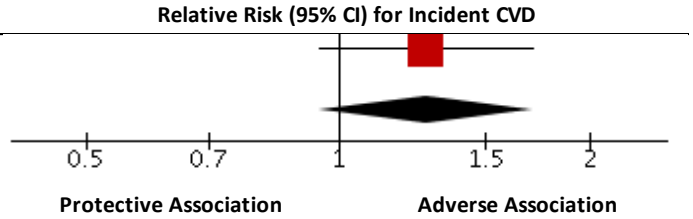


Figure S4. Relation between intake of berries and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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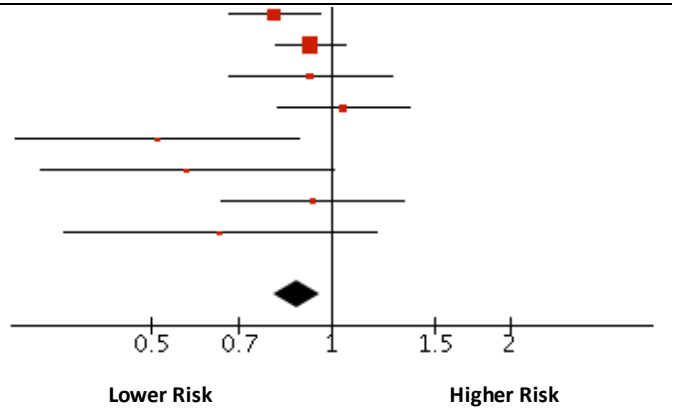
CITRUS FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Japan Public Health Center - Takachi 2008	77,891	1,386	25.1%	0.80 [0.67, 0.96]
NHS & HPFS -Joshipura 2009 - Mod. CHO	109,788	3,892	41.5%	0.92 [0.80, 1.06]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	7.9%	0.92 [0.67, 1.26]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	12.0%	1.05 [0.81, 1.36]
Jidni Medical School - Yamada 2011 - F	6,476	218	2.6%	0.51 [0.30, 0.89]
Jidni Medical School - Yamada 2011 - M	4,147	270	2.4%	0.57 [0.32, 1.01]
PREDIMED- Buil-Cosiales 2016	7,216	342	6.3%	0.93 [0.66, 1.33]
SUN - Buil-Cosiales - 2017	17,007	112	2.1%	0.65 [0.35, 1.19]
Total (95% CI) [Fixed Effets]	**222,525**	**6,220**	**100.0%**	**0.88 [0.80, 0.96]**

Heterogeneity: Chi² = 10.49, df = 7 (P = 0.16); I² = 33%

Test for overall effect: Z = 2.95 (P = 0.003)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Japan Public Health Center - Takachi 2008	77,891	1,386	22.9%	0.80 [0.67, 0.96]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	11.5%	0.92 [0.67, 1.26]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	27.9%	0.92 [0.80, 1.06]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	15.3%	1.05 [0.81, 1.36]
Jidni Medical School - Yamada 2011 - M	4,147	270	4.3%	0.57 [0.32, 1.01]
Jidni Medical School - Yamada 2011 - F	6,476	218	4.6%	0.51 [0.30, 0.89]
PREDIMED- Buil-Cosiales 2016	7,216	342	9.6%	0.93 [0.66, 1.33]
SUN - Buil-Cosiales - 2017	17,007	112	3.8%	0.65 [0.35, 1.19]
Total (95% CI) [Random Effects]	**222,525**	**6,220**	**100.0%**	**0.86 [0.76, 0.97]**

Heterogeneity: Tau² = 0.01; Chi² = 10.49, df = 7 (P = 0.16); I² = 33%

Test for overall effect: Z = 2.40 (P = 0.02)

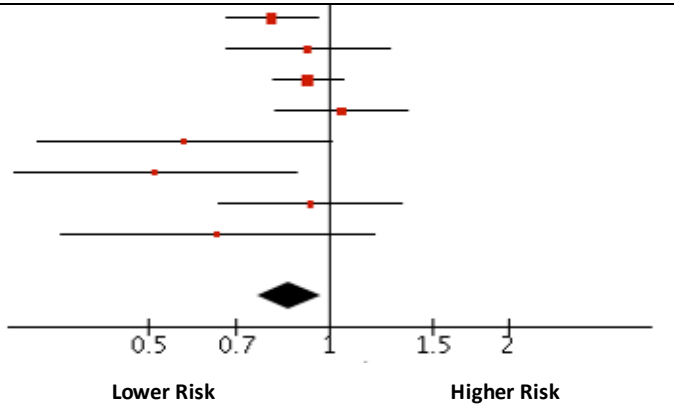


Figure S5. Relation between citrus fruit intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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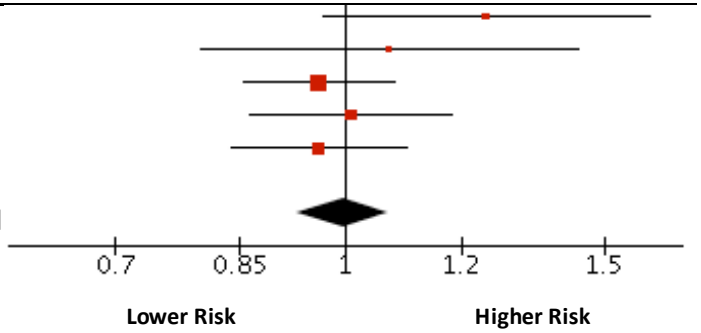
FRUIT JUICE AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	8.0%	1.25 [0.97, 1.61]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	6.0%	1.07 [0.80, 1.44]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	37.5%	0.96 [0.85, 1.08]
EPIC Potsdam - Von Ruesten 2013	23,531	363	21.1%	1.01 [0.86, 1.18]
EPIC NL and MORGEN - Scheffers 2019	34,560	3,801	27.5%	0.96 [0.84, 1.10]
Total (95% CI)	**167,879**	**8,056**	**100.0%**	**1.00 [0.93, 1.07]**

Heterogeneity: Tau² = 0.00; Chi² = 3.86, df = 4 (P = 0.42); I² = 0%

Test for overall effect: Z = 0.06 (P = 0.95)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	8.0%	1.25 [0.97, 1.61]
NHS & HPFS - Joshipura 2009 - Low CHO	-	-	6.0%	1.07 [0.80, 1.44]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	37.5%	0.96 [0.85, 1.08]
EPIC Potsdam - Von Ruesten 2013	23,531	363	21.1%	1.01 [0.86, 1.18]
EPIC NL and MORGEN - Scheffers 2019	34,560	3,801	27.5%	0.96 [0.84, 1.10]
Total (95% CI)	**167,879**	**8,056**	**100.0%**	**1.00 [0.93, 1.07]**

Heterogeneity: Tau² = 0.00; Chi² = 3.86, df = 4 (P = 0.42); I² = 0%

Test for overall effect: Z = 0.06 (P = 0.95)

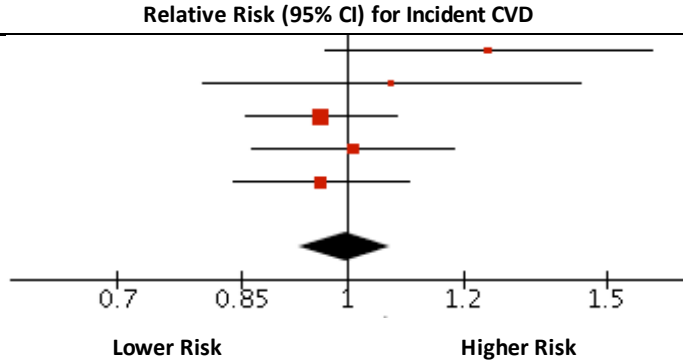


Figure S6. Relation between fruit juice intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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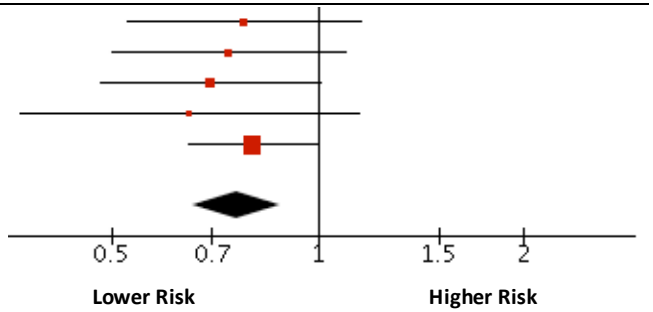
POMMES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,176	1,004	14.5%	0.78 [0.53, 1.15]
Framingham Offspring Study - Jacques 2015	2,880	518	14.5%	0.74 [0.50, 1.10]
PREDIMED- Buil-Cosiales 2016	7,216	342	16.1%	0.70 [0.48, 1.01]
SUN - Buil-Cosiales - 2017	17,007	112	6.9%	0.65 [0.37, 1.15]
NutriNet-Sante - Adriouch 2018	84,158	602	48.0%	0.80 [0.65, 1.00]
Total (95% CI)	**149,437**	**2,578**	**100.0%**	**0.76 [0.66, 0.88]**

Heterogeneity: Chi² = 0.77, df = 4 (P = 0.94); I² = 0%

Test for overall effect: Z = 3.58 (P = 0.0003)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,176	1,004	14.5%	0.78 [0.53, 1.15]
Framingham Offspring Study - Jacques 2015	2,880	518	14.5%	0.74 [0.50, 1.10]
PREDIMED- Buil-Cosiales 2016	7,216	342	16.1%	0.70 [0.48, 1.01]
SUN - Buil-Cosiales - 2017	17,007	112	6.9%	0.65 [0.37, 1.15]
NutriNet-Sante - Adriouch 2018	84,158	602	48.0%	0.80 [0.65, 1.00]
Total (95% CI) [Random Effects]	**149,437**	**2,578**	**100.0%**	**0.76 [0.66, 0.88]**

Heterogeneity: Tau² = 0.00; Chi² = 0.77, df = 4 (P = 0.94); I² = 0%

Test for overall effect: Z = 3.58 (P = 0.0003)

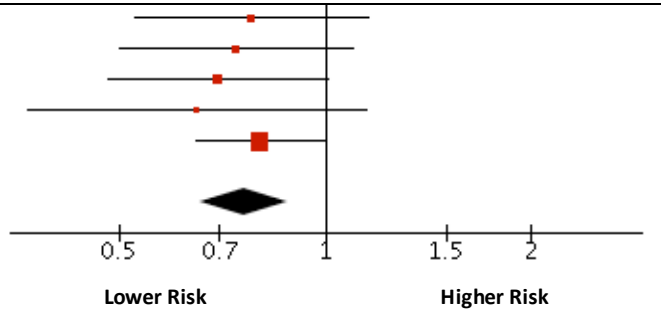


Figure S7. Relation between pommes intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

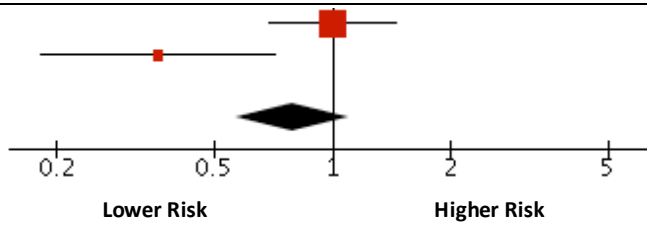
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ALLIUM VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	729	77.2%	1.00 [0.69, 1.45]
Theran Lipid and Glucose - Bahadoran 2017	2,369	79	22.8%	0.36 [0.18, 0.72]
Total (95% CI)	**40,814**	**808**	**100.0%**	**0.79 [0.57, 1.10]**

Heterogeneity: Chi² = 6.56, df = 1 (P = 0.01); I² = 85%
Test for overall effect: Z = 1.39 (P = 0.16)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	729	54.20%	1.00 [0.69, 1.45]
Theran Lipid and Glucose - Bahadoran 2017	2,369	79	45.80%	0.36 [0.18, 0.72]
Total (95% CI) [Random Effects]	**40,814**	**808**	**100.00%**	**0.63 [0.23, 1.70]**

Heterogeneity: Tau² = 0.44; Chi² = 6.56, df = 1 (P = 0.01); I² = 85%
Test for overall effect: Z = 0.92 (P = 0.36)

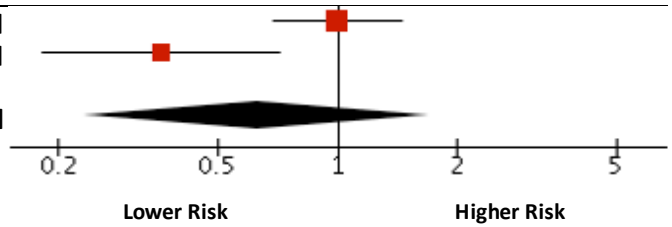


Figure S8. Relation between intake of allium vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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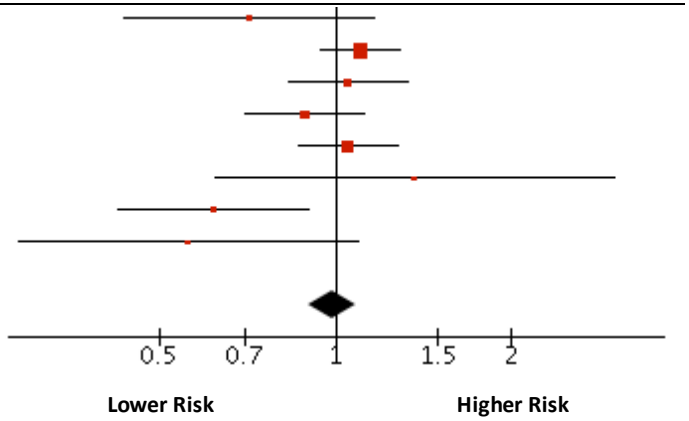
CRUCIFEROUS VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	729	3.5%	0.71 [0.44, 1.16]
Japan Public Health Center - Takachi 2008	77,891	1,386	34.4%	1.11 [0.94, 1.29]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	15.3%	1.05 [0.83, 1.33]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	15.3%	0.89 [0.70, 1.12]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	22.0%	1.05 [0.86, 1.28]
EPIC Potsdam - Von Ruesten 2013	23,531	363	1.4%	1.36 [0.62, 2.99]
PREDIMED- Buil-Cosiales 2016	7,216	342	6.1%	0.62 [0.43, 0.90]
SUN - Buil-Cosiales - 2017	17,007	112	1.9%	0.56 [0.29, 1.09]
Total (95% CI)	**273,878**	**6,824**	**100.0%**	**0.99 [0.90, 1.08]**

Heterogeneity: Chi² = 14.65, df = 7 (P = 0.04); I² = 52%

Test for overall effect: Z = 0.28 (P = 0.78)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	729	7.2%	0.71 [0.44, 1.16]
Japan Public Health Center - Takachi 2008	77,891	1,386	21.7%	1.11 [0.94, 1.29]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	16.8%	0.89 [0.70, 1.12]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	19.2%	1.05 [0.86, 1.28]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	16.8%	1.05 [0.83, 1.33]
EPIC Potsdam - Von Ruesten 2013	23,531	363	3.3%	1.36 [0.62, 2.99]
PREDIMED- Buil-Cosiales 2016	7,216	342	10.5%	0.62 [0.43, 0.90]
SUN - Buil-Cosiales - 2017	17,007	112	4.4%	0.56 [0.29, 1.09]
Total (95% CI) [Random Effects]	**273,878**	**6,824**	**100.0%**	**0.93 [0.80, 1.09]**

Heterogeneity: Tau² = 0.02; Chi² = 14.65, df = 7 (P = 0.04); I² = 52%

Test for overall effect: Z = 0.91 (P = 0.36)

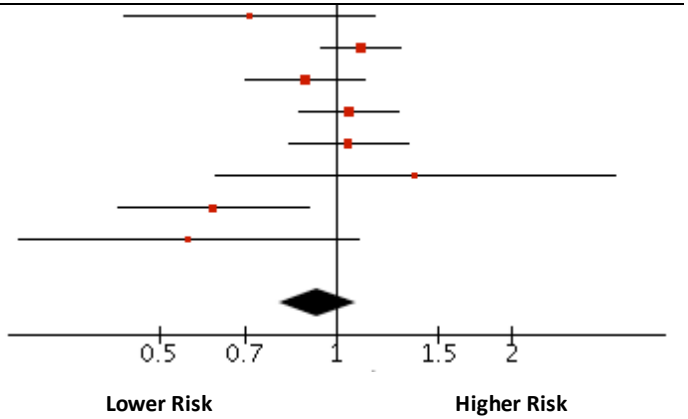


Figure S9. Relation between intake of cruciferous vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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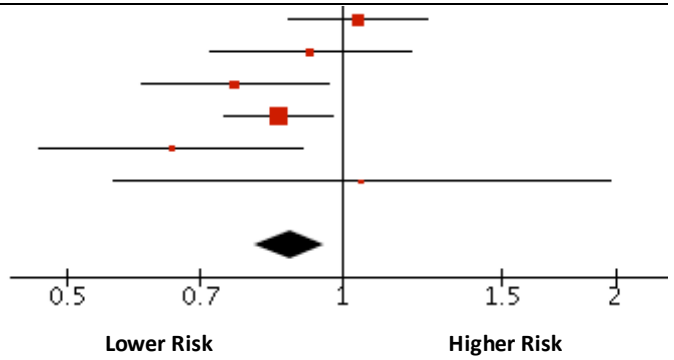
GREEN LEAFY VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Japan Public Health Center - Takachi 2008	77,891	1,386	24.7%	1.04 [0.87, 1.24]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	11.8%	0.92 [0.72, 1.19]
NHS & HPFS -Joshipura 2009 - Low. CHO	-	-	13.9%	0.76 [0.60, 0.97]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	40.8%	0.85 [0.74, 0.98]
PREDIMED- Buil-Cosiales 2016	7,216	342	6.9%	0.65 [0.47, 0.91]
SUN - Buil-Cosiales - 2017	17,007	112	2.0%	1.05 [0.56, 1.97]
Total (95% CI)	**211,902**	**5,732**	**100.0%**	**0.87 [0.76, 0.99]**

Heterogeneity: Chi² = 8.69, df = 5 (P = 0.12); I² = 42%

Test for overall effect: Z = 2.93 (P = 0.003)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Japan Public Health Center - Takachi 2008	77,891	1,386	24.7%	1.04 [0.87, 1.24]
NHS & HPFS - Joshipura 2009 - High CHO	109,788	3,892	11.8%	0.92 [0.72, 1.19]
NHS & HPFS -Joshipura 2009 - Low. CHO	-	-	13.9%	0.76 [0.60, 0.97]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	40.8%	0.85 [0.74, 0.98]
PREDIMED- Buil-Cosiales 2016	7,216	342	6.9%	0.65 [0.47, 0.91]
SUN - Buil-Cosiales - 2017	17,007	112	2.0%	1.05 [0.56, 1.97]
Total (95% CI)	**211,902**	**5,732**	**100.0%**	**0.87 [0.76, 0.99]**

Heterogeneity: Tau² = 0.01; Chi² = 8.69, df = 5 (P = 0.12); I² = 42%

Test for overall effect: Z = 2.16 (P = 0.03)

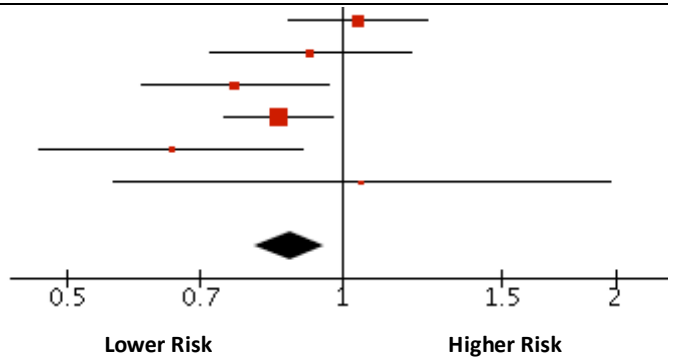


Figure S10. Relation between intake of green leafy vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

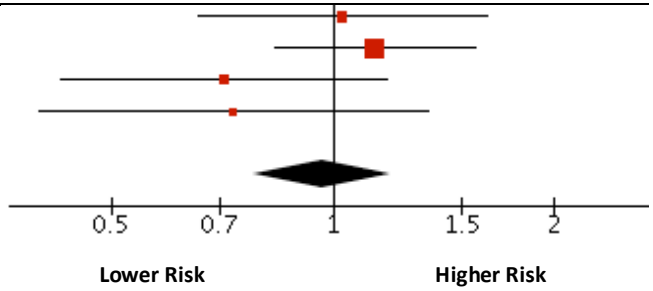
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TOMATOES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (b) - tomatoes	38,445	729	22.7%	1.03 [0.66, 1.62]
WHS - Sesso 2003 (b) - tomato juice	-	-	47.0%	0.71 [0.43, 1.18]
WHS - Sesso 2003 (b) - tomato based products	-	-	17.8%	1.14 [0.83, 1.56]
SUN - Buil-Cosiales - 2017	17,007	112	12.5%	0.73 [0.40, 1.35]
Total (95% CI)	**55,452**	**841**	**100.0%**	**0.97 [0.78, 1.20]**

Heterogeneity: Chi² = 3.31, df = 3 (P = 0.35); I² = 9%
Test for overall effect: Z = 0.29 (P = 0.77)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (b) - tomatoes	38,445	729	23.5%	1.03 [0.66, 1.62]
WHS - Sesso 2003 (b) - tomato based products	-	-	18.8%	0.71 [0.43, 1.18]
WHS - Sesso 2003 (b) - tomato juice	-	-	44.2%	1.14 [0.83, 1.56]
SUN - Buil-Cosiales - 2017	17,007	112	13.5%	0.73 [0.40, 1.35]
Total (95% CI) [Random Effects]	**55,452**	**841**	**100.0%**	**0.96 [0.76, 1.21]**

Heterogeneity: Tau² = 0.01; Chi² = 3.31, df = 3 (P = 0.35); I² = 9%
Test for overall effect: Z = 0.35 (P = 0.73)

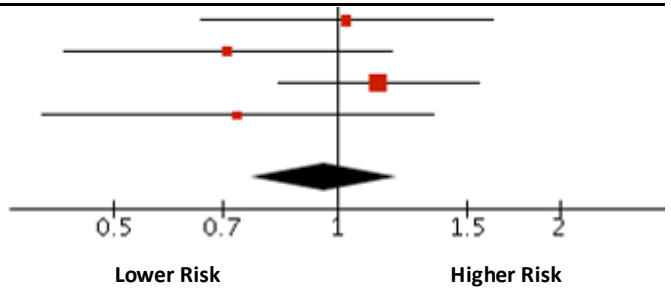
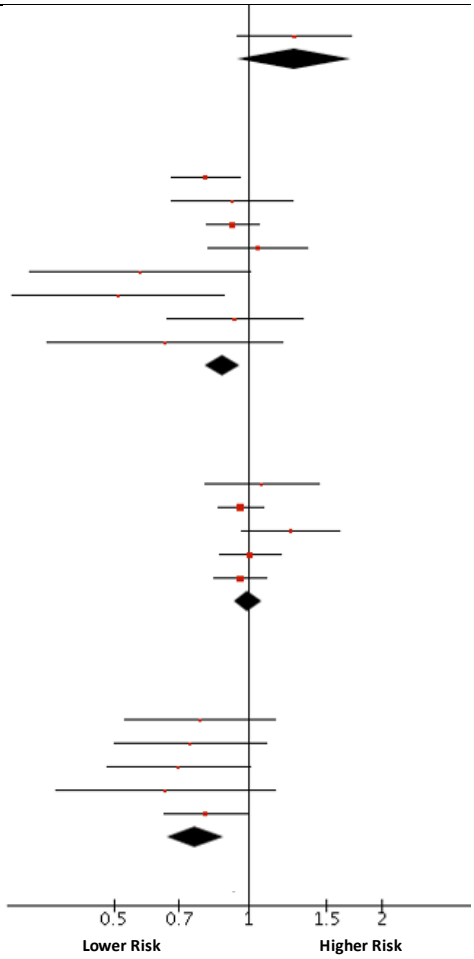


Figure S11. Relation between tomato intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Berries				
WHS - Sesso 2007	38,176	1,004	3.1%	1.27 [0.95, 1.71]
Subtotal (95% CI)	**38,176**	**1,004**	**3.1%**	**1.27 [0.95, 1.71]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 1.60 (P = 0.11)				
Citrus				
Japan Public Health Center - Takachi 2008	77,891	1,386	8.5%	0.80 [0.67, 0.96]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	2.7%	0.92 [0.67, 1.26]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	14.1%	0.92 [0.80, 1.06]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	4.1%	1.05 [0.81, 1.36]
Jidni Medical School - Yamada 2011 - M	4,147	270	0.8%	0.57 [0.32, 1.01]
Jidni Medical School - Yamada 2011 - F	6,476	218	0.9%	0.51 [0.30, 0.89]
PREDIMED- Buil-Cosiales 2016	7,216	342	2.1%	0.93 [0.66, 1.33]
SUN - Buil-Cosiales - 2017	17,007	112	0.7%	0.65 [0.35, 1.19]
Subtotal (95% CI)	**222,525**	**6,220**	**33.9%**	**0.88 [0.80, 0.96]**
Heterogeneity: Chi² = 10.49, df = 7 (P = 0.16); I² = 33%				
Test for overall effect: Z = 2.95 (P = 0.003)				
Fruit juice				
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	3.1%	1.07 [0.80, 1.44]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	19.2%	0.96 [0.85, 1.08]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	4.1%	1.25 [0.97, 1.61]
EPIC Potsdam - Von Ruesten 2013	23,531	363	10.8%	1.01 [0.86, 1.18]
EPIC NL and MORGEN - Scheffers 2019	34,560	3,801	14.1%	0.96 [0.84, 1.10]
Subtotal (95% CI)	**167,879**	**8,056**	**51.2%**	**1.00 [0.93, 1.07]**
Heterogeneity: Chi² = 3.86, df = 4 (P = 0.42); I² = 0%				
Test for overall effect: Z = 0.06 (P = 0.95)				
Pommes				
WHS - Sesso 2003 (a)	38,176	1,004	1.7%	0.78 [0.53, 1.15]
Framingham Offspring Study - Jacques 2015	2,880	518	1.7%	0.74 [0.50, 1.10]
PREDIMED- Buil-Cosiales 2016	7,216	342	1.9%	0.70 [0.48, 1.01]
SUN - Buil-Cosiales - 2017	17,007	112	0.8%	0.65 [0.37, 1.15]
NutriNet-Sante - Adriouch 2018	84,158	602	5.7%	0.80 [0.65, 1.00]
Subtotal (95% CI)	**149,437**	**2,578**	**11.9%**	**0.76 [0.66, 0.88]**
Heterogeneity: Chi² = 0.77, df = 4 (P = 0.94); I² = 0%				
Test for overall effect: Z = 3.58 (P = 0.0003)				
Test for subgroup differences: Chi² = 16.75, df = 3 (P = 0.0008), I² = 82.1%				



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B. Random Effects

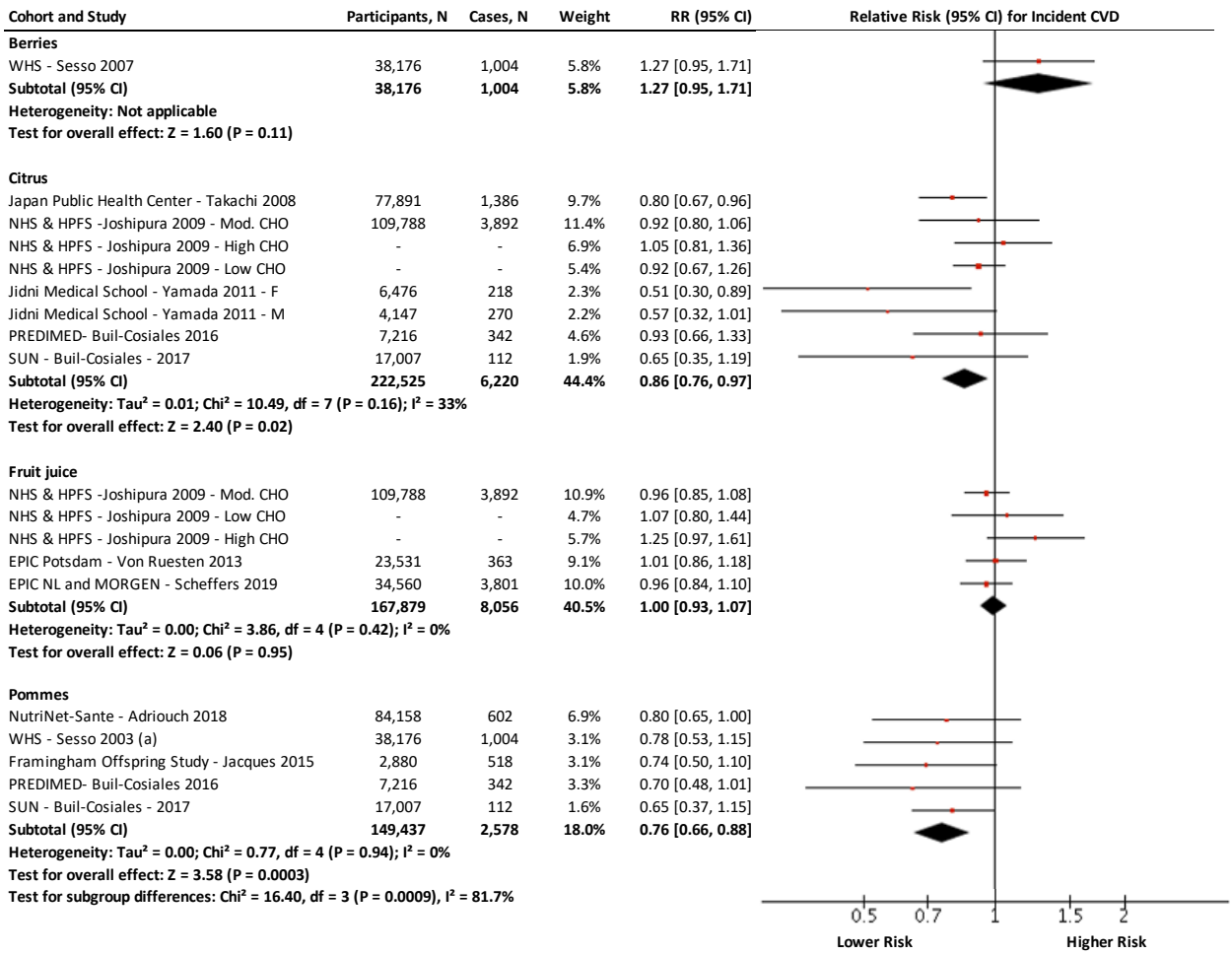
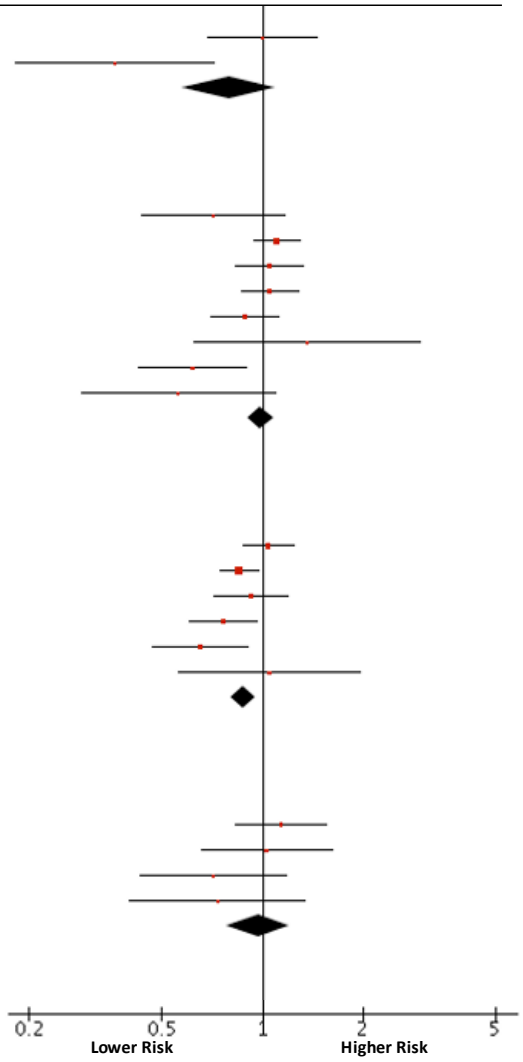


Figure S12. Relation between sources of fruit and CVD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
WHS - Sesso 2003 (a)	38,445	729	2.6%	1.00 [0.69, 1.45]
Theran Lipid and Glucose - Bahadoran 2017	2,369	79	0.8%	0.36 [0.18, 0.72]
Subtotal (95% CI)	**40,814**	**808**	**3.3%**	**0.79 [0.57, 1.10]**
Heterogeneity: Chi² = 6.56, df = 1 (P = 0.01); I² = 85%				
Test for overall effect: Z = 1.39 (P = 0.16)				
Cruciferous				
WHS - Sesso 2003 (a)	38,445	729	1.5%	0.71 [0.44, 1.16]
Japan Public Health Center - Takachi 2008	77,891	1,386	14.6%	1.11 [0.94, 1.29]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	6.5%	1.05 [0.83, 1.33]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	9.3%	1.05 [0.86, 1.28]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	6.5%	0.89 [0.70, 1.12]
EPIC Potsdam - Von Ruesten 2013	23,531	363	0.6%	1.36 [0.62, 2.99]
PREDIMED- Buil-Cosiales 2016	7,216	342	2.6%	0.62 [0.43, 0.90]
SUN - Buil-Cosiales - 2017	17,007	112	0.8%	0.56 [0.29, 1.09]
Subtotal (95% CI)	**273,878**	**6,824**	**42.3%**	**0.99 [0.90, 1.08]**
Heterogeneity: Chi² = 14.65, df = 7 (P = 0.04); I² = 52%				
Test for overall effect: Z = 0.28 (P = 0.78)				
Green Leafy				
Japan Public Health Center - Takachi 2008	77,891	1,386	11.5%	1.04 [0.87, 1.24]
NHS & HPFS -Joshipura 2009 - Mod. CHO	-	-	19.0%	0.85 [0.74, 0.98]
NHS & HPFS - Joshipura 2009 - High CHO	-	-	5.5%	0.92 [0.72, 1.19]
NHS & HPFS - Joshipura 2009 - Low CHO	109,788	3,892	6.5%	0.76 [0.60, 0.97]
PREDIMED- Buil-Cosiales 2016	7,216	342	3.2%	0.65 [0.47, 0.91]
SUN - Buil-Cosiales - 2017	17,007	112	0.9%	1.05 [0.56, 1.97]
Subtotal (95% CI)	**211,902**	**5,732**	**46.6%**	**0.88 [0.80, 0.96]**
Heterogeneity: Chi² = 8.69, df = 5 (P = 0.12); I² = 42%				
Test for overall effect: Z = 2.93 (P = 0.003)				
Tomatoes				
WHS - Sesso 2003 (b) - tomato juice	38,445	729	3.6%	1.14 [0.83, 1.56]
WHS - Sesso 2003 (b) - tomatoes	-	-	1.8%	1.03 [0.66, 1.62]
WHS - Sesso 2003 (b) - tomato based products	-	-	1.4%	0.71 [0.43, 1.18]
SUN - Buil-Cosiales - 2017	17,007	112	1.0%	0.73 [0.40, 1.35]
Subtotal (95% CI)	**55,452**	**841**	**7.7%**	**0.97 [0.78, 1.20]**
Heterogeneity: Chi² = 3.31, df = 3 (P = 0.35); I² = 9%				
Test for overall effect: Z = 0.29 (P = 0.77)				
Test for subgroup differences: Chi² = 4.34, df = 3 (P = 0.23), I² = 30.8%				



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B. Random Effects

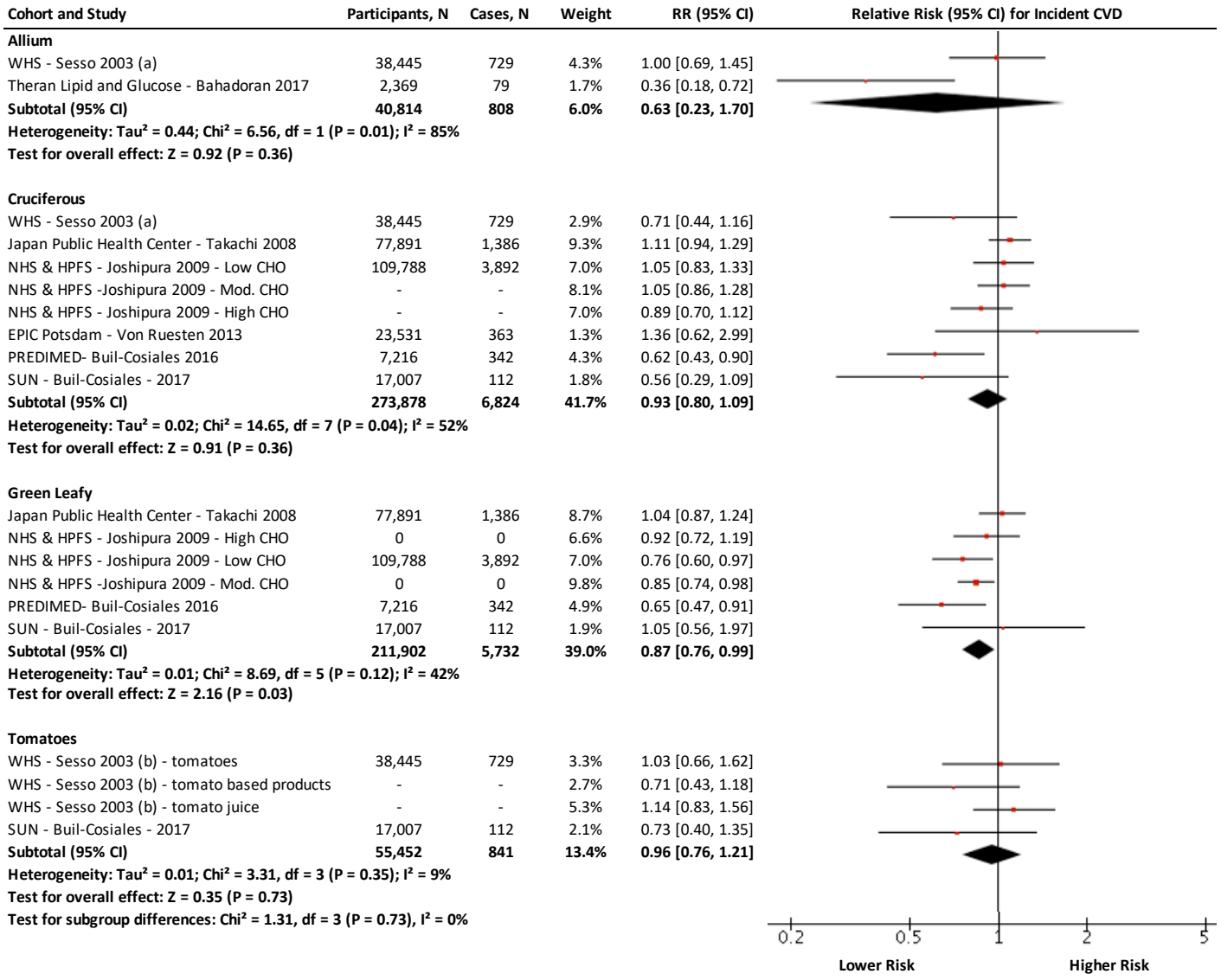


Figure S13. Relation between sources of vegetables and CVD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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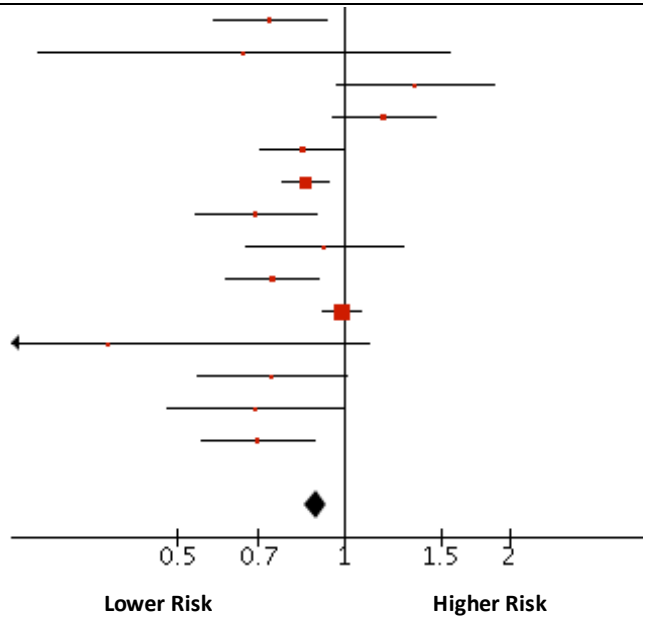
TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
National Health & Nutrition - Bazzano 2002	9,608	1,145	4.1%	0.73 [0.58, 0.93]
Kuopio IHD Risk - Rissanen 2003	1,950	115	0.3%	0.66 [0.28, 1.56]
Odyssey - Genkinger 2004	6,151	378	2.1%	1.35 [0.97, 1.88]
Health and Lifestyle Survey - Kvaavik 2010	4,866	431	4.9%	1.19 [0.96, 1.47]
Shanghai Women Health - Nechuta 2010	71,243	755	7.4%	0.84 [0.71, 1.01]
EPIC - Leenders 2013	451,151	5,125	23.9%	0.85 [0.77, 0.94]
Health Survey of England - Oyebode 2014	65,226	1,554	3.5%	0.69 [0.54, 0.89]
British Regional Heart - Atkins 2014	3,328	327	2.1%	0.92 [0.66, 1.29]
NIPPON DATA80 - Okuda 2015	9,112	823	6.0%	0.74 [0.61, 0.90]
Migrant Study - Hjartaker 2015	9,766	4,595	37.3%	0.99 [0.92, 1.07]
PREDIMED- Buil-Cosiales 2016	7,216	104	0.2%	0.37 [0.12, 1.11]
HAPIEE - Stefler 2016	19,263	438	2.3%	0.74 [0.54, 1.01]
PURE - Miller 2017	135,335	1,649	1.7%	0.69 [0.48, 1.00]
Health and Living Status of Elderly - Lin 2017	4,176	-	4.1%	0.70 [0.55, 0.88]
Total (95% CI)	**798,391**	**17,439**	**100.0%**	**0.89 [0.85, 0.93]**

Heterogeneity: Chi² = 40.92, df = 13 (P < 0.0001); I² = 68%

Test for overall effect: Z = 4.77 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
National Health & Nutrition - Bazzano 2002	9,608	1,145	8.0%	0.73 [0.58, 0.93]
Kuopio IHD Risk - Rissanen 2003	1,950	115	1.3%	0.66 [0.28, 1.56]
Odyssey - Genkinger 2004	6,151	378	5.7%	1.35 [0.97, 1.88]
Shanghai Women Health - Nechuta 2010	71,243	755	9.7%	0.84 [0.71, 1.01]
Health and Lifestyle Survey - Kvaavik 2010	4,866	431	8.6%	1.19 [0.96, 1.47]
EPIC - Leenders 2013	451,151	5,125	12.0%	0.85 [0.77, 0.94]
British Regional Heart - Atkins 2014	3,328	327	5.7%	0.92 [0.66, 1.29]
Health Survey of England - Oyebode 2014	65,226	1,554	7.5%	0.69 [0.54, 0.89]
Migrant Study - Hjartaker 2015	9,766	4,595	12.5%	0.99 [0.92, 1.07]
NIPPON DATA80 - Okuda 2015	9,112	823	9.1%	0.74 [0.61, 0.90]
HAPIEE - Stefler 2016	19,263	438	6.1%	0.74 [0.54, 1.01]
PREDIMED- Buil-Cosiales 2016	7,216	104	0.9%	0.37 [0.12, 1.11]
PURE - Miller 2017	135,335	1,649	5.0%	0.69 [0.48, 1.00]
Health and Living Status of Elderly - Lin 2017	4,176	-	8.0%	0.70 [0.55, 0.88]
Total (95% CI) [Random Effects]	**798,391**	**17,439**	**100.0%**	**0.84 [0.76, 0.94]**

Heterogeneity: Tau² = 0.02; Chi² = 40.92, df = 13 (P < 0.0001); I² = 68%

Test for overall effect: Z = 3.17 (P = 0.002)

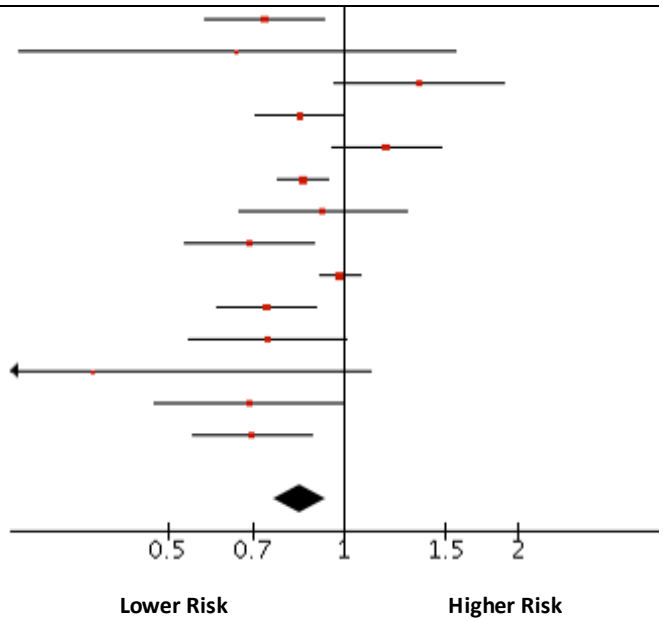


Figure S14. Relation between total fruit and vegetable intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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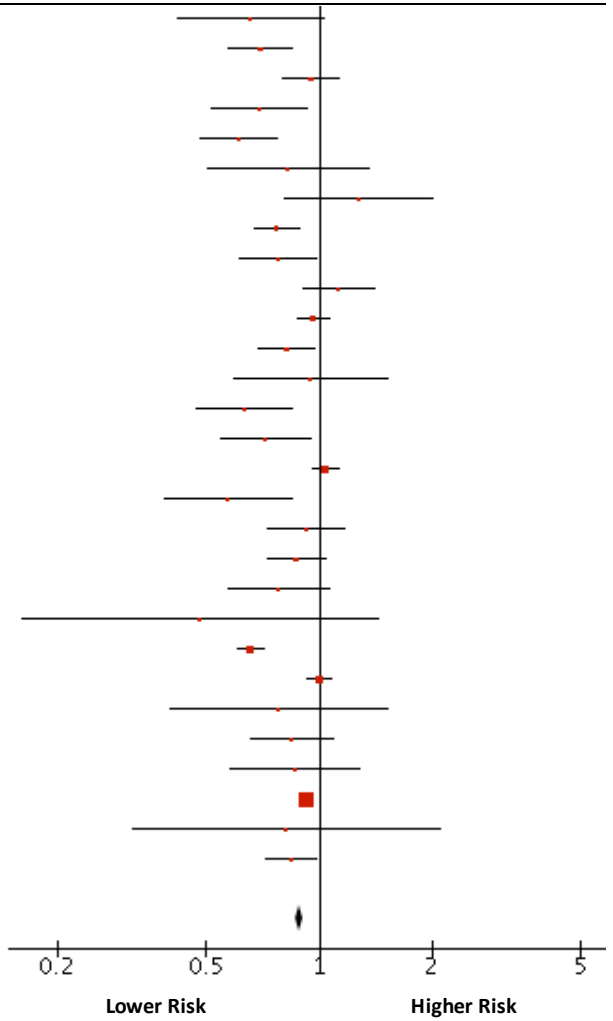
FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	0.3%	0.66 [0.42, 1.03]
Health Food Shoppers - Appleby 2002 - F	6,416	611	1.6%	0.70 [0.57, 0.85]
Health Food Shoppers - Appleby 2002 - M	4,325	591	2.0%	0.95 [0.80, 1.13]
Melbourne Collaborative Cohort - Harriss 2007	40,653	697	0.7%	0.69 [0.51, 0.93]
EPIC Diabetes - Nothlings 2008	10,262	517	1.1%	0.61 [0.48, 0.78]
Takayama Study - Nakamura 2008 - F	15,724	184	0.3%	0.83 [0.51, 1.35]
Takayama Study - Nakamura 2008 - M	13,355	200	0.3%	1.27 [0.81, 2.00]
JACC - Nagura 2009	59,845	2,243	3.3%	0.77 [0.67, 0.88]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	1.1%	0.78 [0.62, 0.99]
NOMAS - Gardener 2011	2,568	314	1.3%	1.13 [0.91, 1.40]
EPIC - Leenders 2013	451,151	5,125	6.4%	0.96 [0.87, 1.06]
Health Survey of England - Oyebode 2014	65,226	1,554	2.0%	0.82 [0.69, 0.98]
British Regional Heart - Atkins 2014	3,328	327	0.3%	0.95 [0.59, 1.52]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	0.7%	0.63 [0.47, 0.85]
MONICA Danish - Tognon 2014	1,849	223	0.8%	0.72 [0.55, 0.95]
Migrant Study - Hjartaker 2015	9,766	4,595	10.0%	1.04 [0.96, 1.13]
UK Women's Cohort - Lai 2015	30,458	286	0.4%	0.57 [0.39, 0.85]
MONICA Switzerland - Vormund 2015 - F	9,196	634	1.1%	0.92 [0.73, 1.17]
MONICA Switzerland - Vormund 2015 - M	8,665	751	2.0%	0.87 [0.73, 1.04]
HAPIEE - Stefler 2016	19,263	438	0.6%	0.78 [0.57, 1.07]
PREDIMED- Buil-Cosiales 2016	7,216	104	0.1%	0.48 [0.16, 1.44]
China Kadoorie Biobank- Du 2017	462,342	6,166	10.0%	0.66 [0.61, 0.71]
DIET-HD - Saglimbene 2017	9,757	515	10.0%	1.00 [0.92, 1.08]
MONICA France - Berard 2017	1,311	41	0.1%	0.78 [0.40, 1.52]
PURE - Miller 2017	135,335	1,649	0.9%	0.84 [0.65, 1.09]
Cooper Center - Shah 2018 - DASH	11,376	249	0.4%	0.86 [0.58, 1.27]
Singapore Chinese Health - Neelakantan 2018	57,078	4,871	39.9%	0.92 [0.89, 0.96]
Renal Transplant Recipients - Sotomayer 2019	400	49	0.1%	0.82 [0.32, 2.10]
NIPPON DATA80 - Kondo 2019	9,115	1,070	2.5%	0.84 [0.72, 0.99]
Total (95% CI)	**1,581,506**	**39,623**	**100.0%**	**0.88 [0.86, 0.91]**

Heterogeneity: Chi² = 136.43, df = 28 (P < 0.00001); I² = 79%

Test for overall effect: Z = 9.70 (P < 0.00001)



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B. Random Effect

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	1.8%	0.66 [0.42, 1.03]
Health Food Shoppers - Appleby 2002 - F	6,416	611	4.2%	0.70 [0.57, 0.85]
Health Food Shoppers - Appleby 2002 - M	4,325	591	4.5%	0.95 [0.80, 1.13]
Melbourne Collaborative Cohort - Harriss 2007	40,653	697	3.0%	0.69 [0.51, 0.93]
EPIC Diabetes - Nothlings 2008	10,262	517	3.7%	0.61 [0.48, 0.78]
Takayama Study - Nakamura 2008 - F	15,724	184	1.6%	0.83 [0.51, 1.35]
Takayama Study - Nakamura 2008 - M	13,355	200	1.8%	1.27 [0.81, 2.00]
JACC - Nagura 2009	59,845	2,243	5.0%	0.77 [0.67, 0.88]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	3.7%	0.78 [0.62, 0.99]
NOMAS - Gardener 2011	2,568	314	4.0%	1.13 [0.91, 1.40]
EPIC - Leenders 2013	451,151	5,125	5.5%	0.96 [0.87, 1.06]
Health Survey of England - Oyebode 2014	65,226	1,554	4.5%	0.82 [0.69, 0.98]
British Regional Heart - Atkins 2014	3,328	327	1.7%	0.95 [0.59, 1.52]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	3.0%	0.63 [0.47, 0.85]
MONICA Danish - Tognon 2014	1,849	223	3.2%	0.72 [0.55, 0.95]
Migrant Study - Hjartaker 2015	9,766	4,595	5.8%	1.04 [0.96, 1.13]
UK Women's Cohort - Lai 2015	30,458	286	2.2%	0.57 [0.39, 0.85]
MONICA Switzerland - Vormund 2015 - F	9,196	634	3.7%	0.92 [0.73, 1.17]
MONICA Switzerland - Vormund 2015 - M	8,665	751	4.5%	0.87 [0.73, 1.04]
HAPIEE - Stefler 2016	19,263	438	2.8%	0.78 [0.57, 1.07]
PREDIMED- Buil-Cosiales 2016	7,216	104	0.4%	0.48 [0.16, 1.44]
China Kadoorie Biobank- Du 2017	462,342	6,166	5.8%	0.66 [0.61, 0.71]
DIET-HD - Saglimbene 2017	9,757	515	5.8%	1.00 [0.92, 1.08]
MONICA France - Berard 2017	1,311	41	1.0%	0.78 [0.40, 1.52]
PURE - Miller 2017	135,335	1,649	3.5%	0.84 [0.65, 1.09]
Cooper Center - Shah 2018 - DASH	11,376	249	2.2%	0.86 [0.58, 1.27]
Singapore Chinese Health - Neelakantan 2018	57,078	4,871	6.1%	0.92 [0.89, 0.96]
Renal Transplant Recipients - Sotomayer 2019	400	49	0.5%	0.82 [0.32, 2.10]
NIPPON DATA80 - Kondo 2019	9,115	1,070	4.8%	0.84 [0.72, 0.99]
Total (95% CI) [Random Effects]	**1,581,506**	**39,623**	**100.0%**	**0.83 [0.77, 0.89]**

Heterogeneity: Tau² = 0.02; Chi² = 136.43, df = 28 (P < 0.00001); I² = 79%

Test for overall effect: Z = 5.10 (P < 0.00001)

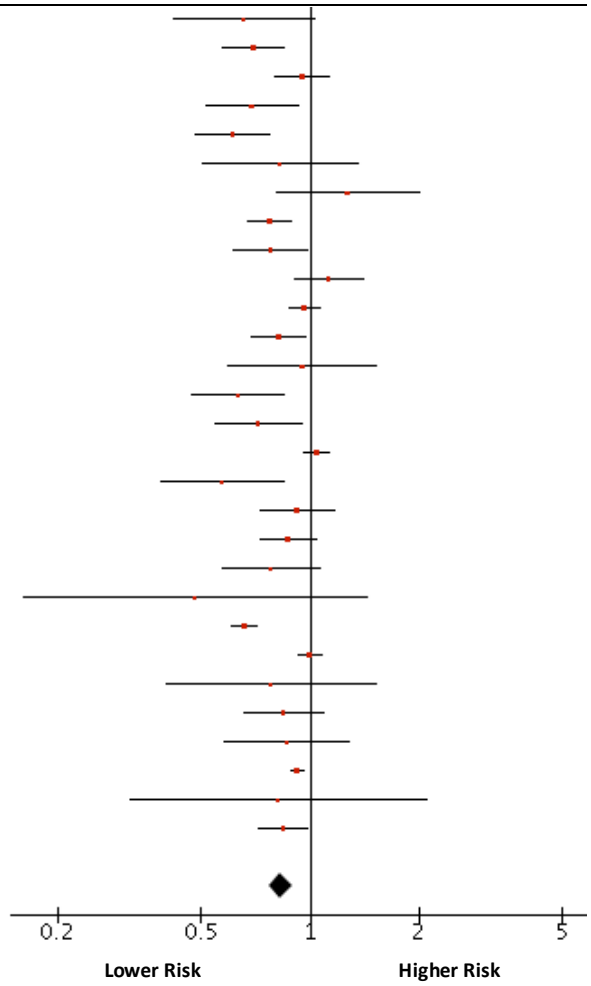


Figure S15. Relation between fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

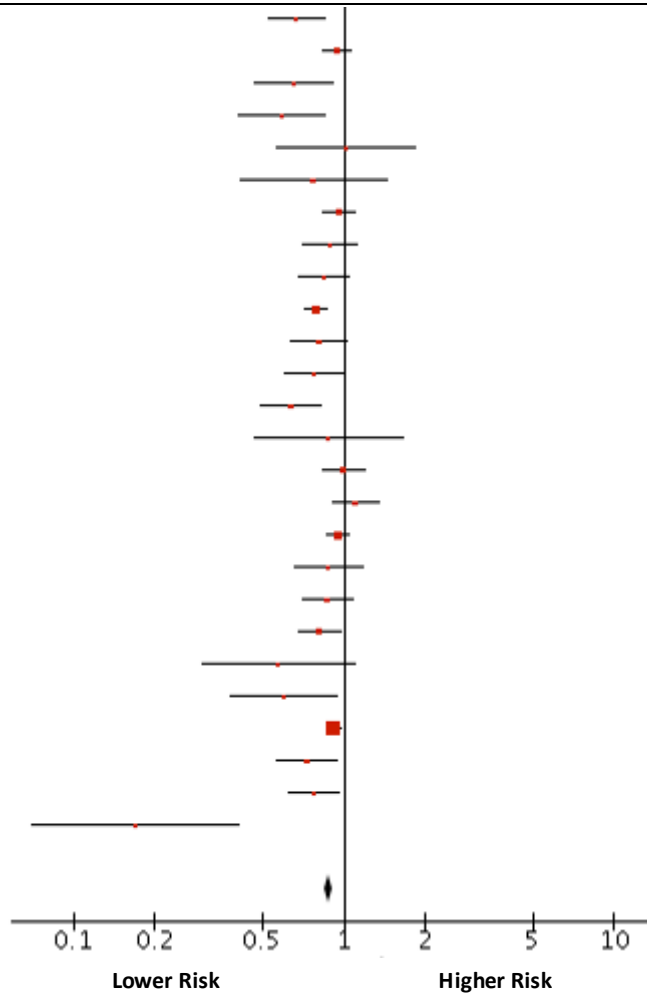
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VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	2.0%	0.67 [0.53, 0.85]
Health Food Shoppers - Appleby 2002	10,471	1,202	7.8%	0.94 [0.84, 1.06]
Melbourne Collaborative Cohort - Harriss 2007	40,653	697	1.0%	0.66 [0.47, 0.92]
EPIC Diabetes - Nothlings 2008	10,262	517	0.8%	0.59 [0.41, 0.85]
Takayama Study - Nakamura 2008 - M	13,355	200	0.3%	1.02 [0.57, 1.84]
Takayama Study - Nakamura 2008 - F	15,724	184	0.3%	0.77 [0.41, 1.44]
JACC - Nagura 2009	59,485	2,243	5.8%	0.96 [0.84, 1.10]
NOMAS - Gardener 2011	2,568	314	2.0%	0.89 [0.70, 1.12]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	2.3%	0.84 [0.68, 1.05]
EPIC - Leenders 2013	451,151	5,125	11.3%	0.79 [0.71, 0.87]
MONICA Danish - Tognon 2014	1,849	223	2.0%	0.81 [0.64, 1.03]
Health Survey of England - Oyebode 2014	65,226	1,554	1.7%	0.78 [0.60, 1.00]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	1.7%	0.64 [0.49, 0.82]
British Regional Heart - Atkins 2014	3,328	327	0.3%	0.88 [0.47, 1.64]
MONICA Switzerland - Vormund 2015 - M	8,665	751	3.5%	1.00 [0.84, 1.19]
MONICA Switzerland - Vormund 2015 - F	9,196	634	2.8%	1.11 [0.91, 1.34]
Migrant Study - Hjartaker 2015	9,766	4,595	11.3%	0.95 [0.86, 1.05]
HAPIEE - Stefler 2016	19,236	438	1.3%	0.88 [0.65, 1.18]
PURE - Miller 2017	135,335	1,649	2.3%	0.87 [0.70, 1.08]
PLSAW - Blekkenhorst 2017	1,226	238	3.5%	0.81 [0.68, 0.97]
MONICA France - Berard 2017	1,311	41	0.3%	0.57 [0.30, 1.09]
NHANES - Conrad 2018	29,133	726	0.5%	0.60 [0.38, 0.94]
Singapore Chinese Health - Neelakantan 2018	57,078	4,871	31.4%	0.91 [0.86, 0.97]
Cooper Center - Shah 2018 - DASH	11,376	249	1.7%	0.73 [0.57, 0.95]
NIPPON DATA80 - Kondo 2019	9,115	1,070	2.3%	0.78 [0.63, 0.97]
Renal Transplant Recipients - Sotomayer 2019	400	49	0.1%	0.17 [0.07, 0.41]
Total (95% CI)	**1,101,435**	**33,516**	**100.0%**	**0.87 [0.85, 0.90]**

Heterogeneity: Chi² = 61.44, df = 25 (P < 0.0001); I² = 59%
Test for overall effect: Z = 7.99 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Men Born in 1913 - Strandhagen 2000	730	226	4.0%	0.67 [0.53, 0.85]
Health Food Shoppers - Appleby 2002	10,471	1,202	6.7%	0.94 [0.84, 1.06]
Melbourne Collaborative Cohort - Harriss 2007	40,653	697	2.6%	0.66 [0.47, 0.92]
EPIC Diabetes - Nothlings 2008	10,262	517	2.2%	0.59 [0.41, 0.85]
Takayama Study - Nakamura 2008 - M	13,355	200	1.0%	1.02 [0.57, 1.84]
Takayama Study - Nakamura 2008 - F	15,724	184	0.9%	0.77 [0.41, 1.44]
JACC - Nagura 2009	59,485	2,243	6.2%	0.96 [0.84, 1.10]
NOMAS - Gardener 2011	2,568	314	4.0%	0.89 [0.70, 1.12]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	4.3%	0.84 [0.68, 1.05]
EPIC - Leenders 2013	451,151	5,125	7.2%	0.79 [0.71, 0.87]
MONICA Danish - Tognon 2014	1,849	223	4.0%	0.81 [0.64, 1.03]
Health Survey of England - Oyebode 2014	65,226	1,554	3.6%	0.78 [0.60, 1.00]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	3.6%	0.64 [0.49, 0.82]
British Regional Heart - Atkins 2014	3,328	327	0.9%	0.88 [0.47, 1.64]
MONICA Switzerland - Vormund 2015 - M	8,665	751	5.2%	1.00 [0.84, 1.19]
MONICA Switzerland - Vormund 2015 - F	9,196	634	4.8%	1.11 [0.91, 1.34]
Migrant Study - Hjartaker 2015	9,766	4,595	7.2%	0.95 [0.86, 1.05]
HAPIEE - Stefler 2016	19,236	438	3.0%	0.88 [0.65, 1.18]
PURE - Miller 2017	135,335	1,649	4.3%	0.87 [0.70, 1.08]
PLSAW - Blekkenhorst 2017	1,226	238	5.2%	0.81 [0.68, 0.97]
MONICA France - Berard 2017	1,311	41	0.9%	0.57 [0.30, 1.09]
NHANES - Conrad 2018	29,133	726	1.6%	0.60 [0.38, 0.94]
Singapore Chinese Health - Neelakantan 2018	57,078	4,871	8.1%	0.91 [0.86, 0.97]
Cooper Center - Shah 2018 - DASH	11,376	249	3.6%	0.73 [0.57, 0.95]
NIPPON DATA80 - Kondo 2019	9,115	1,070	4.3%	0.78 [0.63, 0.97]
Renal Transplant Recipients - Sotomayer 2019	400	49	0.50%	0.17 [0.07, 0.41]
Total (95% CI) [Random Effects]	**1,101,435**	**33,516**	**100.0%**	**0.83 [0.78, 0.89]**

Heterogeneity: Tau² = 0.01; Chi² = 61.44, df = 25 (P < 0.0001); I² = 59%
Test for overall effect: Z = 5.63 (P < 0.00001)

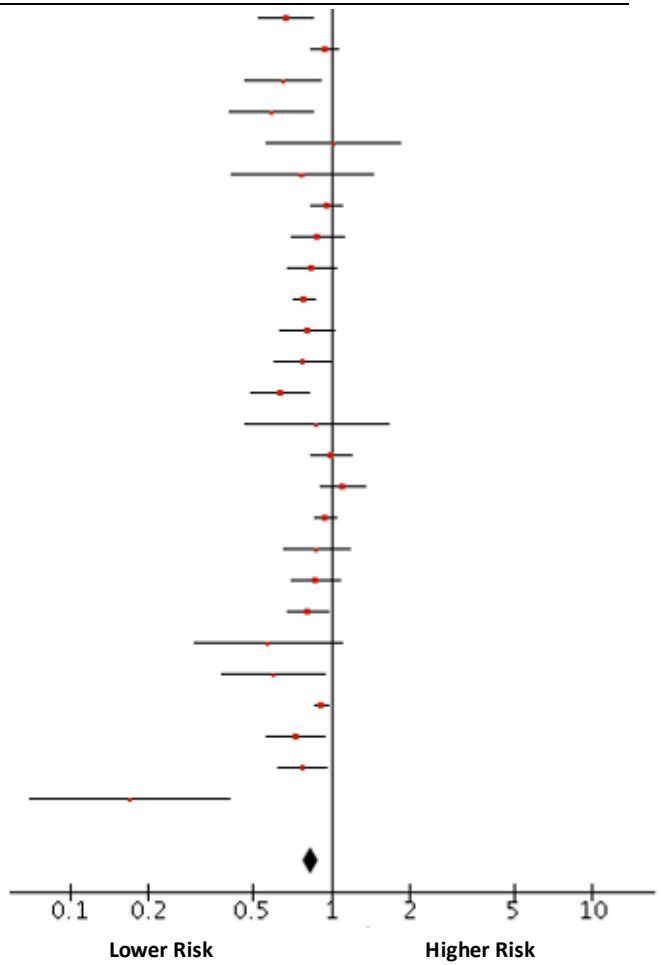
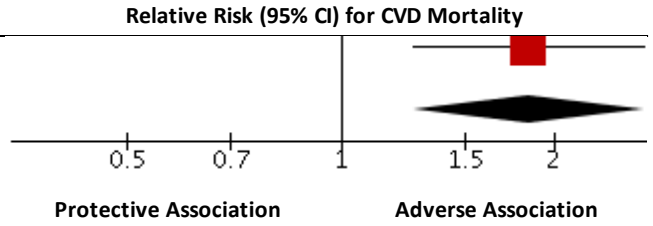


Figure S16. Relation between vegetable intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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APRICOTS AND CARDIOVASCULAR DISEASE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
DIET-HD - Saglimbene 2017	9,757	515	100.00%	1.84 [1.27, 2.67]
Total (95% CI)	**9,757**	**515**	**100.00%**	**1.84 [1.27, 2.67]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 3.21 (P = 0.001)				



Supplementary Figure 17. Relation between intake of apricots and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

BANANAS AND CARDIOVASCULAR DISEASE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Migrant Study - Hjartaker 2015 - banana	9,766	4,595	100.0%	1.06 [0.87, 1.29]
Total (95% CI)	**9,766**	**4,595**	**100.0%**	**1.06 [0.87, 1.29]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.60 (P = 0.55)				

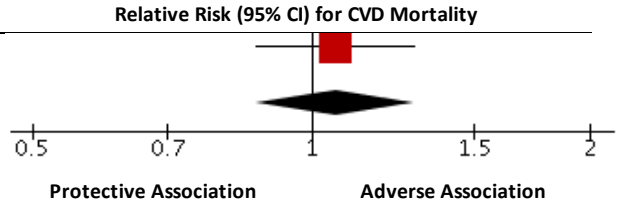


Figure S18. Relation between intake of bananas and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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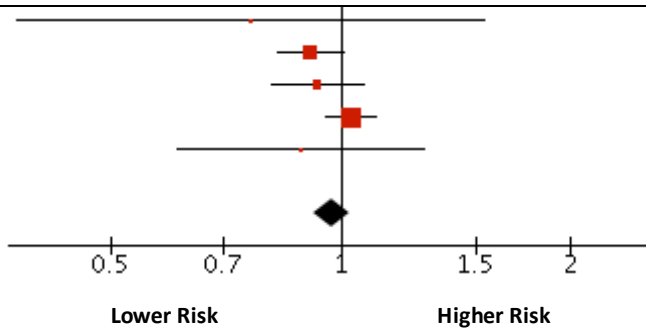
BERRIES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2007	38,176	204	0.8%	0.76 [0.38, 1.55]
Iowa WHS - Mink 2007 - strawberries	34,492	2,316	32.6%	0.91 [0.83, 1.01]
Iowa WHS - Mink 2007 - blueberries	-	-	19.0%	0.93 [0.81, 1.07]
Migrant Study - Hjartaker 2015	9,766	4,595	44.5%	1.03 [0.95, 1.11]
UK Women's Cohort - Lai 2015	30,458	286	3.0%	0.89 [0.61, 1.29]
Total (95% CI)	**112,892**	**7,401**	**100.0%**	**0.97 [0.92, 1.03]**

Heterogeneity: Chi² = 4.68, df = 4 (P = 0.32); I² = 15%

Test for overall effect: Z = 1.04 (P = 0.30)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007 - strawberries	34,492	2,316	32.6%	0.91 [0.83, 1.01]
WHS - Sesso 2007	38,176	204	0.8%	0.76 [0.38, 1.55]
Iowa WHS - Mink 2007 - blueberries	-	-	19.0%	0.93 [0.81, 1.07]
Migrant Study - Hjartaker 2015	9,766	4,595	44.5%	1.03 [0.95, 1.11]
UK Women's Cohort - Lai 2015	30,458	286	3.0%	0.89 [0.61, 1.29]
Total (95% CI) [Random Effects]	**112,892**	**7,401**	**100.0%**	**0.97 [0.90, 1.03]**

Heterogeneity: Tau² = 0.00; Chi² = 4.68, df = 4 (P = 0.32); I² = 15%

Test for overall effect: Z = 1.06 (P = 0.29)

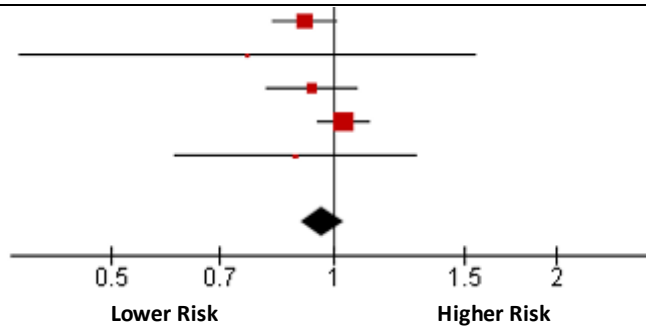


Figure S19. Relation between intake of berries and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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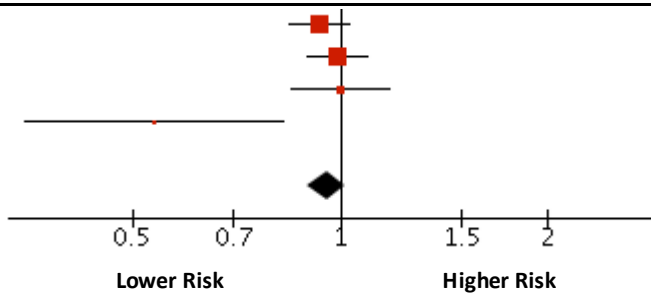
CITRUS FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007 - grapefruit	34,492	2,316	34.8%	0.93 [0.85, 1.03]
Iowa WHS - Mink 2007 - oranges	-	-	34.8%	0.99 [0.90, 1.09]
Migrant Study - Hjartaker 2015	9,766	4,595	24.0%	1.00 [0.85, 1.18]
UK Women's Cohort - Lai 2015	30,458	286	6.5%	0.54 [0.35, 0.83]
Total (95% CI)	**74,716**	**7,197**	**100.0%**	**0.95 [0.90, 1.02]**

Heterogeneity: Chi² = 7.85, df = 3 (P = 0.05); I² = 62%

Test for overall effect: Z = 1.45 (P = 0.15)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007 - oranges	34,492	2,316	34.8%	0.99 [0.90, 1.09]
Iowa WHS - Mink 2007 - grapefruit	-	-	34.8%	0.93 [0.85, 1.03]
Migrant Study - Hjartaker 2015	9,766	4,595	24.0%	1.00 [0.85, 1.18]
UK Women's Cohort - Lai 2015	30,458	286	6.5%	0.54 [0.35, 0.83]
Total (95% CI) [Random Effects]	**74,716**	**7,197**	**100.0%**	**0.93 [0.83, 1.05]**

Heterogeneity: Tau² = 0.01; Chi² = 7.85, df = 3 (P = 0.05); I² = 62%

Test for overall effect: Z = 1.12 (P = 0.26)

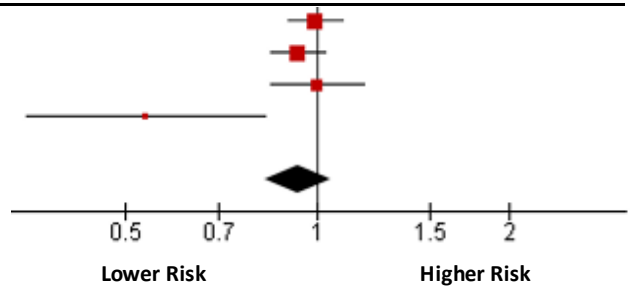


Figure S20. Relation between citrus fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

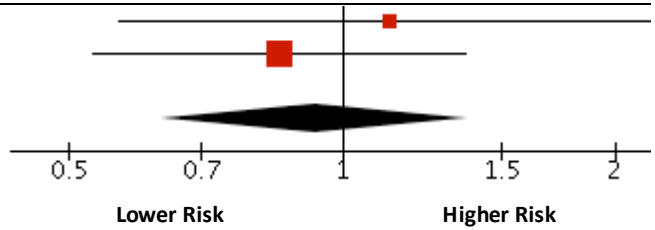
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DRIED FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts H C P - Gaziano 1995	1,299	161	32.0%	1.13 [0.57, 2.24]
UK Women's Cohort - Lai 2015	30,458	286	68.0%	0.85 [0.53, 1.36]
Total (95% CI)	**31,757**	**447**	**100.0%**	**0.93 [0.63, 1.37]**

Heterogeneity: Chi² = 0.44, df = 1 (P = 0.51); I² = 0%
Test for overall effect: Z = 0.36 (P = 0.72)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts H C P - Gaziano 1995	1,299	161	32.0%	1.13 [0.57, 2.24]
UK Women's Cohort - Lai 2015	30,458	286	68.0%	0.85 [0.53, 1.36]
Total (95% CI) [Random Effects]	**31,757**	**447**	**100.0%**	**0.93 [0.63, 1.37]**

Heterogeneity: Tau² = 0.00; Chi² = 0.44, df = 1 (P = 0.51); I² = 0%
Test for overall effect: Z = 0.36 (P = 0.72)

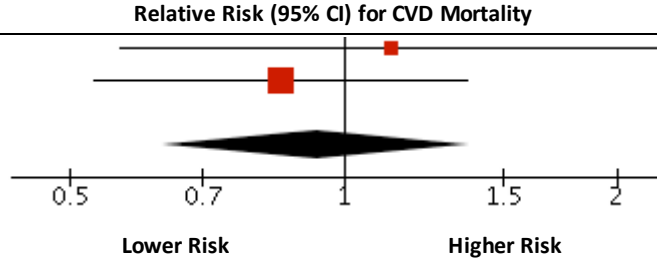


Figure S21. Relation between dried fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity

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FRUIT JUICE AND CARDIOVASCULAR DISEASE MORTALITY

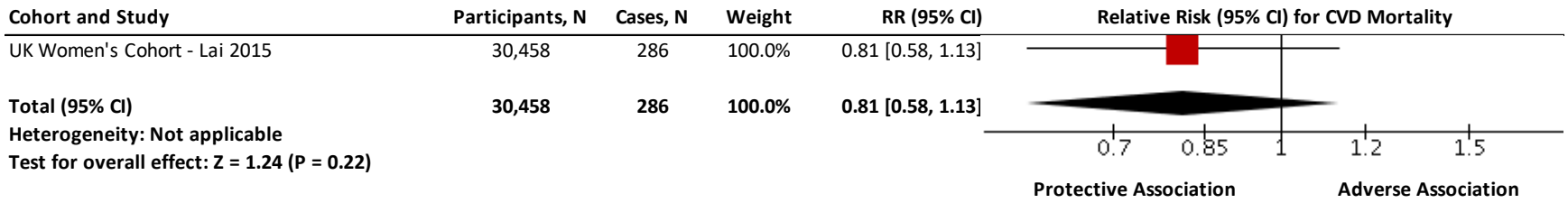


Figure S22. Relation between fruit juice intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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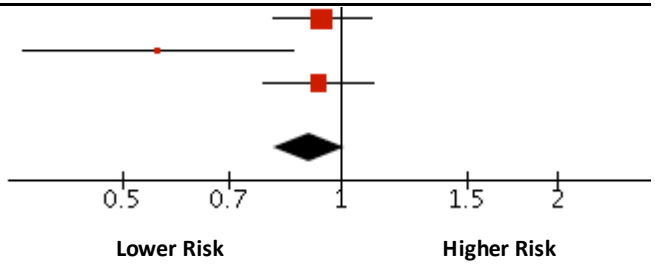
GRAPES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007 - grapes and raisins	34,492	2,316	52.0%	0.94 [0.81, 1.10]
Migrant Study - Hjartaker 2015 - grapes	9,766	4,595	6.9%	0.93 [0.78, 1.11]
UK Women's Cohort - Lai 2015 - grapes	30,458	286	41.1%	0.56 [0.36, 0.86]
Total (95% CI)	**74,716**	**7,197**	**100.0%**	**0.90 [0.81, 1.01]**

Heterogeneity: Chi^2 = 5.12, df = 2 (P = 0.08); I^2 = 61%

Test for overall effect: Z = 1.73 (P = 0.08)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007 - grapes and raisins	34,492	2,316	43.3%	0.94 [0.81, 1.10]
Migrant Study - Hjartaker 2015 - grapes	9,766	4,595	40.5%	0.93 [0.78, 1.11]
UK Women's Cohort - Lai 2015 - grapes	30,458	286	16.2%	0.56 [0.36, 0.86]
Total (95% CI) [Random Effects]	**74,716**	**7,197**	**100.0%**	**0.86 [0.70, 1.06]**

Heterogeneity: Tau^2 = 0.02; Chi^2 = 5.12, df = 2 (P = 0.08); I^2 = 61%

Test for overall effect: Z = 1.42 (P = 0.15)

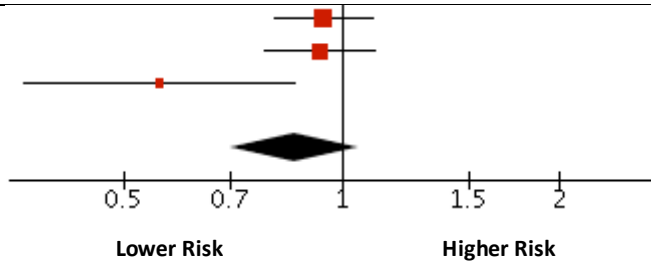


Figure S23. Relation between intake of grapes and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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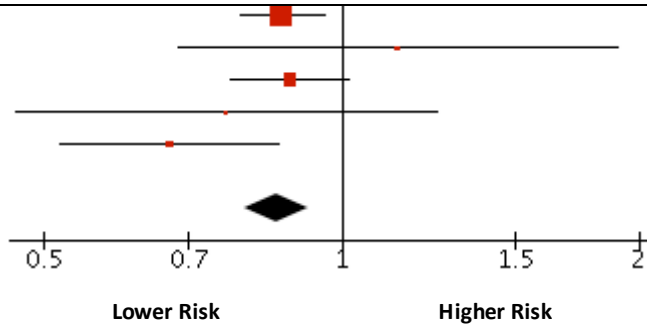
POMMES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007	34,492	2,316	57.6%	0.87 [0.79, 0.96]
UK Women's Cohort - Lai 2015	30,458	286	2.1%	1.14 [0.68, 1.90]
Migrant Study - Hjartaker 2015	9,766	4,595	29.4%	0.89 [0.77, 1.02]
Hodgson et al. 2016	1,456	235	2.3%	0.76 [0.47, 1.25]
DIET-HD - Saglimbene 2017	9,757	515	8.5%	0.67 [0.52, 0.86]
Total (95% CI)	**85,929**	**7,947**	100.0%	**0.86 [0.80, 0.92]**

Heterogeneity: Chi² = 5.30, df = 4 (P = 0.26); I² = 25%

Test for overall effect: Z = 4.04 (P < 0.0001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007	34,492	2,316	46.3%	0.87 [0.79, 0.96]
UK Women's Cohort - Lai 2015	30,458	286	3.8%	1.14 [0.68, 1.90]
Migrant Study - Hjartaker 2015	9,766	4,595	32.7%	0.89 [0.77, 1.02]
Hodgson et al. 2016	1,456	235	4.1%	0.76 [0.47, 1.25]
DIET-HD - Saglimbene 2017	9,757	515	13.2%	0.67 [0.52, 0.86]
Total (95% CI) [Random Effects]	**85,929**	**7,947**	**100.0%**	**0.85 [0.77, 0.94]**

Heterogeneity: Tau² = 0.00; Chi² = 5.30, df = 4 (P = 0.26); I² = 25%

Test for overall effect: Z = 3.15 (P = 0.002)

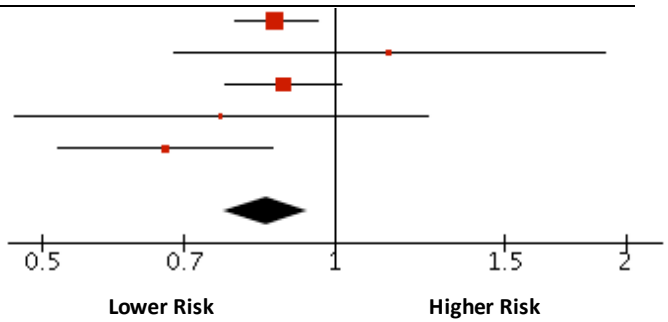


Figure S24. Relation between pommes fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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ALLIUM VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
PLSAW - Blekkenhorst 2017	1,226	238	100.0%	0.33 [0.22, 0.49]
Total (95% CI)	**1,226**	**238**	**100.0%**	**0.33 [0.22, 0.49]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 5.50 (P < 0.00001)				

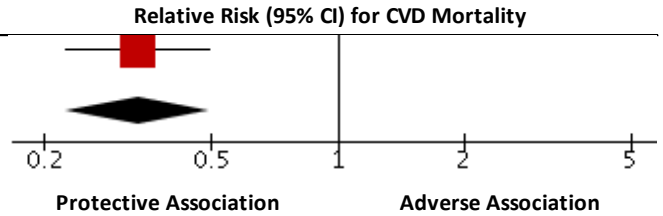


Figure S25. Relation between intake allium vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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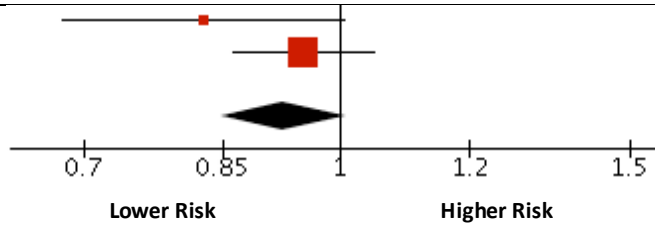
CARROTS AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Zutphen Elderly - Buijsse 2008- Carrots	559	197	20.0%	0.83 [0.68, 1.01]
Miigrant Study - Hjartaker 2015 - carrots	9,766	4,595	80.0%	0.95 [0.86, 1.05]
Total (95% CI)	**10,325**	**4,792**	**100.0%**	**0.92 [0.85, 1.01]**

Heterogeneity: Chi² = 1.57, df = 1 (P = 0.21); I² = 36%

Test for overall effect: Z = 1.74 (P = 0.08)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Zutphen Elderly - Buijsse 2008- Carrots	559	197	30.9%	0.83 [0.68, 1.01]
Miigrant Study - Hjartaker 2015 - carrots	9,766	4,595	69.1%	0.95 [0.86, 1.05]
Total (95% CI) [Random Effects]	**10,325**	**4,792**	**100.0%**	**0.91 [0.80, 1.03]**

Heterogeneity: Tau² = 0.00; Chi² = 1.57, df = 1 (P = 0.21); I² = 36%

Test for overall effect: Z = 1.44 (P = 0.15)

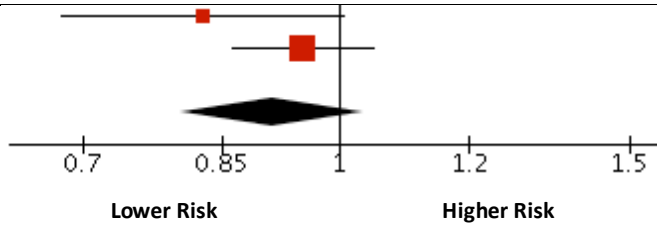


Figure S26. Relation between carrots intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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CELERY AND CARDIOVASCULAR DISEASE MORTALITY

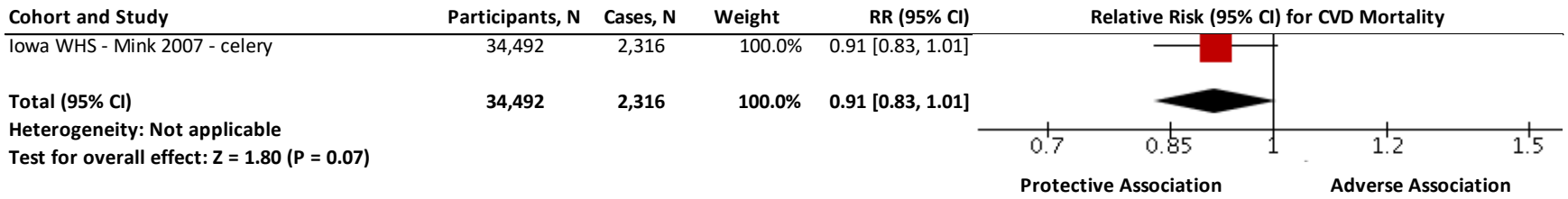


Figure S27. Relation between celery intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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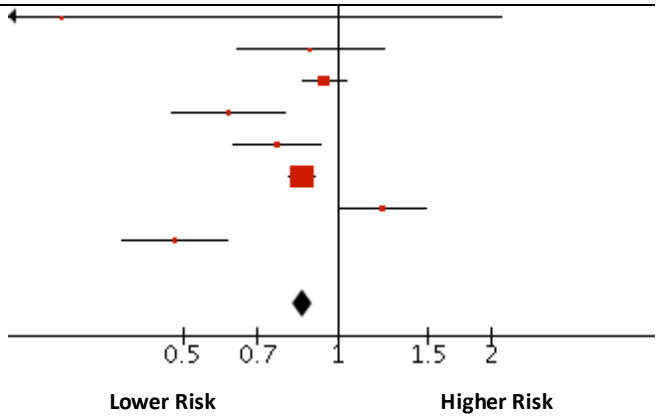
CRUCIFEROUS VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	0.1%	0.29 [0.04, 2.10]
Odyssey - Genkinger 2004	6,151	378	1.8%	0.89 [0.64, 1.24]
Iowa WHS - Mink 2007	34,492	2,316	21.3%	0.94 [0.85, 1.04]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	3.2%	0.61 [0.47, 0.79]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	5.3%	0.76 [0.63, 0.93]
Migrant Study - Hjartaker 2015 - cauliflower	9,766	4,595	59.2%	0.85 [0.80, 0.90]
Migrant Study - Hjartaker 2015 - cabbage	-	-	5.3%	1.22 [1.00, 1.49]
PLSAW - Blekkenhorst 2017	1,226	238	3.7%	0.48 [0.38, 0.61]
Total (95% CI)	**187,730**	**13,081**	**100.0%**	**0.85 [0.82, 0.89]**

Heterogeneity: Chi² = 48.35, df = 7 (P < 0.00001); I² = 86%

Test for overall effect: Z = 6.79 (P < 0.00001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1299	161	0.6%	0.29 [0.04, 2.10]
Odyssey - Genkinger 2004	6,151	378	10.2%	0.89 [0.64, 1.24]
Iowa WHS - Mink 2007	34,492	2,316	17.1%	0.94 [0.85, 1.04]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	12.5%	0.61 [0.47, 0.79]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	14.3%	0.76 [0.63, 0.93]
Migrant Study - Hjartaker 2015 - cauliflower	9,766	4,595	17.8%	0.85 [0.80, 0.90]
Migrant Study - Hjartaker 2015 - cabbage	-	-	14.3%	1.22 [1.00, 1.49]
PLSAW - Blekkenhorst 2017	1,226	238	13.1%	0.48 [0.38, 0.61]
Total (95% CI) [Random Effects]	**187,730**	**13,081**	**100.0%**	**0.80 [0.68, 0.94]**

Heterogeneity: Tau² = 0.04; Chi² = 48.35, df = 7 (P < 0.00001); I² = 86%

Test for overall effect: Z = 2.75 (P = 0.006)

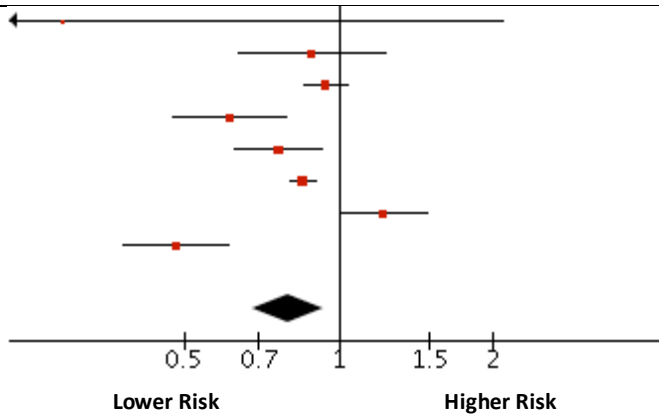


Figure S28. Relation between intake of cruciferous vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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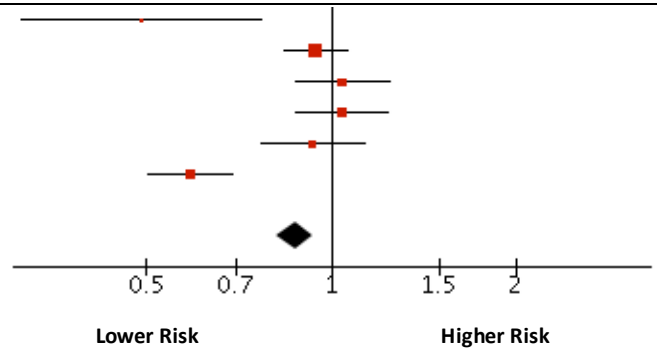
GREEN LEAFY VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	2.3%	0.49 [0.31, 0.77]
Health Food Shoppers - Appleby 2002	10,741	282	34.4%	0.94 [0.84, 1.06]
MONICA Switzerland - Vormund 2015 - M	8,665	751	15.3%	1.04 [0.87, 1.24]
Migrant Study - Hjartaker 2015	9,766	4,595	16.3%	1.04 [0.88, 1.23]
MONICA Switzerland - Vormund 2015 - F	9,196	634	12.4%	0.93 [0.77, 1.13]
PLSAW - Blekkenhorst 2017	1,226	238	19.3%	0.59 [0.50, 0.69]
Total (95% CI)	**40,893**	**6,661**	100.0%	0.87 [0.81, 0.94]

Heterogeneity: Chi² = 40.44, df = 5 (P < 0.00001); I² = 88%

Test for overall effect: Z = 3.86 (P = 0.0001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	10.4%	0.49 [0.31, 0.77]
Health Food Shoppers - Appleby 2002	10,741	282	19.0%	0.94 [0.84, 1.06]
Migrant Study - Hjartaker 2015	9,766	4,595	17.8%	1.04 [0.88, 1.23]
MONICA Switzerland - Vormund 2015 - M	8,665	751	17.6%	1.04 [0.87, 1.24]
MONICA Switzerland - Vormund 2015 - F	9,196	634	17.1%	0.93 [0.77, 1.13]
PLSAW - Blekkenhorst 2017	1,226	238	18.1%	0.59 [0.50, 0.69]
Total (95% CI) [Random Effects]	**40,893**	**6,661**	**100.0%**	**0.84 [0.68, 1.03]**

Heterogeneity: Tau² = 0.06; Chi² = 40.44, df = 5 (P < 0.00001); I² = 88%

Test for overall effect: Z = 1.68 (P = 0.09)

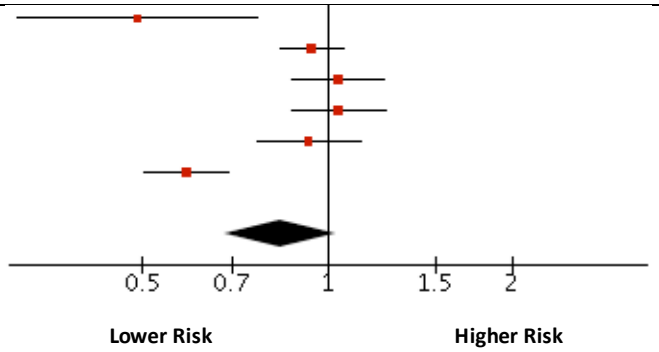


Figure S29. Relation between intake of green leafy vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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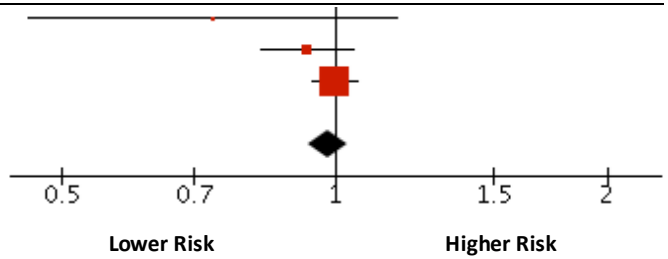
TOMATOES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	1.2%	0.73 [0.46, 1.17]
Iowa WHS - Mink 2007	34,492	2,316	19.8%	0.93 [0.83, 1.05]
Migrant Study - Hjartaker 2015	9,766	4,595	79.0%	1.00 [0.94, 1.06]
Total (95% CI)	**45,557**	**7,072**	**100.0%**	**0.98 [0.93, 1.04]**

Heterogeneity: Chi² = 2.59, df = 2 (P = 0.27); I² = 23%

Test for overall effect: Z = 0.66 (P = 0.51)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	2.5%	0.73 [0.46, 1.17]
Iowa WHS - Mink 2007	34,492	2,316	30.0%	0.93 [0.83, 1.05]
Migrant Study - Hjartaker 2015	9,766	4,595	67.6%	1.00 [0.94, 1.06]
Total (95% CI) [Random Effects]	**45,557**	**7,072**	**100.0%**	**0.97 [0.90, 1.05]**

Heterogeneity: Tau² = 0.00; Chi² = 2.59, df = 2 (P = 0.27); I² = 23%

Test for overall effect: Z = 0.75 (P = 0.45)

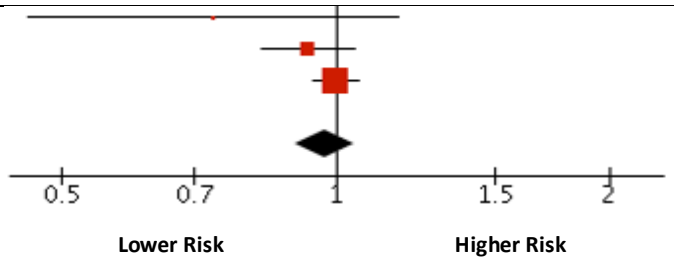
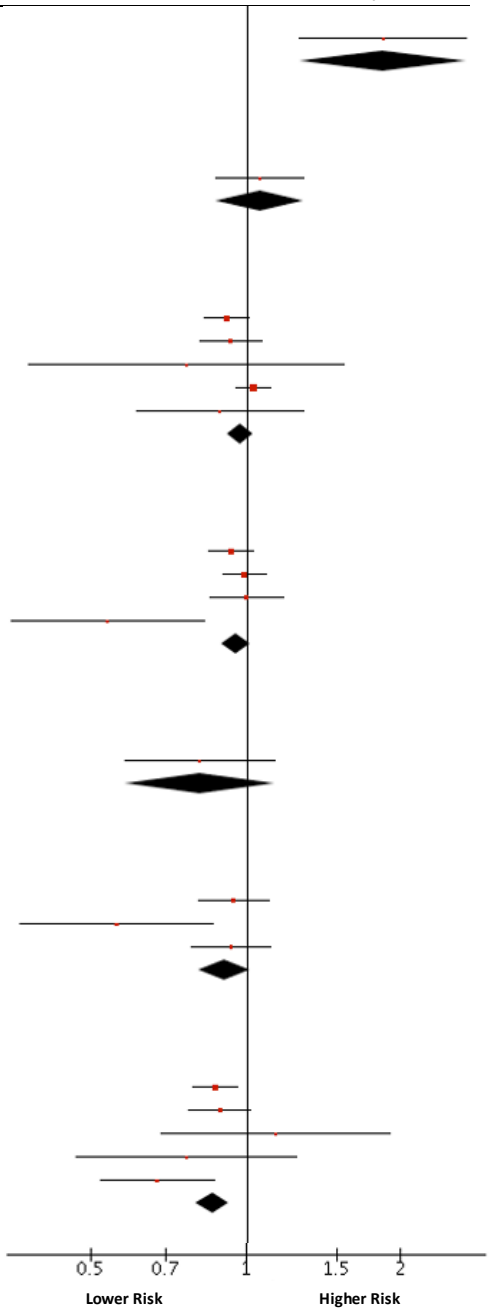


Figure S30. Relation between tomato intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Apricots				
DIET-HD - Saglimbene 2017	9,757	515	0.8%	1.84 [1.27, 2.67]
Subtotal (95% CI)	**9,757**	**515**	**0.8%**	**1.84 [1.27, 2.67]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 3.21 (P = 0.001)				
Bananas				
Migrant Study - Hjartaker 2015 - banana	9,766	4,595	3.0%	1.06 [0.87, 1.29]
Subtotal (95% CI)	**9,766**	**4,595**	**3.0%**	**1.06 [0.87, 1.29]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.60 (P = 0.55)				
Berries				
Iowa WHS - Mink 2007 - strawberries	34,492	2,316	11.8%	0.91 [0.83, 1.01]
Iowa WHS - Mink 2007 - blueberries	-	-	6.0%	0.93 [0.81, 1.07]
WHS - Sesso 2007	38,176	204	0.2%	0.76 [0.38, 1.55]
Migrant Study - Hjartaker 2015	9,766	4,595	18.5%	1.03 [0.95, 1.11]
UK Women's Cohort - Lai 2015	30,458	286	0.8%	0.89 [0.61, 1.29]
Subtotal (95% CI)	**112,892**	**7,401**	**37.4%**	**0.97 [0.92, 1.03]**
Heterogeneity: Chi² = 4.68, df = 4 (P = 0.32); I² = 15%				
Test for overall effect: Z = 1.04 (P = 0.30)				
Citrus				
Iowa WHS - Mink 2007 - grapefruit	34,492	2,316	11.8%	0.93 [0.85, 1.03]
Iowa WHS - Mink 2007 - oranges	-	-	11.8%	0.99 [0.90, 1.09]
Migrant Study - Hjartaker 2015	9,766	4,595	4.1%	1.00 [0.85, 1.18]
UK Women's Cohort - Lai 2015	30,458	286	0.6%	0.54 [0.35, 0.83]
Subtotal (95% CI)	**74,716**	**7,197**	**28.4%**	**0.95 [0.90, 1.02]**
Heterogeneity: Chi² = 7.85, df = 3 (P = 0.05); I² = 62%				
Test for overall effect: Z = 1.45 (P = 0.15)				
Fruit Juice				
UK Women's Cohort - Lai 2015	30,458	286	1.0%	0.81 [0.58, 1.13]
Subtotal (95% CI)	**30,458**	**286**	**1.0%**	**0.81 [0.58, 1.13]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 1.24 (P = 0.22)				
Grapes				
Iowa WHS - Mink 2007 - grapes and raisins	34,492	2,316	4.6%	0.94 [0.81, 1.10]
UK Women's Cohort - Lai 2015 - grapes	30,458	286	0.6%	0.56 [0.36, 0.86]
Migrant Study - Hjartaker 2015 - grapes	9,766	4,595	3.7%	0.93 [0.78, 1.11]
Subtotal (95% CI)	**74,716**	**7,197**	**8.9%**	**0.90 [0.81, 1.01]**
Heterogeneity: Chi² = 5.12, df = 2 (P = 0.08); I² = 61%				
Test for overall effect: Z = 1.73 (P = 0.08)				
Pommes				
Iowa WHS - Mink 2007	34,492	2,316	11.8%	0.87 [0.79, 0.96]
Migrant Study - Hjartaker 2015	9,766	4,595	6.0%	0.89 [0.77, 1.02]
UK Women's Cohort - Lai 2015	30,458	286	0.4%	1.14 [0.68, 1.90]
Hodgson et al. 2016	1,456	235	0.5%	0.76 [0.47, 1.25]
DIET-HD - Saglimbene 2017	9,757	515	1.8%	0.67 [0.52, 0.86]
Subtotal (95% CI)	**85,929**	**7,947**	**20.5%**	**0.86 [0.80, 0.92]**
Heterogeneity: Chi² = 5.30, df = 4 (P = 0.26); I² = 25%				
Test for overall effect: Z = 4.04 (P < 0.0001)				
Test for subgroup differences: Chi² = 22.57, df = 6 (P = 0.0010), I² = 73.4%				



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B. Random Effects

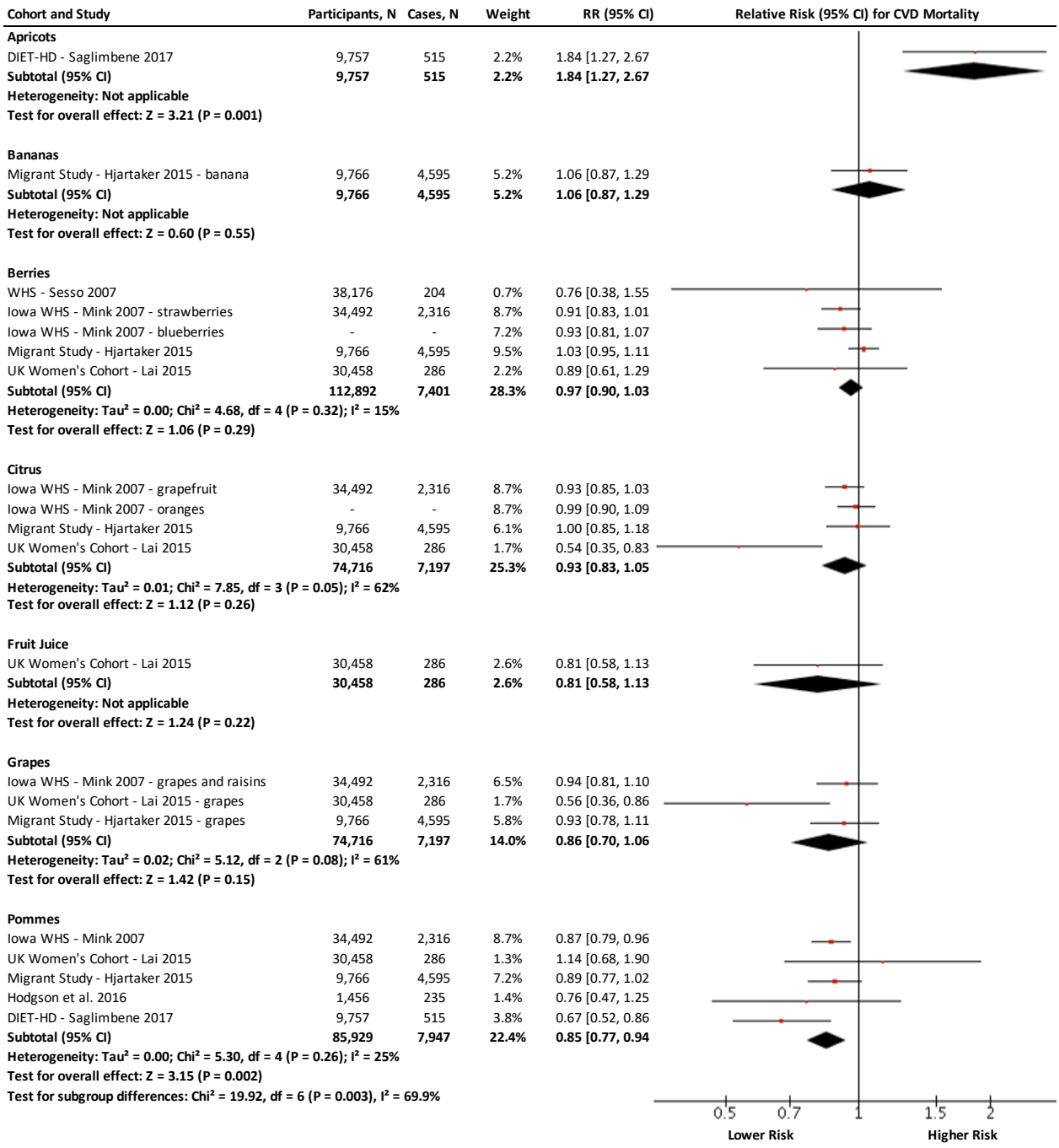
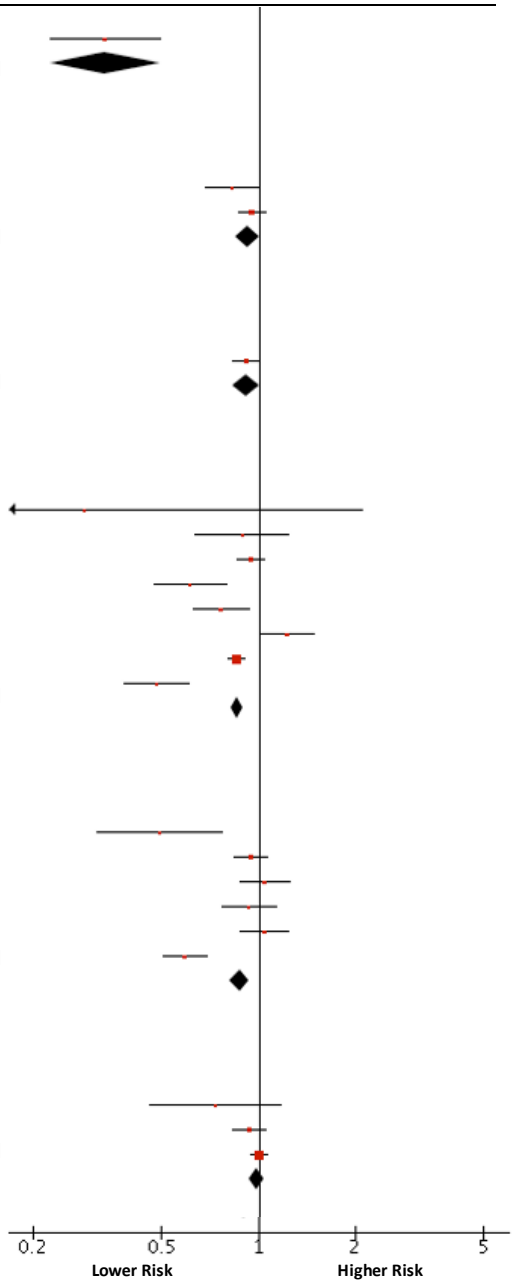


Figure S31. Relation between sources of fruit and CVD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
1.11.1 Allium				
PLSAW - Blekkenhorst 2017	1,226	238	0.5%	0.33 [0.22, 0.49]
Subtotal (95% CI)	**1,226**	**238**	**0.5%**	**0.33 [0.22, 0.49]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 5.50 (P < 0.00001)				
1.11.2 Carrots				
Zutphen Elderly - Buijsse 2008- Carrots	559	197	2.0%	0.83 [0.68, 1.01]
Miigrant Study - Hjartaker 2015 - carrots	9,766	4,595	8.0%	0.95 [0.86, 1.05]
Subtotal (95% CI)	**10,325**	**4,792**	**10.0%**	**0.92 [0.85, 1.01]**
Heterogeneity: Tau² = 0.00; Chi² = 1.57, df = 1 (P = 0.21); I² = 36%				
Test for overall effect: Z = 1.44 (P = 0.15)				
1.11.3 Celery				
Iowa WHS - Mink 2007 - celery	34,492	2,316	8.0%	0.91 [0.83, 1.01]
Subtotal (95% CI)	**34,492**	**2,316**	**8.0%**	**0.91 [0.83, 1.01]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 1.80 (P = 0.07)				
1.11.4 Cruciferous				
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	0.0%	0.29 [0.04, 2.10]
Odyssey - Genkinger 2004	6,151	378	0.7%	0.89 [0.64, 1.24]
Iowa WHS - Mink 2007	34,492	2,316	8.0%	0.94 [0.85, 1.04]
Shanghai Women Health - Zhang 2011 (a)	73,360	3,442	1.2%	0.61 [0.47, 0.79]
Shanghai Men Health - Zhang 2011 (a)	61,436	1,951	2.0%	0.76 [0.63, 0.93]
Migrant Study - Hjartaker 2015 - cabbage	-	-	2.0%	1.22 [1.00, 1.49]
Migrant Study - Hjartaker 2015 - cauliflower	9,766	4,595	22.2%	0.85 [0.80, 0.90]
PLSAW - Blekkenhorst 2017	1,226	238	1.4%	0.48 [0.38, 0.61]
Subtotal (95% CI)	**187,730**	**13,081**	**37.4%**	**0.85 [0.82, 0.89]**
Heterogeneity: Tau² = 0.04; Chi² = 48.35, df = 7 (P < 0.00001); I² = 86%				
Test for overall effect: Z = 2.75 (P = 0.006)				
1.11.5 Green leafy				
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	0.4%	0.49 [0.31, 0.77]
Health Food Shoppers - Appleby 2002	10,741	282	5.5%	0.94 [0.84, 1.06]
MONICA Switzerland - Vormund 2015 - M	8,665	751	2.5%	1.04 [0.87, 1.24]
MONICA Switzerland - Vormund 2015 - F	9,196	634	2.0%	0.93 [0.77, 1.13]
Migrant Study - Hjartaker 2015	9,766	4,595	2.6%	1.04 [0.88, 1.23]
PLSAW - Blekkenhorst 2017	1,226	238	3.1%	0.59 [0.50, 0.69]
Subtotal (95% CI)	**40,893**	**6,661**	**16.1%**	**0.87 [0.81, 0.94]**
Heterogeneity: Tau² = 0.06; Chi² = 40.44, df = 5 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 1.68 (P = 0.09)				
1.11.6 Tomatoes				
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	0.3%	0.73 [0.46, 1.17]
Iowa WHS - Mink 2007	34,492	2,316	5.5%	0.93 [0.83, 1.05]
Migrant Study - Hjartaker 2015	9,766	4,595	22.2%	1.00 [0.94, 1.06]
Subtotal (95% CI)	**45,557**	**7,072**	**28.0%**	**0.98 [0.93, 1.04]**
Heterogeneity: Tau² = 0.00; Chi² = 2.59, df = 2 (P = 0.27); I² = 23%				
Test for overall effect: Z = 0.75 (P = 0.45)				
Test for subgroup differences: Chi² = 41.70, df = 5 (P < 0.00001), I² = 88.0%				



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B. Random Effects

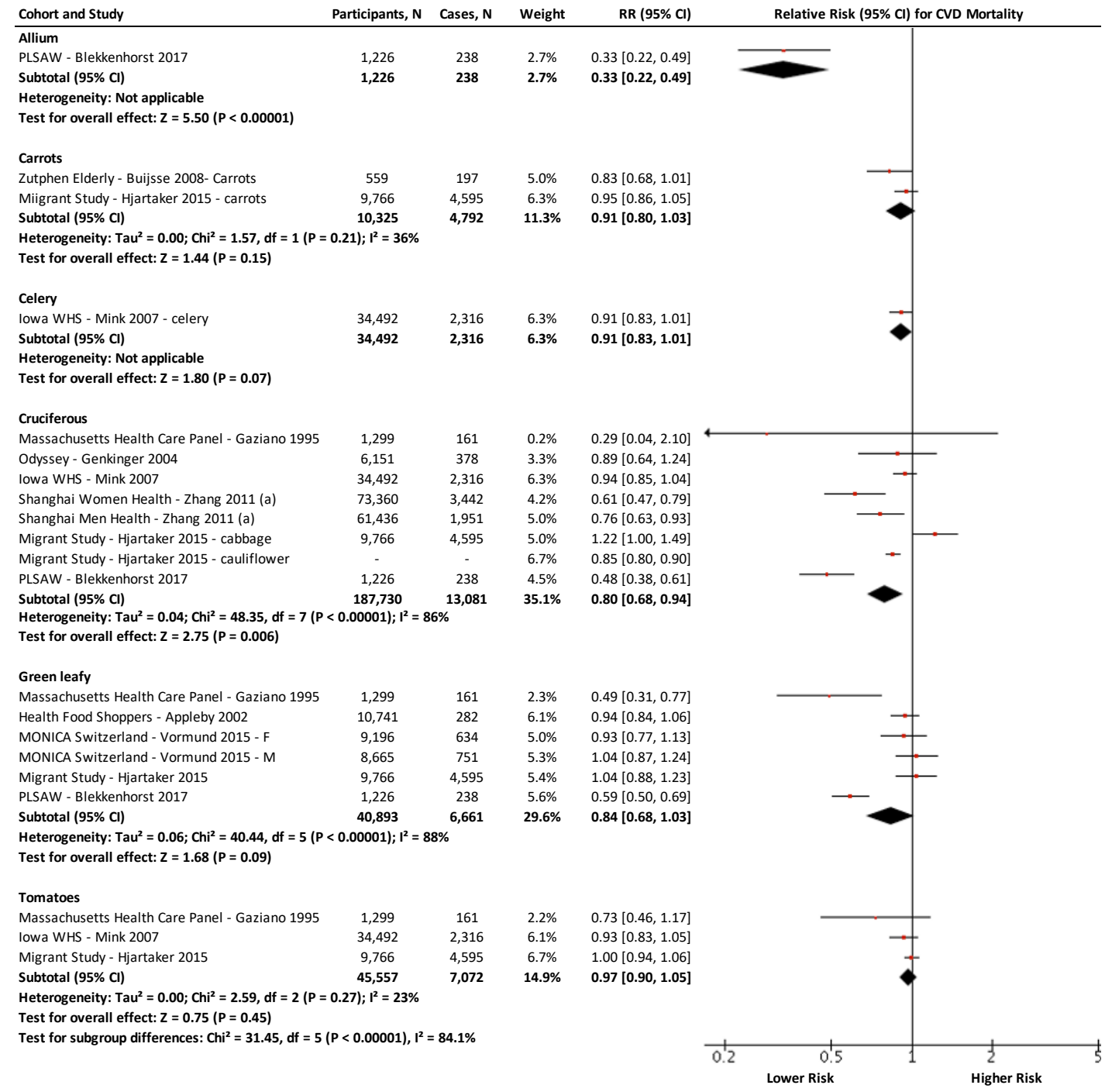


Figure S32. Relation between sources of vegetables and CVD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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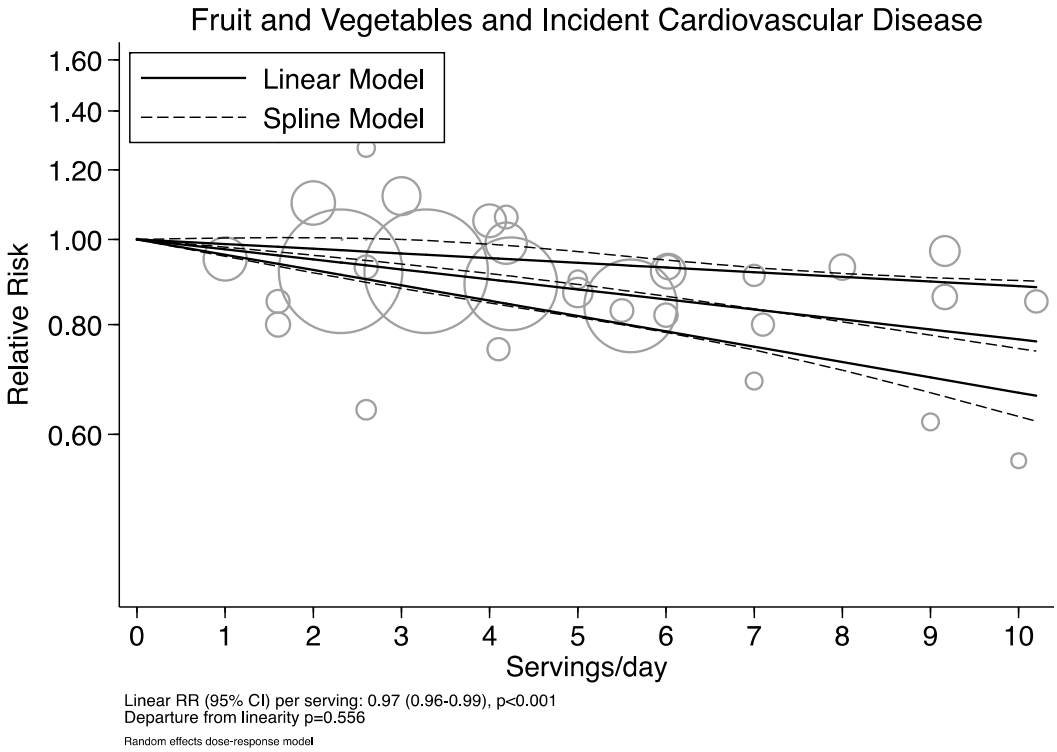


Figure S33. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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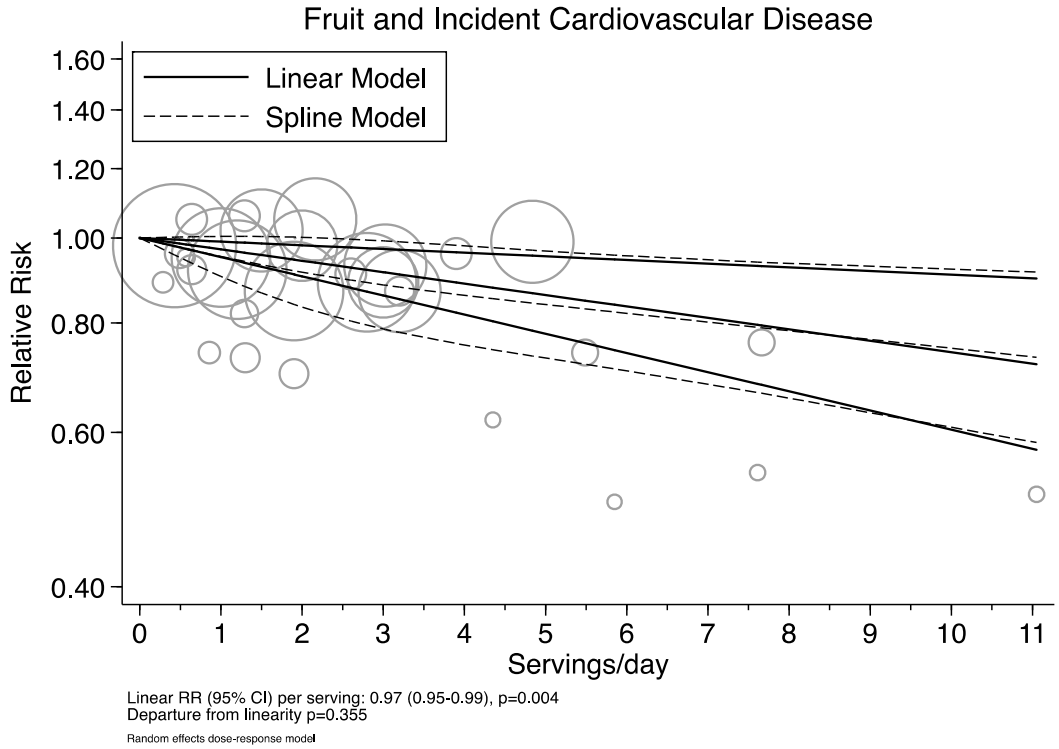


Figure S34. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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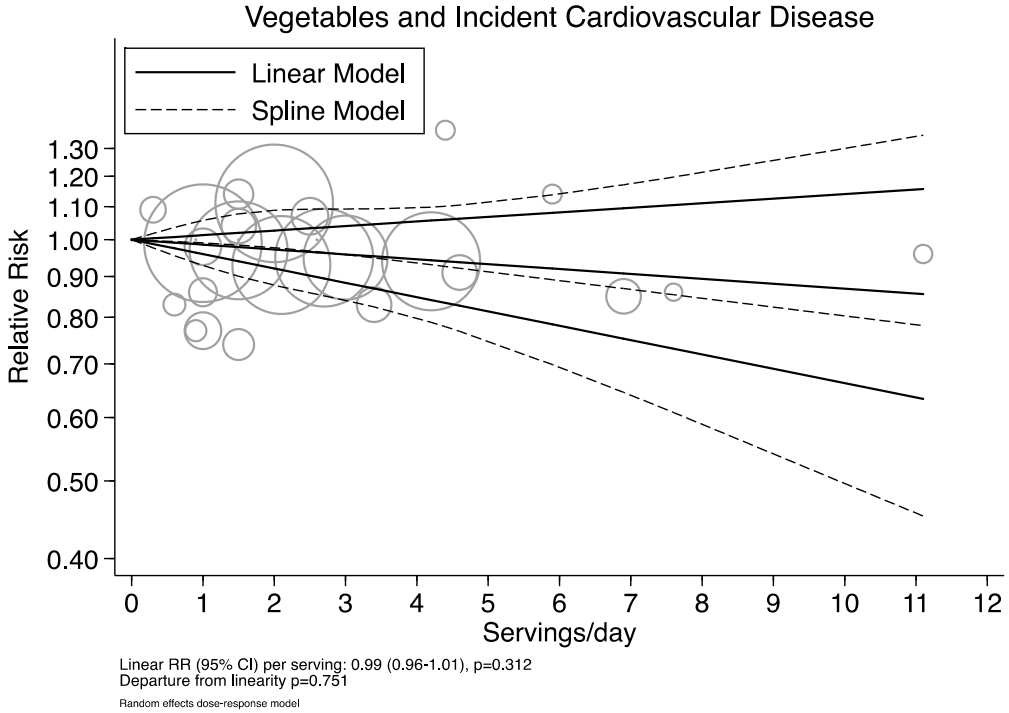


Figure S35. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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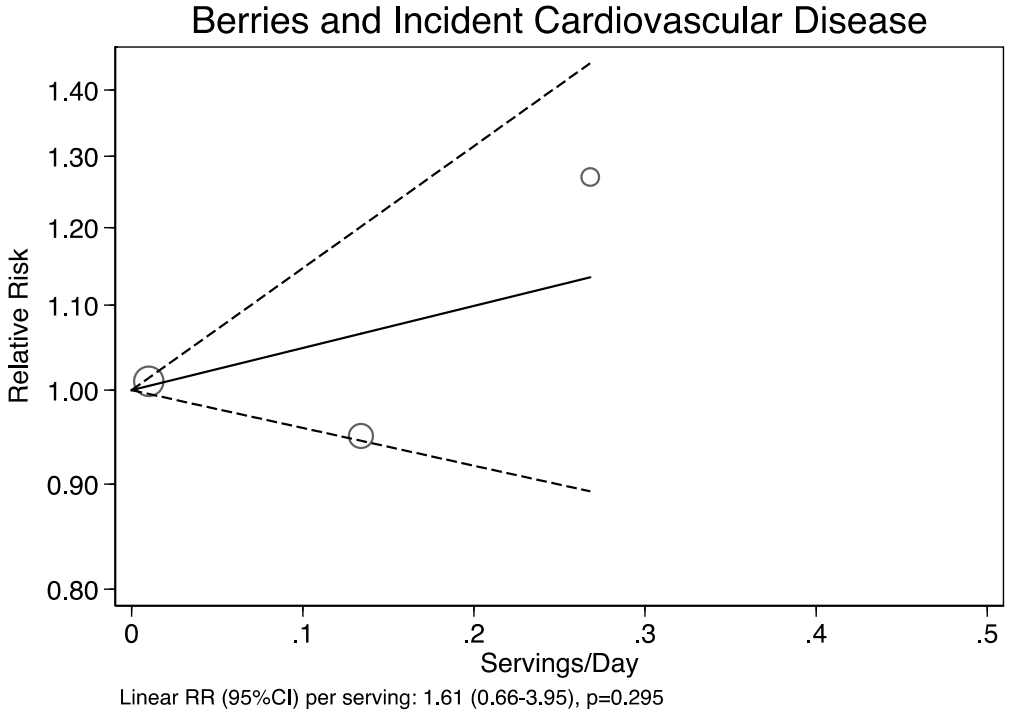


Figure S36. Linear dose-response relation between increasing berries intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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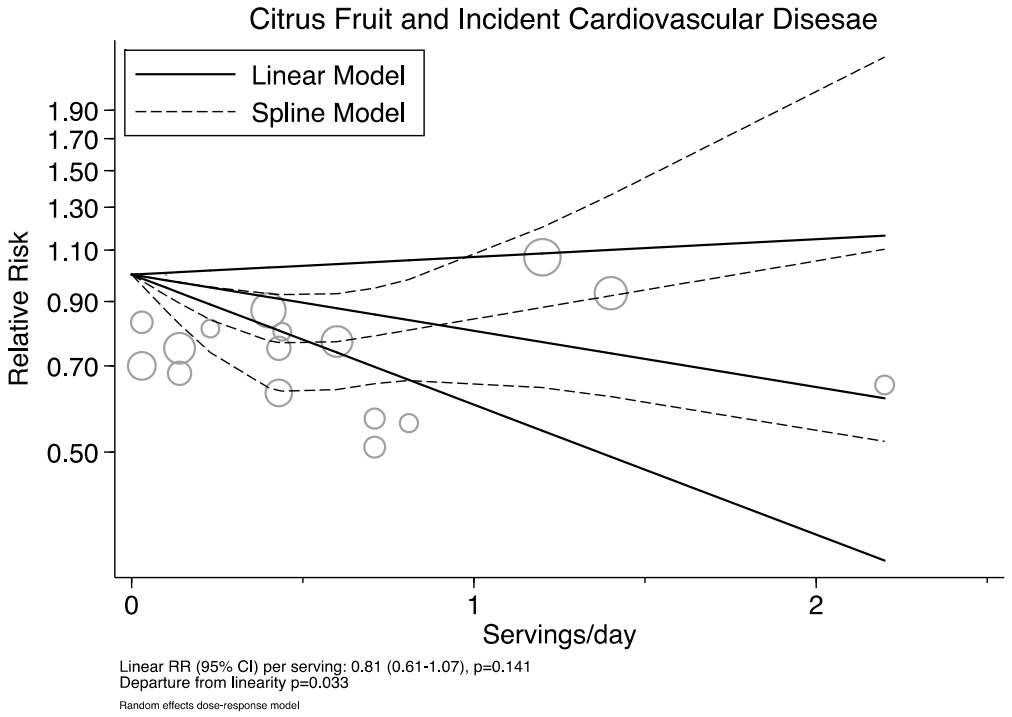


Figure S37. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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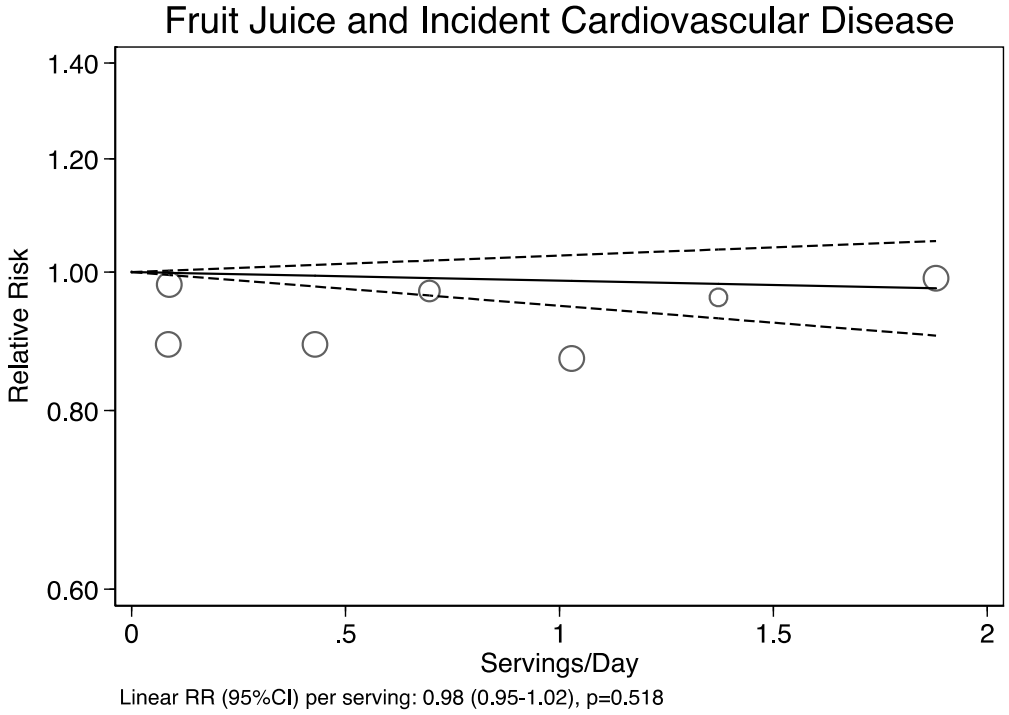


Figure S38. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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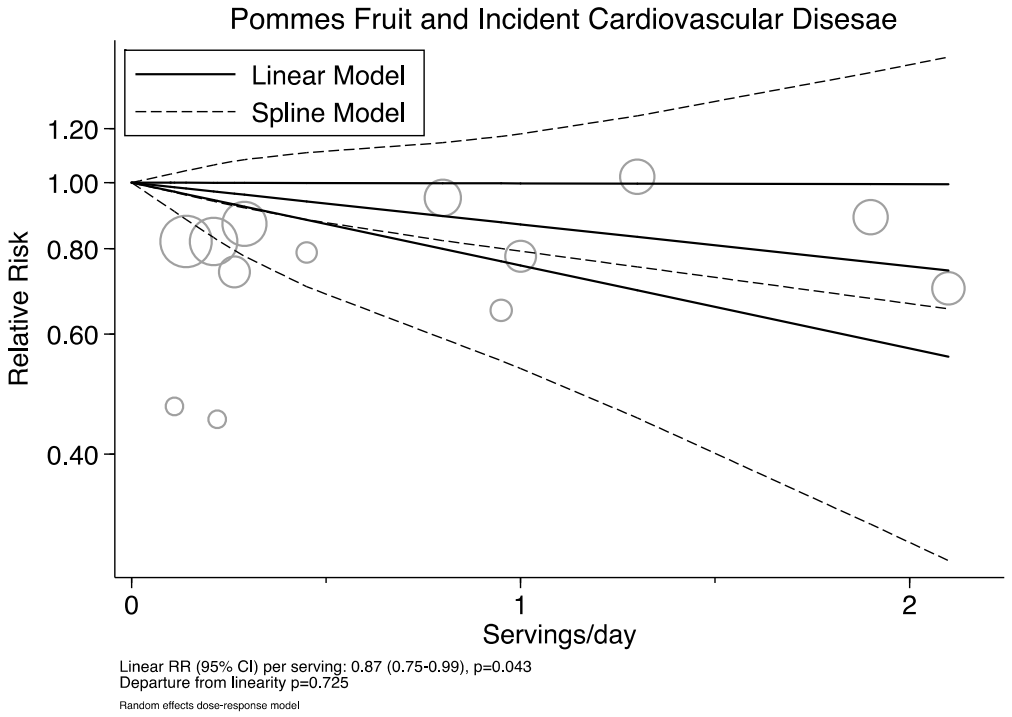


Figure S39. Linear dose-response relation between increasing pommes intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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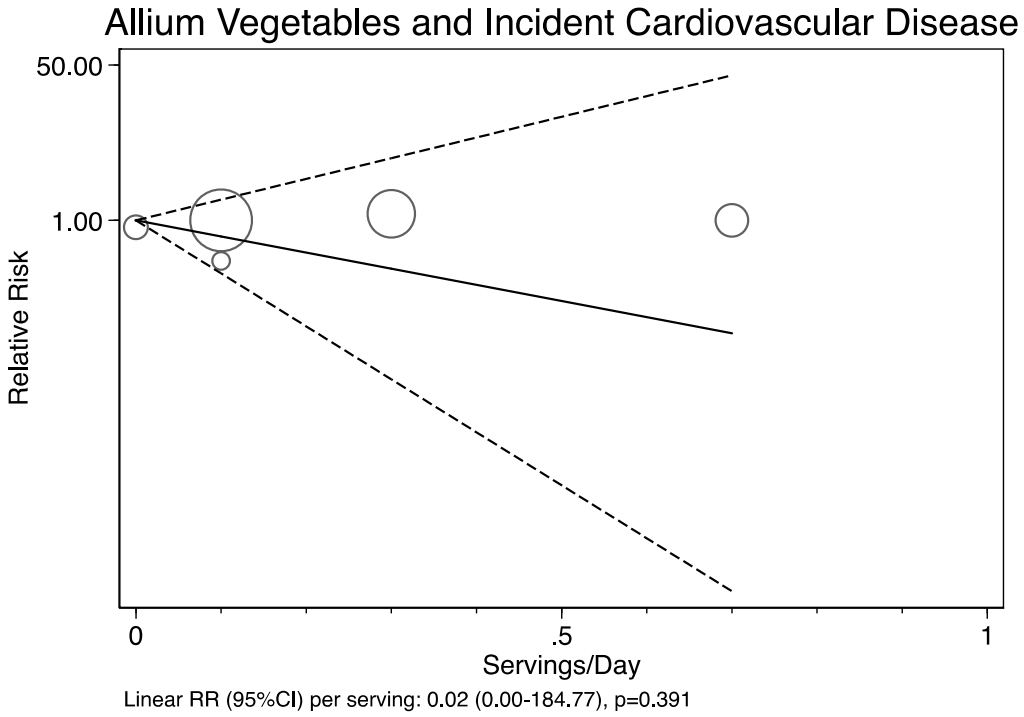


Figure S40. Linear dose-response relation between increasing intake of allium vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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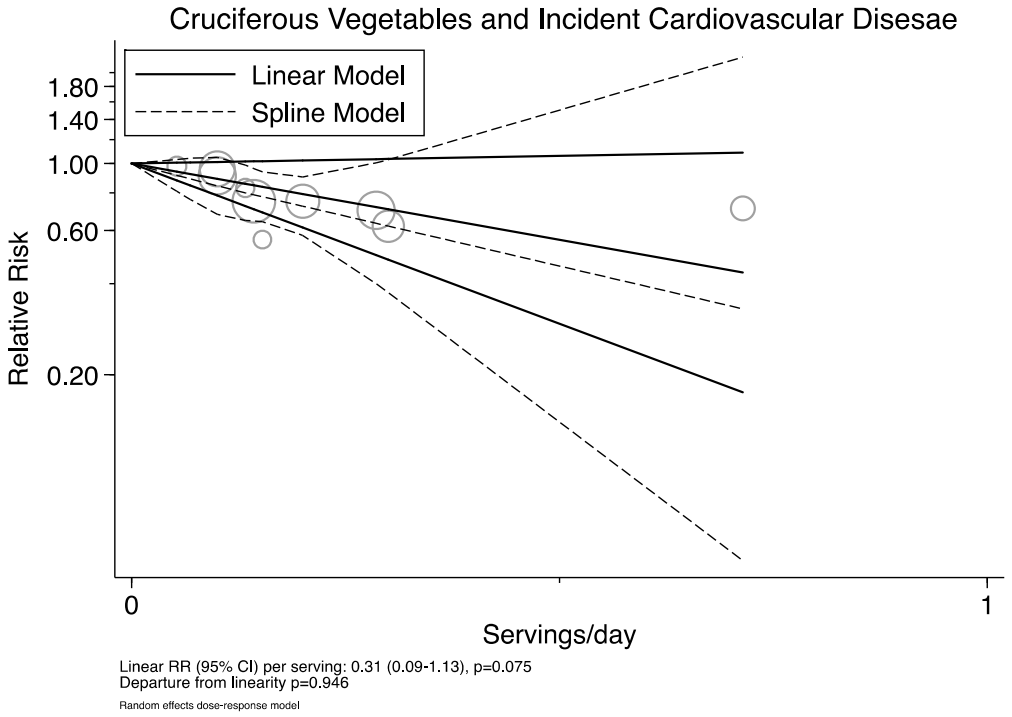


Figure S41. Linear dose-response relation between increasing intake of cruciferous vegetables and incidence of cardiovascular disease y. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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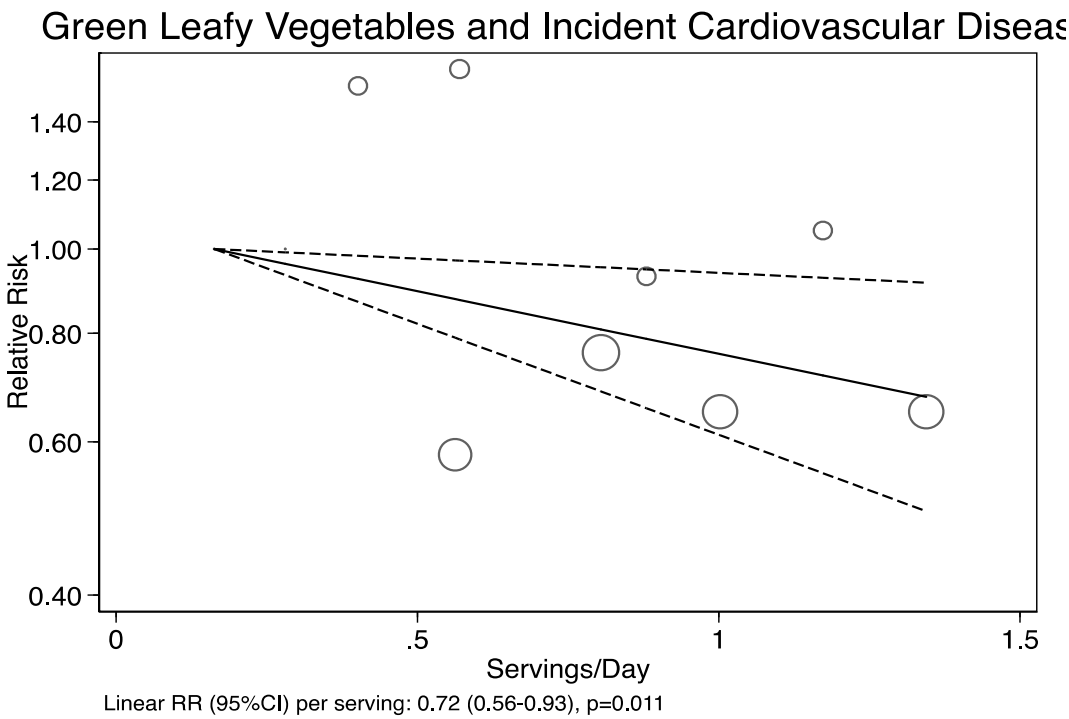


Figure S42. Linear dose-response relation between increasing intake of green leafy vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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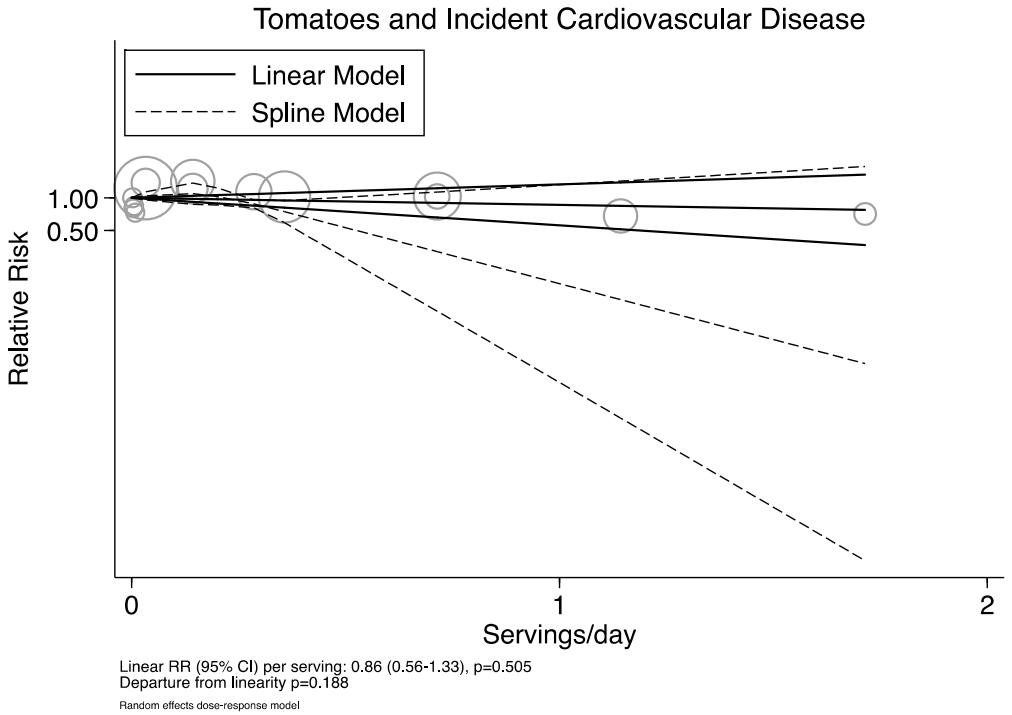


Figure S43. Linear and cubic-spline dose-response relation between increasing tomato intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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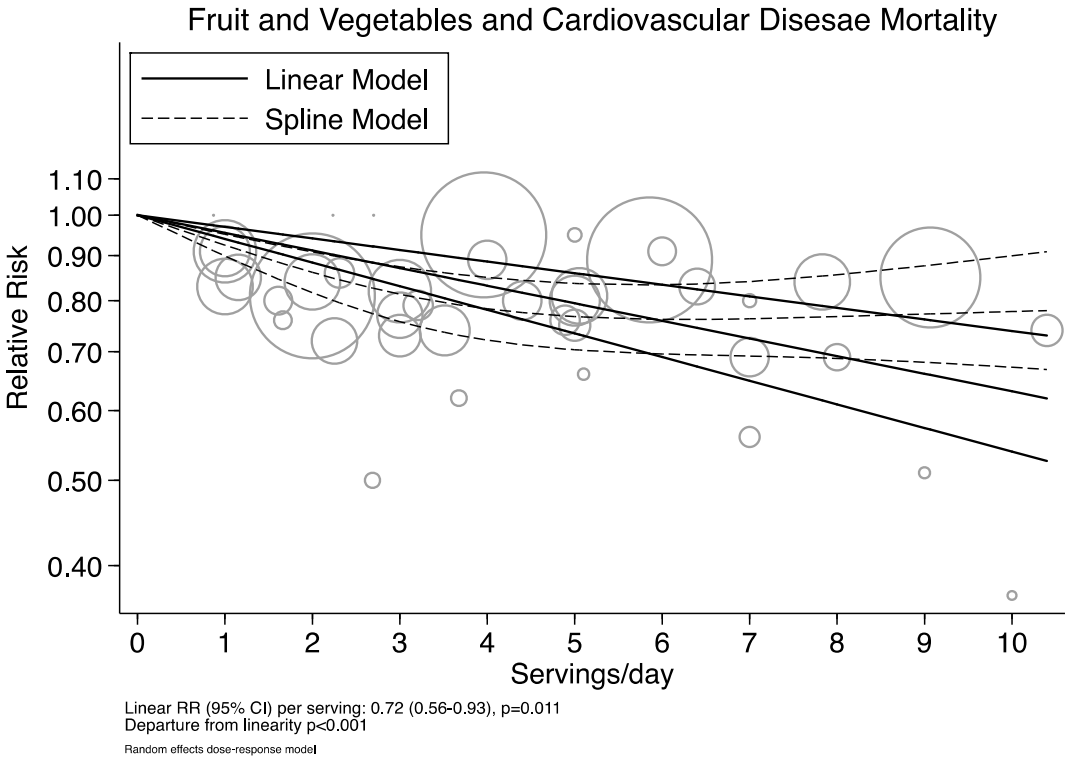


Figure S44. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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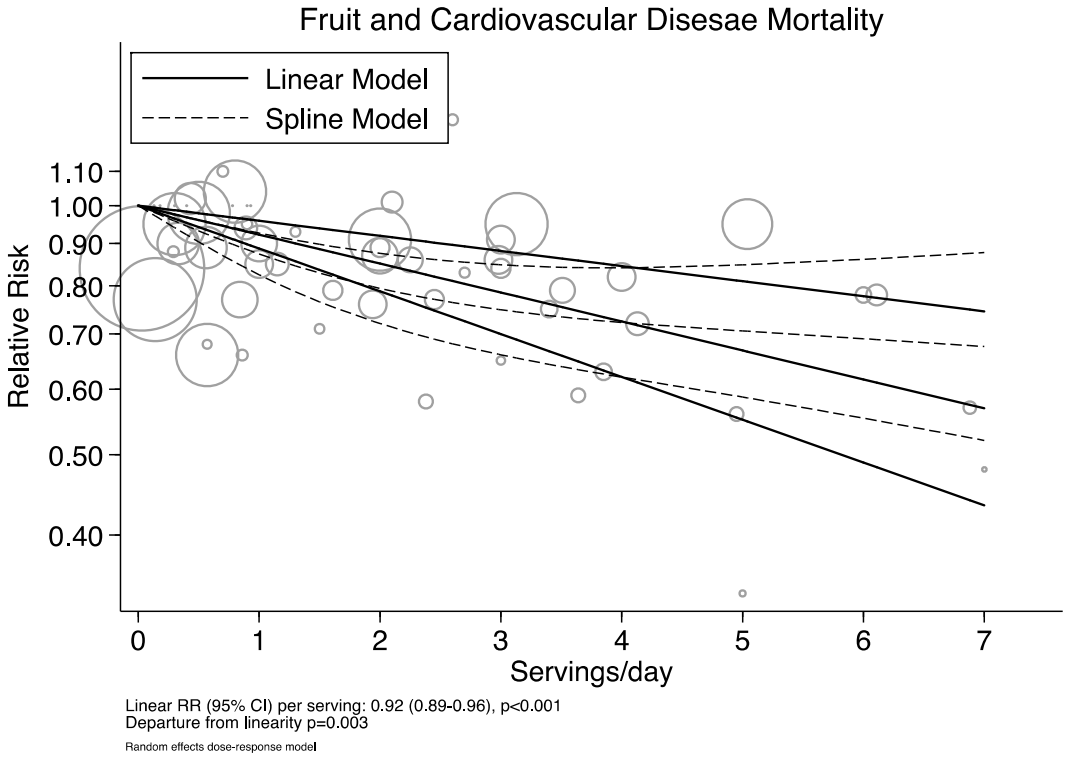


Figure S45. Linear and cubic-spline dose-response relation between increasing fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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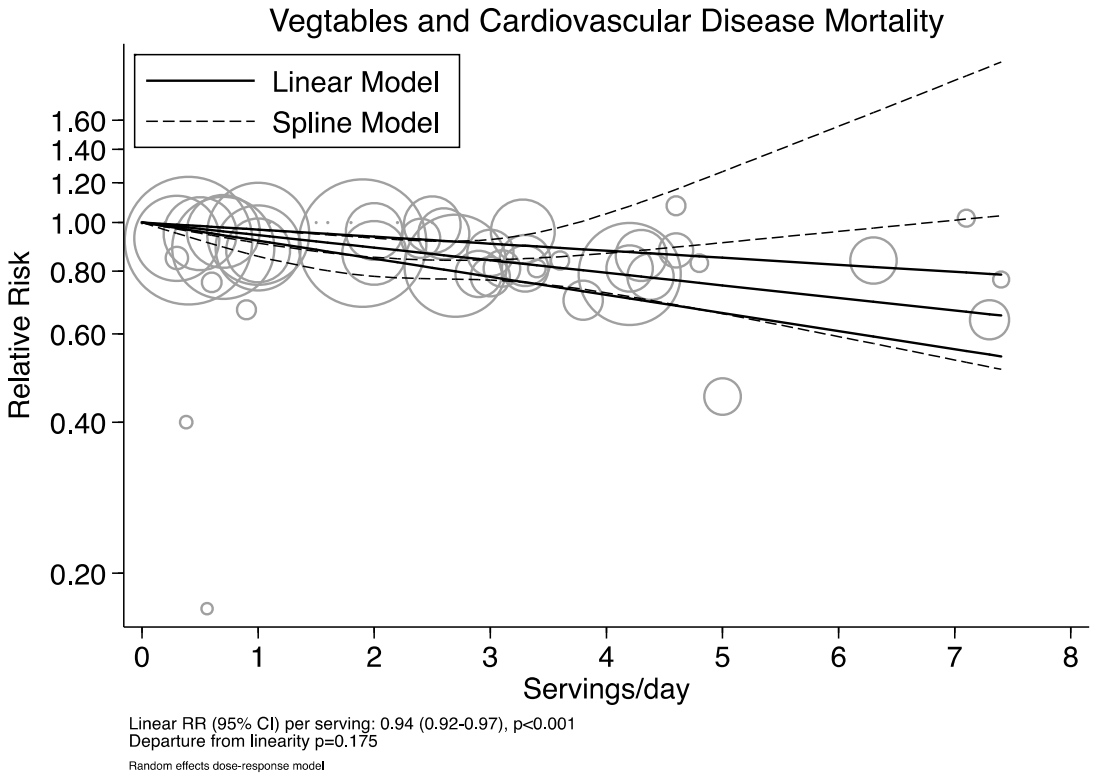


Figure S46. Linear and cubic-spline dose-response relation between increasing intake of vegetables and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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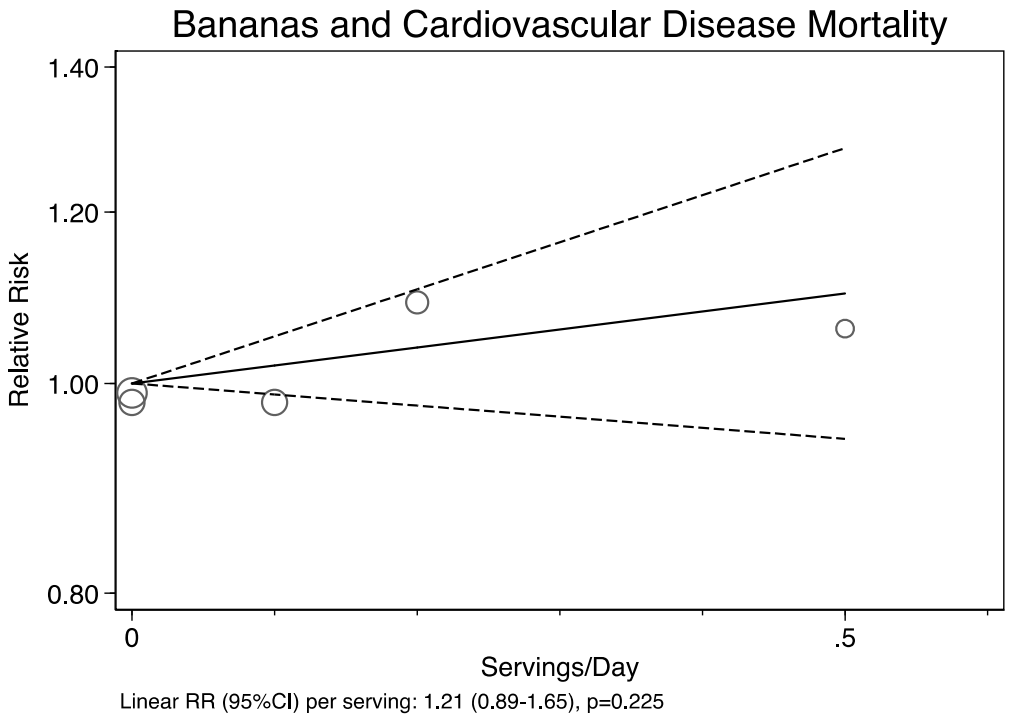


Figure S47. Linear dose-response relation between increasing banana intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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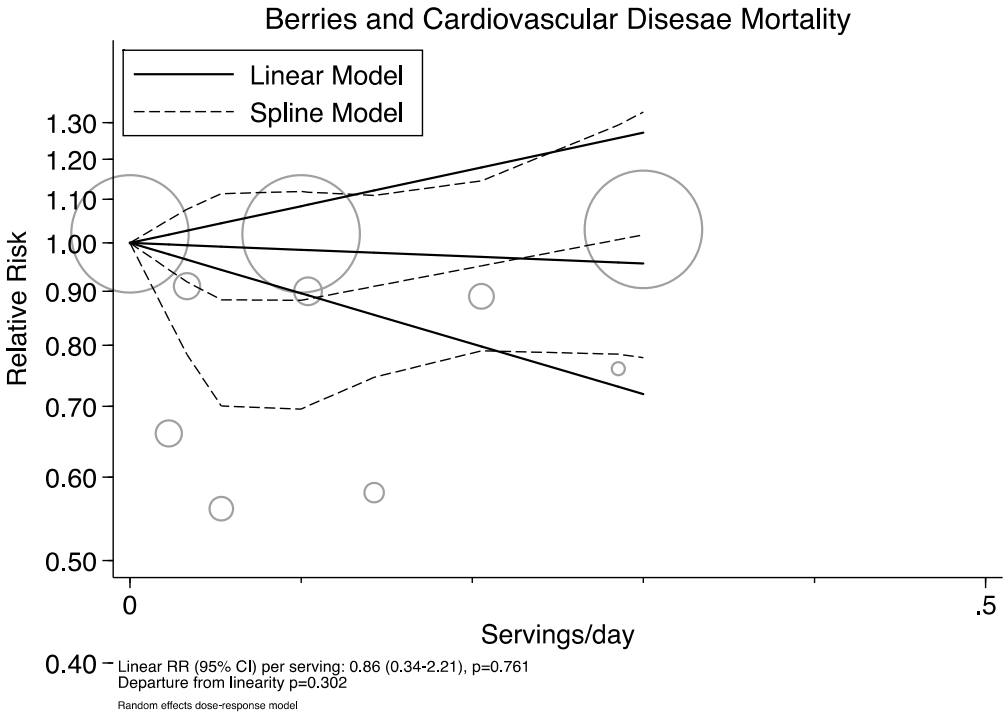


Figure S48. Linear and cubic-spline dose-response relation between increasing berry fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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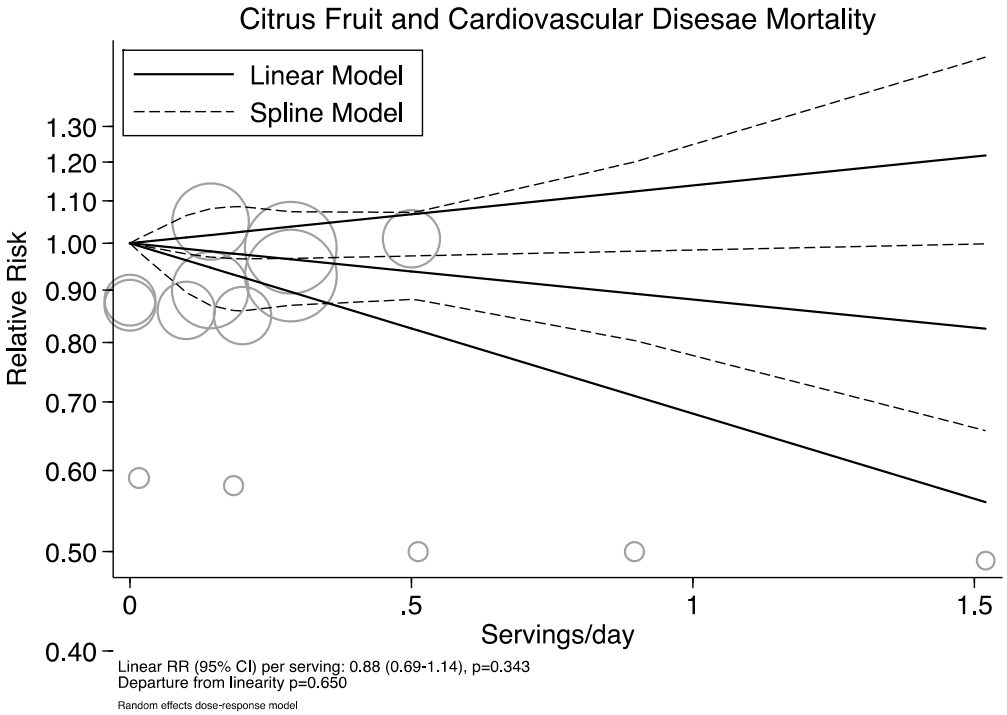


Figure S49. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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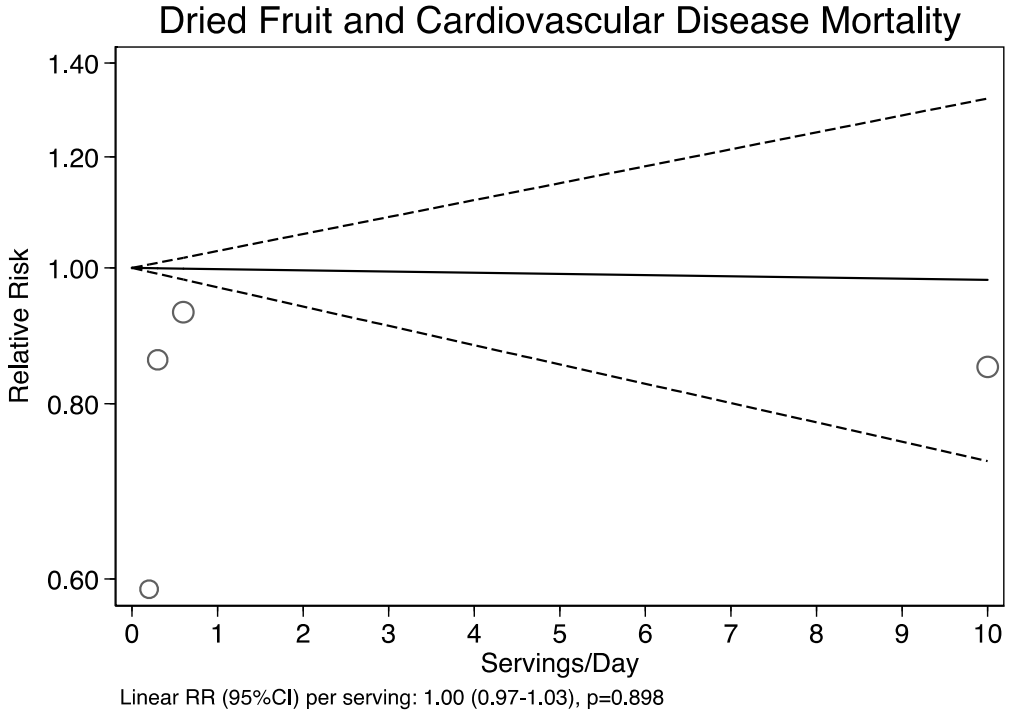


Figure S50. Linear and cubic-spline dose-response relation between increasing dried fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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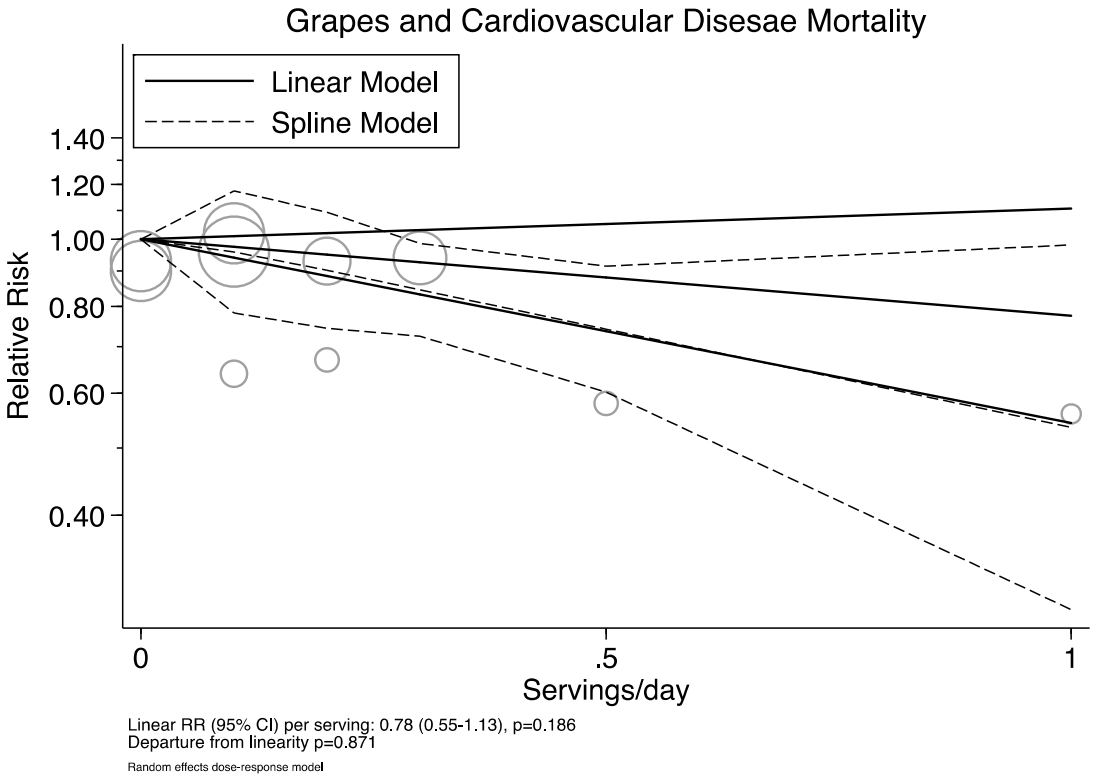


Figure S51. Linear and cubic-spline dose-response relation between increasing grapes intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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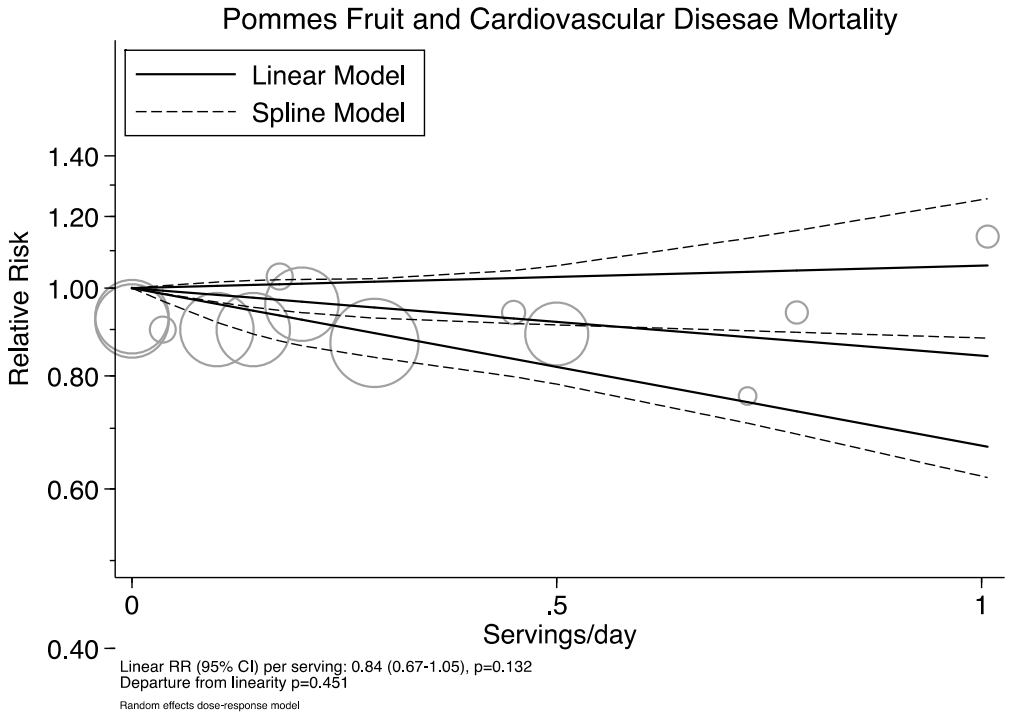


Figure S52. Linear and cubic-spline dose-response relation between increasing pommes intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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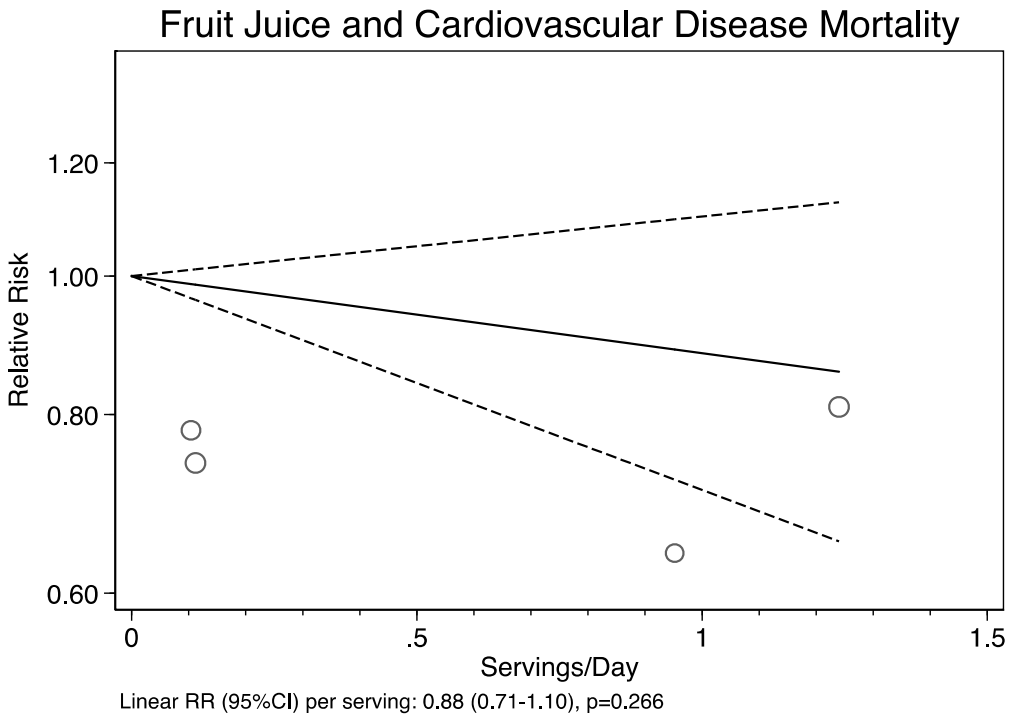


Figure S53. Linear dose-response relation between increasing fruit juice intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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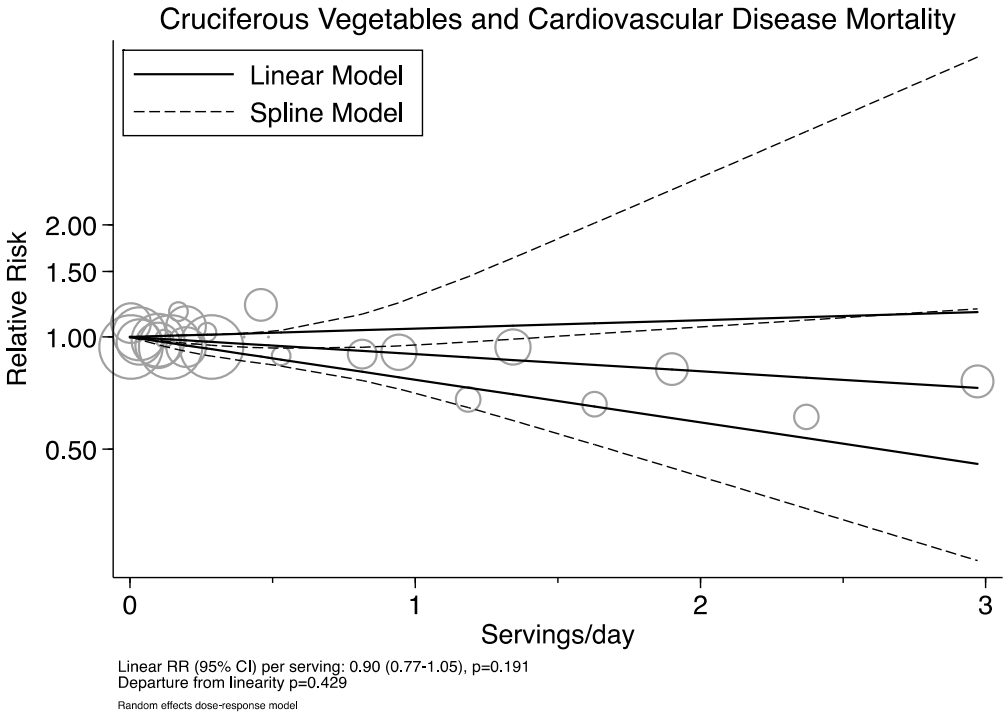


Figure S54. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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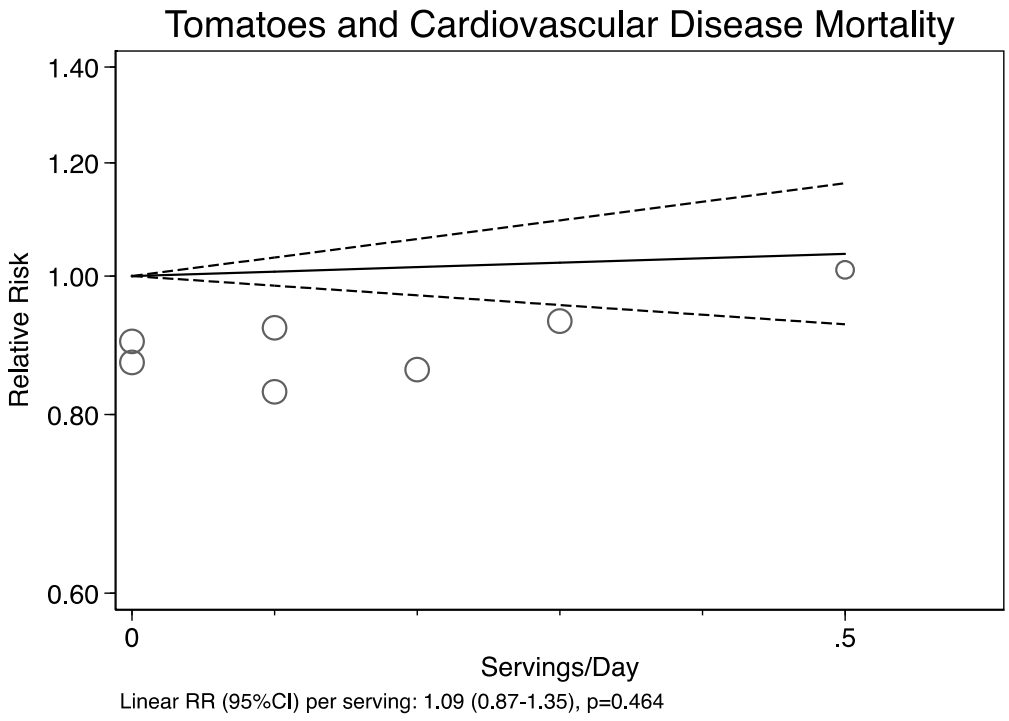


Figure S55. Linear dose-response relation between increasing tomato intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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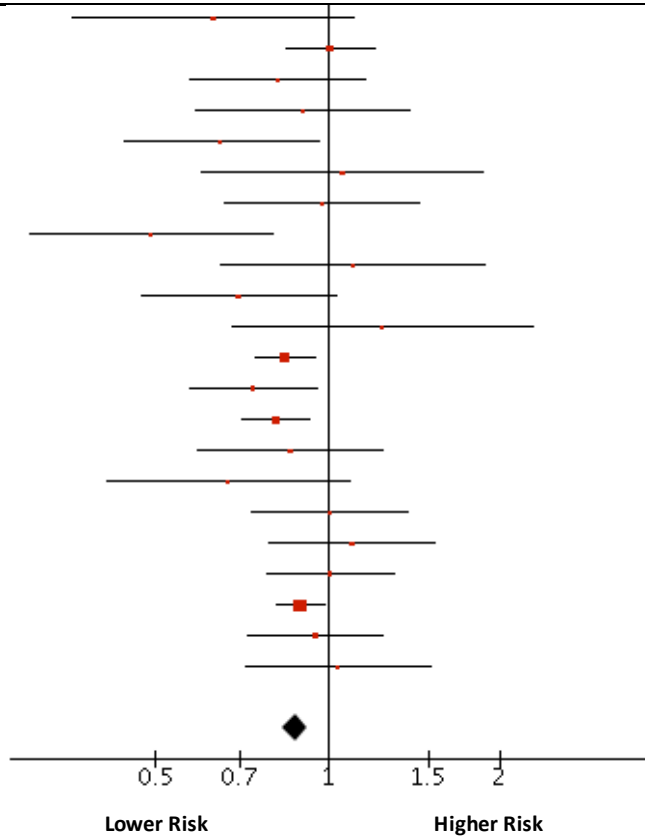
TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	126	0.8%	0.63 [0.36, 1.11]
National Health & Nutrition - Bazzano 2002	9,608	1,786	8.2%	1.01 [0.85, 1.20]
ARIC - Steffen 2003	11,940	535	2.0%	0.82 [0.58, 1.17]
EPIC Norway - Bingham 2008	11,134	678	1.4%	0.90 [0.59, 1.39]
Swedish National Farm Register - Holmberg 2009	1,738	138	1.7%	0.65 [0.44, 0.96]
PRIME - Dauchet 2010 - never smokers	2,410	145	0.8%	1.06 [0.60, 1.87]
PRIME - Dauchet 2010 - former smokers	3,353	140	1.7%	0.98 [0.66, 1.45]
PRIME - Dauchet 2010 - current smokers	2,297	230	1.1%	0.49 [0.30, 0.80]
EPIC Italy - Bendinelli 2011	29,689	144	0.9%	1.11 [0.65, 1.88]
MORGEN - Oude Griep 2011 (b)	20,069	245	1.7%	0.70 [0.47, 1.03]
Japan Diabetes Complications Study - Tanaka 201	1,414	96	0.7%	1.25 [0.68, 2.29]
HPFS - Bhupathiraju 2013	42,135	3,607	18.4%	0.84 [0.75, 0.95]
Health and Wellbeing Surveillance - Gunnell 2013	14,890	538	3.9%	0.74 [0.57, 0.96]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	13.5%	0.81 [0.71, 0.93]
Shanghai Men Health - Yu 2014	67,211	148	1.8%	0.86 [0.59, 1.25]
Shanghai Women Health - Yu 2014	55,242	217	1.1%	0.67 [0.41, 1.09]
British Regional Heart - Atkins 2014	3,328	307	2.6%	1.01 [0.74, 1.38]
SABRE - Eriksen 2015 - European	1,090	207	2.3%	1.11 [0.79, 1.54]
SABRE - Eriksen 2015 - South Asian	1,006	313	3.9%	1.01 [0.78, 1.30]
CCHS - Kobylecki 2015	78,527	2,823	26.5%	0.90 [0.81, 0.99]
PURE - Miller 2017	135,335	2,143	3.4%	0.95 [0.72, 1.25]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	1.8%	1.04 [0.72, 1.51]
Total (95% CI)	**619,182**	**17,987**	**100.0%**	**0.88 [0.83, 0.92]**

Heterogeneity: Chi² = 25.25, df = 21 (P = 0.24); I² = 17%

Test for overall effect: Z = 5.12 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	126	1.2%	0.63 [0.36, 1.11]
National Health & Nutrition - Bazzano 2002	9,608	1,786	9.0%	1.01 [0.85, 1.20]
ARIC - Steffen 2003	11,940	535	2.9%	0.82 [0.58, 1.17]
EPIC Norway - Bingham 2008	11,134	678	2.0%	0.90 [0.59, 1.39]
Swedish National Farm Register - Holmberg 2009	1,738	138	2.4%	0.65 [0.44, 0.96]
PRIME - Dauchet 2010 - never smokers	2,410	145	1.2%	1.06 [0.60, 1.87]
PRIME - Dauchet 2010 - former smokers	3,353	140	2.4%	0.98 [0.66, 1.45]
PRIME - Dauchet 2010 - current smokers	2,297	230	1.5%	0.49 [0.30, 0.80]
EPIC Italy - Bendinelli 2011	29,689	144	1.3%	1.11 [0.65, 1.88]
MORGEN - Oude Griep 2011 (b)	20,069	245	2.4%	0.70 [0.47, 1.03]
Japan Diabetes Complications Study - Tanaka 201	1,414	96	1.0%	1.25 [0.68, 2.29]
HPFS - Bhupathiraju 2013	42,135	3,607	14.8%	0.84 [0.75, 0.95]
Health and Wellbeing Surveillance - Gunnell 2013	14,890	538	5.0%	0.74 [0.57, 0.96]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	12.5%	0.81 [0.71, 0.93]
Shanghai Men Health - Yu 2014	67,211	148	2.6%	0.86 [0.59, 1.25]
Shanghai Women Health - Yu 2014	55,242	217	1.5%	0.67 [0.41, 1.09]
British Regional Heart - Atkins 2014	3,328	307	3.5%	1.01 [0.74, 1.38]
SABRE - Eriksen 2015 - European	1,090	207	3.2%	1.11 [0.79, 1.54]
SABRE - Eriksen 2015 - South Asian	1,006	313	5.0%	1.01 [0.78, 1.30]
CCHS - Kobylecki 2015	78,527	2,823	17.7%	0.90 [0.81, 0.99]
PURE - Miller 2017	135,335	2,143	4.5%	0.95 [0.72, 1.25]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	2.6%	1.04 [0.72, 1.51]
Total (95% CI) [Random Effects]	**619,182**	**17,987**	**100.0%**	**0.88 [0.82, 0.93]**

Heterogeneity: Tau² = 0.00; Chi² = 25.25, df = 21 (P = 0.24); I² = 17%

Test for overall effect: Z = 4.11 (P < 0.0001)

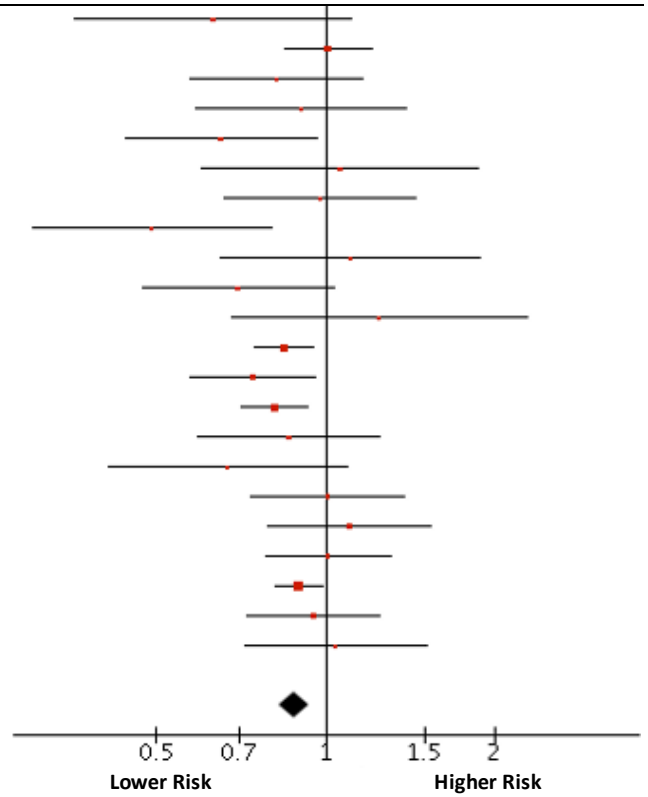


Figure S56. Relation between total fruit and vegetables intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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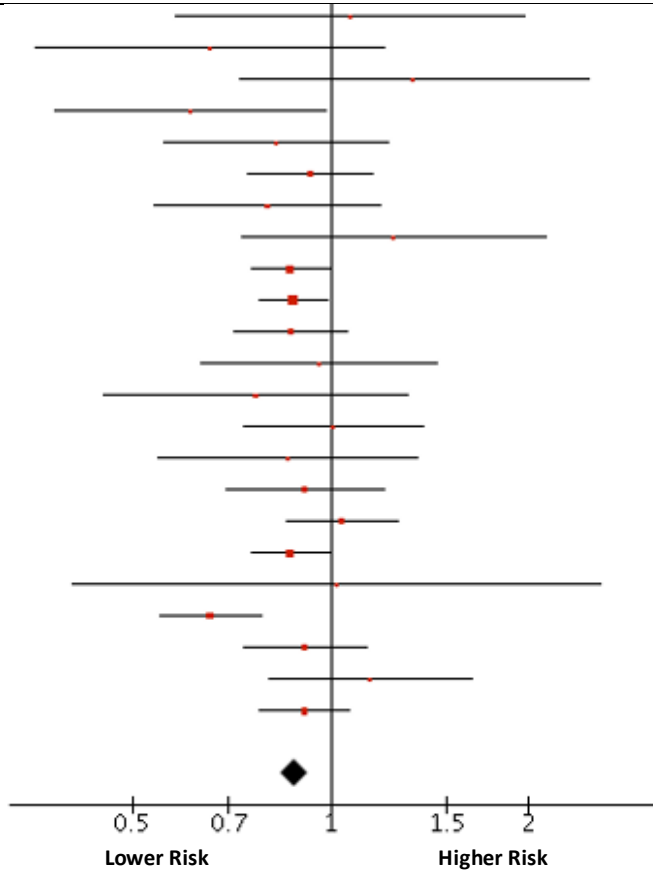
FRUIT AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Adventis Health Study - Fraser -1992	26,473	134	0.6%	1.07 [0.58, 1.97]
WHS - Liu 2000	39,127	126	0.6%	0.66 [0.36, 1.21]
PRIME - Dauchet 2010 - never smokers	2,410	79	0.6%	1.34 [0.73, 2.45]
PRIME - Dauchet 2010 - current smokers	2,297	148	1.1%	0.61 [0.38, 0.98]
PRIME - Dauchet 2010 - former smokers	3,353	140	1.5%	0.83 [0.56, 1.22]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	5.1%	0.93 [0.75, 1.16]
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	1.5%	0.80 [0.54, 1.19]
EPIC Italy - Bendinelli 2011	29,689	144	0.8%	1.25 [0.73, 2.12]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	12.6%	0.87 [0.76, 1.00]
HPFS - Bhupathiraju 2013	42,135	3,607	17.1%	0.88 [0.78, 0.99]
ATBC - Simila 2013	21,955	4,379	6.2%	0.87 [0.71, 1.06]
Shanghai Men Health - Yu 2014	55,424	217	1.4%	0.96 [0.64, 1.45]
Shanghai Women Health - Yu 2014	67,211	148	0.8%	0.77 [0.45, 1.31]
MONICA Danish - Tognon 2014	1,849	161	2.4%	1.01 [0.74, 1.38]
British Regional Heart - Atkins 2014	3,328	307	1.2%	0.86 [0.55, 1.35]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	3.1%	0.91 [0.69, 1.20]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	6.2%	1.04 [0.86, 1.27]
CCHS - Kobylecki 2015	78,527	2,823	12.6%	0.87 [0.76, 1.00]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.3%	1.02 [0.41, 2.56]
China Kadoorie Biobank- Du 2016	451,665	2,551	7.6%	0.66 [0.55, 0.78]
PURE - Miller 2017	135,335	2,143	5.1%	0.91 [0.74, 1.13]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	1.9%	1.15 [0.81, 1.64]
EPIC NL and MORGEN - Scheffers 2019	34,560	2,135	9.6%	0.91 [0.78, 1.07]
Total (95% CI)	**1,170,021**	**23,856**	**100.0%**	**0.88 [0.84, 0.92]**

Heterogeneity: Chi² = 24.96, df = 22 (P = 0.30); I² = 12%

Test for overall effect: Z = 5.11 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Adventis Health Study - Fraser -1992	26,473	134	0.8%	1.07 [0.58, 1.97]
WHS - Liu 2000	39,127	126	0.8%	0.66 [0.36, 1.21]
PRIME - Dauchet 2010 - current smokers	2,297	148	1.3%	0.61 [0.38, 0.98]
PRIME - Dauchet 2010 - former smokers	3,353	140	1.9%	0.83 [0.56, 1.22]
PRIME - Dauchet 2010 - never smokers	2,410	79	0.8%	1.34 [0.73, 2.45]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	5.6%	0.93 [0.75, 1.16]
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	1.9%	0.80 [0.54, 1.19]
EPIC Italy - Bendinelli 2011	29,689	144	1.0%	1.25 [0.73, 2.12]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	11.4%	0.87 [0.76, 1.00]
HPFS - Bhupathiraju 2013	42,135	3,607	14.0%	0.88 [0.78, 0.99]
ATBC - Simila 2013	21,955	4,379	6.5%	0.87 [0.71, 1.06]
Shanghai Men Health - Yu 2014	55,424	217	1.7%	0.96 [0.64, 1.45]
Shanghai Women Health - Yu 2014	67,211	148	1.0%	0.77 [0.45, 1.31]
MONICA Danish - Tognon 2014	1,849	161	2.8%	1.01 [0.74, 1.38]
British Regional Heart - Atkins 2014	3,328	307	1.4%	0.86 [0.55, 1.35]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	3.6%	0.91 [0.69, 1.20]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	6.5%	1.04 [0.86, 1.27]
CCHS - Kobylecki 2015	78,527	2,823	11.4%	0.87 [0.76, 1.00]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.4%	1.02 [0.41, 2.56]
China Kadoorie Biobank- Du 2016	451,665	2,551	7.8%	0.66 [0.55, 0.78]
PURE - Miller 2017	135,335	2,143	5.6%	0.91 [0.74, 1.13]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	2.3%	1.15 [0.81, 1.64]
EPIC NL and MORGEN - Scheffers 2019	34,560	2,135	9.4%	0.91 [0.78, 1.07]
Total (95% CI) [Random Effects]	**1,170,021**	**23,856**	**100.0%**	**0.88 [0.84, 0.93]**

Heterogeneity: Tau² = 0.00; Chi² = 24.96, df = 22 (P = 0.30); I² = 12%

Test for overall effect: Z = 4.43 (P < 0.00001)

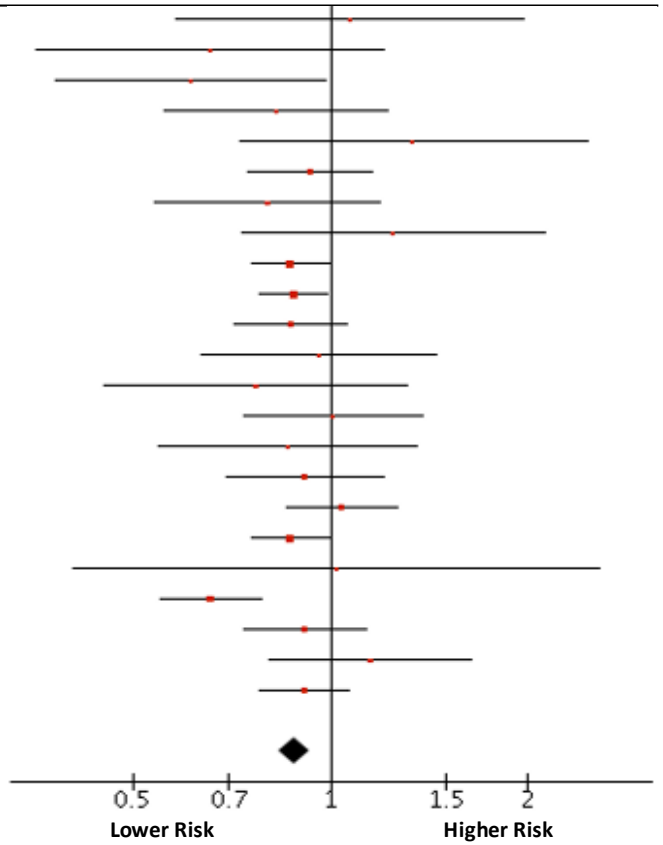


Figure S57. Relation between fruit intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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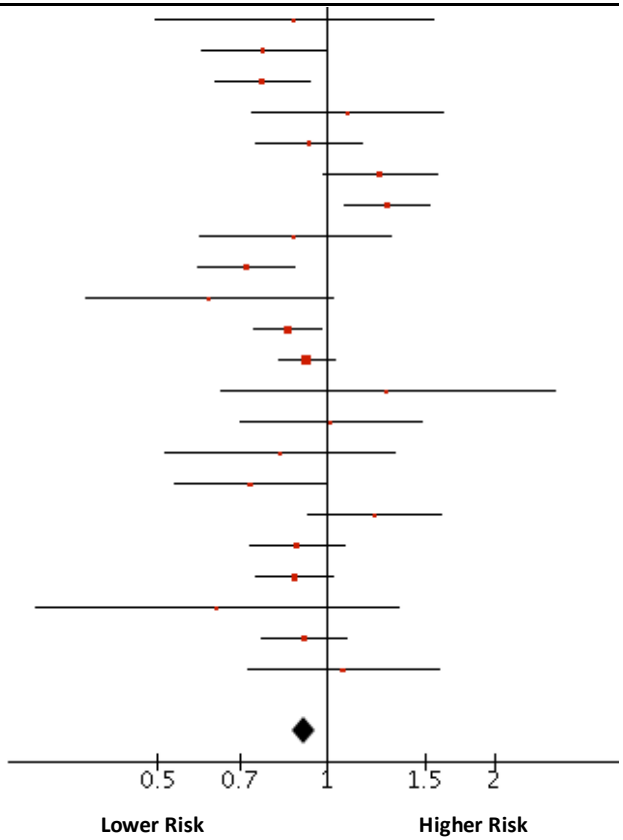
VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	126	0.7%	0.88 [0.50, 1.55]
Physicians Health Study - Liu 2001	15,520	1,148	3.6%	0.77 [0.60, 0.99]
ATBC - Hirvonen 2001	25,373	1,122	6.1%	0.77 [0.63, 0.94]
Danish Diet Cancer Health - Hansen 2010 - F	25,065	255	1.5%	1.09 [0.74, 1.62]
Danish Diet Cancer Health - Hansen 2010 - M	28,318	820	5.0%	0.93 [0.75, 1.16]
PRIME - Dauchet 2010 - never smokers	2,410	79	4.2%	1.25 [0.98, 1.58]
PRIME - Dauchet 2010 - former smokers	3,353	140	7.5%	1.28 [1.08, 1.53]
MORGEN - Oude Griep 2010	20,069	245	1.5%	0.88 [0.59, 1.30]
PRIME - Dauchet 2010 - current smokers	2,297	148	6.1%	0.72 [0.59, 0.87]
EPIC Italy - Bendinelli 2011	29,689	144	0.9%	0.62 [0.37, 1.03]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	12.4%	0.85 [0.74, 0.98]
HPFS - Bhupathiraju 2013	42,135	3,607	16.9%	0.92 [0.82, 1.04]
British Regional Heart - Atkins 2014	3,328	307	0.5%	1.28 [0.65, 2.55]
Shanghai Men Health - Yu 2014	55,424	217	1.7%	1.02 [0.70, 1.48]
Shanghai Women Health - Yu 2014	67,211	148	1.1%	0.83 [0.52, 1.32]
MONICA Danish - Tognon 2014	1,849	161	2.4%	0.73 [0.54, 1.00]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	3.1%	1.22 [0.93, 1.61]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	6.1%	0.89 [0.73, 1.08]
CCHS - Kobylecki 2015	78,527	2,823	9.5%	0.88 [0.75, 1.03]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.4%	0.64 [0.30, 1.34]
PURE - Miller 2017	135,335	2,143	7.5%	0.91 [0.77, 1.09]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	1.5%	1.07 [0.72, 1.59]
Total (95% CI)	**696,330**	**17,172**	**100.0%**	**0.92 [0.87, 0.96]**

Heterogeneity: Chi² = 44.99, df = 21 (P = 0.002); I² = 53%

Test for overall effect: Z = 3.59 (P = 0.0003)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Liu 2000	39,127	126	1.6%	0.88 [0.50, 1.55]
Physicians Health Study - Liu 2001	15,520	1,148	4.9%	0.77 [0.60, 0.99]
ATBC - Hirvonen 2001	25,373	1,122	6.3%	0.77 [0.63, 0.94]
Danish Diet Cancer Health - Hansen 2010 - F	25,065	255	2.9%	1.09 [0.74, 1.62]
Danish Diet Cancer Health - Hansen 2010 - M	28,318	820	5.8%	0.93 [0.75, 1.16]
PRIME - Dauchet 2010 - never smokers	2,410	79	5.4%	1.25 [0.98, 1.58]
PRIME - Dauchet 2010 - former smokers	3,353	140	6.8%	1.28 [1.08, 1.53]
MORGEN - Oude Griep 2010	20,069	245	2.9%	0.88 [0.59, 1.30]
PRIME - Dauchet 2010 - current smokers	2,297	148	6.3%	0.72 [0.59, 0.87]
EPIC Italy - Bendinelli 2011	29,689	144	1.9%	0.62 [0.37, 1.03]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	7.8%	0.85 [0.74, 0.98]
HPFS - Bhupathiraju 2013	42,135	3,607	8.3%	0.92 [0.82, 1.04]
British Regional Heart - Atkins 2014	3,328	307	1.2%	1.28 [0.65, 2.55]
Shanghai Men Health - Yu 2014	55,424	217	3.1%	1.02 [0.70, 1.48]
Shanghai Women Health - Yu 2014	67,211	148	2.2%	0.83 [0.52, 1.32]
MONICA Danish - Tognon 2014	1,849	161	3.9%	0.73 [0.54, 1.00]
CCHS - Kobylecki 2015	78,527	2,823	7.3%	0.88 [0.75, 1.03]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	4.6%	1.22 [0.93, 1.61]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	6.3%	0.89 [0.73, 1.08]
PREDIMED- Buil-Cosiales 2016	7,216	118	1.0%	0.64 [0.30, 1.34]
PURE - Miller 2017	135,335	2,143	6.8%	0.91 [0.77, 1.09]
Japan Public Health Centre - Yoshizaki 2019	16,498	839	2.9%	1.07 [0.72, 1.59]
Total (95% CI) [Random Effects]	**696,330**	**17,172**	**100.0%**	**0.92 [0.85, 0.99]**

Heterogeneity: Tau² = 0.02; Chi² = 44.99, df = 21 (P = 0.002); I² = 53%

Test for overall effect: Z = 2.14 (P = 0.03)

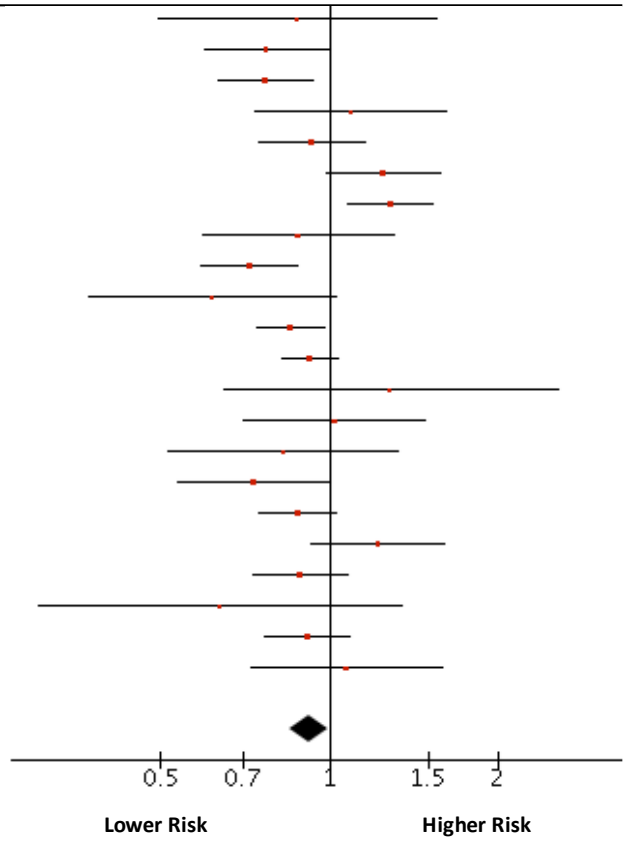


Figure S58. Relation between intake of vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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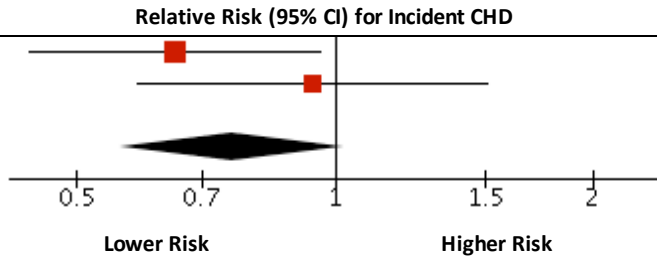
BANANAS AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Women Health - Yu 2014 - bananas	67,211	148	59.0%	0.94 [0.59, 1.51]
Shanghai Men Health - Yu 2014 - bananas	55,424	217	41.0%	0.65 [0.44, 0.96]
Total (95% CI)	**122,635**	**365**	**100.0%**	**0.76 [0.56, 1.02]**

Heterogeneity: Chi² = 1.40, df = 1 (P = 0.24); I² = 29%

Test for overall effect: Z = 1.81 (P = 0.07)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Women Health - Yu 2014 - bananas	67,211	148	43.6%	0.94 [0.59, 1.51]
Shanghai Men Health - Yu 2014 - bananas	55,424	217	56.4%	0.65 [0.44, 0.96]
Total (95% CI) [Random Effects]	**122,635**	**365**	**100.0%**	**0.76 [0.53, 1.10]**

Heterogeneity: Tau² = 0.02; Chi² = 1.40, df = 1 (P = 0.24); I² = 29%

Test for overall effect: Z = 1.47 (P = 0.14)

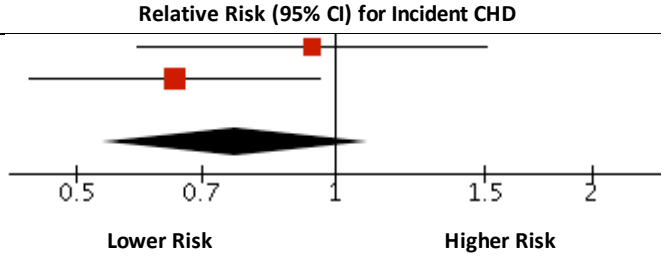


Figure S59. Relation between intake of bananas and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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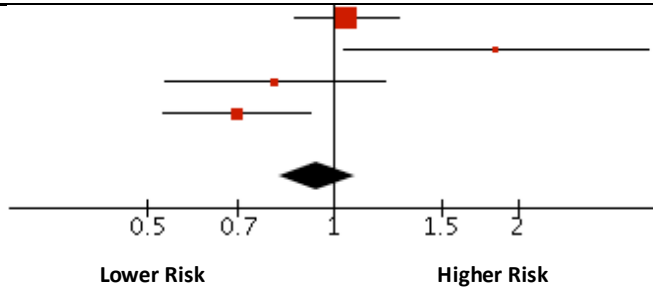
BERRIES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2001	25,373	1,122	53.9%	1.05 [0.86, 1.28]
WHS - Sesso 2007	38,176	289	6.4%	1.84 [1.04, 3.25]
MORGEN - Oude Griep 2011 (b)	20,069	233	12.2%	0.80 [0.53, 1.21]
REGARDS - Goetz 2016 (a)	16,678	589	27.5%	0.70 [0.53, 0.92]
Total (95% CI)	**100,296**	**2,233**	**100.0%**	**0.94 [0.82, 1.09]**

Heterogeneity: Chi² = 11.72, df = 3 (P = 0.008); I² = 74%

Test for overall effect: Z = 0.81 (P = 0.42)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2001	25,373	1,122	31.7%	1.05 [0.86, 1.28]
WHS - Sesso 2007	38,176	289	17.1%	1.84 [1.04, 3.25]
MORGEN - Oude Griep 2011 (b)	20,069	233	22.7%	0.80 [0.53, 1.21]
REGARDS - Goetz 2016 (a)	16,678	589	28.5%	0.70 [0.53, 0.92]
Total (95% CI) [Random Effects]	**100,296**	**2,233**	**100.0%**	**0.97 [0.70, 1.34]**

Heterogeneity: Tau² = 0.08; Chi² = 11.72, df = 3 (P = 0.008); I² = 74%

Test for overall effect: Z = 0.20 (P = 0.84)

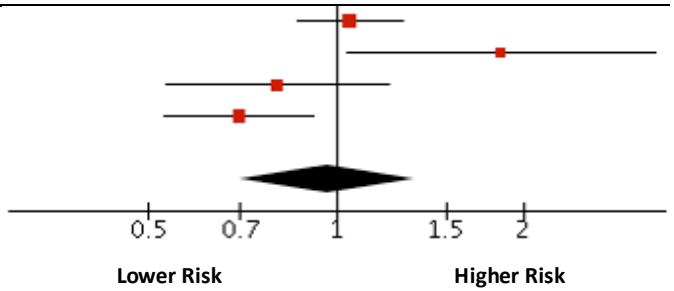


Figure S60. Relation between intake of berries and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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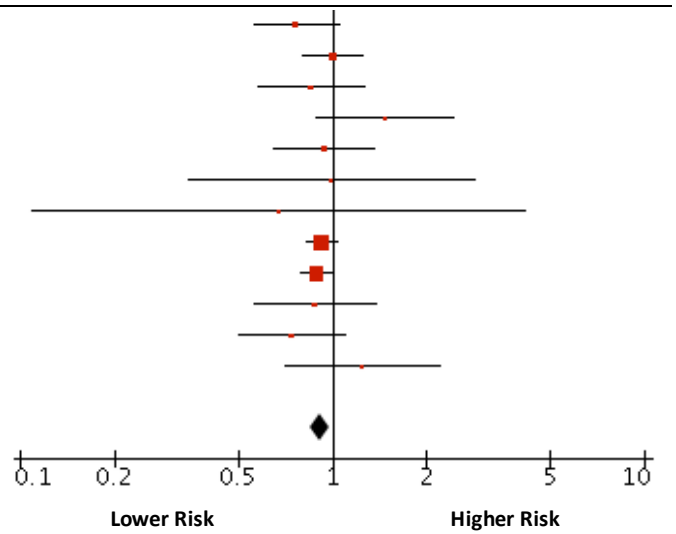
CITRUS FRUIT AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
PRIME - Dauchet 2004	8,087	133	4.9%	0.76 [0.56, 1.04]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	10.3%	1.00 [0.81, 1.24]
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	3.1%	0.85 [0.58, 1.26]
EPIC Italy - Bendinelli 2011	29,689	144	1.8%	1.48 [0.89, 2.46]
MORGEN - Oude Griep 2011 (b)	20,069	233	3.4%	0.94 [0.65, 1.37]
Jidni Medical School - Yamada 2011 - M	4,147	53	0.4%	0.99 [0.34, 2.85]
Jidni Medical School - Yamada 2011 - F	6,476	23	0.1%	0.67 [0.11, 4.15]
HPFS - Bhupathiraju 2013	42,135	3,607	34.5%	0.92 [0.82, 1.04]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	34.5%	0.89 [0.79, 1.00]
Shanghai Women Health - Yu 2014	67,211	148	2.3%	0.88 [0.56, 1.38]
Shanghai Men Health - Yu 2014	55,424	217	3.1%	0.74 [0.50, 1.10]
PREDIMED- Buil-Cosiales 2016	7,216	118	1.5%	1.25 [0.71, 2.20]
Total (95% CI)	**364,978**	**8,333**	**100.0%**	**0.91 [0.85, 0.98]**

Heterogeneity: Chi² = 8.17, df = 11 (P = 0.70); I² = 0%

Test for overall effect: Z = 2.60 (P = 0.009)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
PRIME - Dauchet 2004	8,087	133	4.9%	0.76 [0.56, 1.04]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	10.3%	1.00 [0.81, 1.24]
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	3.1%	0.85 [0.58, 1.26]
MORGEN - Oude Griep 2011 (b)	20,069	233	3.4%	0.94 [0.65, 1.37]
Jidni Medical School - Yamada 2011 - M	4,147	53	0.4%	0.99 [0.34, 2.85]
Jidni Medical School - Yamada 2011 - F	6,476	23	0.1%	0.67 [0.11, 4.15]
EPIC Italy - Bendinelli 2011	29,689	144	1.8%	1.48 [0.89, 2.46]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	34.5%	0.89 [0.79, 1.00]
HPFS - Bhupathiraju 2013	42,135	3,607	34.5%	0.92 [0.82, 1.04]
Shanghai Women Health - Yu 2014	67,211	148	2.3%	0.88 [0.56, 1.38]
Shanghai Men Health - Yu 2014	55,424	217	3.1%	0.74 [0.50, 1.10]
PREDIMED- Buil-Cosiales 2016	7,216	118	1.5%	1.25 [0.71, 2.20]
Total (95% CI) [Random Effects]	**364,978**	**8,333**	**100.0%**	**0.91 [0.85, 0.98]**

Heterogeneity: Chi² = 8.17, df = 11 (P = 0.70); I² = 0%

Test for overall effect: Z = 2.60 (P = 0.009)

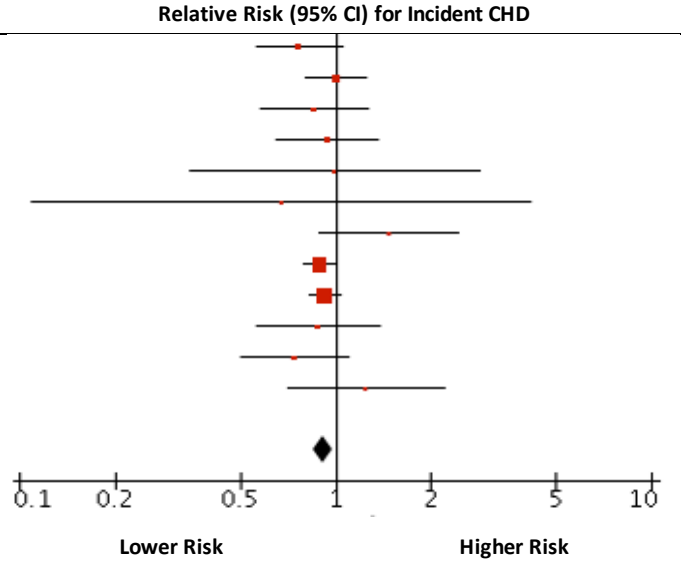


Figure S61. Relation between citrus fruit intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

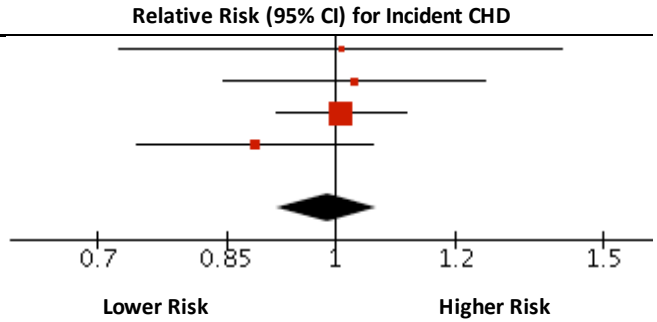
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FRUIT JUICE AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	5.3%	1.01 [0.72, 1.41]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	15.2%	1.03 [0.85, 1.25]
ATBC - Simila 2013	21,955	4,379	60.8%	1.01 [0.92, 1.11]
EPIC NL and MORGEN - Scheffers 2019	34,560	2,135	18.8%	0.89 [0.74, 1.06]
Total (95% CI)	**109,898**	**7,589**	**100.0%**	**0.99 [0.92, 1.07]**

Heterogeneity: Chi^2 = 1.83, df = 3 (P = 0.61); I^2 = 0%
Test for overall effect: Z = 0.29 (P = 0.77)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	5.3%	1.01 [0.72, 1.41]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	15.2%	1.03 [0.85, 1.25]
ATBC - Simila 2013	21,955	4,379	60.8%	1.01 [0.92, 1.11]
EPIC NL and MORGEN - Scheffers 2019	34,560	2,135	18.8%	0.89 [0.74, 1.06]
Total (95% CI) [Random Effects]	**109,898**	**7,589**	**100.0%**	**0.99 [0.92, 1.07]**

Heterogeneity: Tau^2 = 0.00; Chi^2 = 1.83, df = 3 (P = 0.61); I^2 = 0%
Test for overall effect: Z = 0.29 (P = 0.77)

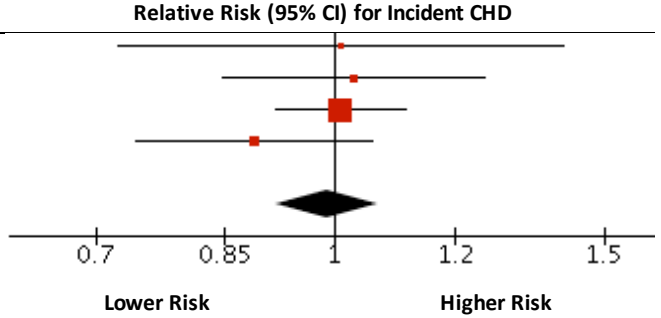


Figure S62. Relation between intake of fruit juice and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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GRAPES AND CORONARY HEART DISEASE INCIDENCE

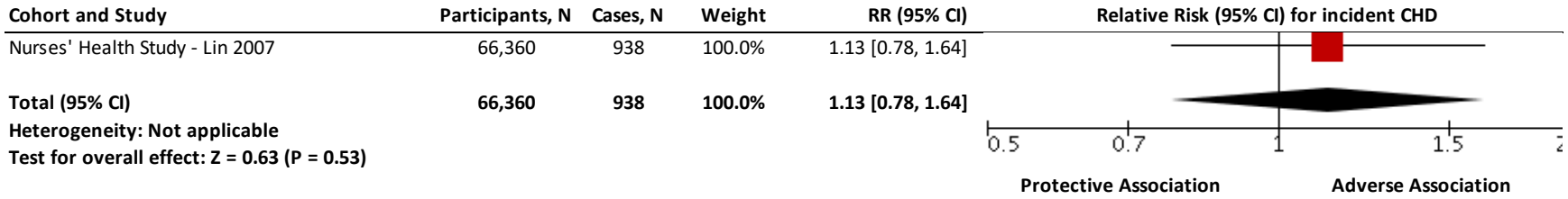


Figure S63. Relation between intake of grapes and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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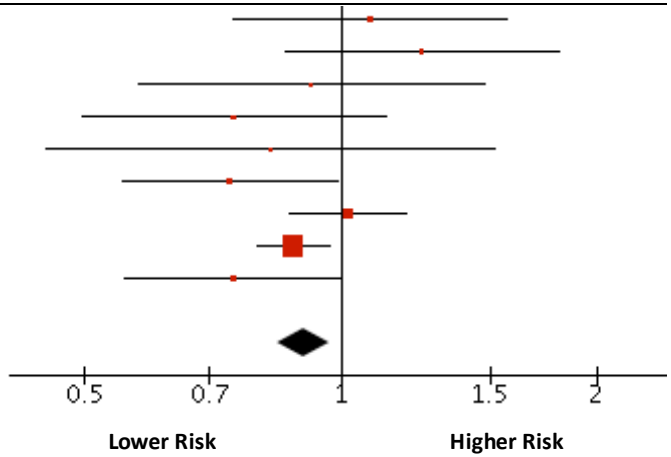
POMMES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Lin 2007	66,360	938	3.7%	1.08 [0.75, 1.57]
MORGEN - Oude Griep 2011 (b)	20,069	245	3.7%	1.25 [0.86, 1.81]
Shanghai Women Health - Yu 2014	67,211	148	2.3%	0.92 [0.58, 1.48]
Shanghai Men Health - Yu 2014	55,424	217	3.0%	0.75 [0.50, 1.13]
PREDIMED- Buil-Cosiales 2016	7,216	118	1.4%	0.83 [0.45, 1.52]
REGARDS - Goetz 2016 (a)	16,678	589	5.9%	0.74 [0.55, 0.99]
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	20.8%	1.02 [0.87, 1.19]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	53.3%	0.88 [0.80, 0.97]
NutriNet-Sante - Adriouch 2018	84,158	309	5.9%	0.75 [0.56, 1.00]
Total (95% CI)	**371,684**	**4,886**	**100.0%**	**0.90 [0.84, 0.97]**

Heterogeneity: Chi² = 10.63, df = 8 (P = 0.22); I² = 25%

Test for overall effect: Z = 2.80 (P = 0.005)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Lin 2007	66,360	938	6.4%	1.08 [0.75, 1.57]
MORGEN - Oude Griep 2011 (b)	20,069	245	6.4%	1.25 [0.86, 1.81]
Shanghai Women Health - Yu 2014	67,211	148	4.2%	0.92 [0.58, 1.48]
Shanghai Men Health - Yu 2014	55,424	217	5.3%	0.75 [0.50, 1.13]
PREDIMED- Buil-Cosiales 2016	7,216	118	2.6%	0.83 [0.45, 1.52]
REGARDS - Goetz 2016 (a)	16,678	589	9.5%	0.74 [0.55, 0.99]
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	22.5%	1.02 [0.87, 1.19]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	33.7%	0.88 [0.80, 0.97]
NutriNet-Sante - Adriouch 2018	84,158	309	9.5%	0.75 [0.56, 1.00]
Total (95% CI) [Random Effects]	**371,684**	**4,886**	**100.0%**	**0.90 [0.82, 1.00]**

Heterogeneity: Tau² = 0.01; Chi² = 10.63, df = 8 (P = 0.22); I² = 25%

Test for overall effect: Z = 1.95 (P = 0.05)

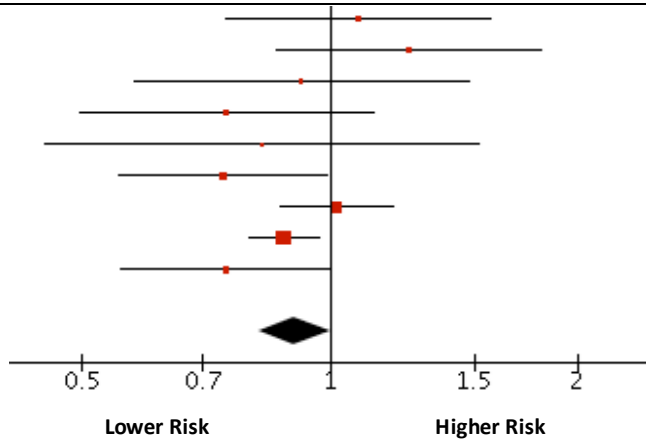


Figure S64. Relation between intake of pommes fruit and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

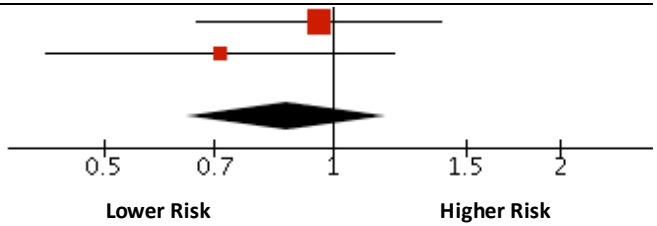
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WATERMELON AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Men Health - Yu 2014 - Watermelon	55,424	217	66.9%	0.96 [0.66, 1.39]
Shanghai Women Health - Yu 2014 - Watermelon	67,211	148	33.1%	0.71 [0.42, 1.21]
Total (95% CI)	**122,635**	**365**	**100.0%**	**0.87 [0.64, 1.18]**

Heterogeneity: Chi² = 0.83, df = 1 (P = 0.36); I² = 0%
Test for overall effect: Z = 0.90 (P = 0.37)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Men Health - Yu 2014 - Watermelon	55,424	217	66.9%	0.96 [0.66, 1.39]
Shanghai Women Health - Yu 2014 - Watermelon	67,211	148	33.1%	0.71 [0.42, 1.21]
Total (95% CI) [Random Effects]	**122,635**	**365**	**100.0%**	**0.87 [0.64, 1.18]**

Heterogeneity: Tau² = 0.00; Chi² = 0.83, df = 1 (P = 0.36); I² = 0%
Test for overall effect: Z = 0.90 (P = 0.37)

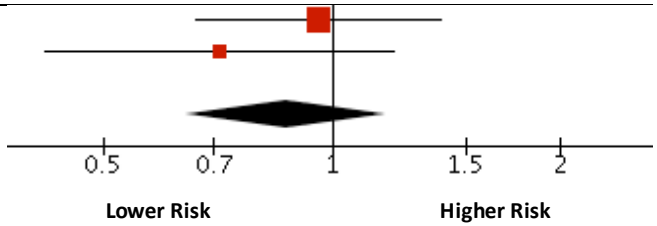


Figure S65. Relation between watermelon intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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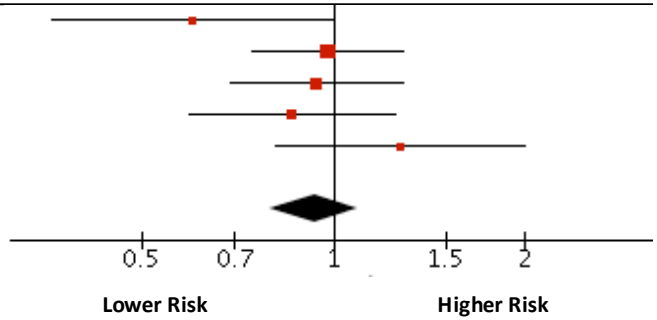
ALLIUM VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Caerphilly Prospective Study - Hertog 1997	1,900	186	9.8%	0.60 [0.36, 1.00]
Nurses' Health Study - Lin 2007	66,360	938	33.7%	0.98 [0.74, 1.29]
MORGEN - Oude Griep 2011 (b)	20,069	245	25.8%	0.94 [0.69, 1.29]
Shanghai Men Health - Yu 2014	55,424	217	18.3%	0.86 [0.59, 1.25]
Shanghai Women Health - Yu 2014	67,211	148	12.5%	1.27 [0.81, 2.00]
Total (95% CI)	**210,964**	**1,734**	**100.0%**	**0.93 [0.80, 1.09]**

Heterogeneity: Chi² = 4.99, df = 4 (P = 0.29); I² = 20%

Test for overall effect: Z = 0.86 (P = 0.39)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Caerphilly Prospective Study - Hertog 1997	1,900	186	11.3%	0.60 [0.36, 1.00]
Nurses' Health Study - Lin 2007	66,360	938	30.5%	0.98 [0.74, 1.29]
MORGEN - Oude Griep 2011 (b)	20,069	245	25.1%	0.94 [0.69, 1.29]
Shanghai Women Health - Yu 2014	67,211	148	14.0%	1.27 [0.81, 2.00]
Shanghai Men Health - Yu 2014	55,424	217	19.2%	0.86 [0.59, 1.25]
Total (95% CI) [Random Effects]	**210,964**	**1,734**	**100.0%**	**0.93 [0.77, 1.11]**

Heterogeneity: Tau² = 0.01; Chi² = 4.99, df = 4 (P = 0.29); I² = 20%

Test for overall effect: Z = 0.80 (P = 0.42)

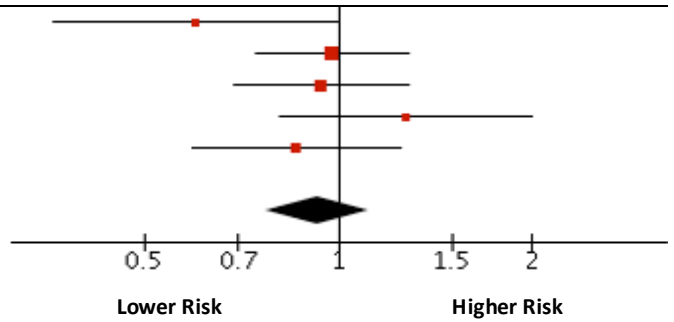


Figure S66. Relation between intake of allium vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

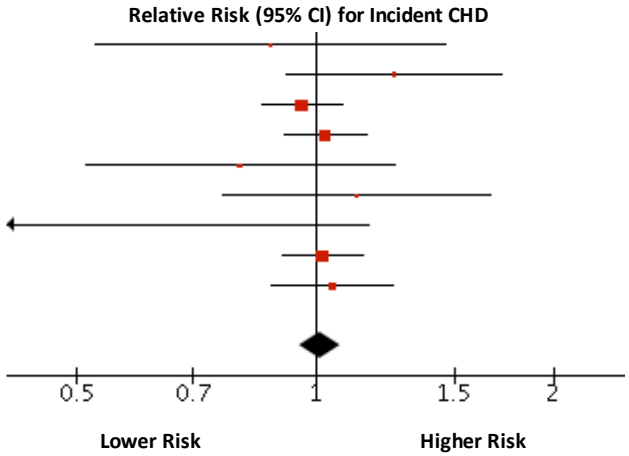
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CRUCIFEROUS VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
EPIC Italy - Bendinelli 2011	29,689	144	1.4%	0.88 [0.53, 1.46]
MORGEN - Oude Griep 2011(b)-green cabbage ve	20,069	245	3.7%	1.26 [0.92, 1.72]
HPFS - Bhupathiraju 2013	42,135	3,607	26.3%	0.96 [0.85, 1.08]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	26.3%	1.03 [0.92, 1.16]
Shanghai Women Health - Yu 2014	67,211	148	1.8%	0.80 [0.51, 1.26]
Shanghai Men Health - Yu 2014	55,424	217	2.4%	1.13 [0.76, 1.67]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.2%	0.32 [0.09, 1.17]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	26.3%	1.02 [0.91, 1.15]
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	11.7%	1.05 [0.88, 1.25]
Total (95% CI)	**347,453**	**9,383**	**100.0%**	**1.01 [0.95, 1.07]**

Heterogeneity: Chi² = 7.55, df = 8 (P = 0.48); I² = 0%
Test for overall effect: Z = 0.38 (P = 0.71)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
MORGEN - Oude Griep 2011(b)-green cabbage ve	20,069	245	3.7%	1.26 [0.92, 1.72]
EPIC Italy - Bendinelli 2011	29,689	144	1.4%	0.88 [0.53, 1.46]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	26.3%	1.03 [0.92, 1.16]
HPFS - Bhupathiraju 2013	42,135	3,607	26.3%	0.96 [0.85, 1.08]
Shanghai Men Health - Yu 2014	55,424	217	2.4%	1.13 [0.76, 1.67]
Shanghai Women Health - Yu 2014	67,211	148	1.8%	0.80 [0.51, 1.26]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.2%	0.32 [0.09, 1.17]
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	11.7%	1.05 [0.88, 1.25]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	26.3%	1.02 [0.91, 1.15]
Total (95% CI) [Random Effects]	**347,453**	**9,383**	**100.0%**	**1.01 [0.95, 1.07]**

Heterogeneity: Tau² = 0.00; Chi² = 7.55, df = 8 (P = 0.48); I² = 0%
Test for overall effect: Z = 0.38 (P = 0.71)

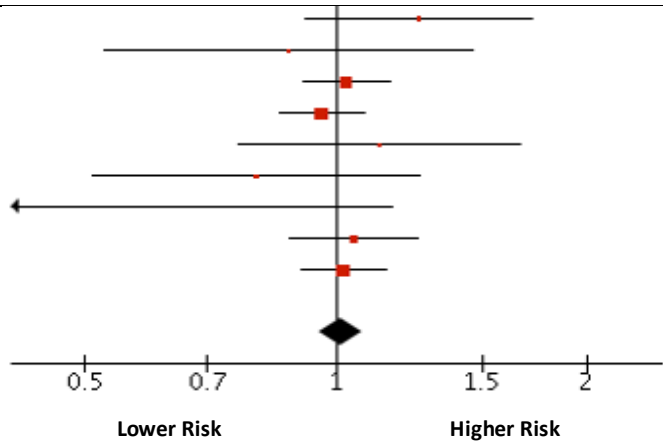


Figure S67. Relation between intake of cruciferous vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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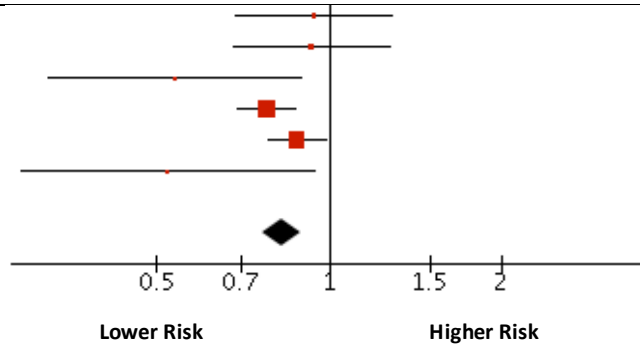
GREEN LEAFY VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
MORGEN - Oude Griep 2011(b) -dark green leafy	20,069	245	5.9%	0.94 [0.69, 1.29]
MORGEN - Oude Griep 2011(b)- lettuce	-	-	5.9%	0.93 [0.68, 1.28]
EPIC Italy - Bendinelli 2011	29,689	144	2.2%	0.54 [0.32, 0.90]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	42.1%	0.78 [0.69, 0.88]
HPFS - Bhupathiraju 2013	42,135	3,607	42.1%	0.88 [0.78, 0.99]
PREDIMED- Buil-Cosiales 2016	7,216	118	1.7%	0.52 [0.29, 0.94]
Total (95% CI)	**170,250**	**6,696**	**100.0%**	**0.82 [0.76, 0.89]**

Heterogeneity: Chi² = 8.30, df = 5 (P = 0.14); I² = 40%

Test for overall effect: Z = 4.95 (P < 0.00001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
MORGEN - Oude Griep 2011(b) - lettuce	20,069	245	11.6%	0.93 [0.68, 1.28]
MORGEN - Oude Griep 2011(b) - dark green leafy	-	-	11.6%	0.94 [0.69, 1.29]
EPIC Italy - Bendinelli 2011	29,689	144	5.1%	0.54 [0.32, 0.90]
HPFS - Bhupathiraju 2013	42,135	3,607	33.9%	0.88 [0.78, 0.99]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	33.9%	0.78 [0.69, 0.88]
PREDIMED- Buil-Cosiales 2016	7,216	118	4.0%	0.52 [0.29, 0.94]
Total (95% CI) [Random Effects]	**170,250**	**6,696**	**100.0%**	**0.82 [0.72, 0.92]**

Heterogeneity: Tau² = 0.01; Chi² = 8.30, df = 5 (P = 0.14); I² = 40%

Test for overall effect: Z = 3.23 (P = 0.001)

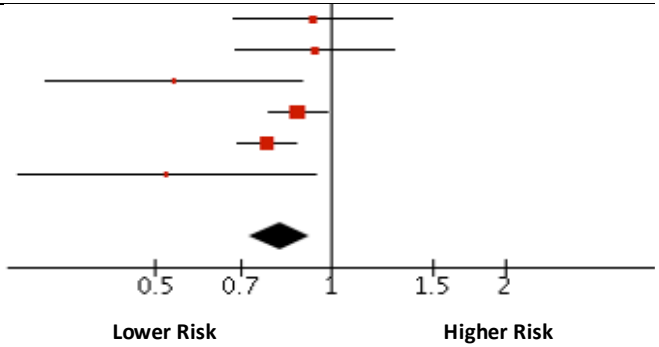


Figure S68. Relation between intake of green leafy vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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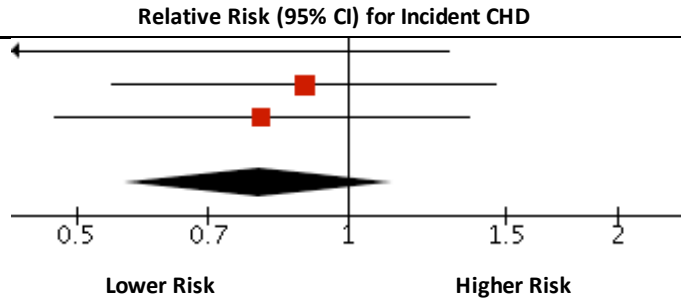
TOMATOES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	201	8.3%	0.39 [0.12, 1.29]
Nurses' Health Study - Lin 2007	66,360	938	49.4%	0.90 [0.55, 1.46]
EPIC Italy - Bendinelli 2011	29,689	144	42.3%	0.80 [0.47, 1.36]
Total (95% CI)	**134,494**	**1,283**	**100.0%**	**0.80 [0.57, 1.13]**

Heterogeneity: Chi² = 1.59, df = 2 (P = 0.45); I² = 0%

Test for overall effect: Z = 1.28 (P = 0.20)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	201	8.3%	0.39 [0.12, 1.29]
Nurses' Health Study - Lin 2007	66,360	938	49.4%	0.90 [0.55, 1.46]
EPIC Italy - Bendinelli 2011	29,689	144	42.3%	0.80 [0.47, 1.36]
Total (95% CI) [Random Effects]	**134,494**	**1,283**	**100.0%**	**0.80 [0.57, 1.13]**

Heterogeneity: Tau² = 0.00; Chi² = 1.59, df = 2 (P = 0.45); I² = 0%

Test for overall effect: Z = 1.28 (P = 0.20)

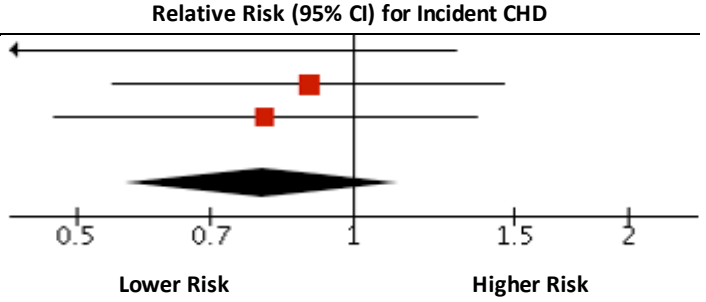
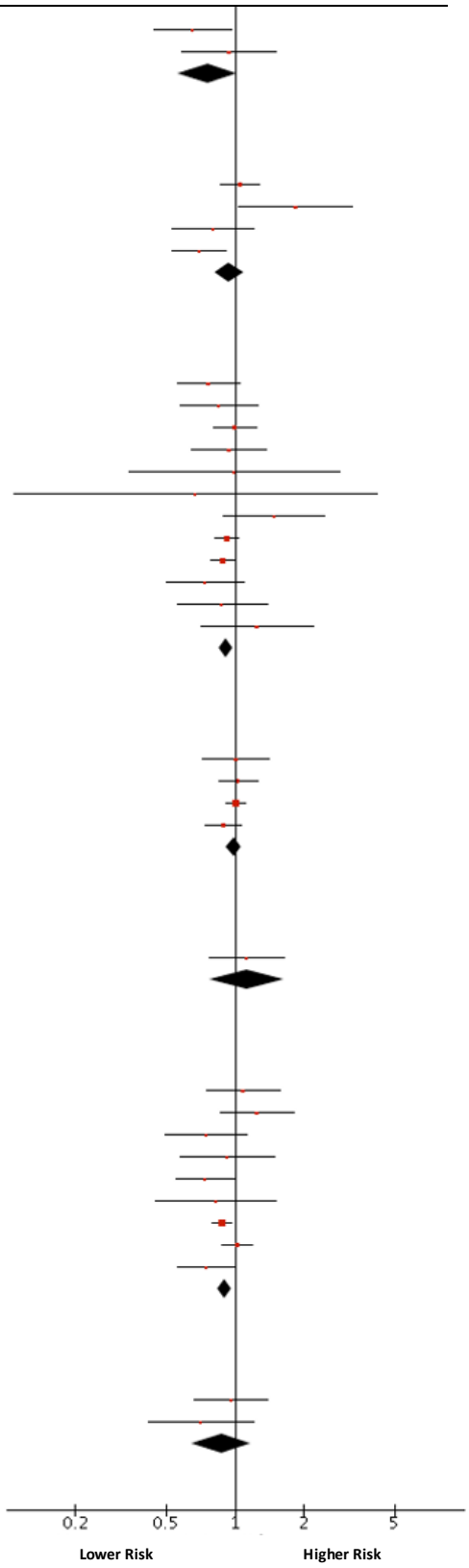


Figure S69. Relation between intake of tomatoes and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Bananas				
Shanghai Men Health - Yu 2014 - bananas	55,424	217	1.0%	0.65 [0.44, 0.96]
Shanghai Women Health - Yu 2014 - bananas	67,211	148	0.7%	0.94 [0.59, 1.51]
Subtotal (95% CI)	122,635	365	1.7%	0.76 [0.56, 1.02]
Heterogeneity: Chi² = 1.40, df = 1 (P = 0.24); I² = 29%				
Test for overall effect: Z = 1.81 (P = 0.07)				
Berries				
ATBC - Hirvonen 2001	25,373	1,122	4.0%	1.05 [0.86, 1.28]
WHS - Sesso 2007	38,176	289	0.5%	1.84 [1.04, 3.25]
MORGEN - Oude Griep 2011 (b)	20,069	233	0.9%	0.80 [0.53, 1.21]
REGARDS - Goetz 2016 (a)	16,678	589	2.0%	0.70 [0.53, 0.92]
Subtotal (95% CI)	100,296	2,233	7.4%	0.94 [0.82, 1.09]
Heterogeneity: Chi² = 11.72, df = 3 (P = 0.008); I² = 74%				
Test for overall effect: Z = 0.81 (P = 0.42)				
Citrus				
PRIME - Dauchet 2004	8,087	133	1.6%	0.76 [0.56, 1.04]
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	1.0%	0.85 [0.58, 1.26]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	3.3%	1.00 [0.81, 1.24]
MORGEN - Oude Griep 2011 (b)	20,069	233	1.1%	0.94 [0.65, 1.37]
Jidni Medical School - Yamada 2011 - M	4,147	53	0.1%	0.99 [0.34, 2.85]
Jidni Medical School - Yamada 2011 - F	6,476	23	0.0%	0.67 [0.11, 4.15]
EPIC Italy - Bendinelli 2011	29,689	144	0.6%	1.48 [0.89, 2.46]
HPFS - Bhupathiraju 2013	42,135	3,607	11.1%	0.92 [0.82, 1.04]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	11.1%	0.89 [0.79, 1.00]
Shanghai Men Health - Yu 2014	55,424	217	1.0%	0.74 [0.50, 1.10]
Shanghai Women Health - Yu 2014	67,211	148	0.8%	0.88 [0.56, 1.38]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.5%	1.25 [0.71, 2.20]
Subtotal (95% CI)	364,978	8,333	32.1%	0.91 [0.85, 0.98]
Heterogeneity: Chi² = 8.17, df = 11 (P = 0.70); I² = 0%				
Test for overall effect: Z = 2.60 (P = 0.009)				
Fruit Juice				
Danish Diet Cancer Health - Hansen 2010 - F	28,318	255	1.4%	1.01 [0.72, 1.41]
Danish Diet Cancer Health - Hansen 2010 - M	25,065	820	4.0%	1.03 [0.85, 1.25]
ATBC - Simila 2013	21,955	4,379	15.9%	1.01 [0.92, 1.11]
EPIC NL and MORGEN - Scheffers 2019	34,560	2,135	4.9%	0.89 [0.74, 1.06]
Subtotal (95% CI)	109,898	7,589	26.2%	0.99 [0.92, 1.07]
Heterogeneity: Chi² = 1.83, df = 3 (P = 0.61); I² = 0%				
Test for overall effect: Z = 0.29 (P = 0.77)				
Grapes				
Nurses' Health Study - Lin 2007	66,360	938	1.1%	1.13 [0.78, 1.64]
Subtotal (95% CI)	66,360	938	1.1%	1.13 [0.78, 1.64]
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.63 (P = 0.53)				
Pommes				
Nurses' Health Study - Lin 2007	66,360	938	1.1%	1.08 [0.75, 1.57]
MORGEN - Oude Griep 2011 (b)	20,069	245	1.1%	1.25 [0.86, 1.81]
Shanghai Men Health - Yu 2014	55,424	217	0.9%	0.75 [0.50, 1.13]
Shanghai Women Health - Yu 2014	67,211	148	0.7%	0.92 [0.58, 1.48]
REGARDS - Goetz 2016 (a)	16,678	589	1.8%	0.74 [0.55, 0.99]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.4%	0.83 [0.45, 1.52]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	15.9%	0.88 [0.80, 0.97]
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	6.2%	1.02 [0.87, 1.19]
NutriNet-Sante - Adriouch 2018	84,158	309	1.8%	0.75 [0.56, 1.00]
Subtotal (95% CI)	371,684	4,886	29.9%	0.90 [0.84, 0.97]
Heterogeneity: Chi² = 10.63, df = 8 (P = 0.22); I² = 25%				
Test for overall effect: Z = 2.80 (P = 0.005)				
Watermelon				
Shanghai Men Health - Yu 2014 - Watermelon	55,424	217	1.1%	0.96 [0.66, 1.39]
Shanghai Women Health - Yu 2014 - Watermel	67,211	148	0.5%	0.71 [0.42, 1.21]
Subtotal (95% CI)	122,635	365	1.6%	0.87 [0.64, 1.18]
Heterogeneity: Chi² = 0.83, df = 1 (P = 0.36); I² = 0%				
Test for overall effect: Z = 0.90 (P = 0.37)				
Test for subgroup differences: Chi² = 6.45, df = 6 (P = 0.37), I² = 7.0%				



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B. Random Effects

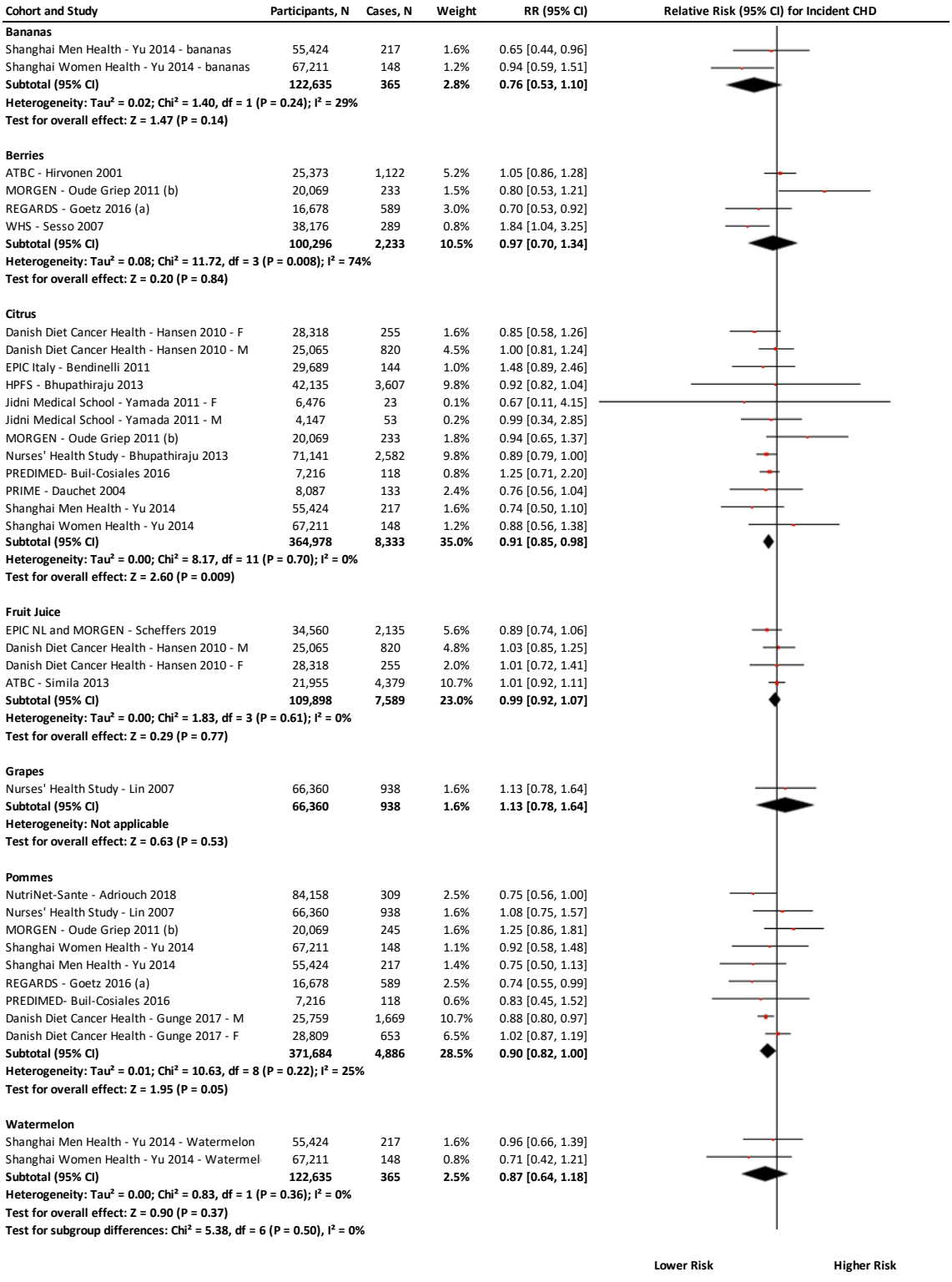
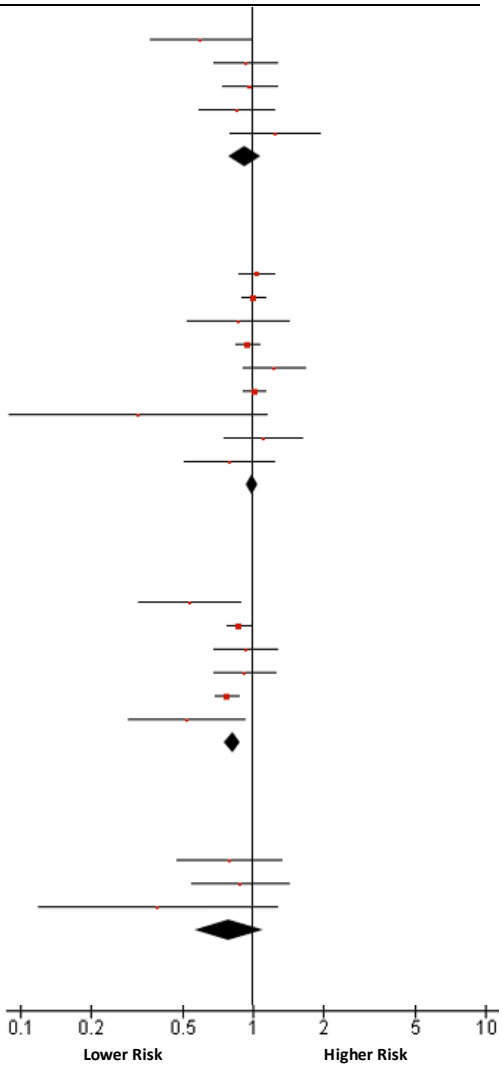


Figure S70. Relation between sources of fruit and CHD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
Caerphilly Prospective Study - Hertog 1997	1,900	186	0.8%	0.60 [0.36, 1.00]
MORGEN - Oude Griep 2011 (b)	20,069	245	2.1%	0.94 [0.69, 1.29]
Nurses' Health Study - Lin 2007	66,360	938	2.7%	0.98 [0.74, 1.29]
Shanghai Men Health - Yu 2014	55,424	217	1.5%	0.86 [0.59, 1.25]
Shanghai Women Health - Yu 2014	67,211	148	1.0%	1.27 [0.81, 2.00]
Subtotal (95% CI)	**210,964**	**1,734**	**8.0%**	**0.93 [0.80, 1.09]**
Heterogeneity: Chi² = 4.99, df = 4 (P = 0.29); I² = 20%				
Test for overall effect: Z = 0.86 (P = 0.39)				
Cruciferous				
Danish Diet Cancer Health - Gunge 2017 - F	28,809	653	6.5%	1.05 [0.88, 1.25]
Danish Diet Cancer Health - Gunge 2017 - M	25,759	1,669	14.6%	1.02 [0.91, 1.15]
EPIC Italy - Bendinelli 2011	29,689	144	0.8%	0.88 [0.53, 1.46]
HPFS - Bhupathiraju 2013	42,135	3,607	14.6%	0.96 [0.85, 1.08]
MORGEN - Oude Griep 2011(b)-green cabbage	20,069	245	2.1%	1.26 [0.92, 1.72]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	14.6%	1.03 [0.92, 1.16]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.1%	0.32 [0.09, 1.17]
Shanghai Men Health - Yu 2014	55,424	217	1.3%	1.13 [0.76, 1.67]
Shanghai Women Health - Yu 2014	67,211	148	1.0%	0.80 [0.51, 1.26]
Subtotal (95% CI)	**347,453**	**9,383**	**55.6%**	**1.01 [0.95, 1.07]**
Heterogeneity: Chi² = 7.55, df = 8 (P = 0.48); I² = 0%				
Test for overall effect: Z = 0.38 (P = 0.71)				
Green leafy				
EPIC Italy - Bendinelli 2011	29,689	144	0.8%	0.54 [0.32, 0.90]
HPFS - Bhupathiraju 2013	42,135	3,607	14.6%	0.88 [0.78, 0.99]
MORGEN - Oude Griep 2011(b) -dark green lea	20,069	245	2.1%	0.94 [0.69, 1.29]
MORGEN - Oude Griep 2011(b)- lettuce	-	-	2.1%	0.93 [0.68, 1.28]
Nurses' Health Study - Bhupathiraju 2013	71,141	2,582	14.6%	0.78 [0.69, 0.88]
PREDIMED- Buil-Cosiales 2016	7,216	118	0.6%	0.52 [0.29, 0.94]
Subtotal (95% CI)	**170,250**	**6,696**	**34.7%**	**0.82 [0.76, 0.89]**
Heterogeneity: Chi² = 8.30, df = 5 (P = 0.14); I² = 40%				
Test for overall effect: Z = 4.95 (P < 0.00001)				
Tomatoes				
EPIC Italy - Bendinelli 2011	29,689	144	0.7%	0.80 [0.47, 1.36]
Nurses' Health Study - Lin 2007	66,360	938	0.8%	0.90 [0.55, 1.46]
WHS - Sesso 2003 (a)	38,445	201	0.1%	0.39 [0.12, 1.29]
Subtotal (95% CI)	**134,494**	**1,283**	**1.7%**	**0.80 [0.57, 1.13]**
Heterogeneity: Chi² = 1.59, df = 2 (P = 0.45); I² = 0%				
Test for overall effect: Z = 1.28 (P = 0.20)				
Test for subgroup differences: Chi² = 17.73, df = 3 (P = 0.0005), I² = 83.1%				



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B. Random Effects

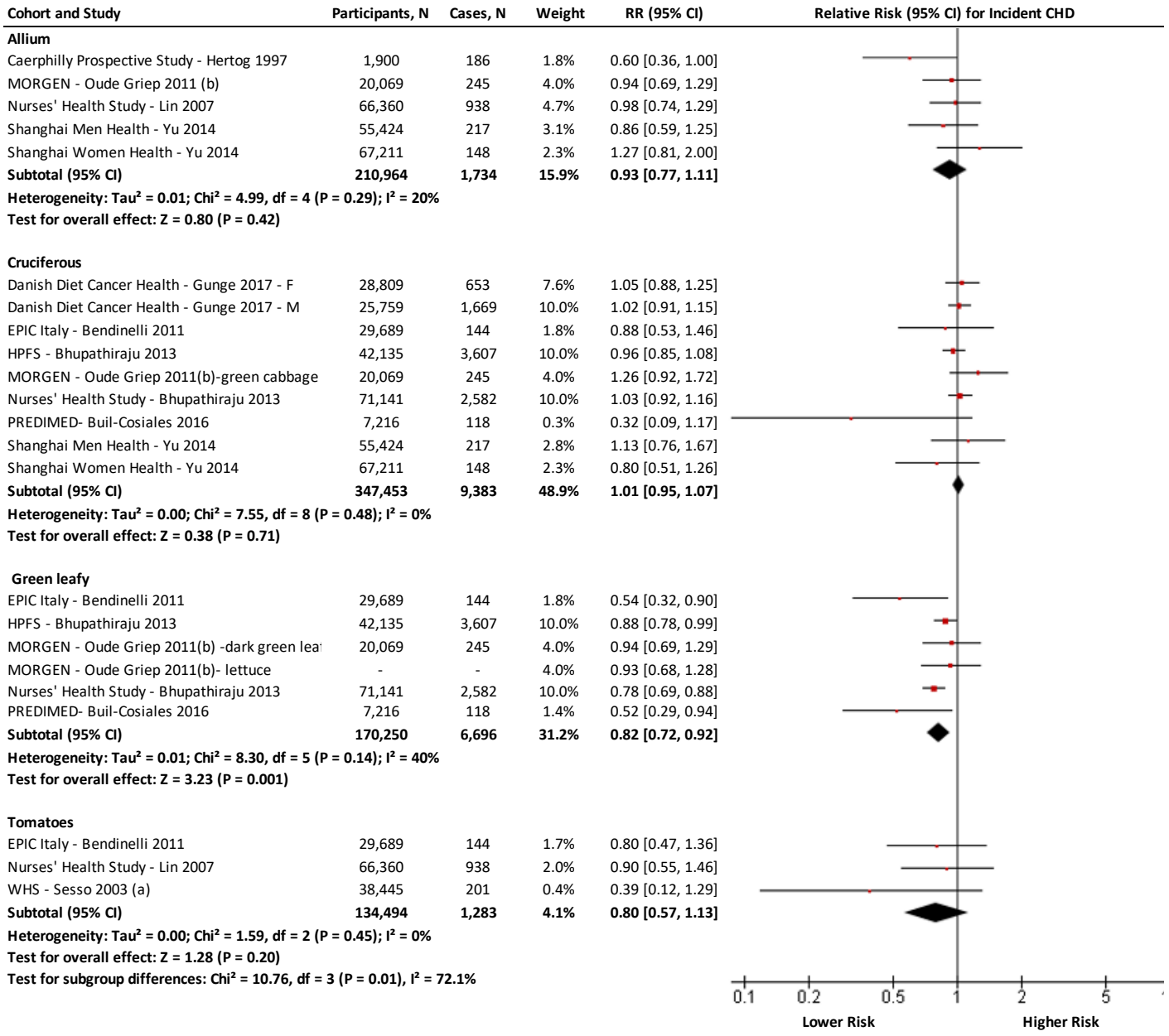


Figure S71. Relation between sources of vegetables and CHD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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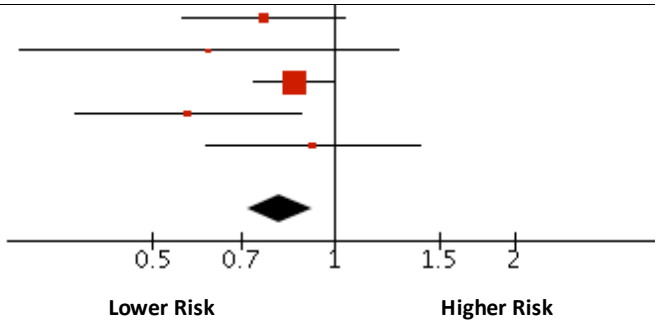
TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
National Health & Nutrition - Bazzano 2002	9,608	639	15.9%	0.76 [0.56, 1.04]
Baltimore Longitudinal Study Aging - Tucker 2005	501	71	3.0%	0.62 [0.30, 1.28]
EPIC - Leenders 2014	451,151	2,139	63.5%	0.86 [0.74, 1.01]
NIPPON DATA80 - Okuda 2015	9,112	165	8.4%	0.57 [0.37, 0.88]
HAPIEE - Stefler 2016	19,263	226	9.2%	0.92 [0.61, 1.39]
Total (95% CI)	**489,635**	**3,240**	**100.0%**	**0.81 [0.72, 0.92]**

Heterogeneity: Chi² = 4.15, df = 4 (P = 0.39); I² = 4%

Test for overall effect: Z = 3.24 (P = 0.001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
National Health & Nutrition - Bazzano 2002	9,608	639	17.0%	0.76 [0.56, 1.04]
Baltimore Longitudinal Study Aging - Tucker 2005	501	71	3.3%	0.62 [0.30, 1.28]
EPIC - Leenders 2014	451,151	2,139	60.6%	0.86 [0.74, 1.01]
NIPPON DATA80 - Okuda 2015	9,112	165	9.2%	0.57 [0.37, 0.88]
HAPIEE - Stefler 2016	19,263	226	10.0%	0.92 [0.61, 1.39]
Total (95% CI) [Random Effects]	**489,635**	**3,240**	**100.0%**	**0.81 [0.71, 0.92]**

Heterogeneity: Tau² = 0.00; Chi² = 4.15, df = 4 (P = 0.39); I² = 4%

Test for overall effect: Z = 3.15 (P = 0.002)

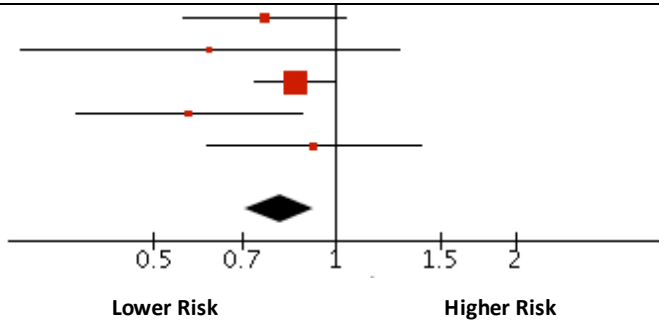


Figure S72. Relation between total fruit and vegetable intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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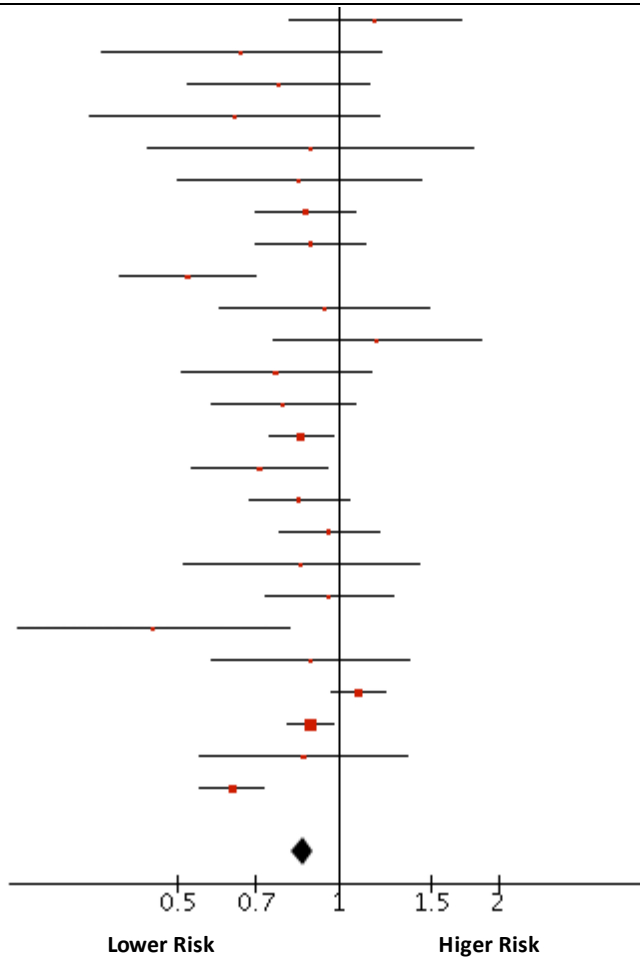
FRUIT AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Adventis Health Study - Fraser -1992	26,473	463	1.5%	1.17 [0.81, 1.70]
Finish Mobile Clinic Health - Knekt 1994 - F	2,748	58	0.6%	0.77 [0.52, 1.14]
Finish Mobile Clinic Health - Knekt 1994 - M	2,385	186	1.4%	0.66 [0.36, 1.21]
Nutrition Status Study - Sahyoun 1996	680	101	0.5%	0.64 [0.34, 1.19]
Oxford Vegetarian - Mann 1997	10,802	64	0.4%	0.89 [0.44, 1.80]
OXCHECK - Whiteman 1999	10,522	144	0.8%	0.84 [0.50, 1.43]
ATBC - Hirvonen 2001	25,373	815	4.6%	0.87 [0.70, 1.08]
Health Food Shoppers - Appleby 2002 - M	6,416	258	3.9%	0.52 [0.39, 0.70]
Health Food Shoppers - Appleby 2002 - F	4,325	347	2.5%	0.89 [0.70, 1.12]
Baltimore Longitudinal Study Aging - Tucker 2005	4,028	298	1.0%	1.19 [0.76, 1.86]
Boyd Orr Cohort - Ness 2005	501	71	1.0%	0.94 [0.60, 1.48]
Melbourne Collaborative Cohort - Harriss 2007	40,653	407	1.3%	0.76 [0.51, 1.15]
JACC - Nagura 2009	59,485	452	2.2%	0.79 [0.57, 1.08]
EPIC - Leenders 2014	1,849	64	11.3%	0.85 [0.51, 1.42]
Singapore Chinese Health - Rebello 2014 - F	451,151	2,139	2.5%	0.85 [0.74, 0.98]
Singapore Chinese Health - Rebello 2014 - M	29,968	638	4.6%	0.71 [0.53, 0.96]
Multiethnic Cohort - Sharma 2014 - F	23,501	1,022	4.6%	0.84 [0.68, 1.05]
MONICA Danish - Tognon 2014	91,751	811	0.8%	0.96 [0.77, 1.19]
Multiethnic Cohort - Sharma 2014 - M	72,866	1,140	2.8%	0.96 [0.73, 1.26]
UK Women's Cohort - Lai 2015	30,458	138	0.6%	0.45 [0.25, 0.81]
NIPPON DATA80 - Okuda 2015	9,112	165	1.1%	0.89 [0.58, 1.37]
Migrant Study - Hjartaker 2015	9,766	2,386	15.4%	1.09 [0.97, 1.23]
Linxian Nutrition - Wang 2016	2,445	355	22.2%	0.89 [0.80, 0.98]
HAPIEE - Stefler 2016	19,263	226	1.0%	0.86 [0.55, 1.35]
China Kadoorie Biobank- Du 2017	462,342	2,038	11.3%	0.63 [0.55, 0.72]
Total (95% CI)	**1,398,863**	**14,786**	**100.0%**	**0.86 [0.82, 0.90]**

Heterogeneity: Chi² = 62.47, df = 24 (P < 0.0001); I² = 62%

Test for overall effect: Z = 6.52 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Adventis Health Study - Fraser -1992	26,473	463	3.4%	1.17 [0.81, 1.70]
Finish Mobile Clinic Health - Knekt 1994 - M	2,748	58	3.2%	0.77 [0.52, 1.14]
Finish Mobile Clinic Health - Knekt 1994 - F	2,385	186	1.7%	0.66 [0.36, 1.21]
Nutrition Status Study - Sahyoun 1996	680	101	1.6%	0.64 [0.34, 1.19]
Oxford Vegetarian - Mann 1997	10,802	64	1.3%	0.89 [0.44, 1.80]
OXCHECK - Whiteman 1999	10,522	144	2.1%	0.84 [0.50, 1.43]
ATBC - Hirvonen 2001	25,373	815	5.6%	0.87 [0.70, 1.08]
Health Food Shoppers - Appleby 2002 - F	6,416	258	4.4%	0.52 [0.39, 0.70]
Health Food Shoppers - Appleby 2002 - M	4,325	347	5.3%	0.89 [0.70, 1.12]
Boyd Orr Cohort - Ness 2005	4,028	298	2.6%	1.19 [0.76, 1.86]
Baltimore Longitudinal Study Aging - Tucker 2005	501	71	2.6%	0.94 [0.60, 1.48]
Melbourne Collaborative Cohort - Harriss 2007	40,653	407	3.0%	0.76 [0.51, 1.15]
JACC - Nagura 2009	59,485	452	4.1%	0.79 [0.57, 1.08]
MONICA Danish - Tognon 2014	1,849	64	2.2%	0.85 [0.51, 1.42]
EPIC - Leenders 2014	451,151	2,139	7.0%	0.85 [0.74, 0.98]
Singapore Chinese Health - Rebello 2014 - F	29,968	638	4.4%	0.71 [0.53, 0.96]
Singapore Chinese Health - Rebello 2014 - M	23,501	1,022	5.6%	0.84 [0.68, 1.05]
Multiethnic Cohort - Sharma 2014 - F	91,751	811	5.6%	0.96 [0.77, 1.19]
Multiethnic Cohort - Sharma 2014 - M	72,866	1,140	4.7%	0.96 [0.73, 1.26]
UK Women's Cohort - Lai 2015	30,458	138	1.8%	0.45 [0.25, 0.81]
NIPPON DATA80 - Okuda 2015	9,112	165	2.8%	0.89 [0.58, 1.37]
Migrant Study - Hjartaker 2015	9,766	2,386	7.4%	1.09 [0.97, 1.23]
Linxian Nutrition - Wang 2016	2,445	355	7.7%	0.89 [0.80, 0.98]
HAPIEE - Stefler 2016	19,263	226	2.6%	0.86 [0.55, 1.35]
China Kadoorie Biobank- Du 2017	462,342	2,038	7.0%	0.63 [0.55, 0.72]
Total (95% CI) [Random Effects]	**1,398,863**	**14,786**	**100.0%**	**0.84 [0.76, 0.91]**

Heterogeneity: Tau² = 0.02; Chi² = 62.47, df = 24 (P < 0.0001); I² = 62%

Test for overall effect: Z = 3.99 (P < 0.0001)

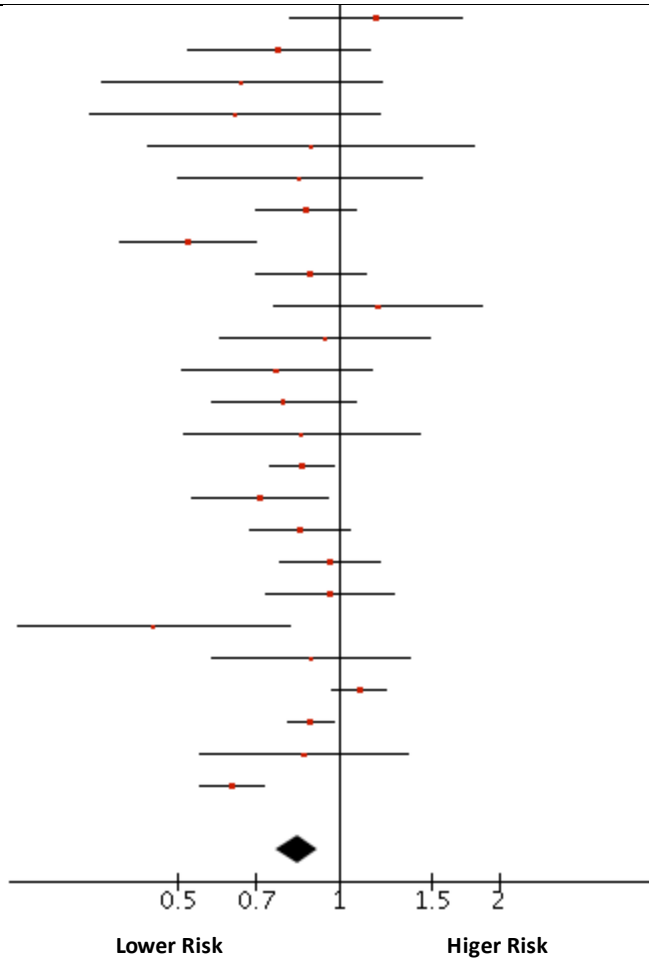


Figure S73. Relation between fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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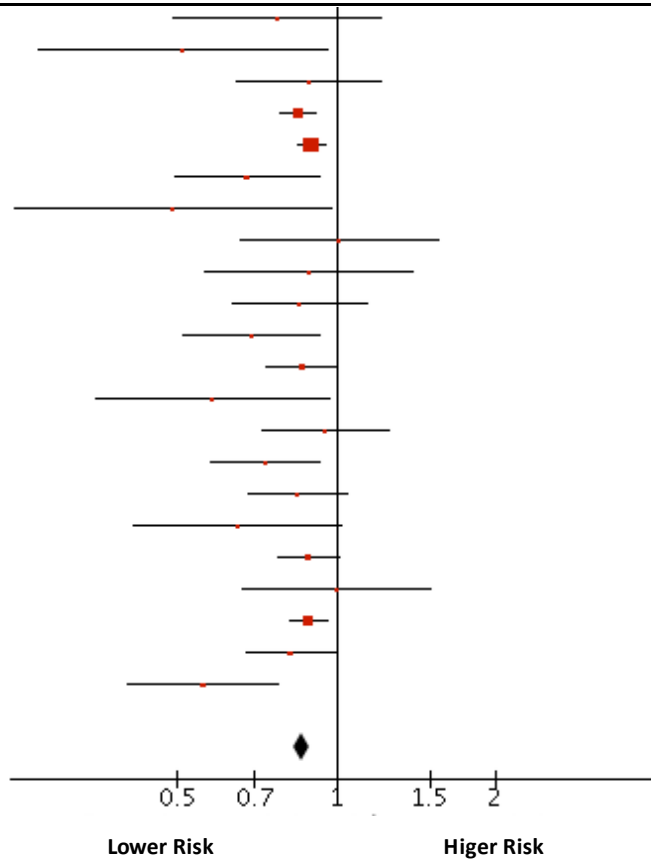
VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	1.1%	0.77 [0.49, 1.21]
Nutrition Status Study - Sahyoun 1996	680	101	0.6%	0.51 [0.27, 0.96]
Finish Mobile Clinic Health - Knekt 1996 - M	29,968	324	2.1%	0.89 [0.65, 1.21]
CPS 11 - Watkins 2000 - F	609,061	4,605	15.8%	0.84 [0.78, 0.91]
CPS 11 - Watkins 2000- M	453,962	9,156	19.5%	0.90 [0.84, 0.95]
ATBC - Hirvonen 2001	25,373	815	2.1%	0.68 [0.49, 0.93]
Baltimore Longitudinal Study Aging - Tucker 2005	501	71	0.5%	0.49 [0.25, 0.98]
Boyd Orr Cohort - Ness 2005	4,028	298	1.2%	1.01 [0.66, 1.55]
Melbourne Collaborative Cohort - Harriss 2007	40,653	407	1.1%	0.89 [0.57, 1.39]
JACC - Nagura 2009	59,485	452	2.4%	0.85 [0.64, 1.14]
Singapore Chinese Health - Rebello 2014 - F	29,968	638	2.4%	0.69 [0.51, 0.93]
EPIC - Leenders 2014	451,151	2,139	6.9%	0.86 [0.74, 1.01]
MONICA Danish - Tognon 2014	1,849	64	0.8%	0.58 [0.35, 0.97]
Multiethnic Cohort - Sharma 2014 - F	91,751	811	2.7%	0.95 [0.72, 1.25]
Multiethnic Cohort - Sharma 2014 - M	72,866	1,140	3.5%	0.73 [0.58, 0.93]
Singapore Chinese Health - Rebello 2014 - M	23,501	1,022	4.1%	0.84 [0.68, 1.05]
NIPPON DATA80 - Okuda 2015	9,112	165	1.1%	0.65 [0.41, 1.02]
Migrant Study - Hjartaker 2015	9,964	2,386	8.4%	0.89 [0.77, 1.02]
HAPIEE - Stefler 2016	19,263	225	1.3%	1.00 [0.66, 1.51]
Linxian Nutrition - Wang 2016	2,445	355	15.8%	0.89 [0.82, 0.96]
PLSAW - Blekkenhorst 2017	1,226	128	4.8%	0.82 [0.67, 1.00]
NHANES - Conrad 2018	29,133	556	2.0%	0.56 [0.40, 0.78]
Total (95% CI)	**1,968,325**	**26,007**	**100.0%**	**0.86 [0.83, 0.89]**

Heterogeneity: Chi² = 26.70, df = 21 (P = 0.18); I² = 21%

Test for overall effect: Z = 8.79 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	1.1%	0.77 [0.49, 1.21]
Nutrition Status Study - Sahyoun 1996	680	101	0.6%	0.51 [0.27, 0.96]
Finish Mobile Clinic Health - Knekt 1996 - M	29,968	324	2.1%	0.89 [0.65, 1.21]
CPS 11 - Watkins 2000 - F	609,061	4,605	15.8%	0.84 [0.78, 0.91]
CPS 11 - Watkins 2000- M	453,962	9,156	19.5%	0.90 [0.84, 0.95]
ATBC - Hirvonen 2001	25,373	815	2.1%	0.68 [0.49, 0.93]
Baltimore Longitudinal Study Aging - Tucker 2005	501	71	0.5%	0.49 [0.25, 0.98]
Boyd Orr Cohort - Ness 2005	4,028	298	1.2%	1.01 [0.66, 1.55]
Melbourne Collaborative Cohort - Harriss 2007	40,653	407	1.1%	0.89 [0.57, 1.39]
JACC - Nagura 2009	59,485	452	2.4%	0.85 [0.64, 1.14]
Singapore Chinese Health - Rebello 2014 - F	29,968	638	2.4%	0.69 [0.51, 0.93]
EPIC - Leenders 2014	451,151	2,139	6.9%	0.86 [0.74, 1.01]
MONICA Danish - Tognon 2014	1,849	64	0.8%	0.58 [0.35, 0.97]
Multiethnic Cohort - Sharma 2014 - F	91,751	811	2.7%	0.95 [0.72, 1.25]
Multiethnic Cohort - Sharma 2014 - M	72,866	1,140	3.5%	0.73 [0.58, 0.93]
Singapore Chinese Health - Rebello 2014 - M	23,501	1,022	4.1%	0.84 [0.68, 1.05]
NIPPON DATA80 - Okuda 2015	9,112	165	1.1%	0.65 [0.41, 1.02]
Migrant Study - Hjartaker 2015	9,964	2,386	8.4%	0.89 [0.77, 1.02]
HAPIEE - Stefler 2016	19,263	225	1.3%	1.00 [0.66, 1.51]
Linxian Nutrition - Wang 2016	2,445	355	15.8%	0.89 [0.82, 0.96]
PLSAW - Blekkenhorst 2017	1,226	128	4.8%	0.82 [0.67, 1.00]
NHANES - Conrad 2018	29,133	556	2.0%	0.56 [0.40, 0.78]
Total (95% CI) [Random Effects]	**1,968,325**	**26,007**	**100.0%**	**0.84 [0.80, 0.88]**

Heterogeneity: Tau² = 0.00; Chi² = 26.70, df = 21 (P = 0.18); I² = 21%

Test for overall effect: Z = 7.10 (P < 0.00001)

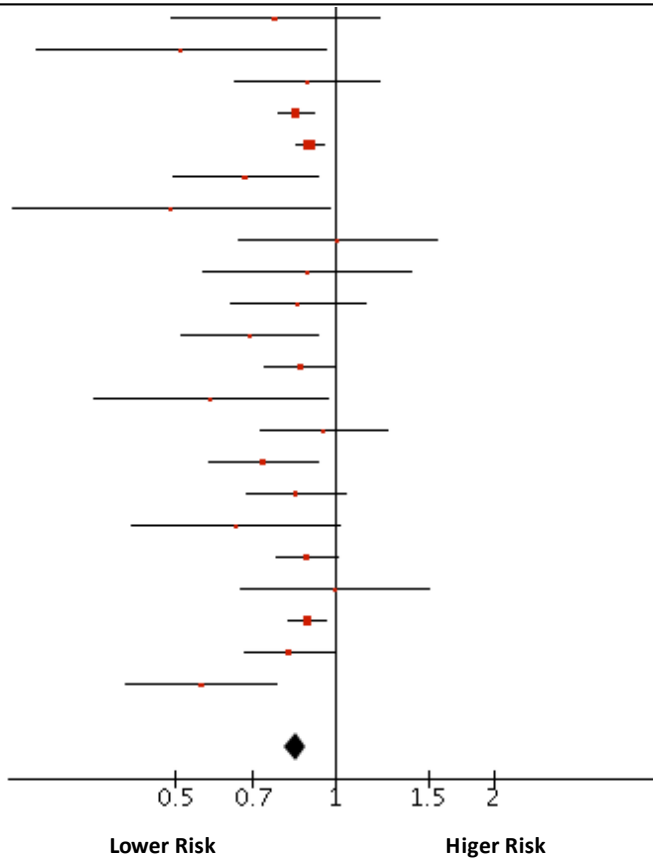


Figure S74. Relation between intake of vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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BANANAS AND CORONARY HEART DISEASE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Migrant Study - Hjartaker 2015	9,964	2,384	100.0%	1.04 [0.81, 1.34]
Total (95% CI)	**9,964**	**2,384**	**100.0%**	**1.04 [0.81, 1.34]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.31 (P = 0.76)				

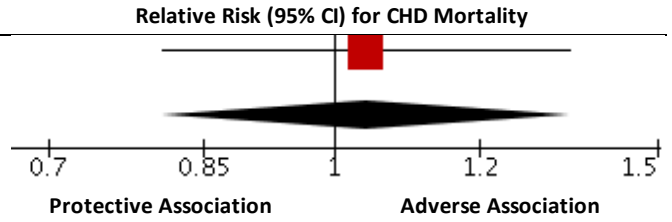


Figure S75. Relation between intake of bananas and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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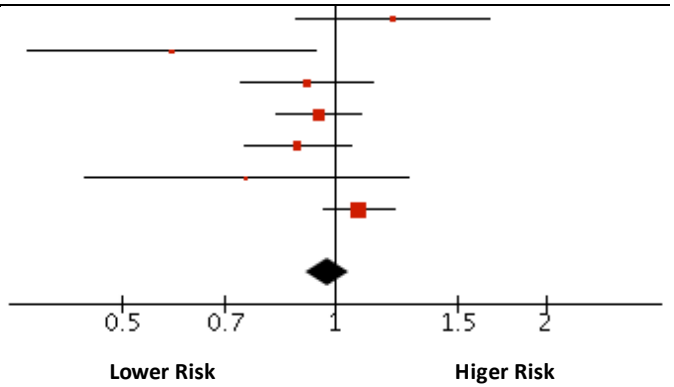
BERRIES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	5.2%	1.21 [0.88, 1.65]
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	2.3%	0.59 [0.37, 0.94]
ATBC - Hirvonen 2001	25,373	815	10.9%	0.91 [0.74, 1.13]
Iowa WHS - Mink 2007 - strawberries	-	-	26.9%	0.95 [0.83, 1.09]
Iowa WHS - Mink 2007 - blueberries	34,492	1,329	16.3%	0.89 [0.74, 1.06]
UK Women's Cohort - Lai 2015	30,458	138	1.8%	0.75 [0.44, 1.27]
Migrant Study - Hjartaker 2015	9,964	2,386	36.6%	1.08 [0.96, 1.22]
Total (95% CI)	**105,420**	**5,141**	**100.0%**	**0.98 [0.91, 1.05]**

Heterogeneity: Chi² = 11.84, df = 6 (P = 0.07); I² = 49%

Test for overall effect: Z = 0.58 (P = 0.56)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	5.00%	0.59 [0.37, 0.94]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	9.50%	1.21 [0.88, 1.65]
ATBC - Hirvonen 2001	25,373	815	15.30%	0.91 [0.74, 1.13]
Iowa WHS - Mink 2007 - blueberries	34,492	1,329	18.70%	0.89 [0.74, 1.06]
Iowa WHS - Mink 2007 - strawberries	-	-	22.60%	0.95 [0.83, 1.09]
Migrant Study - Hjartaker 2015	9,964	2,386	24.70%	1.08 [0.96, 1.22]
UK Women's Cohort - Lai 2015	30,458	138	4.10%	0.75 [0.44, 1.27]
Total (95% CI) [Random Effects]	**105,420**	**5,141**	**100.00%**	**0.95 [0.85, 1.07]**

Heterogeneity: Tau² = 0.01; Chi² = 11.84, df = 6 (P = 0.07); I² = 49%

Test for overall effect: Z = 0.82 (P = 0.41)

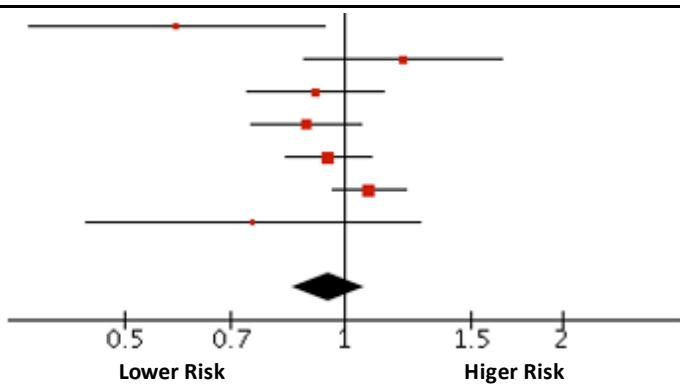


Figure S76. Relation between intake of berries and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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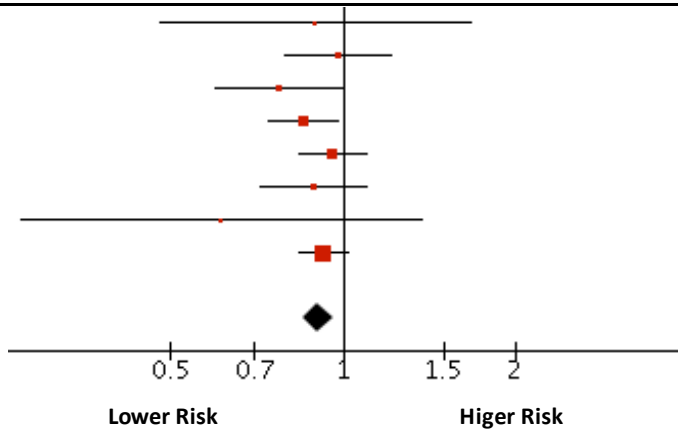
CITRUS FRUIT AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nutrition Status Study - Sahyoun 1996	680	101	0.9%	0.90 [0.48, 1.68]
JACC - Iso 2007 - M	43,031	602	7.9%	0.98 [0.79, 1.22]
JACC - Iso 2007 - F	59,504	398	5.6%	0.77 [0.60, 0.99]
Iowa WHS - Mink 2007 - grapefruit	34,492	1,329	19.5%	0.85 [0.74, 0.98]
Iowa WHS - Mink 2007 - oranges	-	-	19.5%	0.96 [0.84, 1.10]
Migrant Study - Hjartaker 2015	9,964	2,386	7.9%	0.89 [0.71, 1.10]
UK Women's Cohort - Lai 2015	30,458	138	0.6%	0.61 [0.27, 1.37]
Linxian Nutrition - Wang 2016	2,445	355	38.2%	0.92 [0.84, 1.02]
Total (95% CI)	**180,574**	**5,309**	**100.0%**	**0.91 [0.85, 0.96]**

Heterogeneity: Chi² = 4.61, df = 7 (P = 0.71); I² = 0%

Test for overall effect: Z = 3.20 (P = 0.001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nutrition Status Study - Sahyoun 1996	680	101	0.9%	0.90 [0.48, 1.68]
JACC - Iso 2007 - F	59,504	398	5.6%	0.77 [0.60, 0.99]
JACC - Iso 2007 - M	43,031	602	7.9%	0.98 [0.79, 1.22]
Iowa WHS - Mink 2007 - grapefruit	34,492	1,329	19.5%	0.85 [0.74, 0.98]
Iowa WHS - Mink 2007 - oranges	-	-	19.5%	0.96 [0.84, 1.10]
Migrant Study - Hjartaker 2015	9,964	2,386	7.9%	0.89 [0.71, 1.10]
UK Women's Cohort - Lai 2015	30,458	138	0.6%	0.61 [0.27, 1.37]
Linxian Nutrition - Wang 2016	2,445	355	38.2%	0.92 [0.84, 1.02]
Total (95% CI) [Random Effects]	**180,574**	**5,309**	**100.0%**	**0.91 [0.85, 0.96]**

Heterogeneity: Tau² = 0.00; Chi² = 4.61, df = 7 (P = 0.71); I² = 0%

Test for overall effect: Z = 3.20 (P = 0.001)

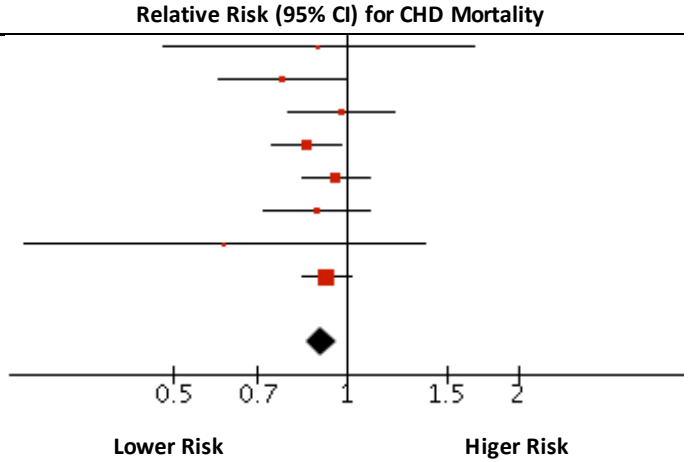


Figure S77. Relation between citrus fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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DRIED FRUIT AND CORONARY HEART DISEASE MORTALITY

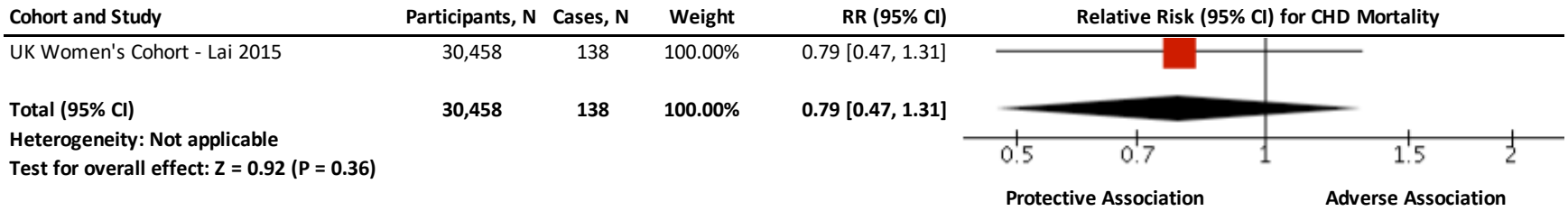


Figure S78. Relation between dried fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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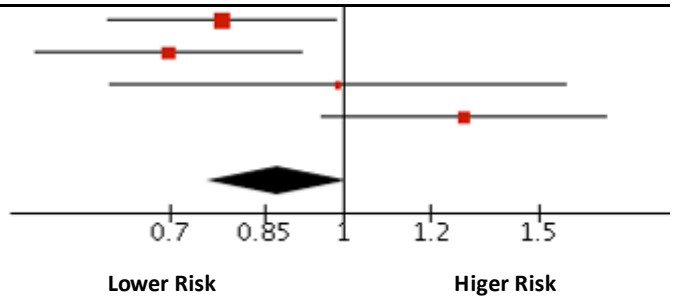
FRUIT JUICE AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - M	41,330	573	38.1%	0.78 [0.62, 0.99]
JACC - Iso 2007 - F	56,482	370	28.0%	0.70 [0.53, 0.92]
UK Women's Cohort - Lai 2015	30,458	138	9.5%	0.99 [0.62, 1.58]
REGARDS - Colin 2019	13,440	168	24.4%	1.32 [1.08 1.62]
Total (95% CI)	**141,710**	**1,249**	**100.0%**	**0.87 [0.75, 1.01]**

Heterogeneity: Chi² = 10.36, df = 3 (P = 0.02); I² = 71%

Test for overall effect: Z = 1.84 (P = 0.07)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - M	41,330	573	26.9%	0.78 [0.62, 0.99]
JACC - Iso 2007 - F	56,482	370	25.7%	0.70 [0.53, 0.92]
UK Women's Cohort - Lai 2015	30,458	138	19.3%	0.99 [0.62, 1.58]
REGARDS - Colin 2019	13,440	168	28.1%	1.32 [1.08 1.62]
Total (95% CI) [Random Effects]	**141,710**	**1,249**	**100.0%**	**0.90 [0.68, 1.19]**

Heterogeneity: Tau² = 0.06; Chi² = 10.36, df = 3 (P = 0.02); I² = 71%

Test for overall effect: Z = 0.74 (P = 0.46)

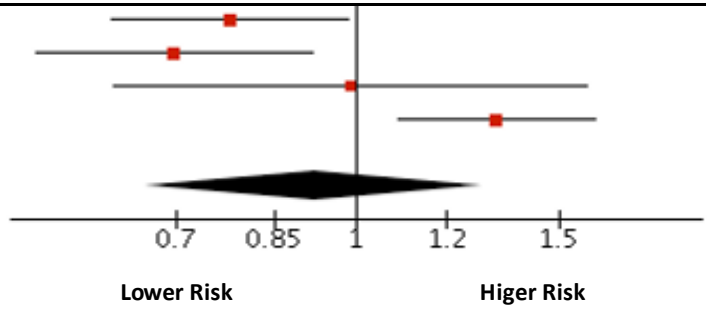


Figure S79. Relation between intake of fruit juice and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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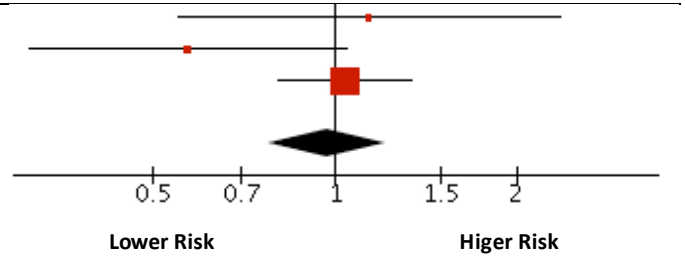
GRAPES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Lin 2007	66,360	324	9.5%	1.14 [0.55, 2.35]
UK Women's Cohort - Lai 2015 - grapes	30,458	138	13.5%	0.57 [0.31, 1.05]
Migrant Study - Hjartaker 2015	9,964	2,384	77.0%	1.04 [0.81, 1.34]
Total (95% CI)	**106,782**	**2,846**	**100.0%**	**0.97 [0.77, 1.21]**

Heterogeneity: Chi² = 3.40, df = 2 (P = 0.18); I² = 41%

Test for overall effect: Z = 0.29 (P = 0.77)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Lin 2007	66,360	324	19.5%	1.14 [0.55, 2.35]
UK Women's Cohort - Lai 2015 - grapes	30,458	138	25.0%	0.57 [0.31, 1.05]
Migrant Study - Hjartaker 2015	9,964	2,384	55.5%	1.04 [0.81, 1.34]
Total (95% CI) [Random Effects]	**106,782**	**2,846**	**100.0%**	**0.91 [0.63, 1.32]**

Heterogeneity: Tau² = 0.05; Chi² = 3.40, df = 2 (P = 0.18); I² = 41%

Test for overall effect: Z = 0.49 (P = 0.63)

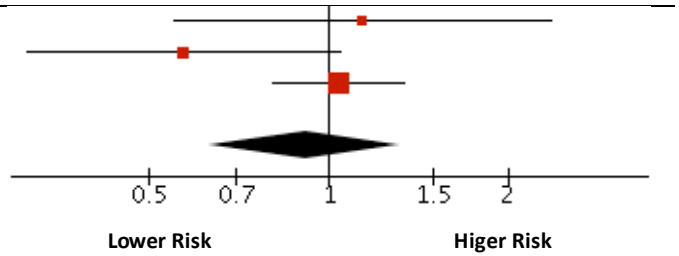


Figure S80. Relation between intake of grapes and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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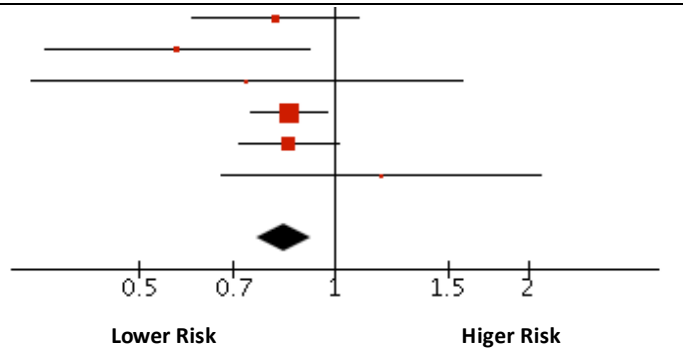
POMMES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	10.9%	0.81 [0.60, 1.09]
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	4.3%	0.57 [0.36, 0.91]
Nurses' Health Study - Lin 2007	66,360	324	1.6%	0.73 [0.34, 1.58]
Iowa WHS - Mink 2007	34,492	1,329	50.0%	0.85 [0.74, 0.98]
Migrant Study - Hjartaker 2015	9,964	2,386	30.3%	0.85 [0.71, 1.02]
UK Women's Cohort - Lai 2015	30,458	138	2.9%	1.19 [0.67, 2.09]
Total (95% CI)	**146,407**	**4,650**	**100.0%**	**0.84 [0.76, 0.92]**

Heterogeneity: Chi² = 4.24, df = 5 (P = 0.52); I² = 0%

Test for overall effect: Z = 3.54 (P = 0.0004)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	4.3%	0.57 [0.36, 0.91]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	10.9%	0.81 [0.60, 1.09]
Iowa WHS - Mink 2007	34,492	1,329	50.0%	0.85 [0.74, 0.98]
Nurses' Health Study - Lin 2007	66,360	324	1.6%	0.73 [0.34, 1.58]
UK Women's Cohort - Lai 2015	30,458	138	2.9%	1.19 [0.67, 2.09]
Migrant Study - Hjartaker 2015	9,964	2,386	30.3%	0.85 [0.71, 1.02]
Total (95% CI) [Random Effects]	**146,407**	**4,650**	**100.0%**	**0.84 [0.76, 0.92]**

Heterogeneity: Tau² = 0.00; Chi² = 4.24, df = 5 (P = 0.52); I² = 0%

Test for overall effect: Z = 3.54 (P = 0.0004)

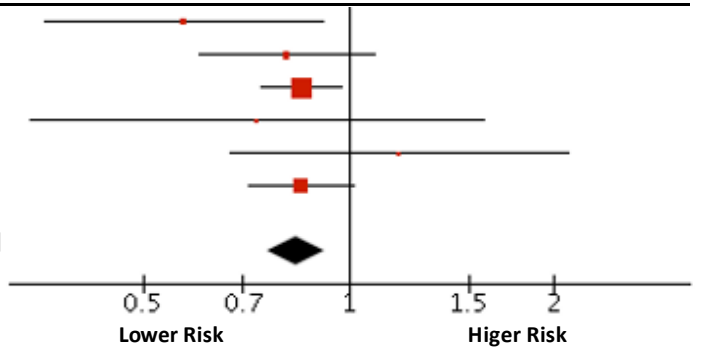


Figure S81. Relation between pommes fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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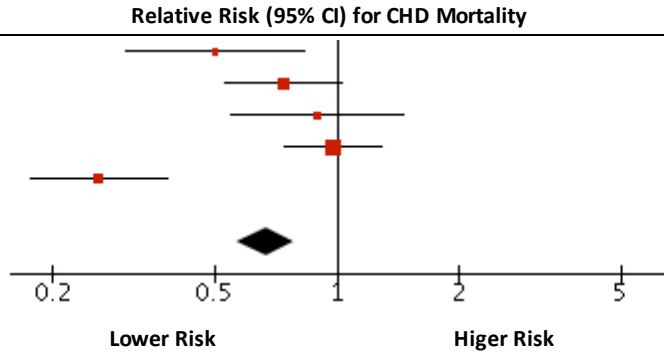
ALLIUM VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	10.5%	0.50 [0.30, 0.83]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	24.5%	0.74 [0.53, 1.03]
Linxian Nutrition - Wang 2016	2,445	355	11.3%	0.98 [0.74, 1.29]
Nurses' Health Study - Lin 2007	66,630	324	36.1%	0.90 [0.55, 1.46]
PLSAW - Blekkenhorst 2017	1,226	128	17.7%	0.26 [0.18, 0.38]
Total (95% CI)	**75,434**	**1,280**	**100.0%**	**0.67 [0.57, 0.79]**

Heterogeneity: Chi² = 32.86, df = 4 (P < 0.00001); I² = 88%

Test for overall effect: Z = 4.80 (P < 0.00001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	18.5%	0.50 [0.30, 0.83]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	20.9%	0.74 [0.53, 1.03]
Linxian Nutrition - Wang 2016	2,445	355	21.6%	0.98 [0.74, 1.29]
Nurses' Health Study - Lin 2007	66,630	324	18.8%	0.90 [0.55, 1.46]
PLSAW - Blekkenhorst 2017	1,226	128	20.2%	0.26 [0.18, 0.38]
Total (95% CI) [Random Effects]	**75,434**	**1,280**	**100.0%**	**0.61 [0.38, 1.00]**

Heterogeneity: Tau² = 0.27; Chi² = 32.86, df = 4 (P < 0.00001); I² = 88%

Test for overall effect: Z = 1.95 (P = 0.05)

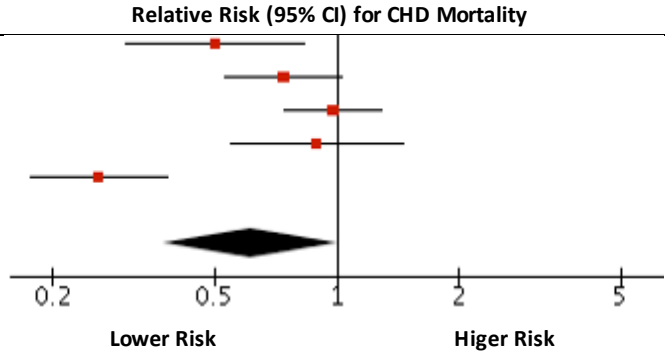
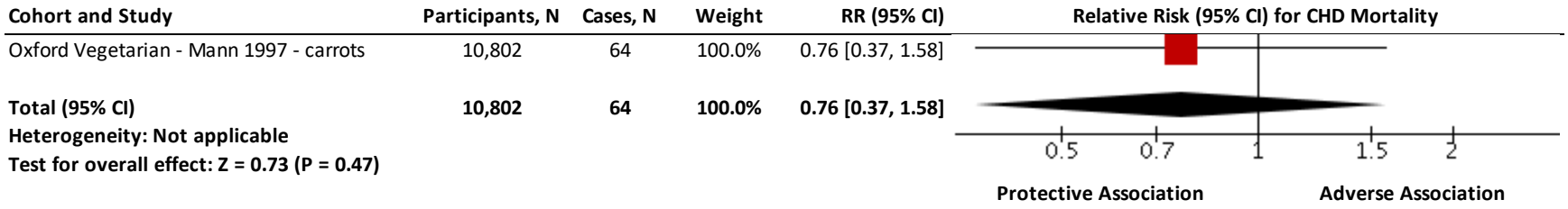


Figure S82. Relation between intake of allium vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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CARROTS AND CORONARY HEART DISEASE MORTALITY



Supplementary Figure 83. Relation between intake of carrots and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

CELERY AND CORONARY HEART DISEASE MORTALITY

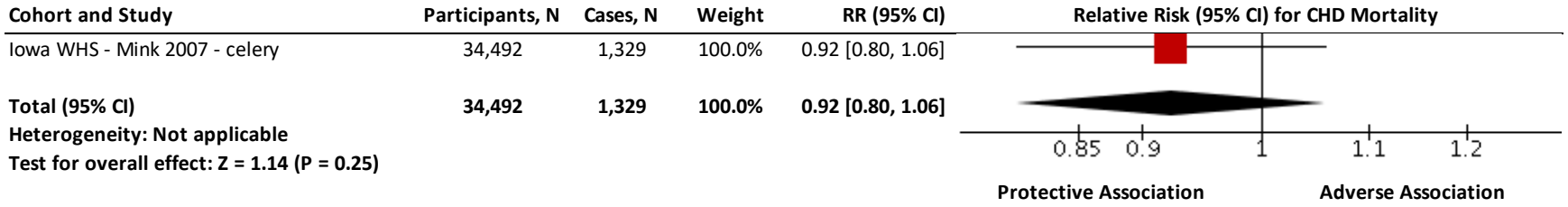


Figure S84. Relation between intake of celery and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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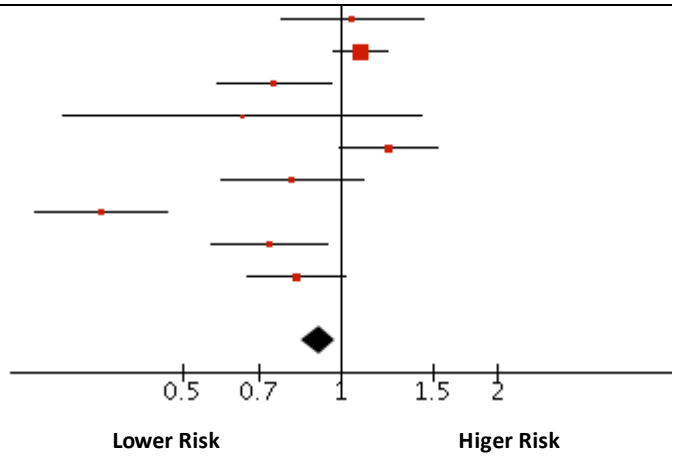
CRUCIFEROUS VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	54,325	396	5.7%	1.05 [0.77, 1.44]
Iowa WHS - Mink 2007	34,492	1,329	40.2%	1.09 [0.97, 1.23]
JACC - Iso 2007 - M	39,486	534	8.6%	0.75 [0.58, 0.97]
Nurses' Health Study - Lin 2007	66,630	324	0.9%	0.65 [0.30, 1.42]
Migrant Study - Hjartaker 2015	9,964	2,386	12.0%	1.23 [0.99, 1.53]
Linxian Nutrition - Wang 2016	2,445	355	5.7%	0.81 [0.59, 1.11]
PLSAW - Blekkenhorst 2017	1,226	128	6.4%	0.35 [0.26, 0.47]
Japan Public Health Center - Mori 2018 - F	47,562	776	8.6%	0.73 [0.57, 0.95]
Japan Public Health Center - Mori 2018 - M	40,642	1,192	12.0%	0.83 [0.67, 1.03]
Total (95% CI)	**296,772**	**7,420**	**100.0%**	**0.91 [0.85, 0.98]**

Heterogeneity: Chi² = 65.50, df = 8 (P < 0.00001); I² = 88%

Test for overall effect: Z = 2.45 (P = 0.01)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	54,325	396	11.0%	1.05 [0.77, 1.44]
Iowa WHS - Mink 2007	34,492	1,329	13.2%	1.09 [0.97, 1.23]
JACC - Iso 2007 - M	39,486	534	11.8%	0.75 [0.58, 0.97]
Nurses' Health Study - Lin 2007	66,630	324	5.4%	0.65 [0.30, 1.42]
Migrant Study - Hjartaker 2015	9,964	2,386	12.3%	1.23 [0.99, 1.53]
Linxian Nutrition - Wang 2016	2,445	355	11.0%	0.81 [0.59, 1.11]
PLSAW - Blekkenhorst 2017	1,226	128	11.3%	0.35 [0.26, 0.47]
Japan Public Health Center - Mori 2018 - M	40,642	1,192	12.3%	0.83 [0.67, 1.03]
Japan Public Health Center - Mori 2018 - F	47,562	776	11.8%	0.73 [0.57, 0.95]
Total (95% CI) [Random Effects]	**296,772**	**7,420**	**100.0%**	**0.81 [0.64, 1.02]**

Heterogeneity: Tau² = 0.11; Chi² = 65.50, df = 8 (P < 0.00001); I² = 88%

Test for overall effect: Z = 1.79 (P = 0.07)

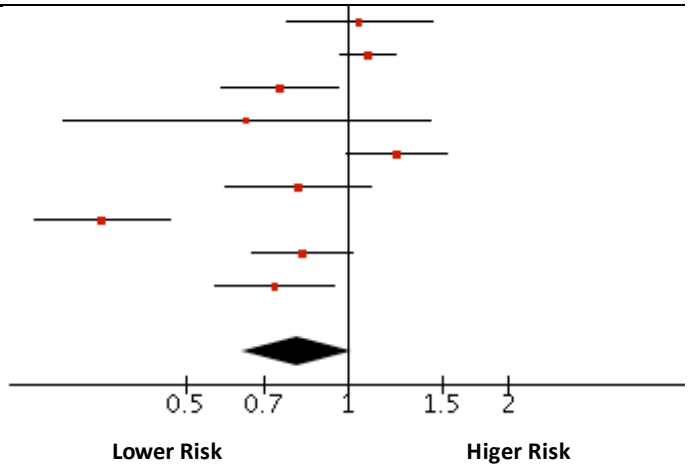


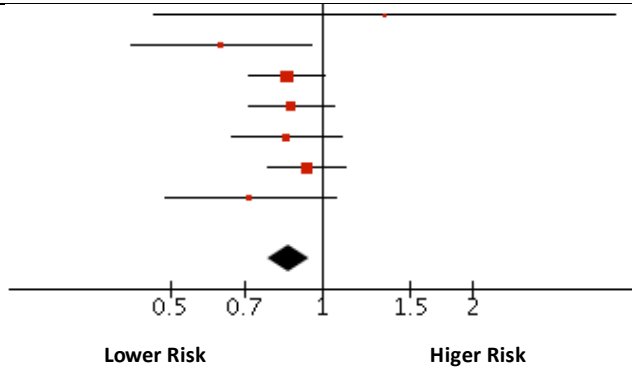
Figure S85. Relation between intake of cruciferous vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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GREEN LEAFY VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Oxford Vegetarian - Mann 1997	10,802	64	0.8%	1.34 [0.46, 3.85]
OXCHECK - Whiteman 1999	10,522	144	5.0%	0.63 [0.42, 0.95]
Health Food Shoppers - Appleby 2002	10,741	605	27.0%	0.85 [0.71, 1.02]
JACC - Iso 2007 - M	43,850	617	21.9%	0.87 [0.71, 1.06]
JACC - Iso 2007 - F	59,809	420	12.9%	0.85 [0.66, 1.10]
Migrant Study - Hjartaker 2015	9,964	2,386	27.0%	0.93 [0.78, 1.11]
Linxian Nutrition - Wang 2016	2,445	355	5.5%	0.72 [0.49, 1.06]
Total (95% CI)				
Heterogeneity: Chi² = 4.47, df = 6 (P = 0.61); I² = 0%	**148,133**	**4,591**	**100.0%**	**0.86 [0.78, 0.94]**
Test for overall effect: Z = 3.25 (P = 0.001)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Oxford Vegetarian - Mann 1997	10,802	64	0.8%	1.34 [0.46, 3.85]
OXCHECK - Whiteman 1999	10,522	144	5.0%	0.63 [0.42, 0.95]
Health Food Shoppers - Appleby 2002	10,741	605	27.0%	0.85 [0.71, 1.02]
JACC - Iso 2007 - M	43,850	617	21.9%	0.87 [0.71, 1.06]
JACC - Iso 2007 - F	59,809	420	12.9%	0.85 [0.66, 1.10]
Migrant Study - Hjartaker 2015	9,964	2,386	27.0%	0.93 [0.78, 1.11]
Linxian Nutrition - Wang 2016	2,445	355	5.5%	0.72 [0.49, 1.06]
Total (95% CI) [Random Effects]	**148,133**	**4,591**	**100.0%**	**0.86 [0.78, 0.94]**
Heterogeneity: Tau² = 0.00; Chi² = 4.47, df = 6 (P = 0.61); I² = 0%				
Test for overall effect: Z = 3.25 (P = 0.001)				

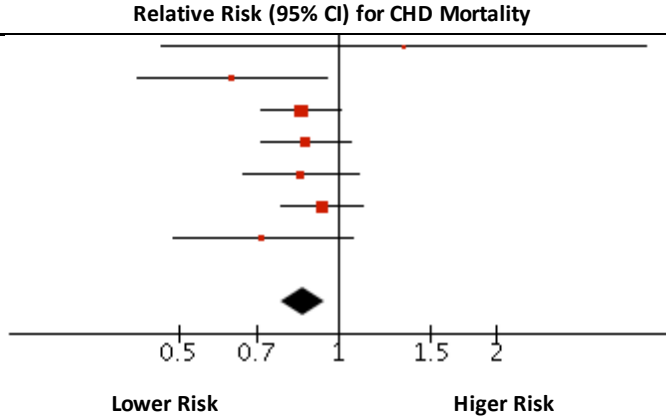


Figure S86. Relation between intake of green leafy vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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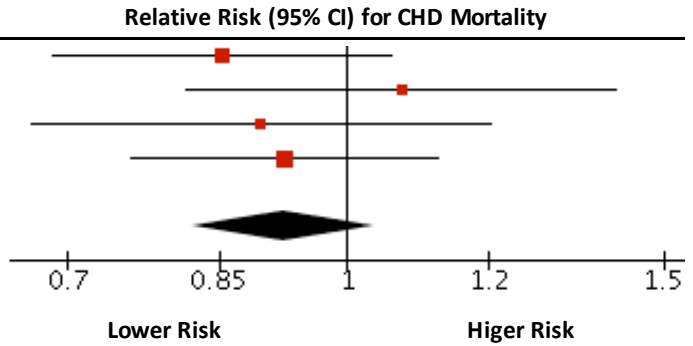
TOMATOES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - tomatoes - M	41,547	568	29.7%	0.85 [0.69, 1.06]
JACC - Iso 2007 - tomatoes - F	56,947	379	18.3%	1.07 [0.82, 1.41]
Nurses' Health Study - Lin 2007	66,630	324	16.0%	0.90 [0.67, 1.20]
Migrant Study - Hjartaker 2015	9,964	2,386	36.0%	0.92 [0.76, 1.12]
Total (95% CI)	**175,088**	**3,657**	**100.0%**	**0.92 [0.82, 1.04]**

Heterogeneity: Chi² = 1.72, df = 3 (P = 0.63); I² = 0%

Test for overall effect: Z = 1.35 (P = 0.18)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - males - tomatoes	41,547	568	29.7%	0.85 [0.69, 1.06]
Nurses' Health Study - Lin 2007	66,630	324	16.0%	0.90 [0.67, 1.20]
JACC - Iso 2007 - tomatoes - F	56,947	379	18.3%	1.07 [0.82, 1.41]
Migrant Study - Hjartaker 2015	9,964	2,386	36.0%	0.92 [0.76, 1.12]
Total (95% CI) [Random Effects]	**175,088**	**3,657**	**100.0%**	**0.92 [0.82, 1.04]**

Heterogeneity: Tau² = 0.00; Chi² = 1.72, df = 3 (P = 0.63); I² = 0%

Test for overall effect: Z = 1.35 (P = 0.18)

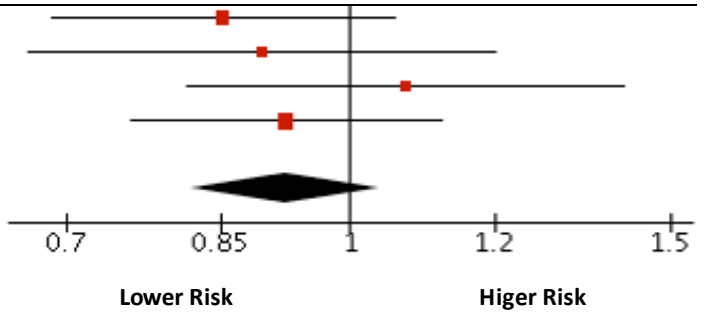
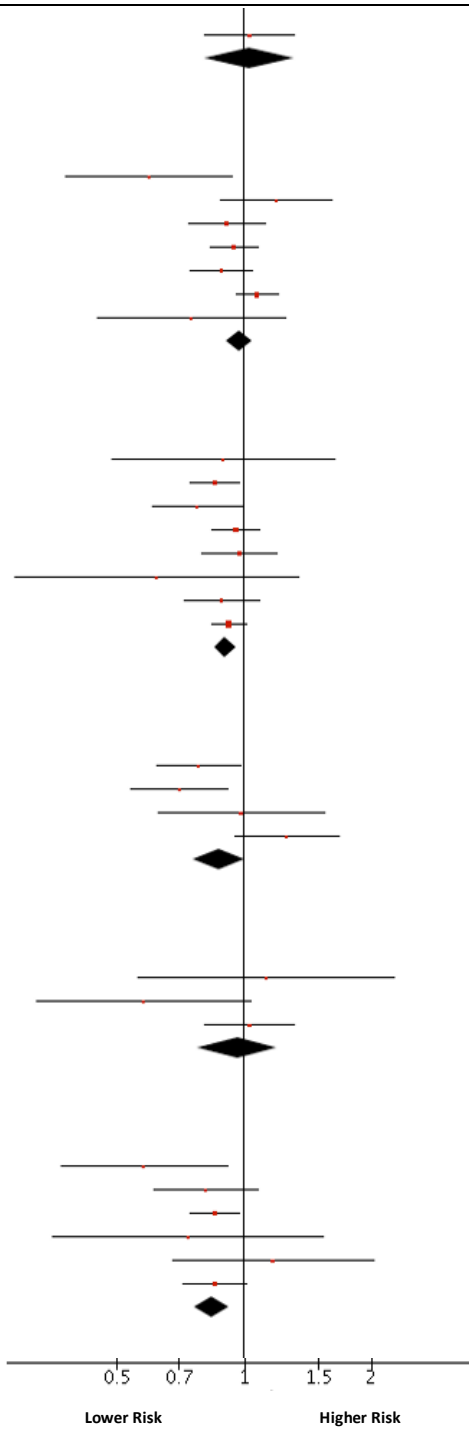


Figure S87. Relation between intake of tomatoes and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Bananas				
Migrant Study - Hjartaker 2015	9,964	2,384	2.3%	1.04 [0.81, 1.34]
Subtotal (95% CI)	**9,964**	**2,384**	**2.3%**	**1.04 [0.81, 1.34]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.31 (P = 0.76)				
Berries				
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	0.7%	0.59 [0.37, 0.94]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	1.5%	1.21 [0.88, 1.65]
ATBC - Hirvonen 2001	25,373	815	3.3%	0.91 [0.74, 1.13]
Iowa WHS - Mink 2007 - strawberries	-	-	8.1%	0.95 [0.83, 1.09]
Iowa WHS - Mink 2007 - blueberries	34,492	1,329	4.9%	0.89 [0.74, 1.06]
Migrant Study - Hjartaker 2015	9,964	2,386	11.0%	1.08 [0.96, 1.22]
UK Women's Cohort - Lai 2015	30,458	138	0.5%	0.75 [0.44, 1.27]
Subtotal (95% CI)	**105,420**	**5,141**	**29.9%**	**0.98 [0.91, 1.05]**
Heterogeneity: Tau² = 0.01; Chi² = 11.84, df = 6 (P = 0.07); I² = 49%				
Test for overall effect: Z = 0.82 (P = 0.41)				
Citrus				
Nutrition Status Study - Sahyoun 1996	680	101	0.4%	0.90 [0.48, 1.68]
Iowa WHS - Mink 2007 - grapefruit	34,492	1,329	8.1%	0.85 [0.74, 0.98]
JACC - Iso 2007 - F	59,504	398	2.3%	0.77 [0.60, 0.99]
Iowa WHS - Mink 2007 - oranges	-	-	8.1%	0.96 [0.84, 1.10]
JACC - Iso 2007 - M	43,031	602	3.3%	0.98 [0.79, 1.22]
UK Women's Cohort - Lai 2015	30,458	138	0.2%	0.61 [0.27, 1.37]
Migrant Study - Hjartaker 2015	9,964	2,386	3.3%	0.89 [0.71, 1.10]
Linxian Nutrition - Wang 2016	2,445	355	15.8%	0.92 [0.84, 1.02]
Subtotal (95% CI)	**180,574**	**5,309**	**41.4%**	**0.91 [0.85, 0.96]**
Heterogeneity: Tau² = 0.00; Chi² = 4.61, df = 7 (P = 0.71); I² = 0%				
Test for overall effect: Z = 3.20 (P = 0.001)				
Fruit Juice				
JACC - Iso 2007 - M	41,330	573	2.7%	0.78 [0.62, 0.99]
JACC - Iso 2007 - F	56,482	370	2.0%	0.70 [0.53, 0.92]
UK Women's Cohort - Lai 2015	30,458	138	0.7%	0.99 [0.62, 1.58]
REGARDS - Colin 2019	13,440	168	1.8%	1.28 [0.96, 1.72]
Subtotal (95% CI)	**141,710**	**1,249**	**7.2%**	**0.87 [0.75, 1.01]**
Heterogeneity: Tau² = 0.06; Chi² = 10.36, df = 3 (P = 0.02); I² = 71%				
Test for overall effect: Z = 0.74 (P = 0.46)				
Grapes				
Nurses' Health Study - Lin 2007	66,360	324	0.3%	1.14 [0.55, 2.35]
UK Women's Cohort - Lai 2015 - grapes	30,458	138	0.4%	0.57 [0.31, 1.05]
Migrant Study - Hjartaker 2015	9,964	2,384	2.3%	1.04 [0.81, 1.34]
Subtotal (95% CI)	**106,782**	**2,846**	**3.0%**	**0.97 [0.77, 1.21]**
Heterogeneity: Tau² = 0.05; Chi² = 3.40, df = 2 (P = 0.18); I² = 41%				
Test for overall effect: Z = 0.49 (P = 0.63)				
Pommes				
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	0.7%	0.57 [0.36, 0.91]
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	1.8%	0.81 [0.60, 1.09]
Iowa WHS - Mink 2007	34,492	1,329	8.1%	0.85 [0.74, 0.98]
Nurses' Health Study - Lin 2007	66,360	324	0.3%	0.73 [0.34, 1.58]
UK Women's Cohort - Lai 2015	30,458	138	0.5%	1.19 [0.67, 2.09]
Migrant Study - Hjartaker 2015	9,964	2,386	4.9%	0.85 [0.71, 1.02]
Subtotal (95% CI)	**146,407**	**4,650**	**16.1%**	**0.84 [0.76, 0.92]**
Heterogeneity: Tau² = 0.00; Chi² = 4.24, df = 5 (P = 0.52); I² = 0%				
Test for overall effect: Z = 3.54 (P = 0.0004)				
Test for subgroup differences: Chi² = 8.23, df = 5 (P = 0.14), I² = 39.3%				



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B. Random Effects

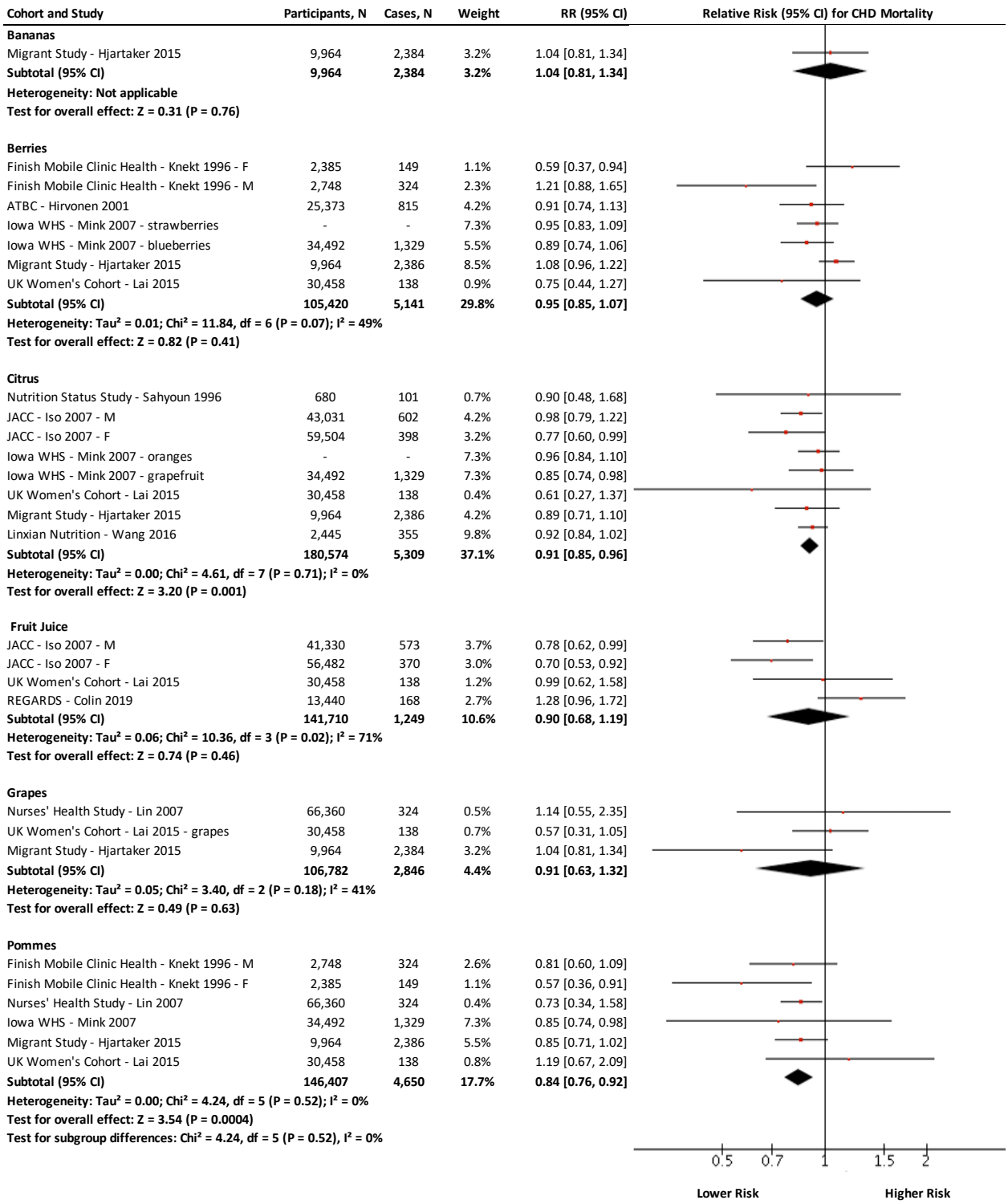
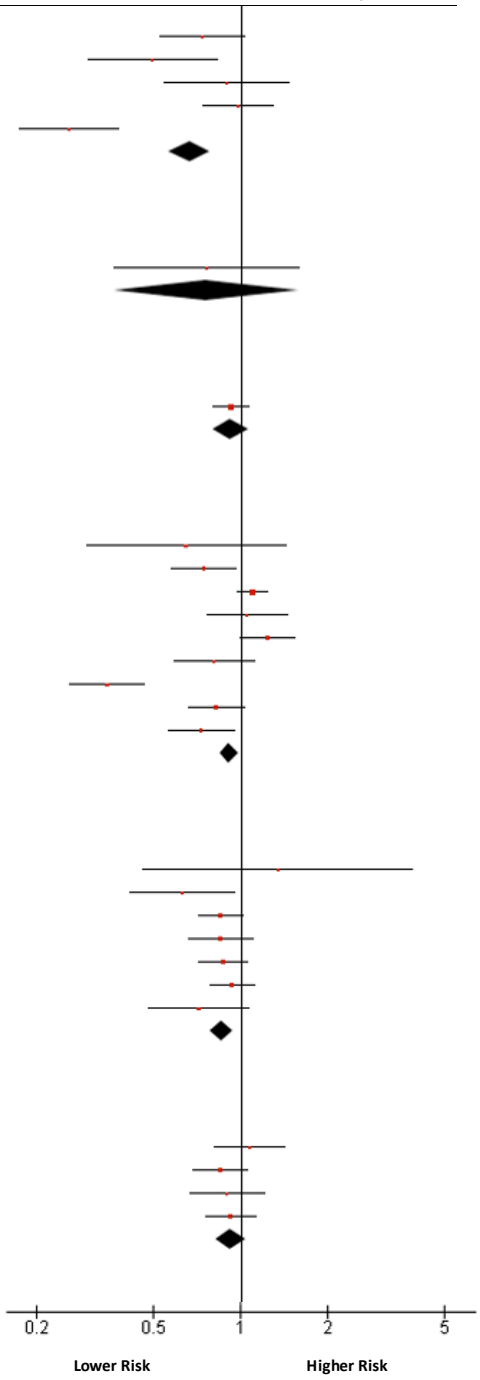


Figure S88. Relation between sources of fruit and CHD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	1.9%	0.74 [0.53, 1.03]
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	0.8%	0.50 [0.30, 0.83]
Nurses' Health Study - Lin 2007	66,630	324	0.9%	0.90 [0.55, 1.46]
Linxian Nutrition - Wang 2016	2,445	355	2.9%	0.98 [0.74, 1.29]
PLSAW - Blekkenhorst 2017	1,226	128	1.4%	0.26 [0.18, 0.38]
Subtotal (95% CI)	**75,434**	**1,280**	**8.0%**	**0.67 [0.57, 0.79]**
Heterogeneity: Chi² = 32.86, df = 4 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 4.80 (P < 0.00001)				
Carrots				
Oxford Vegetarian - Mann 1997 - carrots	10,802	64	0.4%	0.76 [0.37, 1.58]
Subtotal (95% CI)	**10,802**	**64**	**0.4%**	**0.76 [0.37, 1.58]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.73 (P = 0.47)				
Celery				
Iowa WHS - Mink 2007 - celery	34,492	1,329	11.5%	0.92 [0.80, 1.06]
Subtotal (95% CI)	**34,492**	**1,329**	**11.5%**	**0.92 [0.80, 1.06]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 1.14 (P = 0.25)				
Cruciferous				
Nurses' Health Study - Lin 2007	66,630	324	0.4%	0.65 [0.30, 1.42]
JACC - Iso 2007 - M	39,486	534	3.3%	0.75 [0.58, 0.97]
Iowa WHS - Mink 2007	34,492	1,329	15.6%	1.09 [0.97, 1.23]
JACC - Iso 2007 - F	54,325	396	2.2%	1.05 [0.77, 1.44]
Migrant Study - Hjartaker 2015	9,964	2,386	4.6%	1.23 [0.99, 1.53]
Linxian Nutrition - Wang 2016	2,445	355	2.2%	0.81 [0.59, 1.11]
PLSAW - Blekkenhorst 2017	1,226	128	2.5%	0.35 [0.26, 0.47]
Japan Public Health Center - Mori 2018 - M	40,642	1,192	4.6%	0.83 [0.67, 1.03]
Japan Public Health Center - Mori 2018 - F	47,562	776	3.3%	0.73 [0.57, 0.95]
Subtotal (95% CI)	**296,772**	**7,420**	**38.8%**	**0.91 [0.85, 0.98]**
Heterogeneity: Chi² = 65.50, df = 8 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 2.45 (P = 0.01)				
Green leafy				
Oxford Vegetarian - Mann 1997	10,802	64	0.2%	1.34 [0.46, 3.85]
OXCHECK - Whiteman 1999	10,522	144	1.3%	0.63 [0.42, 0.95]
Health Food Shoppers - Appleby 2002	10,741	605	6.9%	0.85 [0.71, 1.02]
JACC - Iso 2007 - F	59,809	420	3.3%	0.85 [0.66, 1.10]
JACC - Iso 2007 - M	43,850	617	5.6%	0.87 [0.71, 1.06]
Migrant Study - Hjartaker 2015	9,964	2,386	6.9%	0.93 [0.78, 1.11]
Linxian Nutrition - Wang 2016	2,445	355	1.4%	0.72 [0.49, 1.06]
Subtotal (95% CI)	**148,133**	**4,591**	**25.7%**	**0.86 [0.78, 0.94]**
Heterogeneity: Chi² = 4.47, df = 6 (P = 0.61); I² = 0%				
Test for overall effect: Z = 3.25 (P = 0.001)				
Tomatoes				
JACC - Iso 2007 - tomatoes - F	56,947	379	2.9%	1.07 [0.82, 1.41]
JACC - Iso 2007 - tomatoes - M	41,547	568	4.6%	0.85 [0.69, 1.06]
Nurses' Health Study - Lin 2007	66,630	324	2.5%	0.90 [0.67, 1.20]
Migrant Study - Hjartaker 2015	9,964	2,386	5.6%	0.92 [0.76, 1.12]
Subtotal (95% CI)	**175,088**	**3,657**	**15.6%**	**0.92 [0.82, 1.04]**
Heterogeneity: Chi² = 1.72, df = 3 (P = 0.63); I² = 0%				
Test for overall effect: Z = 1.35 (P = 0.18)				
Test for subgroup differences: Chi² = 13.08, df = 5 (P = 0.02), I² = 61.8%				



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
Finish Mobile Clinic Health - Knekt 1996 - M	2,748	324	3.6%	0.74 [0.53, 1.03]
Finish Mobile Clinic Health - Knekt 1996 - F	2,385	149	2.5%	0.50 [0.30, 0.83]
Nurses' Health Study - Lin 2007	66,630	324	2.6%	0.90 [0.55, 1.46]
Linxian Nutrition - Wang 2016	2,445	355	4.0%	0.98 [0.74, 1.29]
PLSAW - Blekkenhorst 2017	1,226	128	3.2%	0.26 [0.18, 0.38]
Subtotal (95% CI)	**75,434**	**1,280**	**15.8%**	**0.61 [0.38, 1.00]**
Heterogeneity: Tau² = 0.27; Chi² = 32.86, df = 4 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 1.95 (P = 0.05)				
Carrots				
Oxford Vegetarian - Mann 1997 - carrots	10,802	64	1.6%	0.76 [0.37, 1.58]
Subtotal (95% CI)	**10,802**	**64**	**1.6%**	**0.76 [0.37, 1.58]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.73 (P = 0.47)				
Celery				
Iowa WHS - Mink 2007 - celery	34,492	1,329	5.0%	0.92 [0.80, 1.06]
Subtotal (95% CI)	**34,492**	**1,329**	**5.0%**	**0.92 [0.80, 1.06]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 1.14 (P = 0.25)				
Cruciferous				
JACC - Iso 2007 - M	39,486	534	4.2%	0.75 [0.58, 0.97]
Iowa WHS - Mink 2007	34,492	1,329	5.1%	1.09 [0.97, 1.23]
JACC - Iso 2007 - F	54,325	396	3.7%	1.05 [0.77, 1.44]
Nurses' Health Study - Lin 2007	66,630	324	1.4%	0.65 [0.30, 1.42]
Migrant Study - Hjartaker 2015	9,964	2,386	4.5%	1.23 [0.99, 1.53]
Linxian Nutrition - Wang 2016	2,445	355	3.7%	0.81 [0.59, 1.11]
PLSAW - Blekkenhorst 2017	1,226	128	3.9%	0.35 [0.26, 0.47]
Japan Public Health Center - Mori 2018 - F	47,562	776	4.2%	0.73 [0.57, 0.95]
Japan Public Health Center - Mori 2018 - M	40,642	1,192	4.5%	0.83 [0.67, 1.03]
Subtotal (95% CI)	**296,772**	**7,420**	**35.2%**	**0.81 [0.64, 1.02]**
Heterogeneity: Tau² = 0.11; Chi² = 65.50, df = 8 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 1.79 (P = 0.07)				
Green leafy				
Oxford Vegetarian - Mann 1997	10,802	64	0.9%	1.34 [0.46, 3.85]
OXCHECK - Whiteman 1999	10,522	144	3.0%	0.63 [0.42, 0.95]
Health Food Shoppers - Appleby 2002	10,741	605	4.8%	0.85 [0.71, 1.02]
JACC - Iso 2007 - F	59,809	420	4.2%	0.85 [0.66, 1.10]
JACC - Iso 2007 - M	43,850	617	4.6%	0.87 [0.71, 1.06]
Migrant Study - Hjartaker 2015	9,964	2,386	4.8%	0.93 [0.78, 1.11]
Linxian Nutrition - Wang 2016	2,445	355	3.2%	0.72 [0.49, 1.06]
Subtotal (95% CI)	**148,133**	**4,591**	**25.4%**	**0.86 [0.78, 0.94]**
Heterogeneity: Tau² = 0.00; Chi² = 4.47, df = 6 (P = 0.61); I² = 0%				
Test for overall effect: Z = 3.25 (P = 0.001)				
Tomatoes				
Nurses' Health Study - Lin 2007	66,630	324	3.9%	0.90 [0.67, 1.20]
JACC - Iso 2007 - tomatoes - M	41,547	568	4.5%	0.85 [0.69, 1.06]
JACC - Iso 2007 - tomatoes - F	56,947	379	4.0%	1.07 [0.82, 1.41]
Migrant Study - Hjartaker 2015	9,964	2,386	4.6%	0.92 [0.76, 1.12]
Subtotal (95% CI)	**175,088**	**3,657**	**17.0%**	**0.92 [0.82, 1.04]**
Heterogeneity: Tau² = 0.00; Chi² = 1.72, df = 3 (P = 0.63); I² = 0%				
Test for overall effect: Z = 1.35 (P = 0.18)				
Test for subgroup differences: Chi² = 4.09, df = 5 (P = 0.54), I² = 0%				

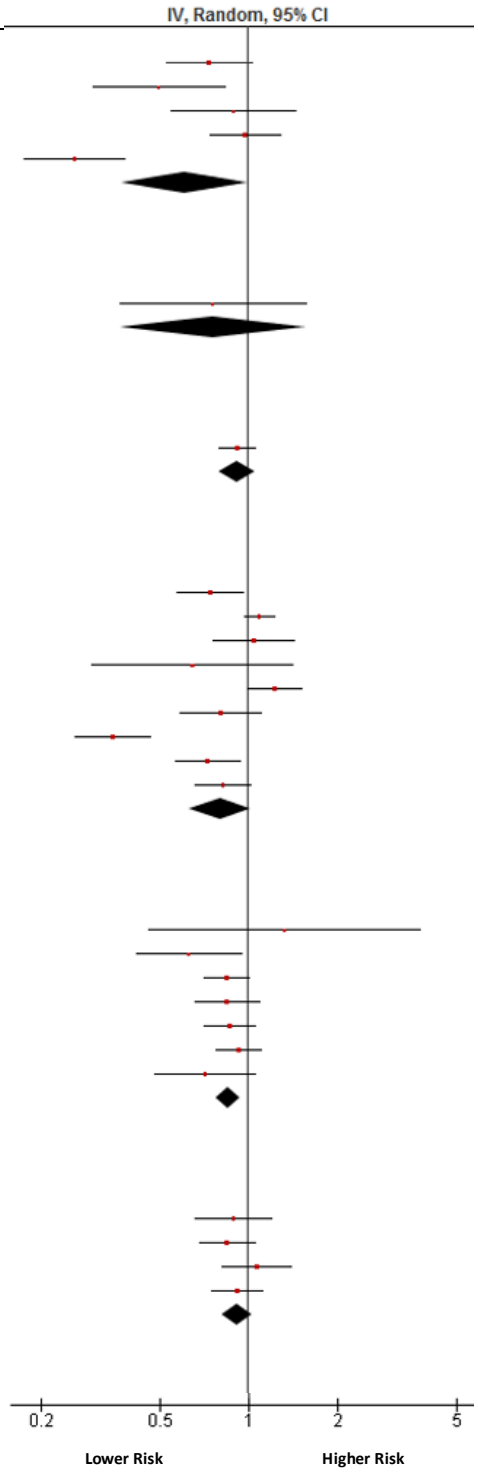


Figure S89. Relation between sources of vegetables and CHD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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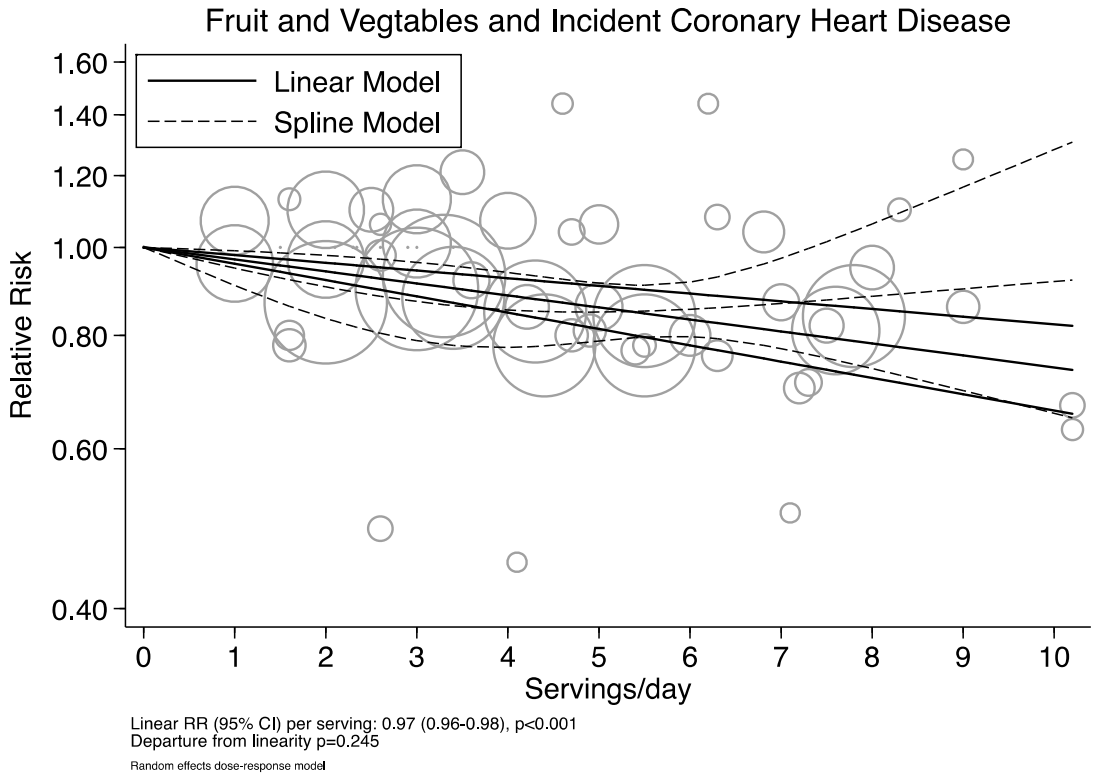


Figure S90. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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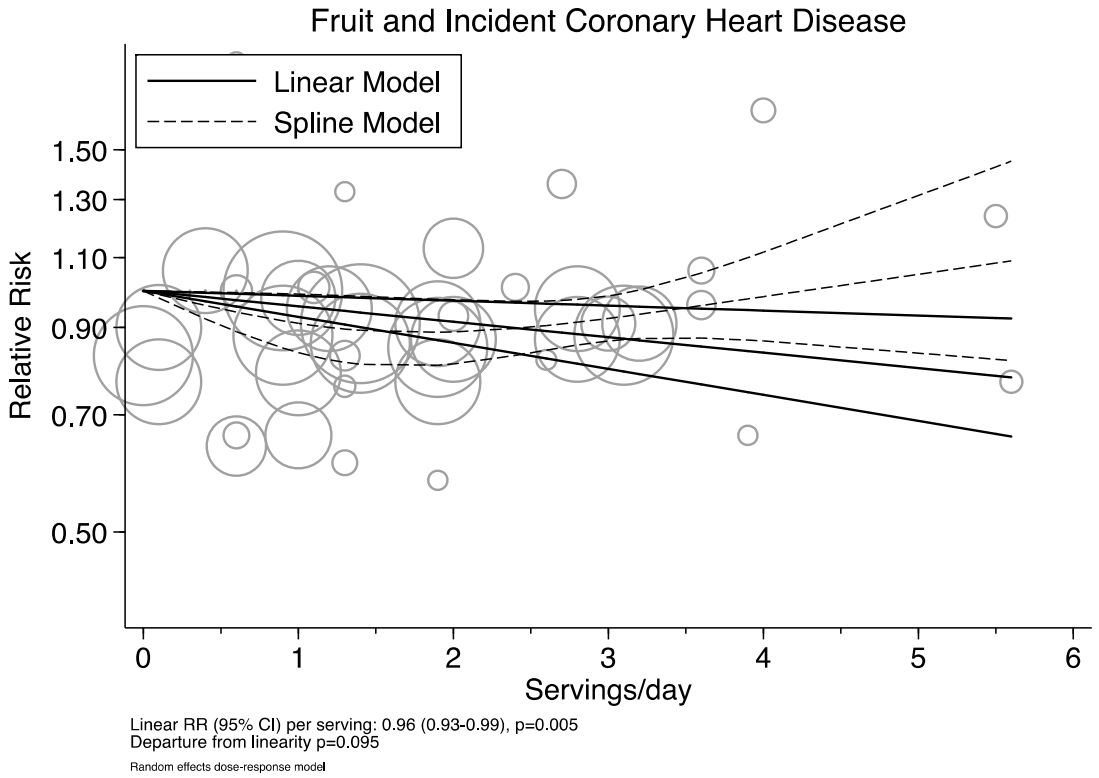


Figure S91. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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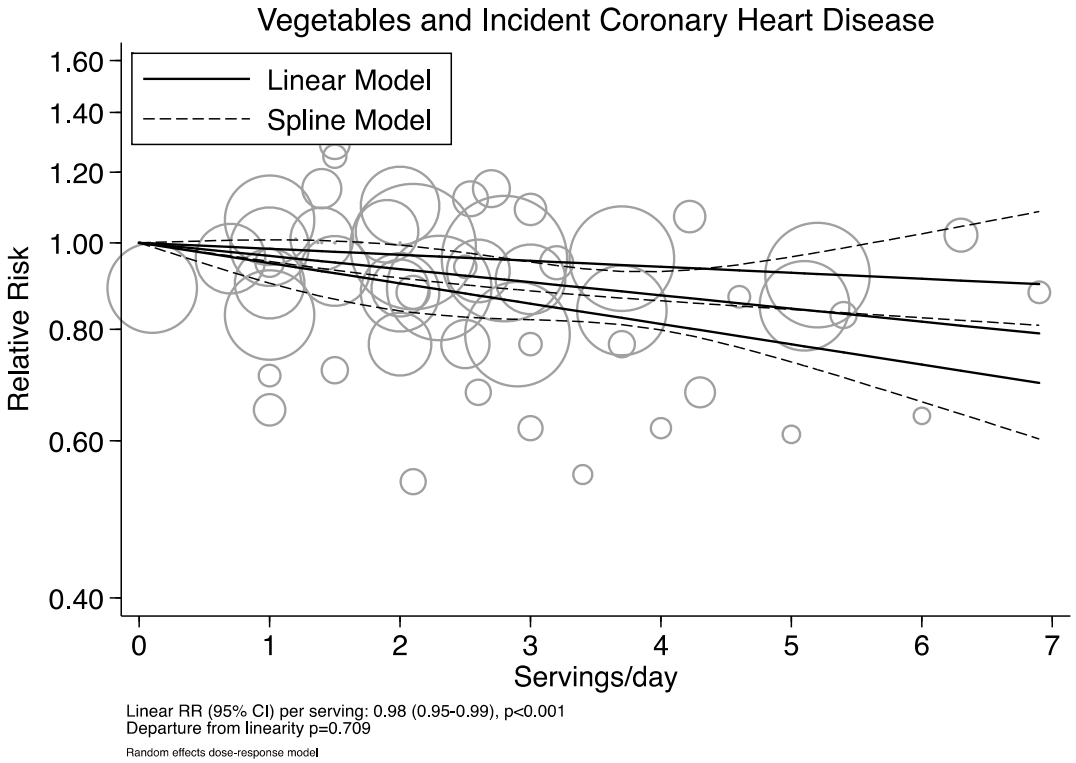


Figure S92. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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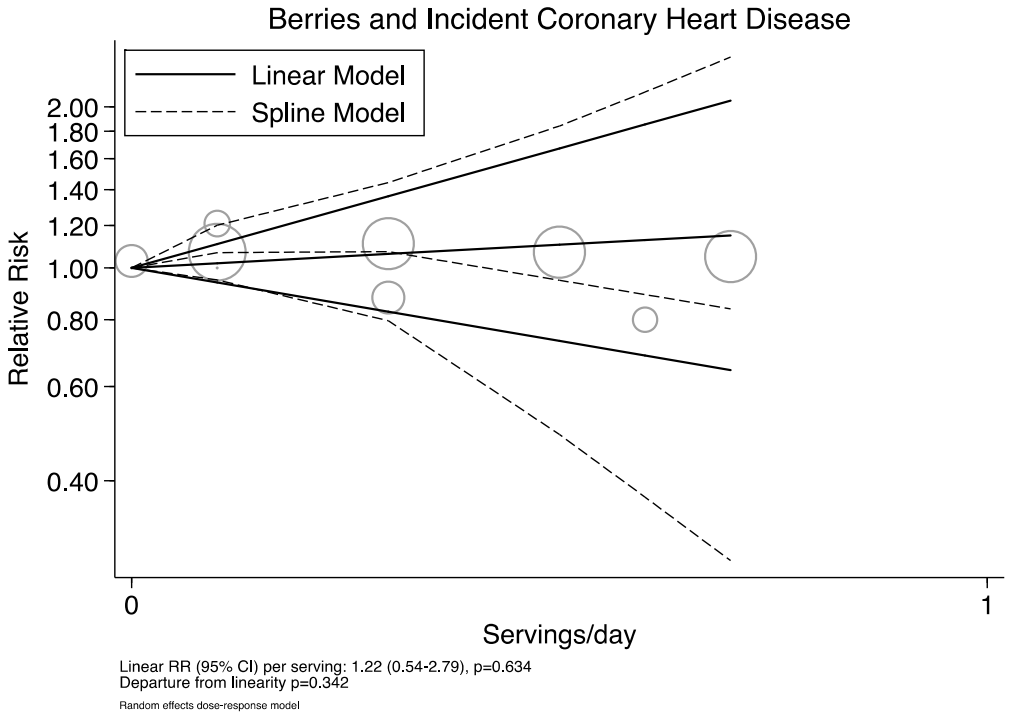


Figure S93. Linear and cubic-spline dose-response relation between increasing berries intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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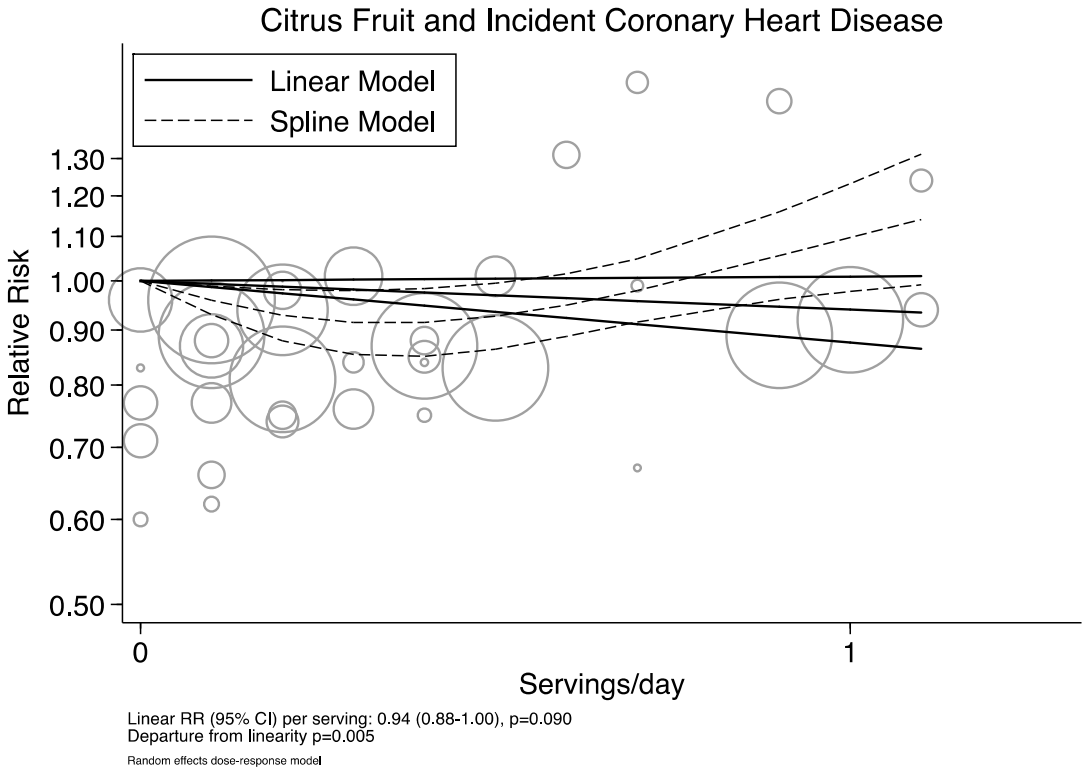


Figure S94. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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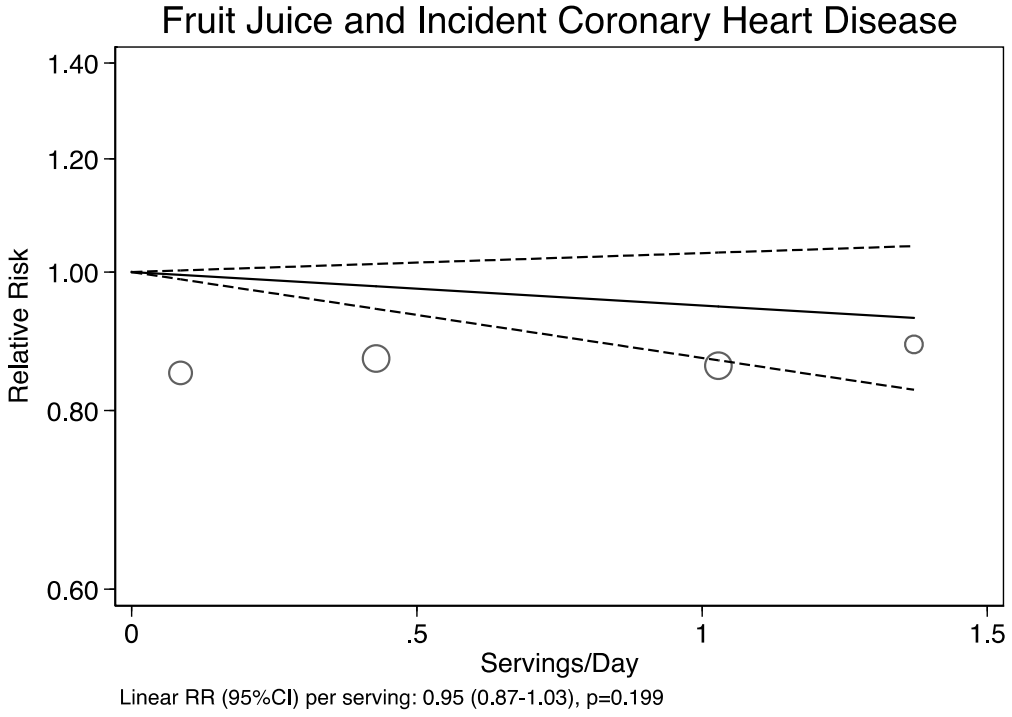


Figure S95. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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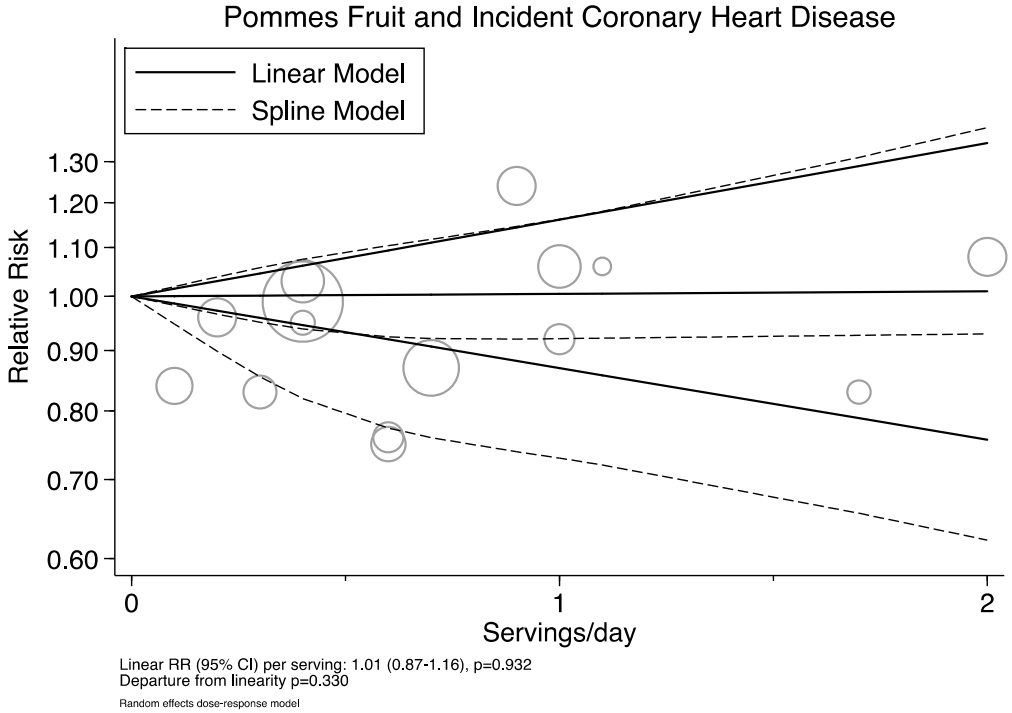


Figure S96. Linear and cubic-spline dose-response relation between increasing pommes intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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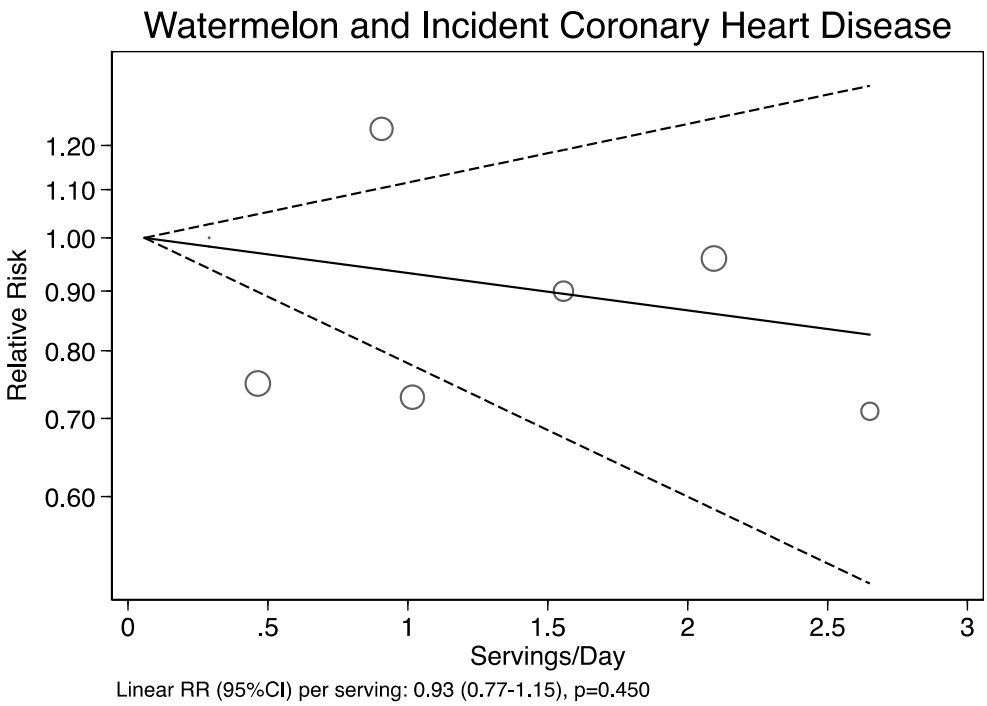


Figure S97. Linear dose-response relation between increasing watermelon intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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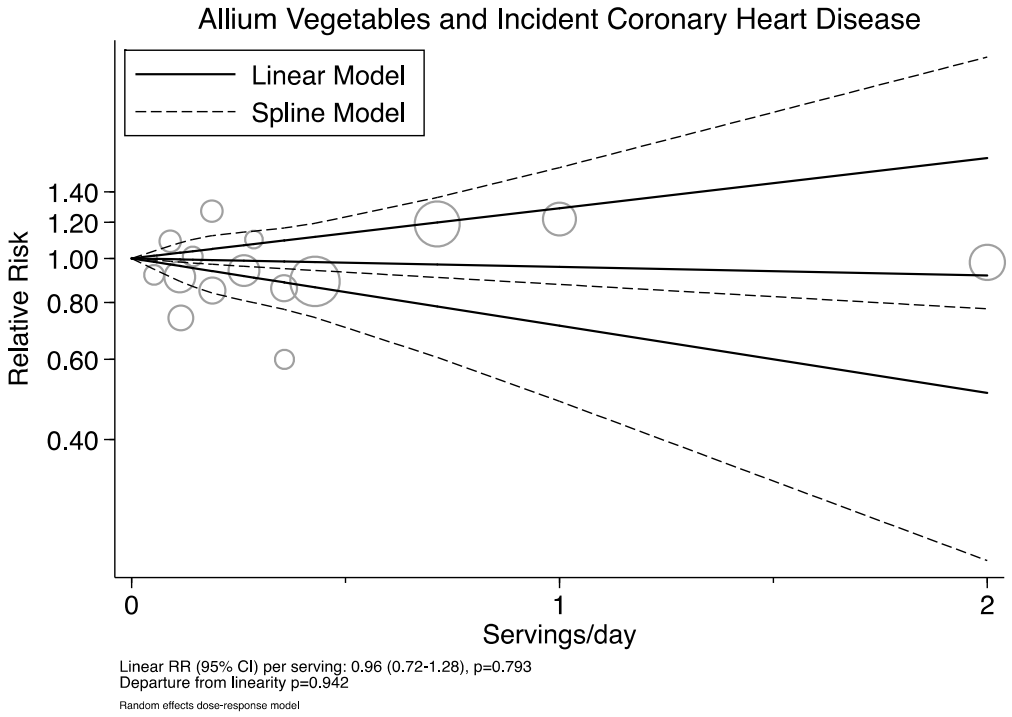


Figure S98. Linear and cubic-spline dose-response relation between increasing intake of allium vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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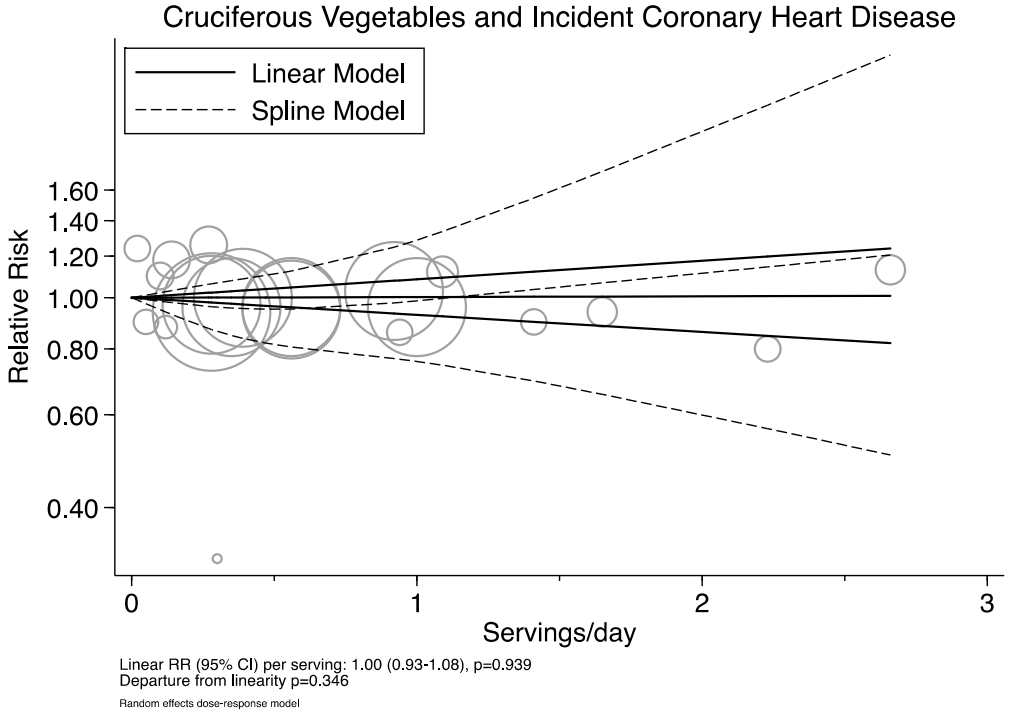


Figure S99. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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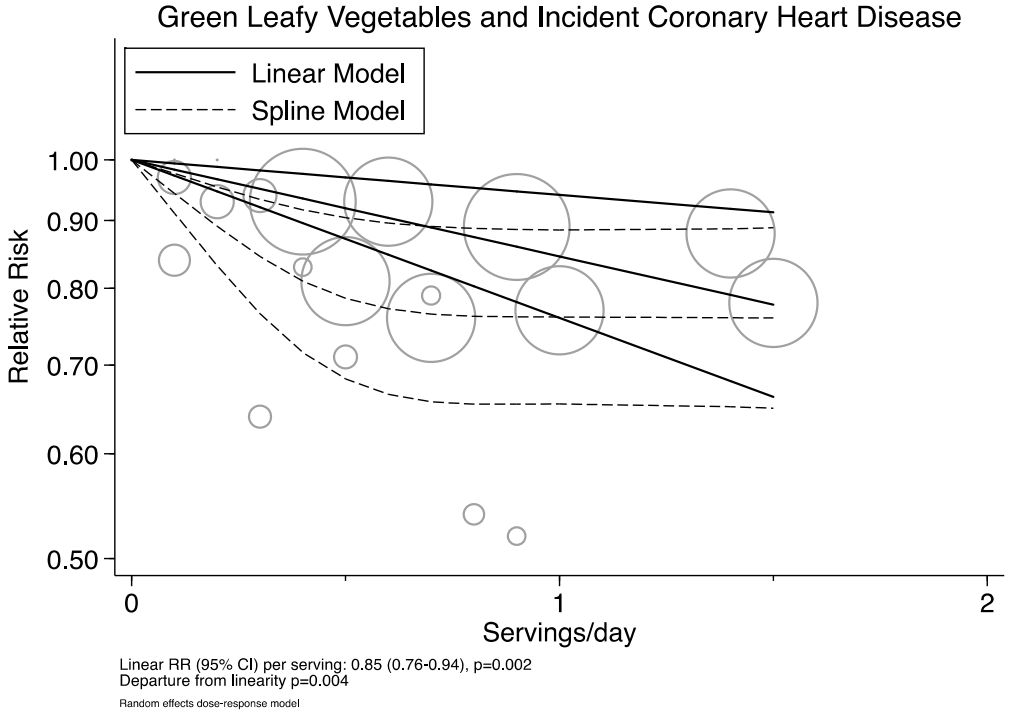


Figure S100. Linear and cubic-spline dose-response relation between increasing intake of green leafy vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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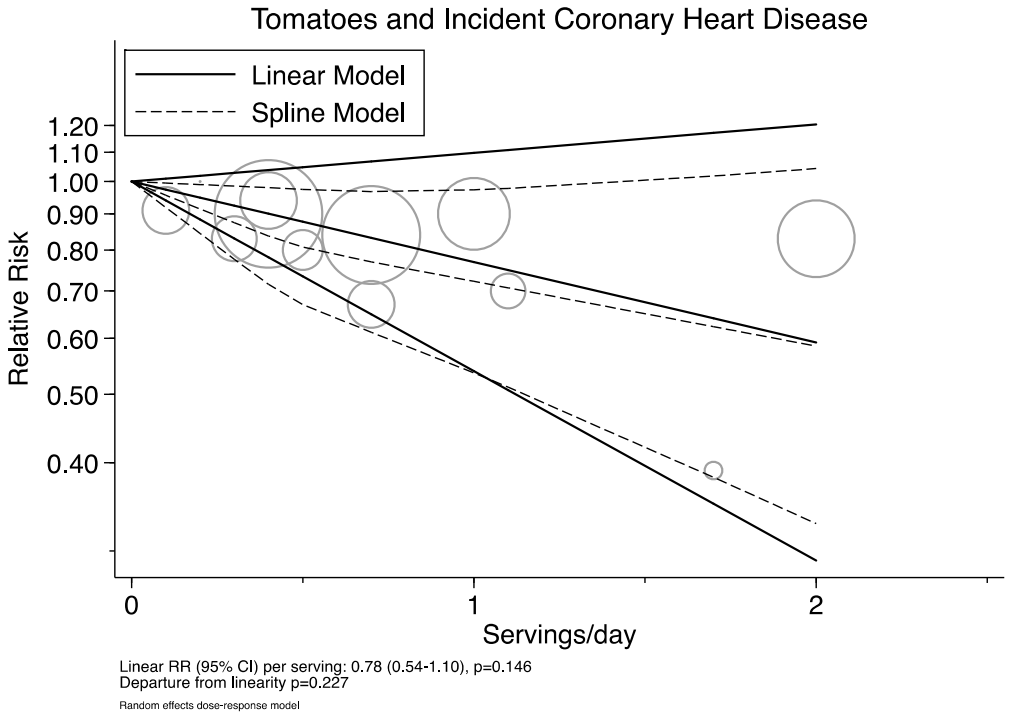


Figure S101. Linear and cubic-spline dose-response relation between increasing tomato intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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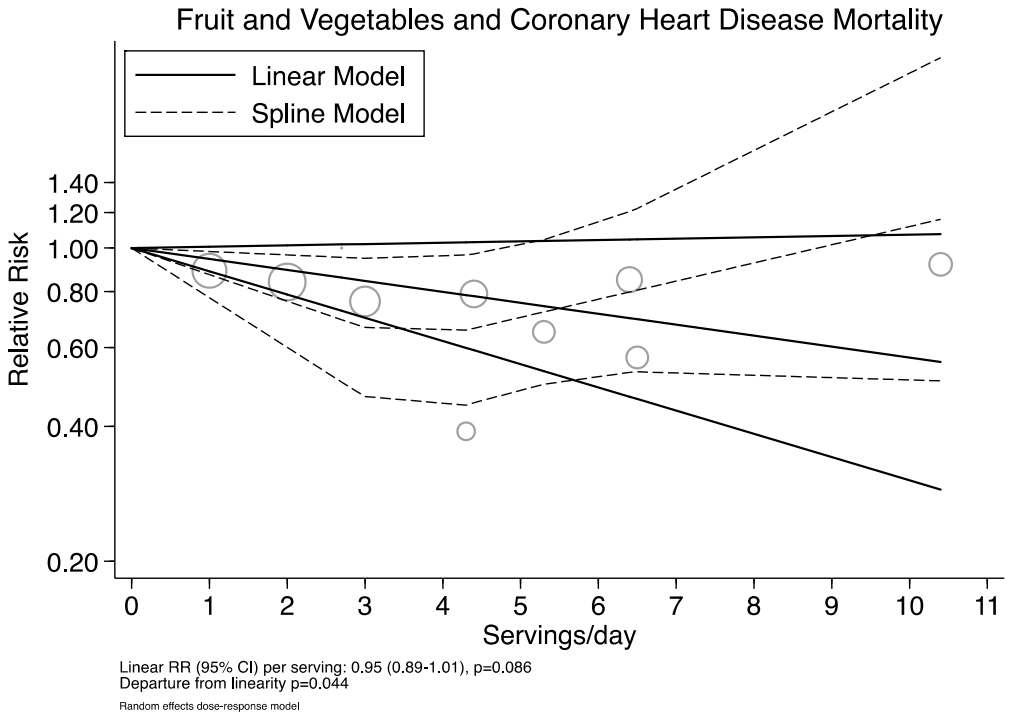


Figure S102. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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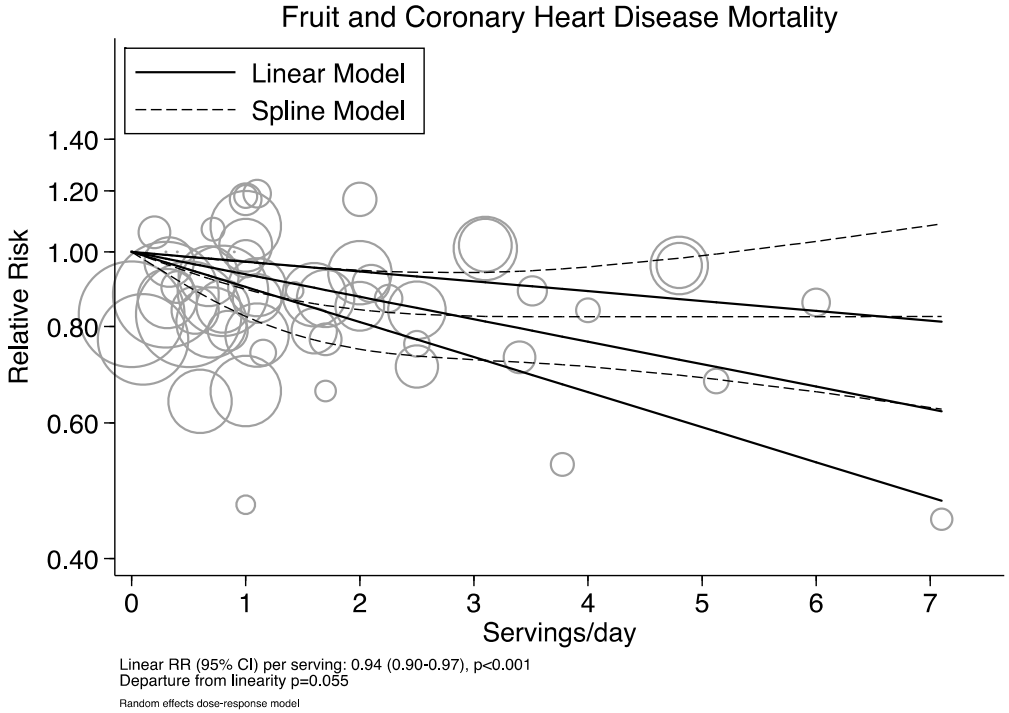


Figure S103. Linear and cubic-spline dose-response relation between increasing fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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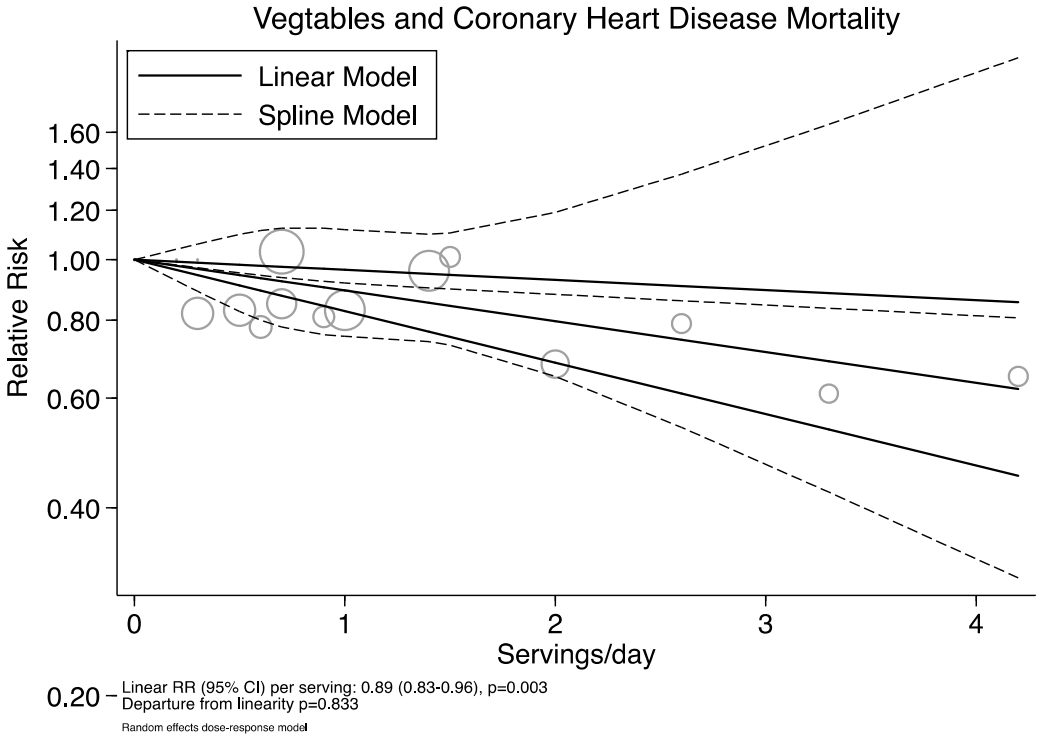


Figure S104. Linear and cubic-spline dose-response relation between increasing intake of vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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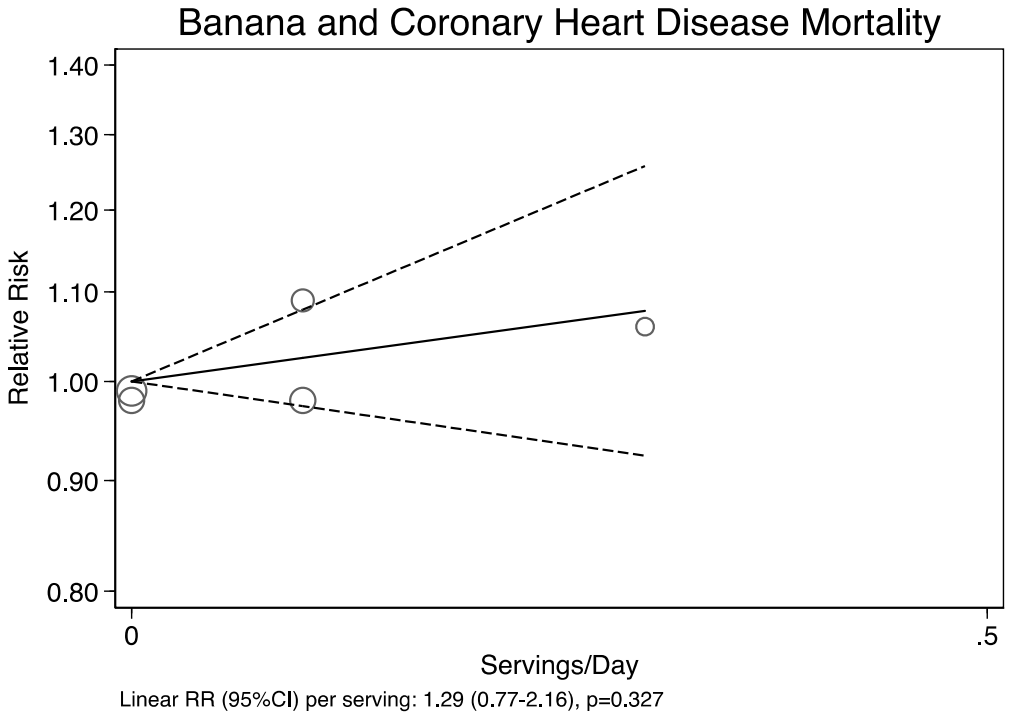


Figure S105. Linear dose-response relation between increasing banana intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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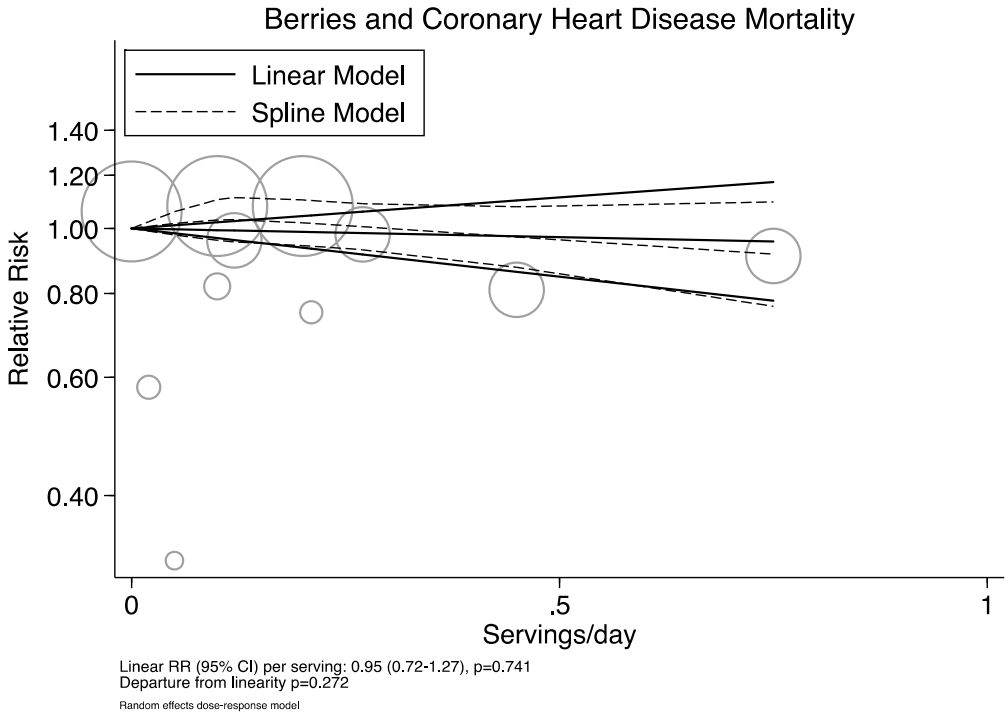


Figure S106. Linear dose-response relation between increasing berries intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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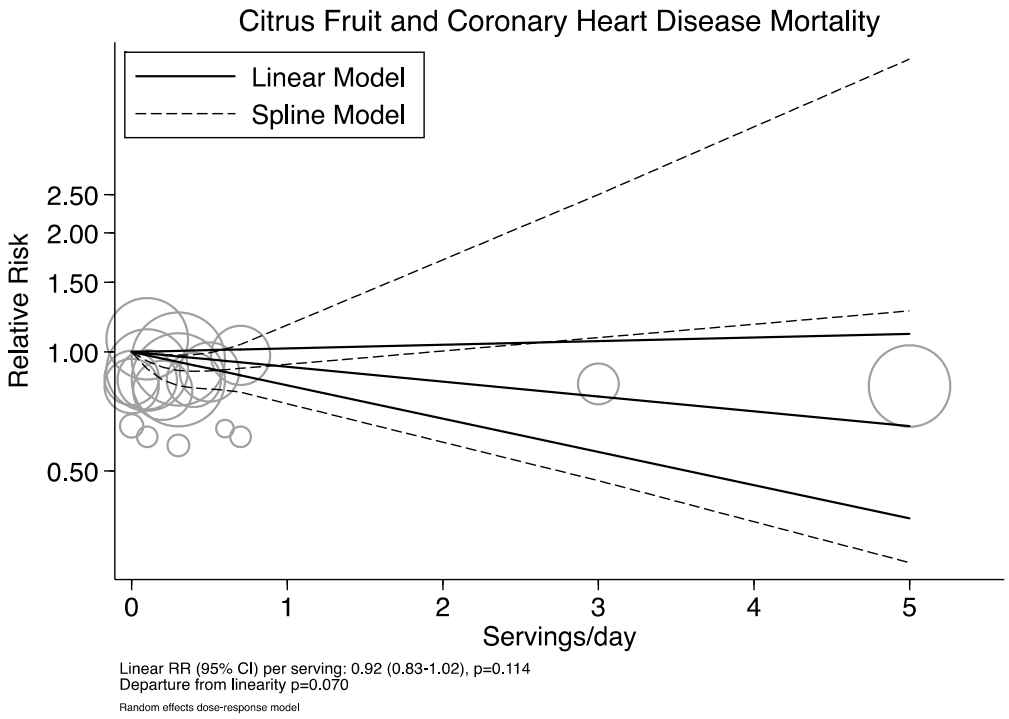


Figure S107. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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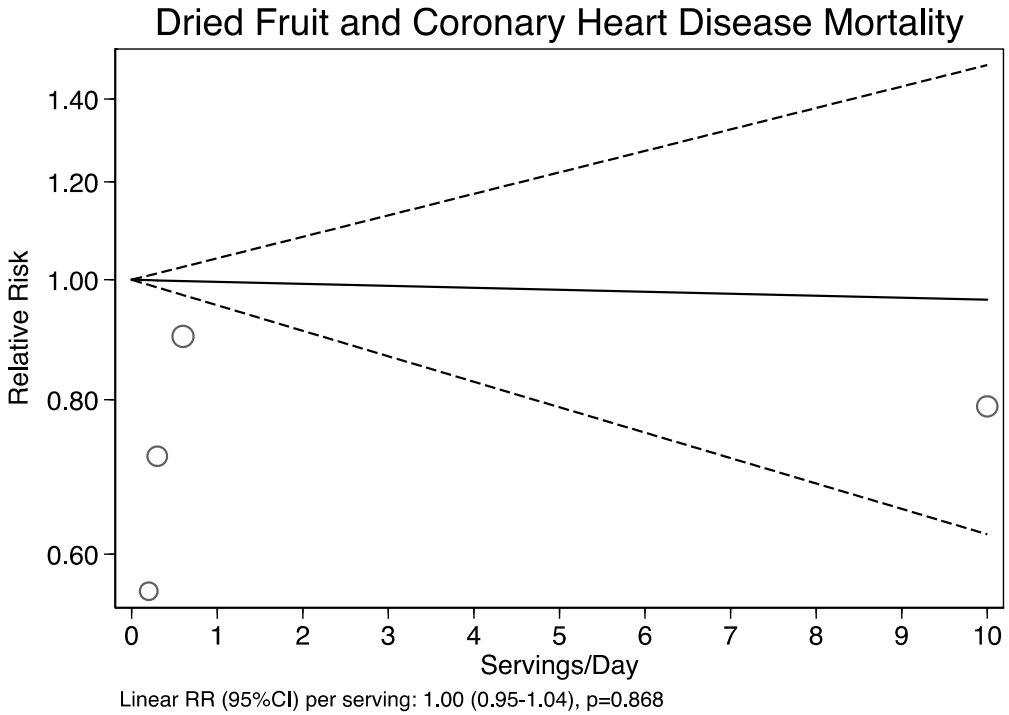


Figure S108. Linear and cubic-spline dose-response relation between increasing dried fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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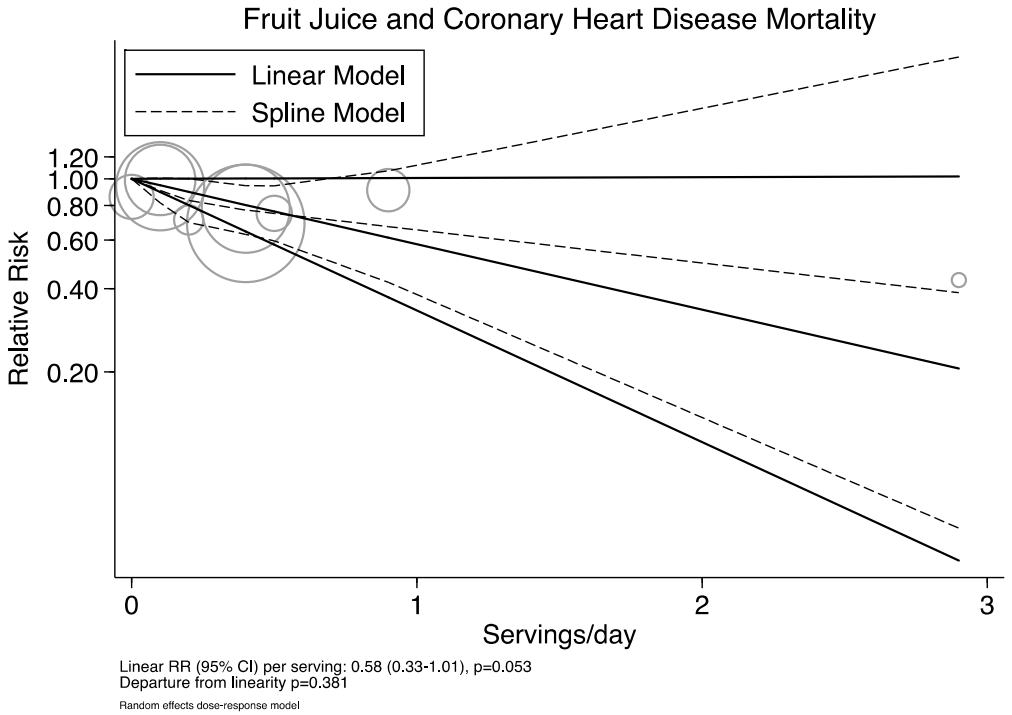


Figure S109. Linear and cubic-spline dose-response relation between increasing fruit juice intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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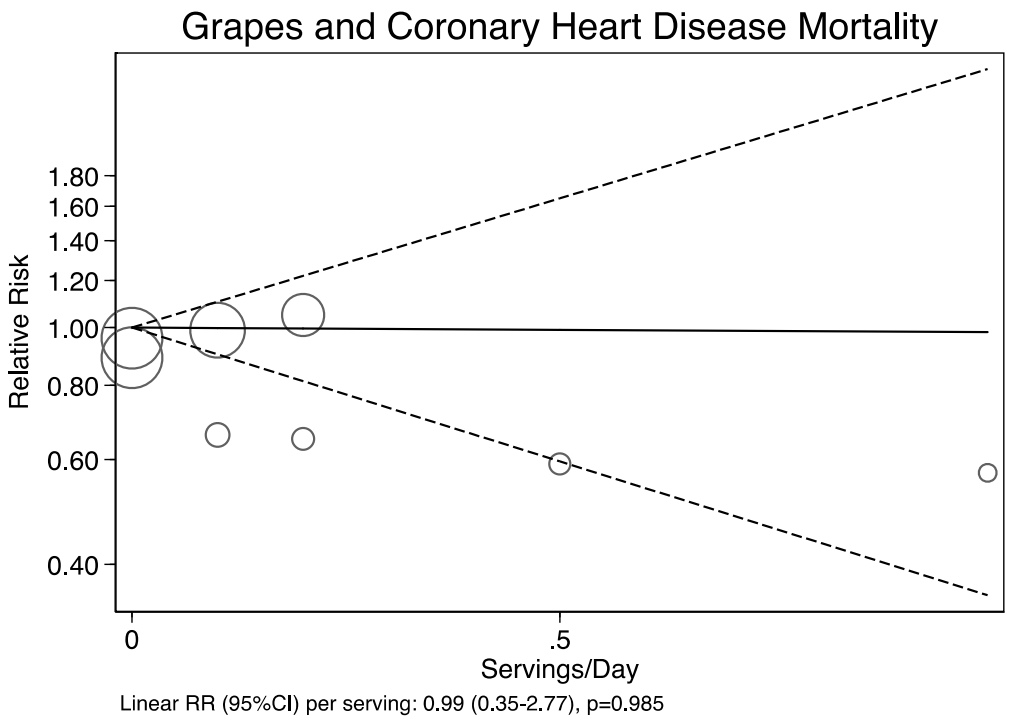


Figure S110. Linear dose-response relation between increasing grape intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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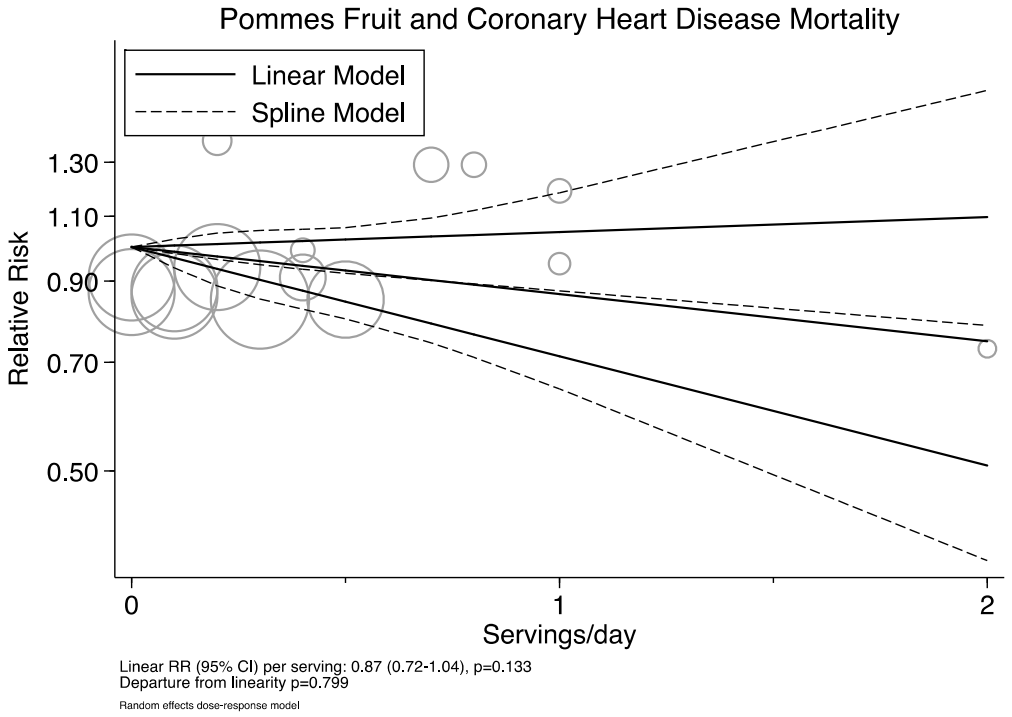


Figure S111. Linear and cubic-spline dose-response relation between increasing pommes intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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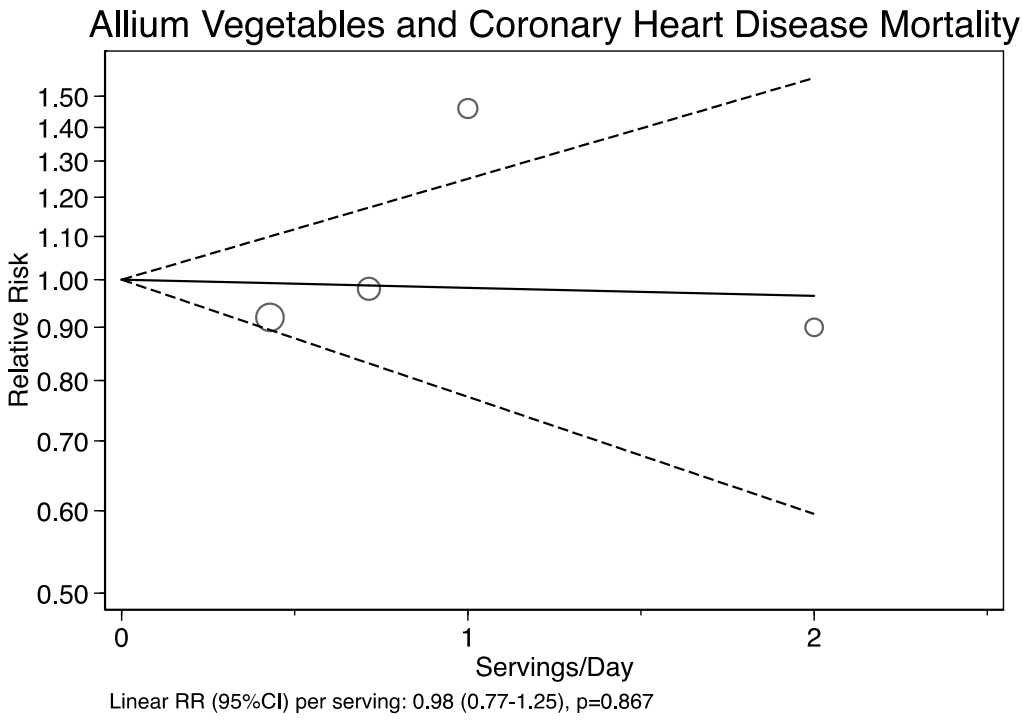


Figure S112. Linear dose-response relation between increasing intake of allium vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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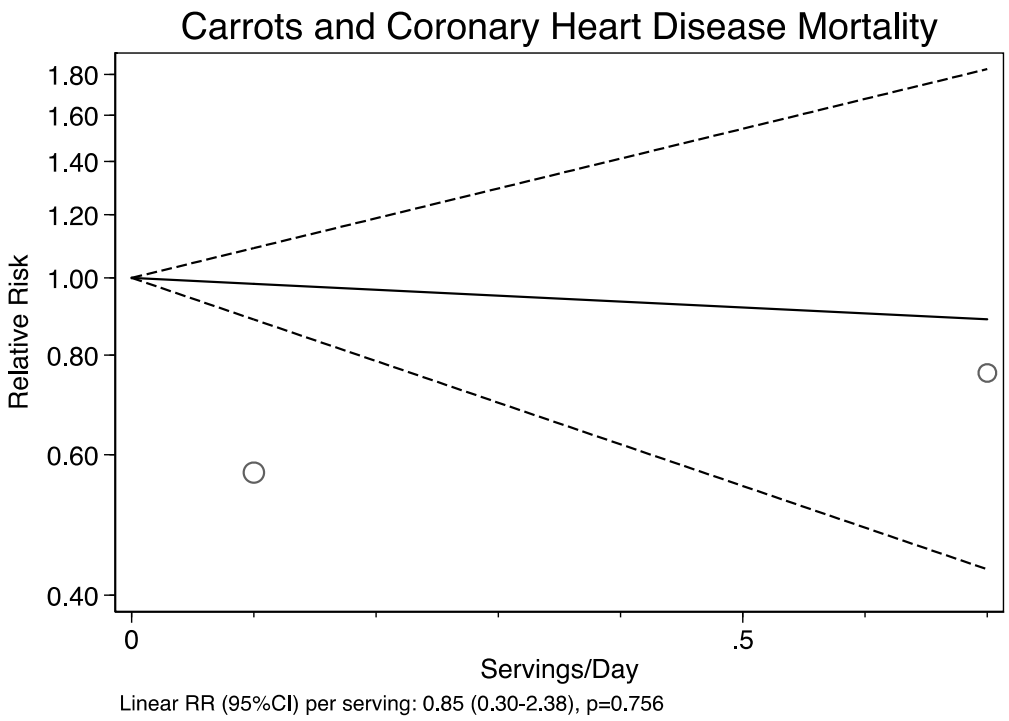


Figure S113. Linear dose-response relation between increasing intake of carrots and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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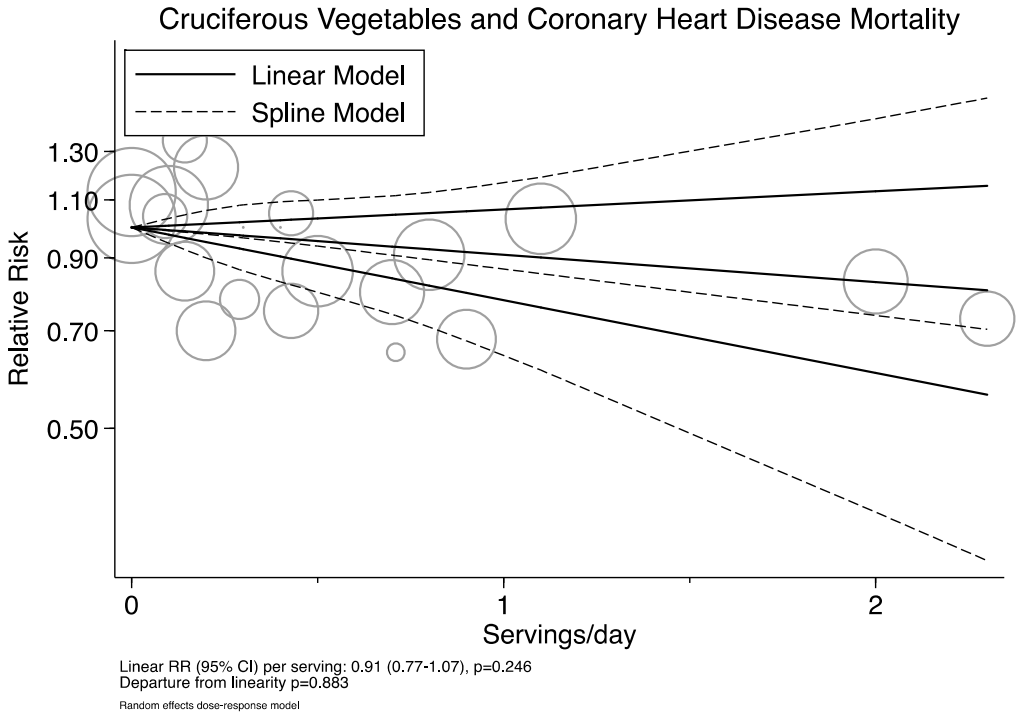


Figure S114. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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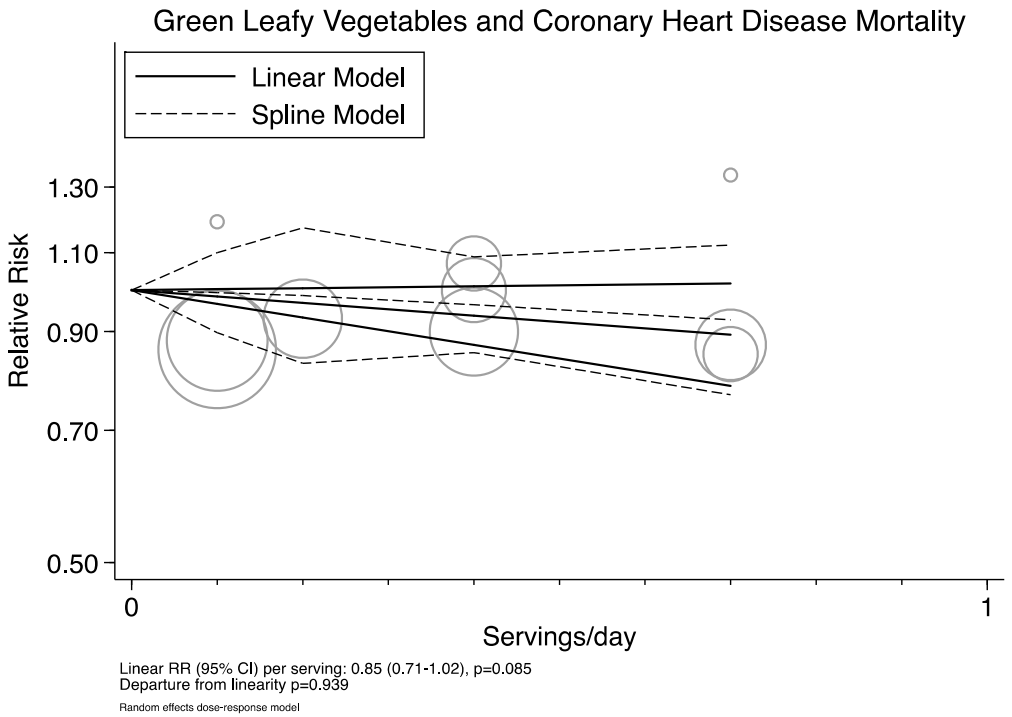


Figure S115. Linear dose-response relation between increasing intake of green leafy vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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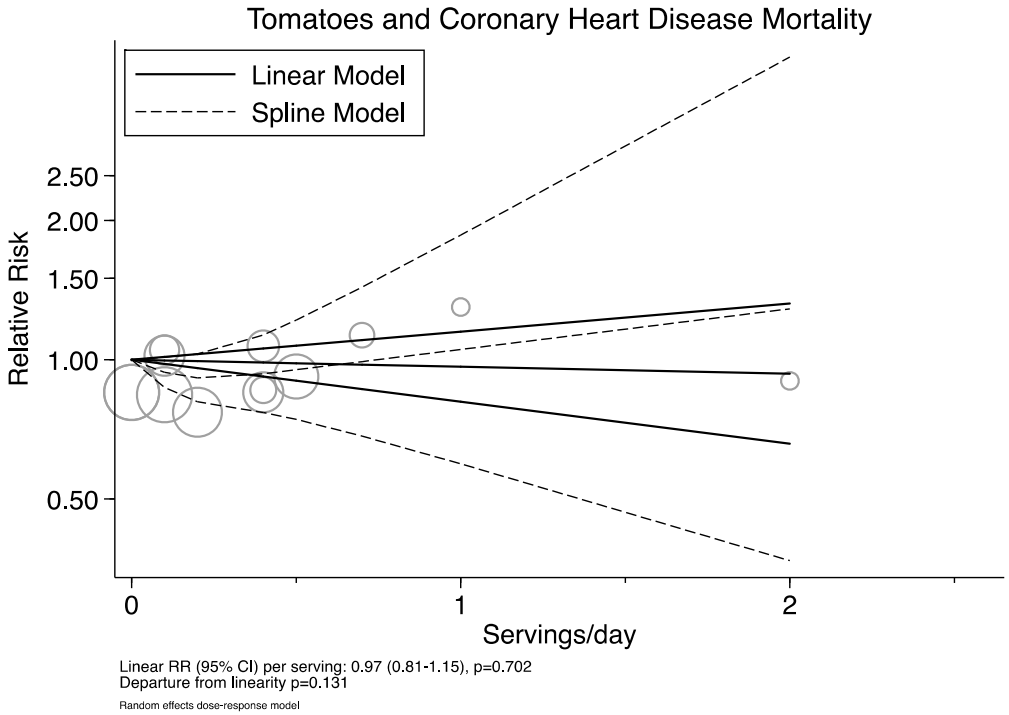


Figure S116. Linear dose-response relation between increasing tomato intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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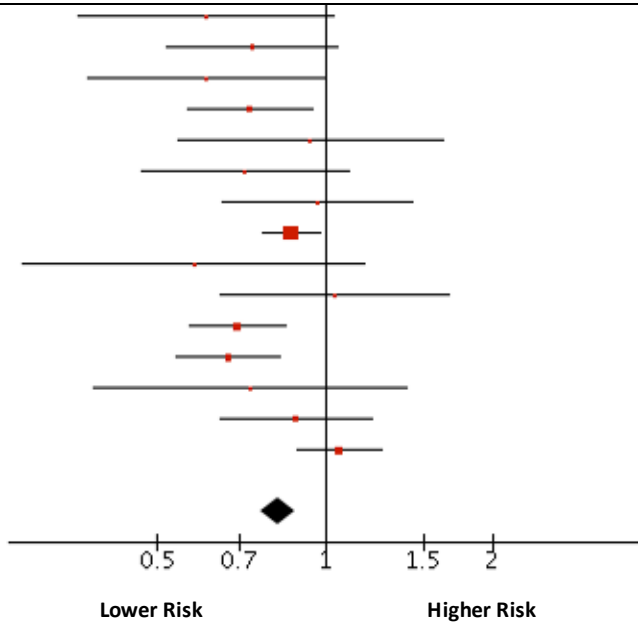
TOTAL FRUIT AND VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	1.6%	0.61 [0.36, 1.04]
Nurses' Health Study - Joshipura 1999	75,596	204	3.7%	0.74 [0.52, 1.05]
HPFS - Joshipura 1999	38,683	336	1.9%	0.61 [0.38, 1.00]
National Health & Nutrition - Bazzano 2002	9,608	888	7.1%	0.73 [0.57, 0.95]
ARIC - Steffen 2003	11,940	214	1.5%	0.94 [0.54, 1.63]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	2.5%	0.72 [0.47, 1.11]
MORGEN - Oude Griep 2011 (a)	20,069	233	3.0%	0.97 [0.66, 1.44]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	33.3%	0.87 [0.77, 0.98]
Japan Diabetes Complications Study - Tanaka 2013	1,414	68	0.9%	0.58 [0.29, 1.18]
Rotterdam - Bos 2014	3,750	545	2.1%	1.04 [0.65, 1.67]
CCHS - Manuel 2015 - F	44,776	842	12.0%	0.70 [0.57, 0.85]
CCHS - Manuel 2015 - M	37,483	709	9.9%	0.67 [0.54, 0.83]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.1%	0.73 [0.38, 1.40]
PURE - Miller 2017	135,335	2,234	4.7%	0.89 [0.65, 1.21]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	14.8%	1.06 [0.89, 1.27]
Total (95% CI)	**532,667**	**11,091**	**100.0%**	**0.82 [0.77, 0.88]**

Heterogeneity: Chi² = 22.28, df = 14 (P = 0.07); I² = 37%

Test for overall effect: Z = 5.61 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	3.0%	0.61 [0.36, 1.04]
Nurses' Health Study - Joshipura 1999	75,596	204	5.8%	0.74 [0.52, 1.05]
HPFS - Joshipura 1999	38,683	336	3.5%	0.61 [0.38, 1.00]
National Health & Nutrition - Bazzano 2002	9,608	888	9.0%	0.73 [0.57, 0.95]
ARIC - Steffen 2003	11,940	214	2.9%	0.94 [0.54, 1.63]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	4.3%	0.72 [0.47, 1.11]
MORGEN - Oude Griep 2011 (a)	20,069	233	5.0%	0.97 [0.66, 1.44]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	16.6%	0.87 [0.77, 0.98]
Japan Diabetes Complications Study - Tanaka 2013	1,414	68	1.8%	0.58 [0.29, 1.18]
Rotterdam - Bos 2014	3,750	545	3.7%	1.04 [0.65, 1.67]
CCHS - Manuel 2015 - F	44,776	842	11.8%	0.70 [0.57, 0.85]
CCHS - Manuel 2015 - M	37,483	709	10.7%	0.67 [0.54, 0.83]
PREDIMED- Buil-Cosiales 2016	7,216	169	2.1%	0.73 [0.38, 1.40]
PURE - Miller 2017	135,335	2,234	6.9%	0.89 [0.65, 1.21]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	12.9%	1.06 [0.89, 1.27]
Total (95% CI) [Random Effects]	**532,667**	**11,091**	**100.0%**	**0.80 [0.73, 0.89]**

Heterogeneity: Tau² = 0.01; Chi² = 22.28, df = 14 (P = 0.07); I² = 37%

Test for overall effect: Z = 4.30 (P < 0.0001)

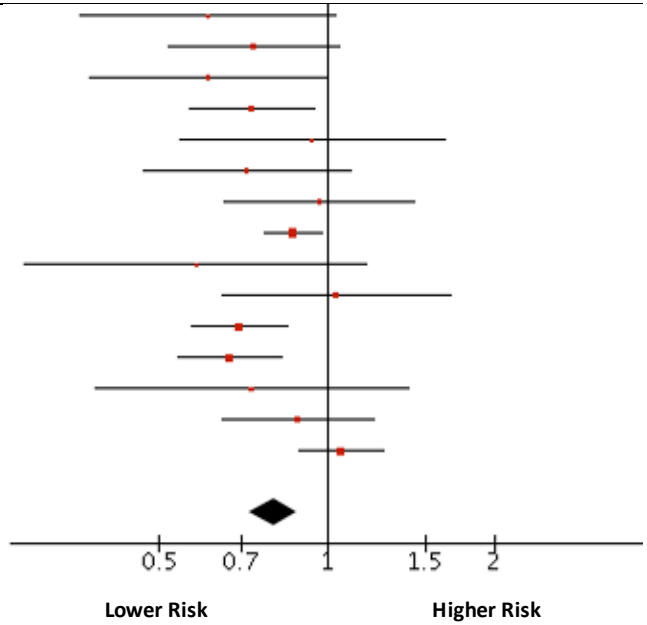


Figure S117. Relation between total fruit and vegetables intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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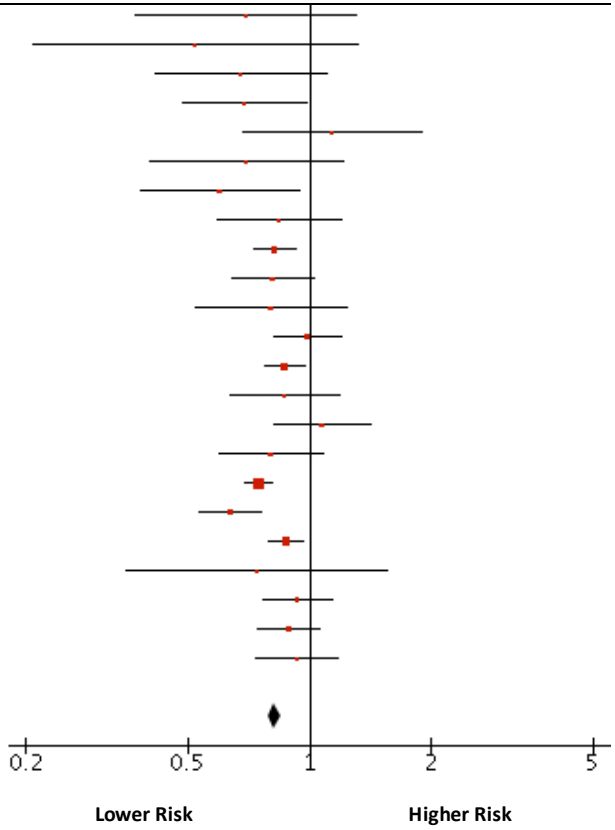
FRUIT AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	0.4%	0.70 [0.37, 1.31]
Zutphen Elderly Study - Keli 1996	552	42	0.2%	0.52 [0.21, 1.31]
HPFS - Joshipura 1999	38,683	366	0.6%	0.68 [0.41, 1.11]
Nurses' Health Study - Joshipura 1999	75,596	204	1.3%	0.69 [0.49, 0.98]
Shibata Study - Yokoyama 2000 - M	880	91	0.6%	1.14 [0.68, 1.90]
Shibata Study - Yokoyama 2000 - F	1,241	105	0.5%	0.70 [0.40, 1.21]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	0.8%	0.60 [0.38, 0.94]
ATBC - Larsson 2009 - intracerebral hemorrhage	26,556	383	1.3%	0.84 [0.59, 1.20]
ATBC - Larsson 2009 - cerebral infraction	-	2,702	11.3%	0.82 [0.73, 0.92]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	2.8%	0.81 [0.64, 1.03]
ATBC - Larsson 2009 - subarachnoid hemorrhage	-	196	0.8%	0.80 [0.52, 1.24]
MONICA Finland - Zhang 2011 (b)	36,686	1,478	4.1%	0.99 [0.81, 1.20]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	11.3%	0.87 [0.77, 0.98]
MONICA Danish - Tognon 2014	1,849	167	1.6%	0.87 [0.64, 1.19]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	2.1%	1.07 [0.82, 1.41]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	1.8%	0.80 [0.60, 1.08]
China Kadoorie Biobank - Du 2016 - ischemic stroke	451,665	3,523	25.3%	0.75 [0.69, 0.81]
China Kadoorie Biobank-Du 2016 -hemorrhagic stroke	-	14,579	5.0%	0.64 [0.53, 0.76]
China Kadoorie Biobank - Du 2016 - other	-	11,054	16.2%	0.88 [0.80, 0.97]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.3%	0.74 [0.35, 1.56]
PURE - Miller 2017	135,335	2,234	4.1%	0.93 [0.77, 1.13]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	5.0%	0.89 [0.74, 1.06]
EPIC NL and MORGEN - Scheffers 2019	34,560	1,135	2.8%	0.93 [0.74, 1.18]
Total (95% CI)	**987,993**	**43,702**	**100.0%**	**0.82 [0.79, 0.85]**

Heterogeneity: Chi² = 33.36, df = 22 (P = 0.06); I² = 34%

Test for overall effect: Z = 9.77 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	0.8%	0.70 [0.37, 1.31]
Zutphen Elderly Study - Keli 1996	552	42	0.4%	0.52 [0.21, 1.31]
HPFS - Joshipura 1999	38,683	366	1.3%	0.68 [0.41, 1.11]
Nurses' Health Study - Joshipura 1999	75,596	204	2.3%	0.69 [0.49, 0.98]
Shibata Study - Yokoyama 2000 - M	880	91	1.2%	1.14 [0.68, 1.90]
Shibata Study - Yokoyama 2000 - F	1,241	105	1.0%	0.70 [0.40, 1.21]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	1.5%	0.60 [0.38, 0.94]
ATBC - Larsson 2009 - intracerebral hemorrhage	26,556	383	2.3%	0.84 [0.59, 1.20]
ATBC - Larsson 2009 - cerebral infraction	-	2,702	9.9%	0.82 [0.73, 0.92]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	4.5%	0.81 [0.64, 1.03]
ATBC - Larsson 2009 - subarachnoid hemorrhage	-	196	1.6%	0.80 [0.52, 1.24]
MONICA Finland - Zhang 2011 (b)	36,686	1,478	5.7%	0.99 [0.81, 1.20]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	9.9%	0.87 [0.77, 0.98]
MONICA Danish - Tognon 2014	1,849	167	2.8%	0.87 [0.64, 1.19]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	3.5%	1.07 [0.82, 1.41]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	3.2%	0.80 [0.60, 1.08]
China Kadoorie Biobank - Du 2016 - ischemic stroke	451,665	3,523	12.8%	0.75 [0.69, 0.81]
China Kadoorie Biobank-Du 2016 -hemorrhagic stroke	-	14,579	6.6%	0.64 [0.53, 0.76]
China Kadoorie Biobank - Du 2016 - other	-	11,054	11.3%	0.88 [0.80, 0.97]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.6%	0.74 [0.35, 1.56]
PURE - Miller 2017	135,335	2,234	5.7%	0.93 [0.77, 1.13]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	6.6%	0.89 [0.74, 1.06]
EPIC NL and MORGEN - Scheffers 2019	34,560	1,135	4.5%	0.93 [0.74, 1.18]
Total (95% CI) [Random Effects]	**987,993**	**43,702**	**100.0%**	**0.83 [0.78, 0.88]**

Heterogeneity: Tau² = 0.01; Chi² = 33.36, df = 22 (P = 0.06); I² = 34%

Test for overall effect: Z = 6.30 (P < 0.00001)

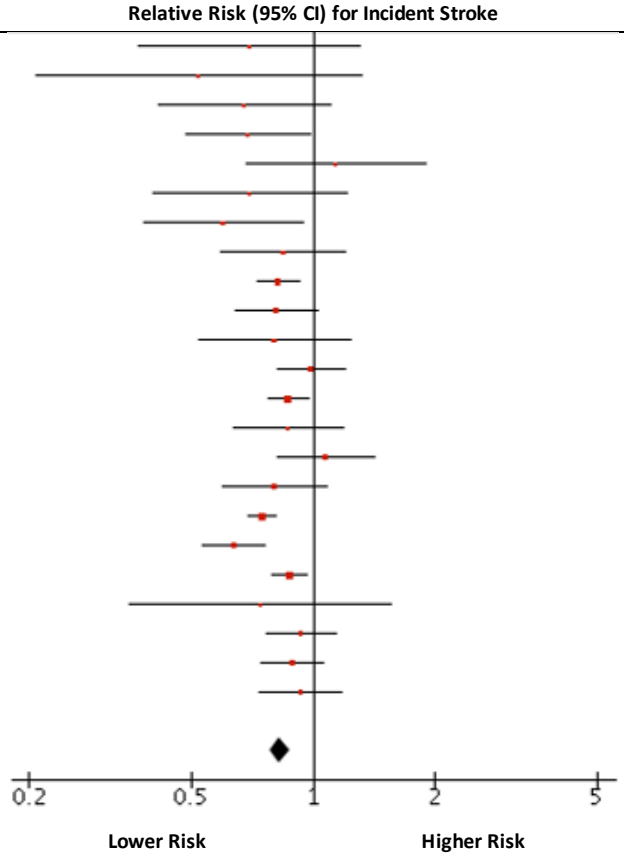


Figure S118. Relation between fruit intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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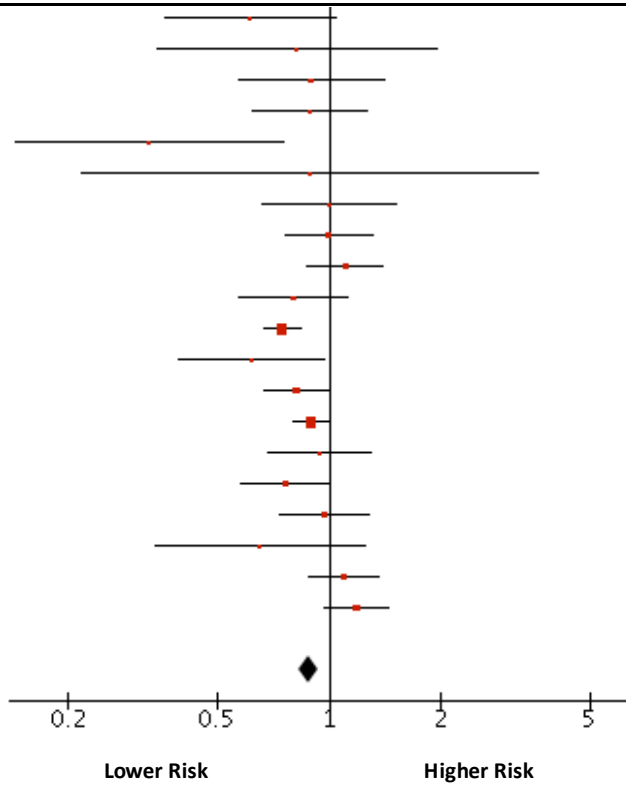
VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	1.1%	0.61 [0.36, 1.04]
Zutphen Elderly Study - Keli 1996	552	42	0.4%	0.82 [0.35, 1.94]
HPFS - Joshipura 1999	38,683	336	1.5%	0.90 [0.57, 1.41]
Nurses' Health Study - Joshipura 1999	75,596	204	2.4%	0.89 [0.62, 1.26]
Shibata Study - Yokoyama 2000 - M	880	91	0.4%	0.33 [0.14, 0.75]
Shibata Study - Yokoyama 2000 - F	1,241	105	0.2%	0.89 [0.22, 3.64]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	1.8%	1.00 [0.66, 1.51]
Miyako Study - Pham 2007	9,651	226	4.0%	1.00 [0.76, 1.32]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	5.5%	1.11 [0.87, 1.40]
ATBC - Larsson 2009 - intracerebral hemorrhage	26,556	383	2.7%	0.80 [0.58, 1.12]
ATBC - Larsson 2009 - cerebral infraction	26,556	2,702	22.0%	0.75 [0.67, 0.84]
ATBC - Larsson 2009 - subarachnoid hemorrhage	26,556	196	1.5%	0.62 [0.39, 0.97]
MONICA Finland - Zhang 2011 (b)	36,686	1,478	7.9%	0.82 [0.67, 1.00]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	22.0%	0.90 [0.80, 1.01]
MONICA Danish - Tognon 2014	1,849	167	3.1%	0.94 [0.69, 1.29]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	4.0%	0.76 [0.58, 1.00]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	4.0%	0.97 [0.74, 1.28]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.7%	0.65 [0.34, 1.24]
PURE - Miller 2017	135,335	2,234	6.6%	1.09 [0.88, 1.36]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	7.9%	1.19 [0.97, 1.44]
Total (95% CI)	**564,531**	**13,607**	**100.0%**	**0.88 [0.83, 0.93]**

Heterogeneity: Chi² = 37.99, df = 19 (P = 0.006); I² = 50%

Test for overall effect: Z = 4.45 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	97	2.4%	0.61 [0.36, 1.04]
Zutphen Elderly Study - Keli 1996	552	42	1.0%	0.82 [0.35, 1.94]
HPFS - Joshipura 1999	38,683	336	3.1%	0.90 [0.57, 1.41]
Nurses' Health Study - Joshipura 1999	75,596	204	4.4%	0.89 [0.62, 1.26]
Shibata Study - Yokoyama 2000 - M	880	91	1.1%	0.33 [0.14, 0.75]
Shibata Study - Yokoyama 2000 - F	1,241	105	0.4%	0.89 [0.22, 3.64]
Danish Diet Cancer Health - Johnsen 2003	54,506	266	3.6%	1.00 [0.66, 1.51]
Miyako Study - Pham 2007	9,651	226	5.9%	1.00 [0.76, 1.32]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	6.9%	1.11 [0.87, 1.40]
ATBC - Larsson 2009 - intracerebral hemorrhage	26,556	383	4.7%	0.80 [0.58, 1.12]
ATBC - Larsson 2009 - cerebral infraction	26,556	2,702	10.5%	0.75 [0.67, 0.84]
ATBC - Larsson 2009 - subarachnoid hemorrhage	26,556	196	3.1%	0.62 [0.39, 0.97]
MONICA Finland - Zhang 2011 (b)	36,686	1,478	8.0%	0.82 [0.67, 1.00]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	10.5%	0.90 [0.80, 1.01]
MONICA Danish - Tognon 2014	1,849	167	5.1%	0.94 [0.69, 1.29]
Malmo Diet Cancer Study- Sonestedt 2015 - F	16,397	-	5.9%	0.76 [0.58, 1.00]
Malmo Diet Cancer Study - Sonestedt 2015 - M	10,048	-	5.9%	0.97 [0.74, 1.28]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.7%	0.65 [0.34, 1.24]
PURE - Miller 2017	135,335	2,234	7.5%	1.09 [0.88, 1.36]
Japan Public Health Centre - Yoshizaki 2019	16,498	197	8.0%	1.19 [0.97, 1.44]
Total (95% CI) [Random Effects]	**564,531**	**13,607**	**100.0%**	**0.89 [0.81, 0.97]**

Heterogeneity: Tau² = 0.02; Chi² = 37.99, df = 19 (P = 0.006); I² = 50%

Test for overall effect: Z = 2.57 (P = 0.01)

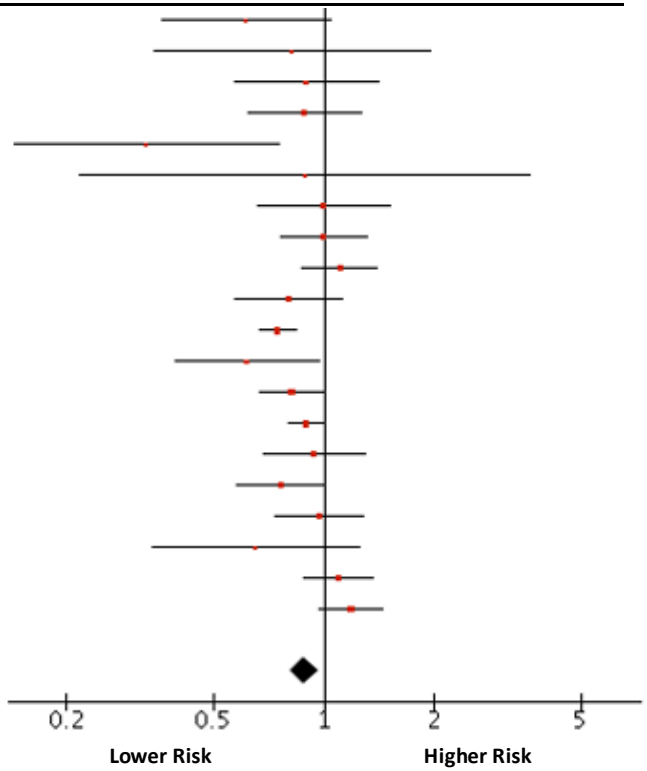


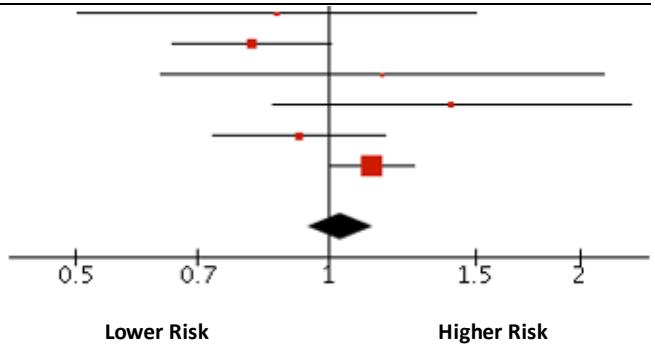
Figure S119. Relation between intake of vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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BERRIES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2000 - intracerebral hemorrage	26,593	95	2.7%	0.81 [0.65, 1.01]
ATBC - Hirvonen 2000 - cerebral infarction	-	736	17.6%	1.16 [0.63, 2.13]
ATBC - Hirvonen 2000 - subarachnoid hemorrage	-	83	2.2%	0.87 [0.50, 1.50]
WHS - Sesso 2007	38,176	339	3.4%	1.40 [0.86, 2.29]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	14.8%	0.92 [0.73, 1.17]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	59.2%	1.13 [1.00, 1.27]
Total (95% CI)	**143,662**	**5,967**	**100.0%**	**1.03 [0.94, 1.13]**
Heterogeneity: Chi² = 9.90, df = 5 (P = 0.08); I² = 50%				
Test for overall effect: Z = 0.72 (P = 0.47)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2000 - cerebral infarction	26,593	736	23.40%	0.81 [0.65, 1.01]
ATBC - Hirvonen 2000 - subarachnoid hemorrage	-	83	6.10%	1.16 [0.63, 2.13]
ATBC - Hirvonen 2000 - intracerebral hemorrage	-	95	7.20%	0.87 [0.50, 1.50]
WHS - Sesso 2007	38,176	339	8.70%	1.40 [0.86, 2.29]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	21.70%	0.92 [0.73, 1.17]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	32.80%	1.13 [1.00, 1.27]
Total (95% CI) [Random Effects]	**143,662**	**5,967**	**100.0%**	**1.00 [0.85, 1.18]**
Heterogeneity: Tau² = 0.02; Chi² = 9.90, df = 5 (P = 0.08); I² = 50%				
Test for overall effect: Z = 0.02 (P = 0.99)				

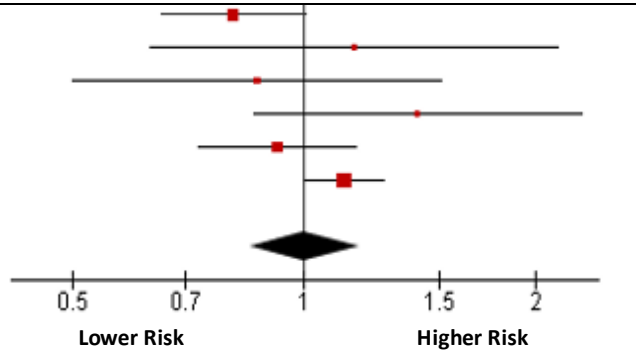


Figure S120. Relation between intake of berries and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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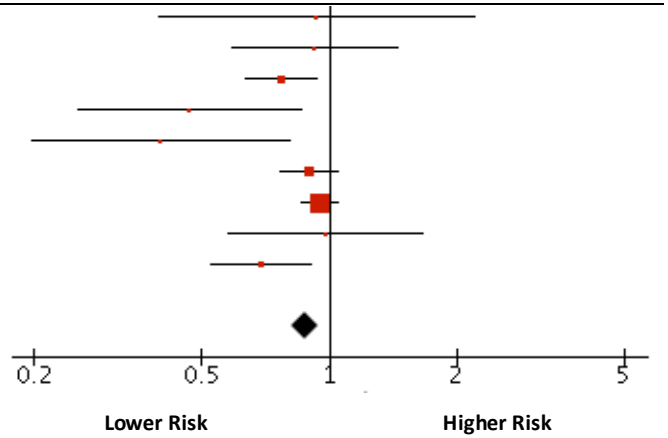
CITRUS FRUIT AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Zutphen Elderly Study - Keli 1996	552	42	0.7%	0.93 [0.39, 2.21]
HPFS - Joshipura 1999	38,683	366	2.5%	0.92 [0.59, 1.45]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	13.1%	0.77 [0.63, 0.94]
Jidni Medical School - Yamada 2011 - F	6,476	183	1.4%	0.47 [0.25, 0.86]
Jidni Medical School - Yamada 2011 - M	4,147	201	1.0%	0.40 [0.20, 0.81]
Nurses' Health Study - Cassidy 2012	69,622	943	20.5%	0.90 [0.77, 1.05]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	52.4%	0.95 [0.86, 1.05]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.8%	0.98 [0.58, 1.66]
REGARDS - Goetz 2016 (b)	20,024	524	6.7%	0.69 [0.52, 0.91]
Total (95% CI)	**225,613**	**7,142**	**100.0%**	**0.88 [0.82, 0.94]**

Heterogeneity: Chi² = 16.43, df = 8 (P = 0.04); I² = 51%

Test for overall effect: Z = 3.59 (P = 0.0003)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Zutphen Elderly Study - Keli 1996	552	42	2.3%	0.93 [0.39, 2.21]
HPFS - Joshipura 1999	38,683	366	7.0%	0.92 [0.59, 1.45]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	18.1%	0.77 [0.63, 0.94]
Jidni Medical School - Yamada 2011 - M	4,147	201	3.3%	0.40 [0.20, 0.81]
Jidni Medical School - Yamada 2011 - F	6,476	183	4.3%	0.47 [0.25, 0.86]
Nurses' Health Study - Cassidy 2012	69,622	943	21.0%	0.90 [0.77, 1.05]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	25.2%	0.95 [0.86, 1.05]
PREDIMED- Buil-Cosiales 2016	7,216	169	5.4%	0.98 [0.58, 1.66]
REGARDS - Goetz 2016 (b)	20,024	524	13.4%	0.69 [0.52, 0.91]
Total (95% CI) [Random Effects]	**225,613**	**7,142**	**100.0%**	**0.82 [0.71, 0.93]**

Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 16.43$, df = 8 (P = 0.04); $I^2 = 51\%$

Test for overall effect: Z = 2.93 (P = 0.003)

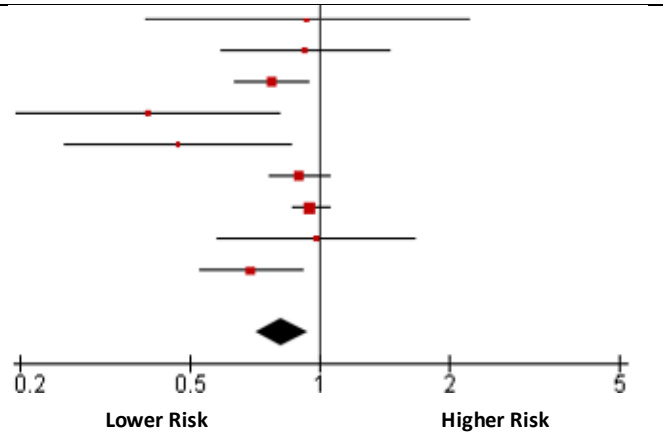


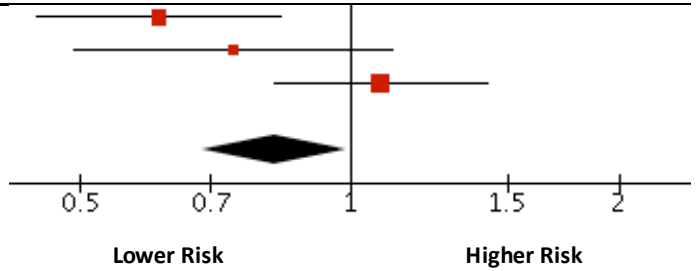
Figure S121. Relation between citrus fruit intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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FRUIT JUICE AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Joshipura 1999	75,596	204	34.6%	0.61 [0.45, 0.84]
HPFS - Joshipura 1999	38,683	366	20.1%	0.74 [0.49, 1.12]
EPIC NL and MORGEN - Scheffers 2019	34,560	1,135	45.2%	1.08 [0.82, 1.43]
Total (95% CI)	**148,839**	**1,705**	**100.0%**	**0.82 [0.68, 0.99]**
Heterogeneity: Chi² = 7.51, df = 2 (P = 0.02); I² = 73%				
Test for overall effect: Z = 2.06 (P = 0.04)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Joshipura 1999	75,596	204	34.40%	0.61 [0.45, 0.84]
HPFS - Joshipura 1999	38,683	366	29.10%	0.74 [0.49, 1.12]
EPIC NL and MORGEN - Scheffers 2019	34,560	1,135	36.50%	1.08 [0.82, 1.43]
Total (95% CI) [Random Effects]	**148,839**	**1,705**	**100.0%**	**0.80 [0.55, 1.15]**
Heterogeneity: Tau² = 0.08; Chi² = 7.51, df = 2 (P = 0.02); I² = 73%				
Test for overall effect: Z = 1.21 (P = 0.23)				

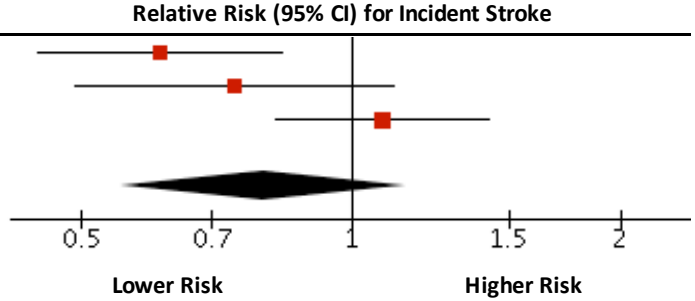


Figure S122. Relation between intake of fruit juice and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

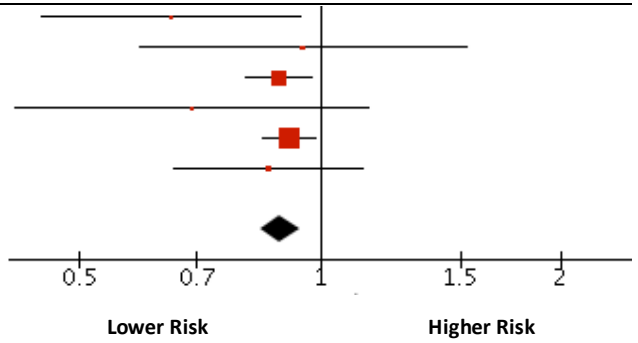
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POMMES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health Exam - Knekt 2000 - M	9,208	445	2.4%	0.65 [0.45, 0.94]
Finish Mobile Clinic Health Exam - Knekt 2000 - F	-	378	1.5%	0.95 [0.59, 1.52]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	35.2%	0.89 [0.80, 0.98]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.3%	0.69 [0.41, 1.15]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	55.0%	0.91 [0.85, 0.99]
NutriNet-Sante - Adriouch 2018	84,158	293	4.5%	0.86 [0.65, 1.13]
Total (95% CI)	**230,881**	**7,657**	**100.0%**	**0.89 [0.84, 0.95]**

Heterogeneity: Chi² = 4.25, df = 5 (P = 0.51); I² = 0%
Test for overall effect: Z = 3.86 (P = 0.0001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health Exam - Knekt 2000 - M	9,208	445	2.4%	0.65 [0.45, 0.94]
Finish Mobile Clinic Health Exam - Knekt 2000 - F	-	378	1.5%	0.95 [0.59, 1.52]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	35.2%	0.89 [0.80, 0.98]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.3%	0.69 [0.41, 1.15]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	55.0%	0.91 [0.85, 0.99]
NutriNet-Sante - Adriouch 2018	84,158	293	4.5%	0.86 [0.65, 1.13]
Total (95% CI) [Random Effects]	**230,881**	**7,657**	**100.0%**	**0.89 [0.84, 0.95]**

Heterogeneity: Tau² = 0.00; Chi² = 4.25, df = 5 (P = 0.51); I² = 0%
Test for overall effect: Z = 3.86 (P = 0.0001)

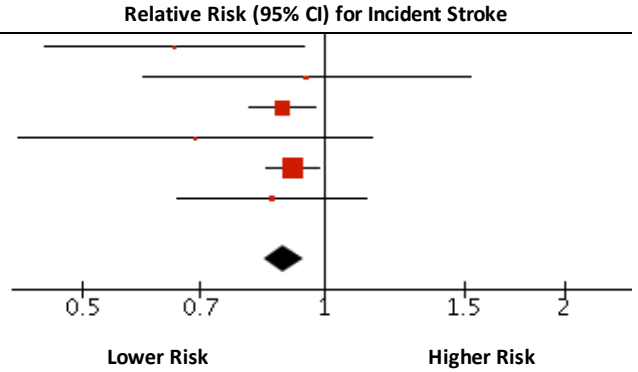


Figure S123. Relation between intake of pommes fruit and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

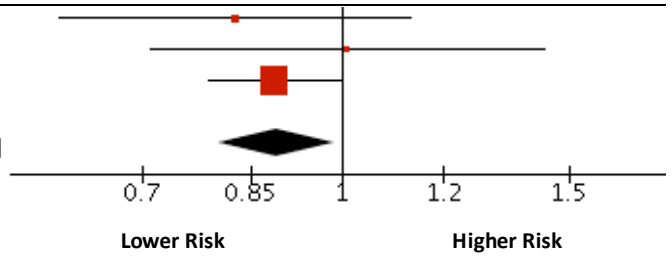
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ALLIUM VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health Exam - Knekt 2000 - M	9,208	445	11.2%	0.83 [0.60, 1.13]
Finish Mobile Clinic Health Exam - Knekt 2000 - F	-	378	8.9%	1.01 [0.71, 1.44]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	79.9%	0.89 [0.79, 1.00]
Total (95% CI)	**84,169**	**4,912**	**100.0%**	**0.89 [0.80, 0.99]**

Heterogeneity: Chi² = 0.71, df = 2 (P = 0.70); I² = 0%
Test for overall effect: Z = 2.17 (P = 0.03)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Finish Mobile Clinic Health Exam - Knekt 2000 - M	9,208	445	11.2%	0.83 [0.60, 1.13]
Finish Mobile Clinic Health Exam - Knekt 2000 - F	-	378	8.9%	1.01 [0.71, 1.44]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	79.9%	0.89 [0.79, 1.00]
Total (95% CI) [Random Effects]	**84,169**	**4,912**	**100.0%**	**0.89 [0.80, 0.99]**

Heterogeneity: Tau² = 0.00; Chi² = 0.71, df = 2 (P = 0.70); I² = 0%
Test for overall effect: Z = 2.17 (P = 0.03)

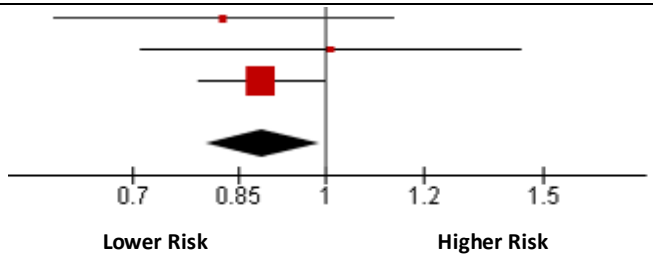


Figure S124. Relation between intake of allium vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

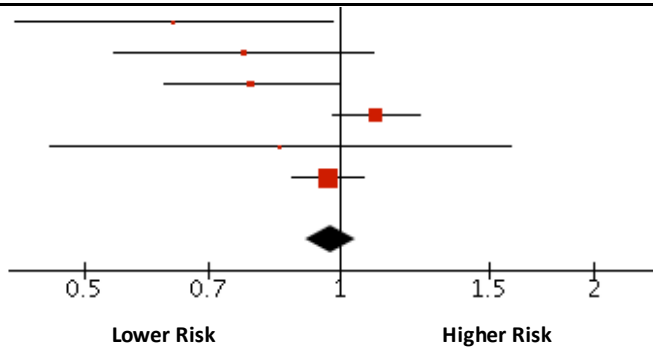
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CRUCIFEROUS VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
HPFS - Joshipura 1999	38,683	336	2.6%	0.64 [0.41, 0.98]
Nurses' Health Study - Joshipura 1999	75,596	204	3.8%	0.77 [0.54, 1.10]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	8.6%	0.79 [0.62, 1.00]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	34.4%	1.11 [0.98, 1.24]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.2%	0.85 [0.46, 1.60]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	49.5%	0.97 [0.88, 1.07]
Total (95% CI)	**255,726**	**7,706**	**100.0%**	**0.98 [0.91, 1.05]**

Heterogeneity: Chi^2 = 13.17, df = 5 (P = 0.02); I^2 = 62%
Test for overall effect: Z = 0.70 (P = 0.49)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Joshipura 1999	75,596	204	11.3%	0.77 [0.54, 1.10]
HPFS - Joshipura 1999	38,683	336	8.5%	0.64 [0.41, 0.98]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	18.0%	0.79 [0.62, 1.00]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	27.9%	1.11 [0.98, 1.24]
PREDIMED- Buil-Cosiales 2016	7,216	169	4.6%	0.85 [0.46, 1.60]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	29.6%	0.97 [0.88, 1.07]
Total (95% CI) [Random Effects]	**255,726**	**7,706**	**100.0%**	**0.91 [0.78, 1.05]**

Heterogeneity: Tau^2 = 0.02; Chi^2 = 13.17, df = 5 (P = 0.02); I^2 = 62%
Test for overall effect: Z = 1.34 (P = 0.18)

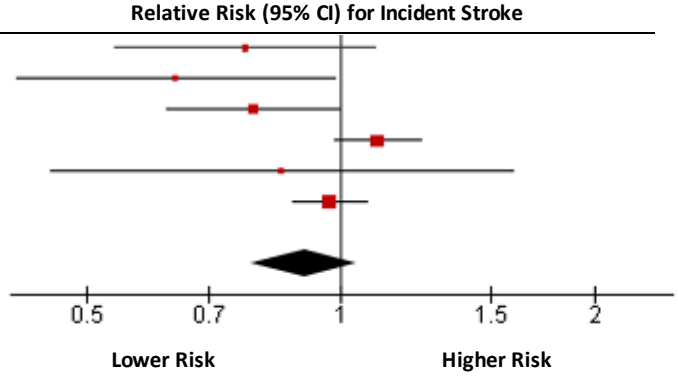


Figure S125. Relation between intake of cruciferous vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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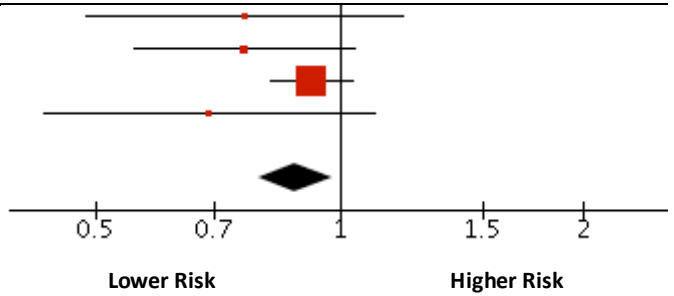
GREEN LEAFY VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
HPFS - Joshipura 1999	38,683	336	5.4%	0.76 [0.49, 1.20]
Nurses' Health Study - Joshipura 1999	75,596	204	11.1%	0.76 [0.56, 1.04]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	78.7%	0.92 [0.82, 1.04]
PREDIMED- Buil-Cosiales 2016	7,216	169	4.9%	0.69 [0.43, 1.11]
Total (95% CI)	**196,456**	**4,798**	**100.0%**	**0.88 [0.79, 0.98]**

Heterogeneity: Chi² = 2.82, df = 3 (P = 0.42); I² = 0%

Test for overall effect: Z = 2.36 (P = 0.02)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Nurses' Health Study - Joshipura 1999	75,596	204	11.1%	0.76 [0.56, 1.04]
HPFS - Joshipura 1999	38,683	336	5.4%	0.76 [0.49, 1.20]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	78.7%	0.92 [0.82, 1.04]
PREDIMED- Buil-Cosiales 2016	7,216	169	4.9%	0.69 [0.43, 1.11]
Total (95% CI) [Random Effects]	**196,456**	**4,798**	**100.0%**	**0.88 [0.79, 0.98]**

Heterogeneity: Tau² = 0.00; Chi² = 2.82, df = 3 (P = 0.42); I² = 0%

Test for overall effect: Z = 2.36 (P = 0.02)

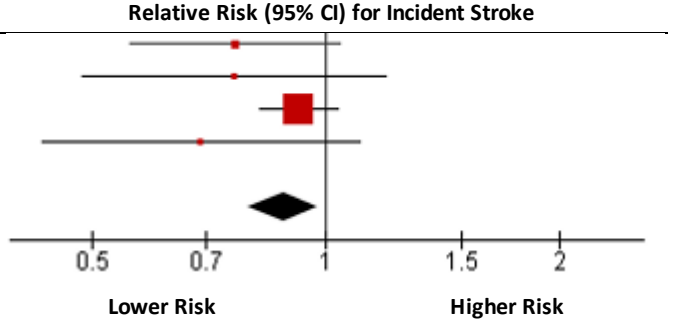


Figure S126. Relation between intake of green leafy vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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TOMATOES AND STROKE INCIDENCE

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (b) - tomato based products	38,445	247	100.0%	0.20 [0.05, 0.82]
Total (95% CI)	**38,445**	**247**	**100.0%**	**0.20 [0.05, 0.82]**

Heterogeneity: Not applicable
Test for overall effect: Z = 2.24 (P = 0.03)

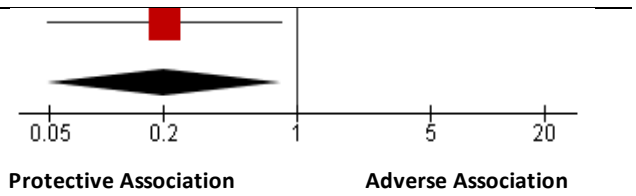
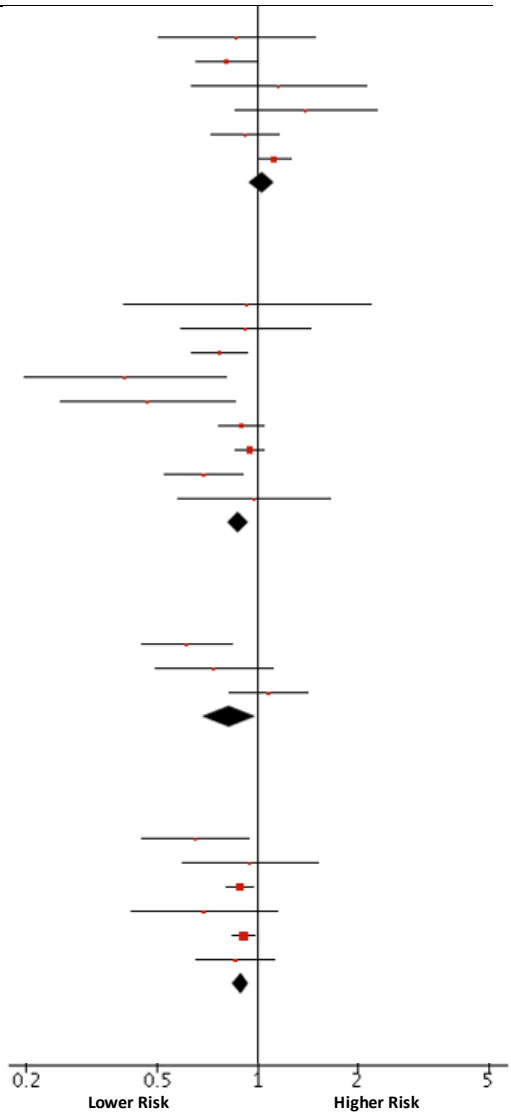


Figure S127. Relation between intake of tomatoes and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I, with values ≥ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Berries				
ATBC - Hirvonen 2000 - intracerebral hemorrag	26,593	95	0.5%	0.87 [0.50, 1.50]
ATBC - Hirvonen 2000 - cerebral infarction	-	736	3.3%	0.81 [0.65, 1.01]
ATBC - Hirvonen 2000 - subarachnoid hemorrag	-	83	0.4%	1.16 [0.63, 2.13]
WHS - Sesso 2007	38,176	339	0.6%	1.40 [0.86, 2.29]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	2.8%	0.92 [0.73, 1.17]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	11.2%	1.13 [1.00, 1.27]
Subtotal (95% CI)	**143,662**	**5,967**	**18.9%**	**1.03 [0.94, 1.13]**
Heterogeneity: Chi² = 9.90, df = 5 (P = 0.08); I² = 50%				
Test for overall effect: Z = 0.72 (P = 0.47)				
Citrus				
Zutphen Elderly Study - Keli 1996	552	42	0.2%	0.93 [0.39, 2.21]
HPFS - Joshipura 1999	38,683	366	0.8%	0.92 [0.59, 1.45]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	4.0%	0.77 [0.63, 0.94]
Jidni Medical School - Yamada 2011 - M	4,147	201	0.3%	0.40 [0.20, 0.81]
Jidni Medical School - Yamada 2011 - F	6,476	183	0.4%	0.47 [0.25, 0.86]
Nurses' Health Study - Cassidy 2012	69,622	943	6.3%	0.90 [0.77, 1.05]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	16.1%	0.95 [0.86, 1.05]
REGARDS - Goetz 2016 (b)	20,024	524	2.1%	0.69 [0.52, 0.91]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.6%	0.98 [0.58, 1.66]
Subtotal (95% CI)	**225,613**	**7,142**	**30.8%**	**0.88 [0.82, 0.94]**
Heterogeneity: Chi² = 16.43, df = 8 (P = 0.04); I² = 51%				
Test for overall effect: Z = 3.59 (P = 0.0003)				
Fruit Juice				
Nurses' Health Study - Joshipura 1999	75,596	204	1.6%	0.61 [0.45, 0.84]
HPFS - Joshipura 1999	38,683	366	0.9%	0.74 [0.49, 1.12]
EPIC NL and MORGEN - Scheffers 2019	34,560	1,135	2.1%	1.08 [0.82, 1.43]
Subtotal (95% CI)	**148,839**	**1,705**	**4.5%**	**0.82 [0.68, 0.99]**
Heterogeneity: Chi² = 7.51, df = 2 (P = 0.02); I² = 73%				
Test for overall effect: Z = 2.06 (P = 0.04)				
Pommes				
Finish Mobile Clinic Health Exam - Knekt 2000 -	9,208	445	1.1%	0.65 [0.45, 0.94]
Finish Mobile Clinic Health Exam - Knekt 2000 -	-	378	0.7%	0.95 [0.59, 1.52]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	16.1%	0.89 [0.80, 0.98]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.6%	0.69 [0.41, 1.15]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	25.2%	0.91 [0.85, 0.99]
NutriNet-Sante - Adriouch 2018	84,158	293	2.1%	0.86 [0.65, 1.13]
Subtotal (95% CI)	**230,881**	**7,657**	**45.8%**	**0.89 [0.84, 0.95]**
Heterogeneity: Chi² = 4.25, df = 5 (P = 0.51); I² = 0%				
Test for overall effect: Z = 3.86 (P = 0.0001)				
Test for subgroup differences: Chi² = 10.20, df = 3 (P = 0.02), I² = 70.6%				



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B. Random Effects

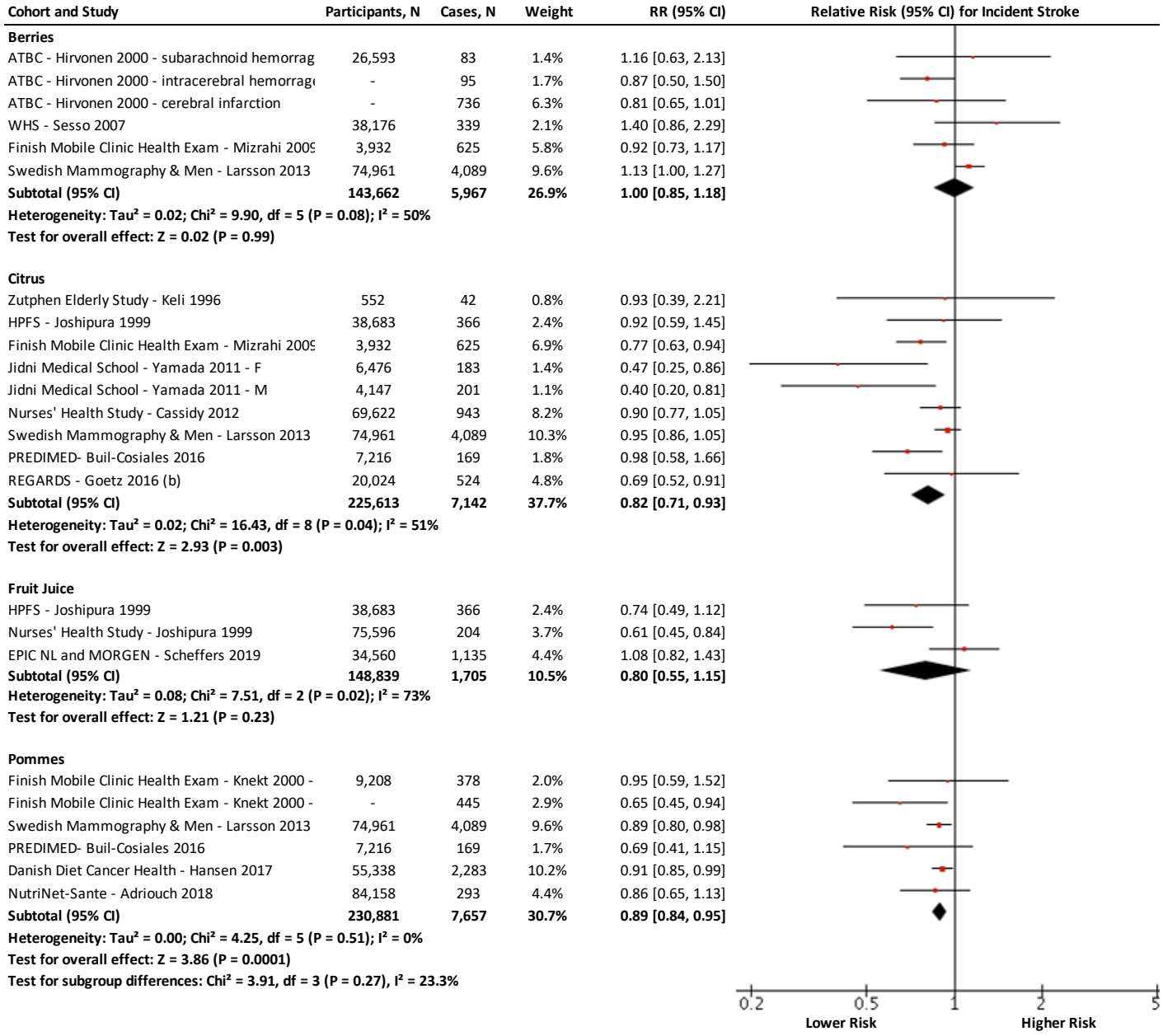
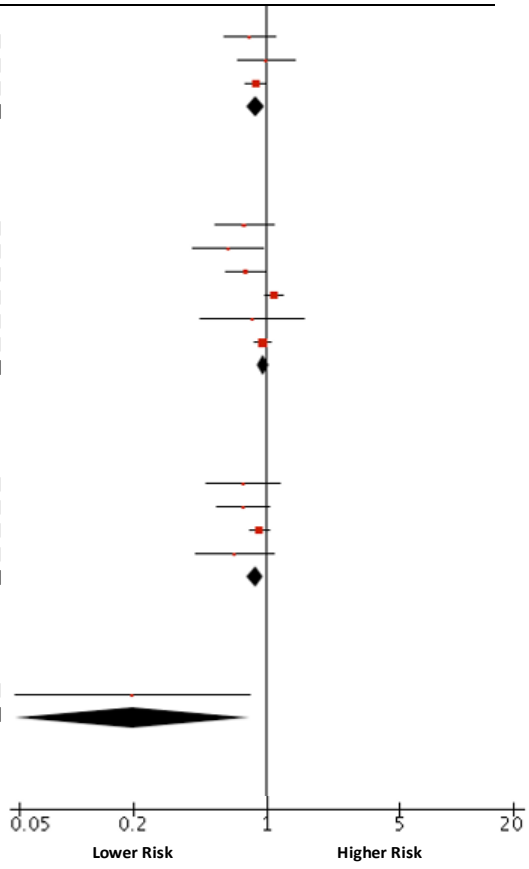


Figure S128. Relation between sources of fruit and stoke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
Finish Mobile Clinic Health Exam - Knekt 2000 - M	9,208	445	2.6%	0.83 [0.60, 1.13]
Finish Mobile Clinic Health Exam - Knekt 2000 - F	-	378	2.0%	1.01 [0.71, 1.44]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	18.4%	0.89 [0.79, 1.00]
Subtotal (95% CI)	**84,169**	**4,912**	**23.0%**	**0.89 [0.80, 0.99]**
Heterogeneity: Chi² = 0.71, df = 2 (P = 0.70); I² = 0%				
Test for overall effect: Z = 2.17 (P = 0.03)				
Cruciferous				
Nurses' Health Study - Joshipura 1999	75,596	204	2.0%	0.77 [0.54, 1.10]
HPFS - Joshipura 1999	38,683	336	1.4%	0.64 [0.41, 0.98]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	4.6%	0.79 [0.62, 1.00]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	18.4%	1.11 [0.98, 1.24]
PREDIMED- Buil-Cosiales 2016	7,216	169	0.6%	0.85 [0.46, 1.60]
Danish Diet Cancer Health - Hansen 2017	55,338	2,283	26.5%	0.97 [0.88, 1.07]
Subtotal (95% CI)	**255,726**	**7,706**	**53.5%**	**0.98 [0.91, 1.05]**
Heterogeneity: Chi² = 13.17, df = 5 (P = 0.02); I² = 62%				
Test for overall effect: Z = 0.70 (P = 0.49)				
Green Leafy				
HPFS - Joshipura 1999	38,683	336	1.3%	0.76 [0.49, 1.20]
Nurses' Health Study - Joshipura 1999	75,596	204	2.6%	0.76 [0.56, 1.04]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	18.4%	0.92 [0.82, 1.04]
PREDIMED- Buil-Cosiales 2016	7,216	169	1.1%	0.69 [0.43, 1.11]
Subtotal (95% CI)	**196,456**	**4,798**	**23.4%**	**0.88 [0.79, 0.98]**
Heterogeneity: Chi² = 2.82, df = 3 (P = 0.42); I² = 0%				
Test for overall effect: Z = 2.36 (P = 0.02)				
Tomatoes				
WHS - Sesso 2003 (b) - tomato based products	38,445	247	0.1%	0.20 [0.05, 0.82]
Subtotal (95% CI)	**38,445**	**247**	**0.1%**	**0.20 [0.05, 0.82]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 2.24 (P = 0.03)				
Test for subgroup differences: Chi² = 8.08, df = 3 (P = 0.04), I² = 62.9%				



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B. Random Effects

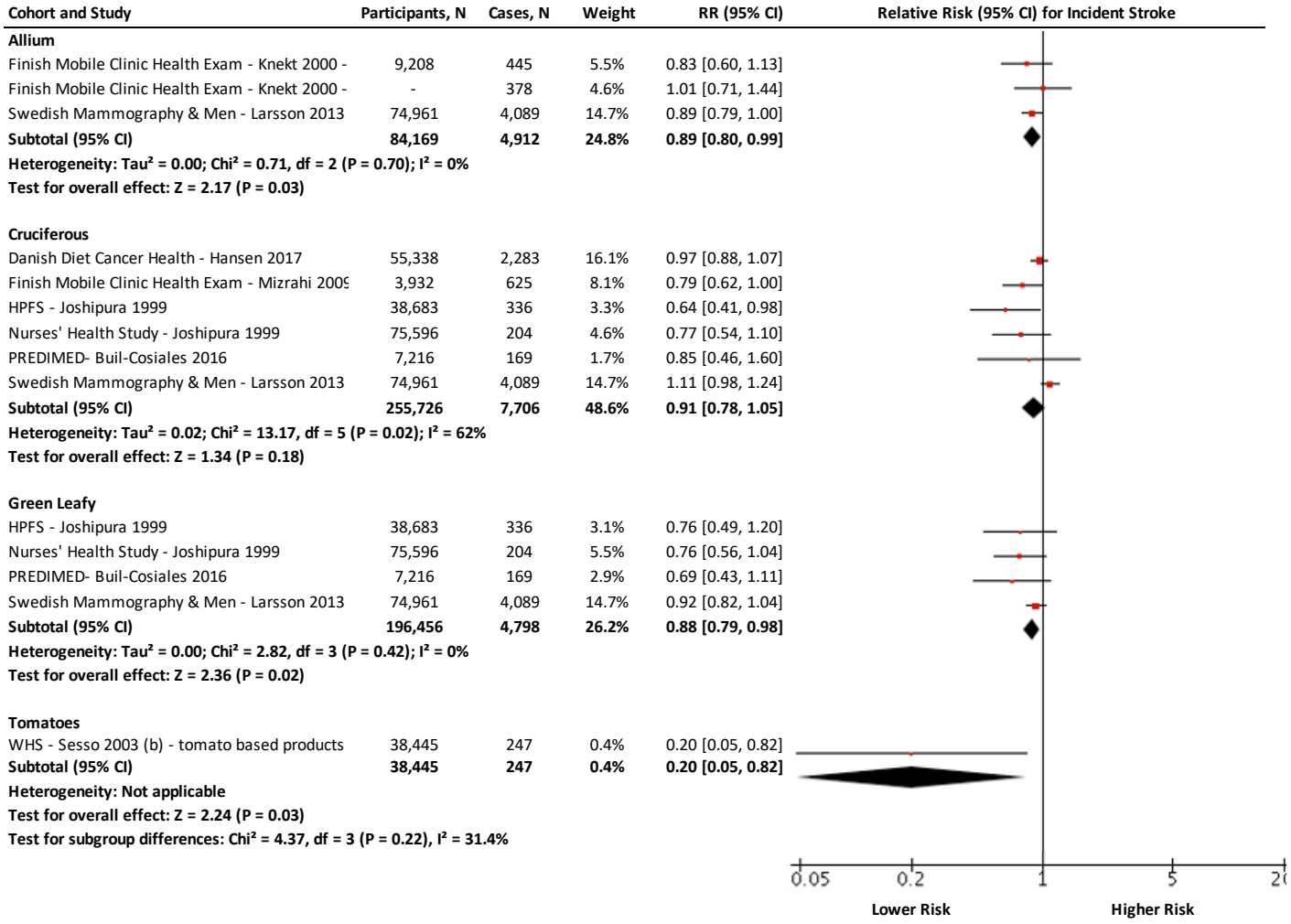


Figure S129. Relation between sources of vegetables and stoke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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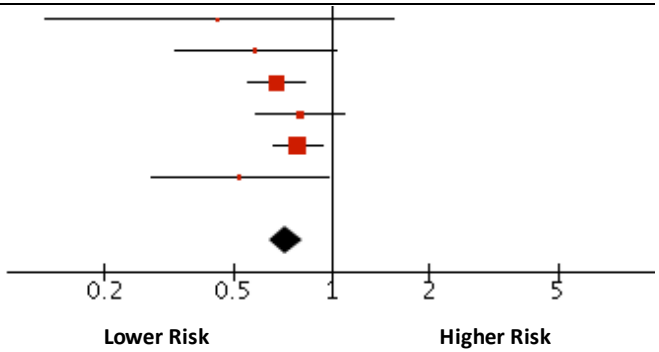
TOTAL FRUIT AND VEGETABLES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	14	0.90%	0.45 [0.13, 1.54]
National Health & Nutrition - Bazzano 2002	9,608	218	4.10%	0.58 [0.33, 1.03]
EPIC - Leenders 2014	451,151	1,291	34.90%	0.68 [0.56, 0.82]
NIPPON DATA80 - Okuda 2015	9,112	385	13.60%	0.80 [0.59, 1.10]
Migrant Study - Hjartaker 2015	9,766	1,034	43.10%	0.79 [0.66, 0.94]
HAPIEE - Stefler 2016	19,263	109	3.40%	0.52 [0.28, 0.98]
Total (95% CI)	**499,732**	**3,051**	**100.0%**	**0.73 [0.65, 0.81]**

Heterogeneity: Chi² = 3.89, df = 5 (P = 0.57); I² = 0%

Test for overall effect: Z = 5.43 (P < 0.00001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Framingham - Gillman 1995	832	14	0.9%	0.45 [0.13, 1.54]
National Health & Nutrition - Bazzano 2002	9,608	218	4.1%	0.58 [0.33, 1.03]
EPIC - Leenders 2014	451,151	1,291	34.9%	0.68 [0.56, 0.82]
NIPPON DATA80 - Okuda 2015	9,112	385	13.6%	0.80 [0.59, 1.10]
Migrant Study - Hjartaker 2015	9,766	1,034	43.1%	0.79 [0.66, 0.94]
HAPIEE - Stefler 2016	19,263	109	3.4%	0.52 [0.28, 0.98]
Total (95% CI) [Random Effects]	**499,732**	**3,051**	**100.0%**	**0.73 [0.65, 0.81]**

Heterogeneity: Tau² = 0.00; Chi² = 3.89, df = 5 (P = 0.57); I² = 0%

Test for overall effect: Z = 5.43 (P < 0.00001)

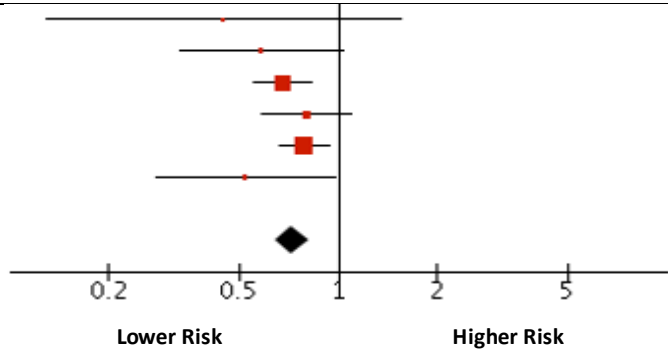


Figure S130. Relation between total fruit and vegetables intake and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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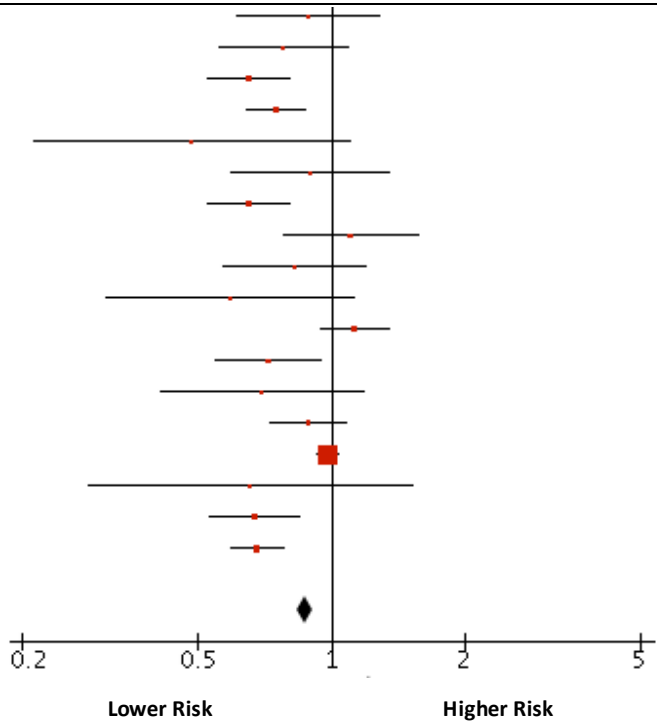
FRUIT AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Health Food Shoppers - Appleby 2002 - M	4,325	142	1.3%	0.89 [0.61, 1.29]
Health Food Shoppers - Appleby 2002 - F	6,416	214	1.6%	0.78 [0.56, 1.09]
Life Span Study - Sauvaget 2003 - M	14,966	692	3.8%	0.65 [0.52, 0.81]
Life Span Study - Sauvaget 2003 - F	23,471	1,234	7.2%	0.75 [0.64, 0.88]
Boyd Orr Cohort - Ness 2005	4,028	83	0.3%	0.48 [0.21, 1.10]
Miyako Study - Pham 2007	9,651	226	1.1%	0.90 [0.59, 1.35]
JACC - Nagura 2009	59,485	1,053	3.8%	0.65 [0.52, 0.81]
Multiethnic Cohort - Sharma 2013 - M	78,410	434	1.4%	1.11 [0.78, 1.57]
Multiethnic Cohort - Sharma 2013 - F	95,618	426	1.3%	0.83 [0.57, 1.20]
MONICA Danish - Tognon 2014	1,849	40	0.4%	0.59 [0.31, 1.12]
EPIC - Leenders 2014	451,151	1,291	5.7%	1.13 [0.95, 1.35]
NIPPON DATA80 - Okuda 2015	9,112	385	2.4%	0.72 [0.55, 0.95]
UK Women's Cohort - Lai 2015	30,458	148	0.6%	0.70 [0.41, 1.18]
Migrant Study - Hjartaker 2015	9,766	1,034	4.6%	0.89 [0.73, 1.08]
Linxian Nutrition - Wang 2016	2,445	452	51.5%	0.98 [0.92, 1.04]
HAPIEE - Stefler 2016	19,263	109	0.3%	0.66 [0.28, 1.53]
China Kadoorie Biobank - Du 2017 - ischemic	462,342	585	3.2%	0.67 [0.53, 0.85]
China Kadoorie Biobank- Du 2017-hemorrhagic	-	2,351	9.5%	0.68 [0.59, 0.78]
Total (95% CI)	**1,282,756**	**10,899**	**100.0%**	**0.87 [0.84, 0.91]**

Heterogeneity: Chi² = 67.81, df = 17 (P < 0.00001); I² = 75%

Test for overall effect: Z = 6.30 (P < 0.00001)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Health Food Shoppers - Appleby 2002 - F	6,416	214	5.2%	0.78 [0.56, 1.09]
Health Food Shoppers - Appleby 2002 - M	4,325	142	4.6%	0.89 [0.61, 1.29]
Life Span Study - Sauvaget 2003 - F	23,471	1,234	8.1%	0.75 [0.64, 0.88]
Life Span Study - Sauvaget 2003 - M	14,966	692	7.1%	0.65 [0.52, 0.81]
Boyd Orr Cohort - Ness 2005	4,028	83	1.5%	0.48 [0.21, 1.10]
Miyako Study - Pham 2007	9,651	226	4.2%	0.90 [0.59, 1.35]
JACC - Nagura 2009	59,485	1,053	7.1%	0.65 [0.52, 0.81]
Multiethnic Cohort - Sharma 2013 - M	78,410	434	4.9%	1.11 [0.78, 1.57]
Multiethnic Cohort - Sharma 2013 - F	95,618	426	4.6%	0.83 [0.57, 1.20]
EPIC - Leenders 2014	451,151	1,291	7.8%	1.13 [0.95, 1.35]
MONICA Danish - Tognon 2014	1,849	40	2.3%	0.59 [0.31, 1.12]
Migrant Study - Hjartaker 2015	9,766	1,034	7.4%	0.89 [0.73, 1.08]
NIPPON DATA80 - Okuda 2015	9,112	385	6.1%	0.72 [0.55, 0.95]
UK Women's Cohort - Lai 2015	30,458	148	3.0%	0.70 [0.41, 1.18]
Linxian Nutrition - Wang 2016	2,445	452	9.4%	0.98 [0.92, 1.04]
HAPIEE - Stefler 2016	19,263	109	1.5%	0.66 [0.28, 1.53]
China Kadoorie Biobank- Du 2017-hemorrhagic	462,342	2,351	8.4%	0.68 [0.59, 0.78]
China Kadoorie Biobank - Du 2017 - ischemic	-	585	6.7%	0.67 [0.53, 0.85]
Total (95% CI) [Random Effects]	**1,282,756**	**10,899**	**100.0%**	**0.79 [0.71, 0.89]**

Heterogeneity: Tau² = 0.03; Chi² = 67.81, df = 17 (P < 0.00001); I² = 75%

Test for overall effect: Z = 4.12 (P < 0.0001)

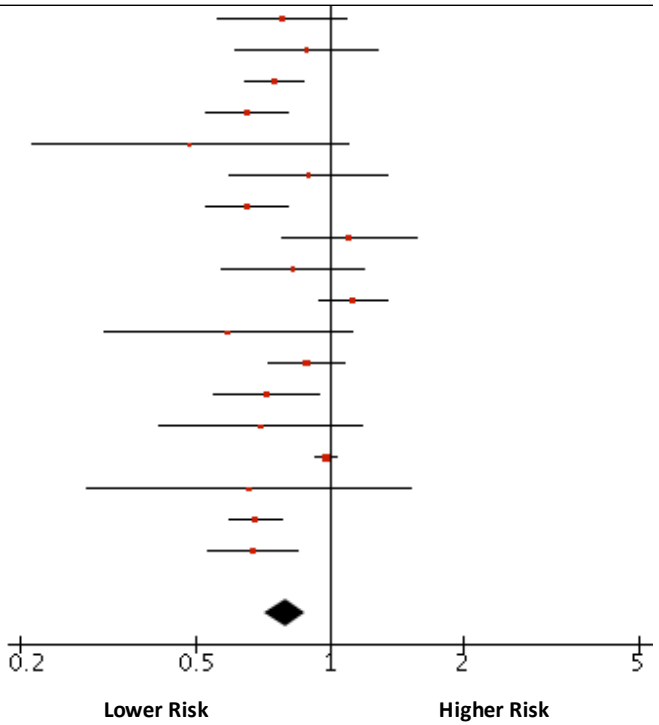


Figure S131. Relation between fruit intake and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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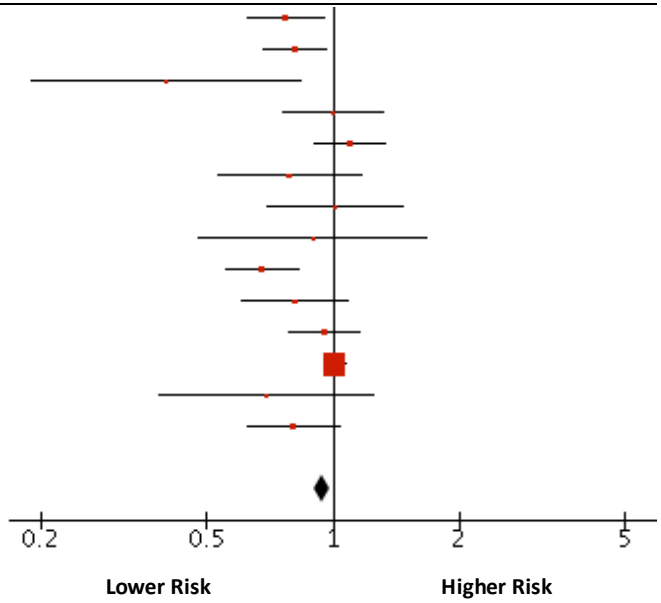
VEGETABLES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Life Span Study - Sauvaget 2003 - M	14,966	692	4.5%	0.77 [0.62, 0.96]
Life Span Study - Sauvaget 2003 - F	23,471	1,234	6.7%	0.81 [0.68, 0.97]
Boyd Orr Cohort - Ness 2005	4,028	83	0.4%	0.40 [0.19, 0.84]
Miyako Study - Pham 2007	9,651	226	2.8%	1.00 [0.76, 1.32]
JACC - Nagura 2009	59,485	1,053	5.4%	1.09 [0.90, 1.33]
Multiethnic Cohort - Sharma 2013 - F	95,618	426	1.3%	0.79 [0.53, 1.16]
Multiethnic Cohort - Sharma 2013 - M	78,410	434	1.5%	1.01 [0.70, 1.47]
MONICA Danish - Tognon 2014	1,849	40	0.5%	0.90 [0.48, 1.68]
EPIC - Leenders 2014	451,151	1,291	5.4%	0.68 [0.56, 0.82]
NIPPON DATA80 - Okuda 2015	9,112	385	2.4%	0.81 [0.60, 1.09]
Migrant Study - Hjartaker 2015	9,766	1,034	5.4%	0.95 [0.78, 1.16]
Linxian Nutrition - Wang 2016	2,445	452	60.0%	1.01 [0.95, 1.07]
HAPIEE - Stefler 2016	19,263	109	0.6%	0.69 [0.38, 1.24]
PLSAW - Blekkenhorst 2017	1,226	92	3.2%	0.80 [0.62, 1.04]
Total (95% CI)	**780,441**	**7,551**	**100.0%**	**0.94 [0.90, 0.99]**

Heterogeneity: Chi² = 34.54, df = 13 (P = 0.0010); I² = 62%

Test for overall effect: Z = 2.58 (P = 0.010)



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B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Life Span Study - Sauvaget 2003 - M	14,966	692	9.0%	0.77 [0.62, 0.96]
Life Span Study - Sauvaget 2003 - F	23,471	1,234	10.3%	0.81 [0.68, 0.97]
Boyd Orr Cohort - Ness 2005	4,028	83	1.7%	0.40 [0.19, 0.84]
Miyako Study - Pham 2007	9,651	226	7.2%	1.00 [0.76, 1.32]
JACC - Nagura 2009	59,485	1,053	9.6%	1.09 [0.90, 1.33]
Multiethnic Cohort - Sharma 2013 - M	78,410	434	5.0%	1.01 [0.70, 1.47]
Multiethnic Cohort - Sharma 2013 - F	95,618	426	4.7%	0.79 [0.53, 1.16]
EPIC - Leenders 2014	451,151	1,291	9.6%	0.68 [0.56, 0.82]
MONICA Danish - Tognon 2014	1,849	40	2.3%	0.90 [0.48, 1.68]
NIPPON DATA80 - Okuda 2015	9,112	385	6.7%	0.81 [0.60, 1.09]
Migrant Study - Hjartaker 2015	9,766	1,034	9.6%	0.95 [0.78, 1.16]
HAPIEE - Stefler 2016	19,263	109	2.5%	0.69 [0.38, 1.24]
Linxian Nutrition - Wang 2016	2,445	452	14.1%	1.01 [0.95, 1.07]
PLSAW - Blekkenhorst 2017	1,226	92	7.7%	0.80 [0.62, 1.04]
Total (95% CI) [Random Effects]	**780,441**	**7,551**	**100.0%**	**0.86 [0.78, 0.96]**

Heterogeneity: Tau² = 0.02; Chi² = 34.54, df = 13 (P = 0.0010); I² = 62%

Test for overall effect: Z = 2.79 (P = 0.005)

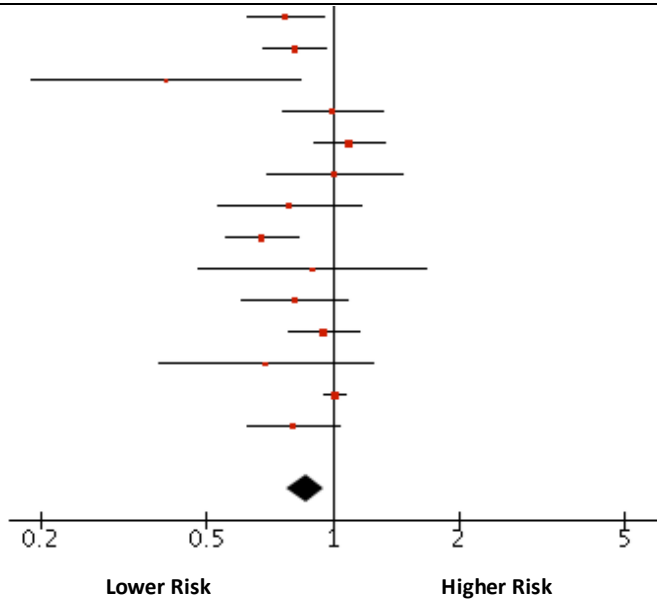


Figure S132. Relation between intake of vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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BANANAS AND STROKE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Migrant Study - Hjartaker 2015 - banana	9,766	1,034	100.0%	1.04 [0.70, 1.54]
Total (95% CI)	**9,766**	**1,034**	**100.0%**	**1.04 [0.70, 1.54]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.20 (P = 0.84)				

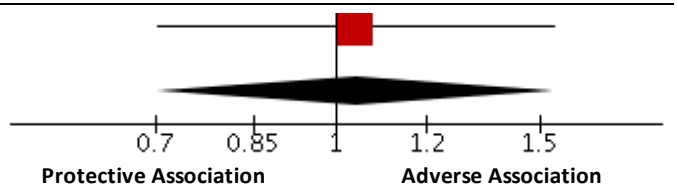


Figure S133. Relation between intake of bananas and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

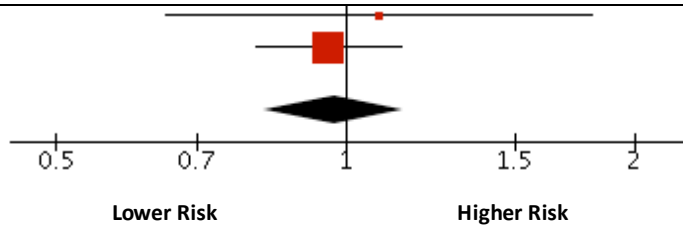
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BERRIES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015	30,458	148	10.7%	1.08 [0.65, 1.80]
Migrant Study - Hjartaker 2015	9,766	1,034	89.3%	0.96 [0.81, 1.15]
Total (95% CI)	**40,224**	**1,182**	**100.0%**	**0.97 [0.82, 1.15]**

Heterogeneity: Chi² = 0.19, df = 1 (P = 0.66); I² = 0%
Test for overall effect: Z = 0.32 (P = 0.75)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015	30,458	148	10.7%	1.08 [0.65, 1.80]
Migrant Study - Hjartaker 2015	9,766	1,034	89.3%	0.96 [0.81, 1.15]
Total (95% CI) [Random Effects]	**40,224**	**1,182**	**100.0%**	**0.97 [0.82, 1.15]**

Heterogeneity: Tau² = 0.00; Chi² = 0.19, df = 1 (P = 0.66); I² = 0%
Test for overall effect: Z = 0.32 (P = 0.75)

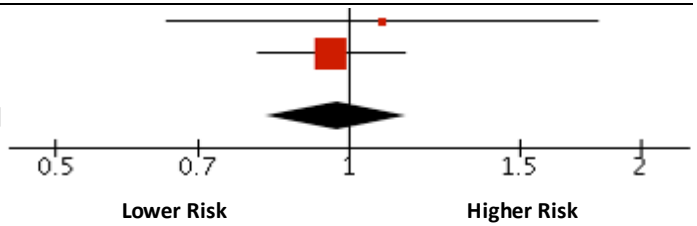


Figure S134. Relation between intake of berries and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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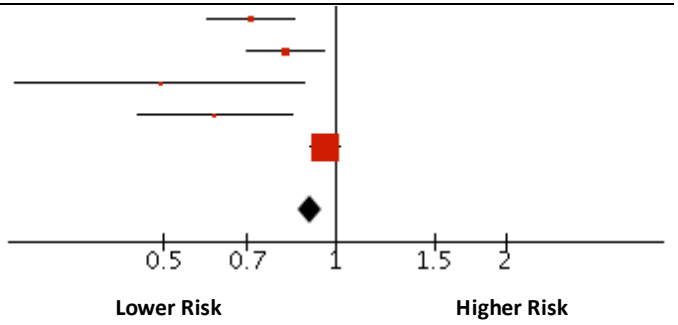
CITRUS FRUIT AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	59,504	1,028	8.6%	0.71 [0.60, 0.85]
JACC - Iso 2007 - M	43,031	1,207	10.8%	0.82 [0.70, 0.96]
UK Women's Cohort - Lai 2015	30,458	148	0.8%	0.49 [0.27, 0.89]
Migrant Study - Hjartaker 2015	9,766	1,034	2.7%	0.61 [0.45, 0.84]
Linxian Nutrition - Wang 2016	2,445	452	77.1%	0.96 [0.91, 1.02]
Total (95% CI)	**145,204**	**3,869**	**100.0%**	**0.90 [0.86, 0.95]**

Heterogeneity: Chi² = 22.75, df = 4 (P = 0.0001); I² = 82%

Test for overall effect: Z = 3.81 (P = 0.0001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - M	43,031	1,207	24.3%	0.82 [0.70, 0.96]
JACC - Iso 2007 - F	59,504	1,028	23.3%	0.71 [0.60, 0.85]
UK Women's Cohort - Lai 2015	30,458	148	7.9%	0.49 [0.27, 0.89]
Migrant Study - Hjartaker 2015	9,766	1,034	16.4%	0.61 [0.45, 0.84]
Linxian Nutrition - Wang 2016	2,445	452	28.1%	0.96 [0.91, 1.02]
Total (95% CI) [Random Effects]	**145,204**	**3,869**	**100.0%**	**0.76 [0.63, 0.92]**

Heterogeneity: Tau² = 0.03; Chi² = 22.75, df = 4 (P = 0.0001); I² = 82%

Test for overall effect: Z = 2.79 (P = 0.005)

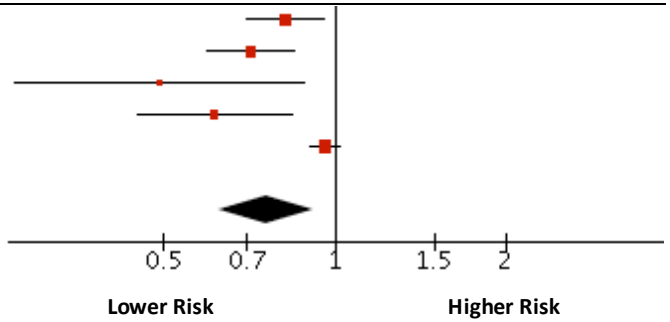


Figure S135. Relation between intake of citrus fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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DRIED FRUIT AND STROKE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015	30,458	152	100.0%	0.95 [0.80, 1.13]
Total (95% CI)	**30,458**	**152**	**100.0%**	**0.95 [0.80, 1.13]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.56 (P = 0.58)				

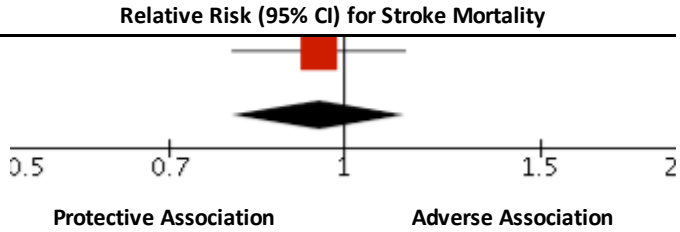


Figure S136. Relation between intake of dried fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

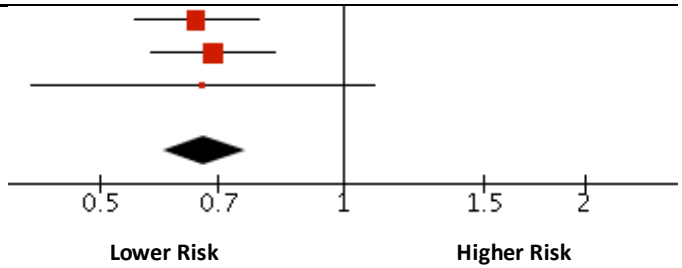
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FRUIT JUICE AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	56,482	953	47.0%	0.66 [0.55, 0.78]
JACC - Iso 2007 - M	41,330	1,131	47.0%	0.69 [0.58, 0.82]
UK Women's Cohort - Lai 2015	30,458	148	6.1%	0.67 [0.41, 1.09]
Total (95% CI)	**128,270**	**2,232**	**100.0%**	**0.67 [0.60, 0.76]**

Heterogeneity: Chi² = 0.15, df = 2 (P = 0.93); I² = 0%
Test for overall effect: Z = 6.41 (P < 0.00001)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	56,482	953	47.0%	0.66 [0.55, 0.78]
JACC - Iso 2007 - M	41,330	1,131	47.0%	0.69 [0.58, 0.82]
UK Women's Cohort - Lai 2015	30,458	148	6.1%	0.67 [0.41, 1.09]
Total (95% CI) [Random Effects]	**128,270**	**2,232**	**100.0%**	**0.67 [0.60, 0.76]**

Heterogeneity: Tau² = 0.00; Chi² = 0.15, df = 2 (P = 0.93); I² = 0%
Test for overall effect: Z = 6.41 (P < 0.00001)

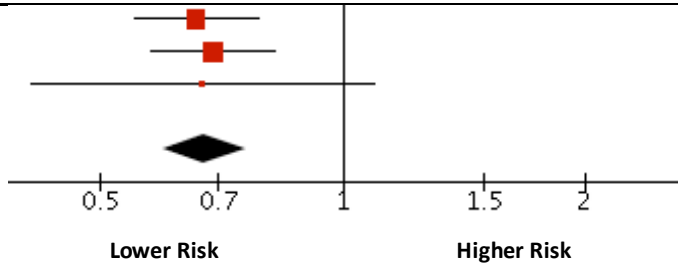


Figure S137. Relation between intake of fruit juice and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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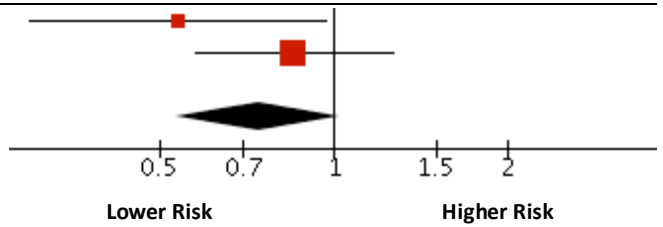
GRAPES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015 - grapes	30,458	148	30.8%	0.54 [0.30, 0.97]
Migrant Study - Hjartaker 2015 - grapes	9,766	1,034	69.2%	0.85 [0.58, 1.26]
Total (95% CI)	**40,224**	**1,182**	**100.0%**	**0.74 [0.53, 1.02]**

Heterogeneity: Chi² = 1.63, df = 1 (P = 0.20); I² = 39%

Test for overall effect: Z = 1.81 (P = 0.07)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015 - grapes	30,458	148	38.2%	0.54 [0.30, 0.97]
Migrant Study - Hjartaker 2015 - grapes	9,766	1,034	61.8%	0.85 [0.58, 1.26]
Total (95% CI) [Random Effects]	**40,224**	**1,182**	**100.0%**	**0.71 [0.46, 1.11]**

Heterogeneity: Tau² = 0.04; Chi² = 1.63, df = 1 (P = 0.20); I² = 39%

Test for overall effect: Z = 1.50 (P = 0.13)

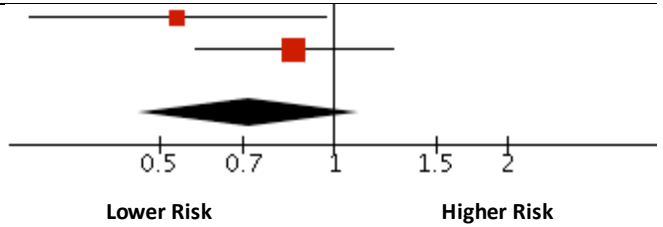


Figure S138. Relation between intake of grapes and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

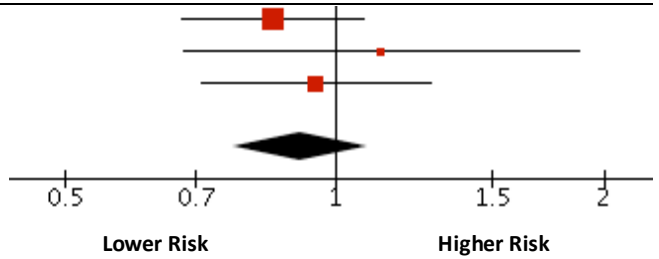
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POMMES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007	34,492	469	54.0%	0.85 [0.67, 1.08]
UK Women's Cohort - Lai 2015	30,458	148	11.5%	1.13 [0.68, 1.88]
Migrant Study - Hjartaker 2015	9,766	1,034	34.5%	0.95 [0.71, 1.28]
Total (95% CI)	**74,716**	**1,651**	**100.0%**	**0.91 [0.77, 1.09]**

Heterogeneity: Chi² = 1.06, df = 2 (P = 0.59); I² = 0%
Test for overall effect: Z = 1.02 (P = 0.31)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Iowa WHS - Mink 2007	34,492	469	54.0%	0.85 [0.67, 1.08]
UK Women's Cohort - Lai 2015	30,458	148	11.5%	1.13 [0.68, 1.88]
Migrant Study - Hjartaker 2015	9,766	1,034	34.5%	0.95 [0.71, 1.28]
Total (95% CI) [Random Effects]	**74,716**	**1,651**	**100.0%**	**0.91 [0.77, 1.09]**

Heterogeneity: Tau² = 0.00; Chi² = 1.06, df = 2 (P = 0.59); I² = 0%
Test for overall effect: Z = 1.02 (P = 0.31)

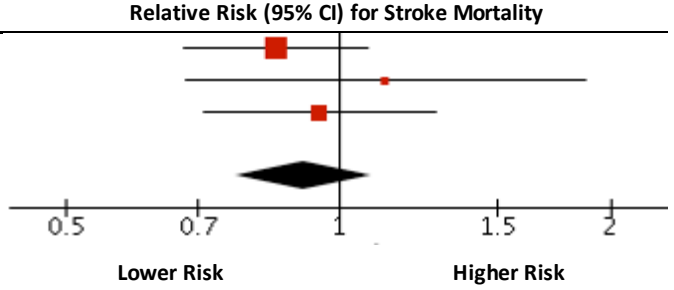


Figure S139. Relation between intake of pommes fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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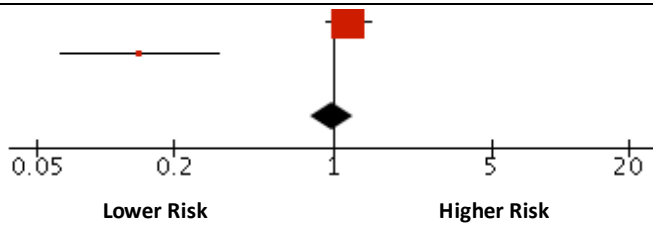
ALLIUM AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Linxian Nutrition - Wang 2016	2,445	452	51.7%	1.17 [0.93, 1.48]
PLSAW - Blekkenhorst 2017	1,226	92	48.3%	0.14 [0.06, 0.31]
Total (95% CI)	**3,671**	**544**	**100.0%**	**0.99 [0.79, 1.24]**

Heterogeneity: Chi² = 24.86, df = 1 (P < 0.00001); I² = 96%

Test for overall effect: Z = 0.07 (P = 0.94)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Linxian Nutrition - Wang 2016	2,445	452	51.7%	1.17 [0.93, 1.48]
PLSAW - Blekkenhorst 2017	1,226	92	48.3%	0.14 [0.06, 0.31]
Total (95% CI) [Random Effects]	**3,671**	**544**	**100.0%**	**0.42 [0.05, 3.38]**

Heterogeneity: Tau² = 2.18; Chi² = 24.86, df = 1 (P < 0.00001); I² = 96%

Test for overall effect: Z = 0.82 (P = 0.41)

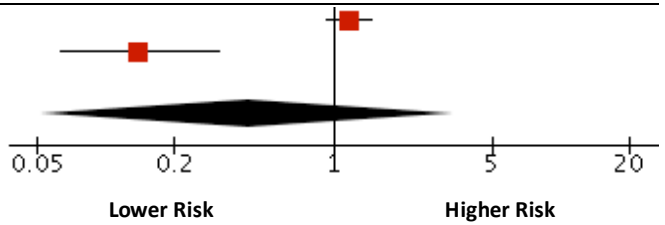


Figure S140. Relation between intake of allium vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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CARROTS AND STROKE MORTALITY

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Miigrant Study - Hjartaker 2015 - carrots	9,766	1,034	100.0%	0.54 [0.48, 0.61]
Total (95% CI)	**9,766**	**1,034**	**100.0%**	**0.54 [0.48, 0.61]**

Heterogeneity: Not applicable
Test for overall effect: Z = 10.33 (P < 0.00001)

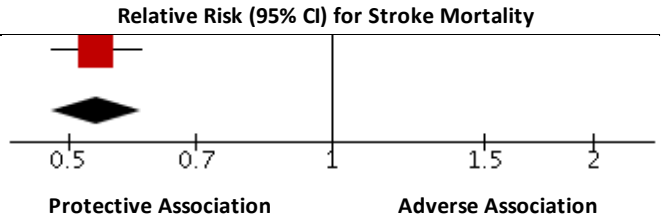


Figure S141. Relation between intake of carrots and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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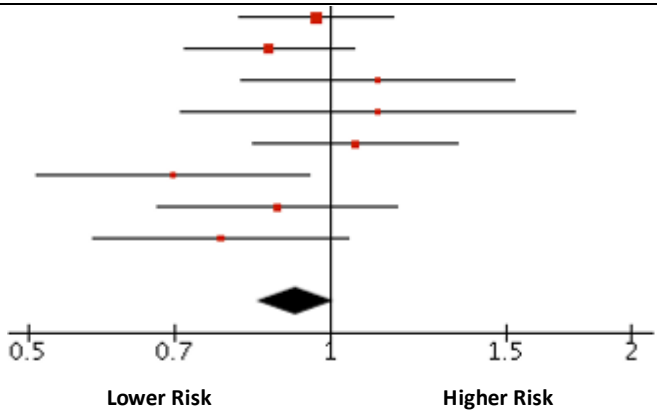
CRUCIFEROUS VEGETABLES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - M	39,486	1,098	25.4%	0.97 [0.81, 1.16]
JACC - Iso 2007 - F	54,325	919	20.6%	0.87 [0.71, 1.06]
Migrant Study - Hjartaker 2015 - cauliflower	9,766	1,034	8.0%	1.12 [0.82, 1.53]
Migrant Study - Hjartaker 2015 - cabbage	-	-	3.9%	1.12 [0.71, 1.75]
Linxian Nutrition - Wang 2016	2,445	452	14.3%	1.06 [0.84, 1.34]
PLSAW - Blekkenhorst 2017	1,226	92	8.0%	0.70 [0.51, 0.95]
Japan Public Health Center - Mori 2018 - M	40,642	856	10.5%	0.89 [0.67, 1.17]
Japan Public Health Center - Mori 2018 - F	47,562	614	9.2%	0.78 [0.58, 1.04]
Total (95% CI)	**195,452**	**5,065**	**100.0%**	**0.92 [0.85, 1.01]**

Heterogeneity: Chi² = 8.55, df = 7 (P = 0.29); I² = 18%

Test for overall effect: Z = 1.75 (P = 0.08)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - M	39,486	1,098	22.2%	0.97 [0.81, 1.16]
JACC - Iso 2007 - F	54,325	919	19.2%	0.87 [0.71, 1.06]
Migrant Study - Hjartaker 2015 - cabbage	9,766	1,034	4.7%	1.12 [0.71, 1.75]
Migrant Study - Hjartaker 2015 - cauliflower	-	-	9.0%	1.12 [0.82, 1.53]
Linxian Nutrition - Wang 2016	2,445	452	14.5%	1.06 [0.84, 1.34]
PLSAW - Blekkenhorst 2017	1,226	92	9.0%	0.70 [0.51, 0.95]
Japan Public Health Center - Mori 2018 - F	47,562	614	10.1%	0.78 [0.58, 1.04]
Japan Public Health Center - Mori 2018 - M	40,642	856	11.3%	0.89 [0.67, 1.17]
Total (95% CI) [Random Effects]	**195,452**	**5,065**	**100.0%**	**0.92 [0.83, 1.02]**

Heterogeneity: Tau² = 0.00; Chi² = 8.55, df = 7 (P = 0.29); I² = 18%

Test for overall effect: Z = 1.57 (P = 0.12)

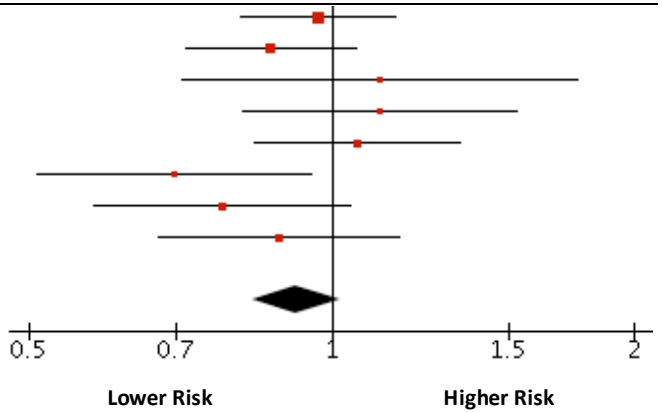


Figure S142. Relation between intake of cruciferous vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq$ 50% indicating substantial heterogeneity.

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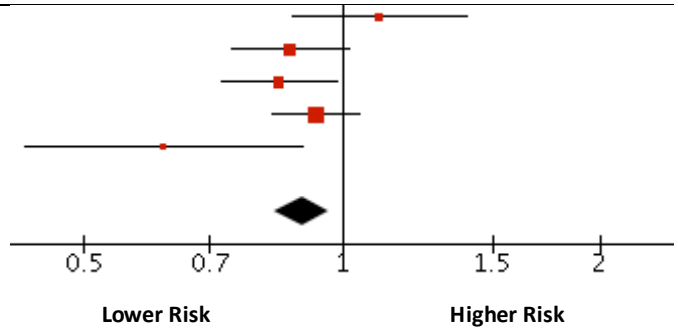
GREEN LEAFY VEGETABLES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Health Food Shoppers - Appleby 2002	10,741	356	10.1%	1.11 [0.87, 1.40]
JACC - Iso 2007 - M	43,850	1,229	22.7%	0.87 [0.74, 1.02]
JACC - Iso 2007 - F	60,169	1,032	22.7%	0.84 [0.72, 0.99]
Migrant Study - Hjartaker 2015	9,766	1,034	40.4%	0.93 [0.83, 1.05]
Linxian Nutrition - Wang 2016	2,445	452	4.0%	0.62 [0.43, 0.90]
Total (95% CI)	**126,971**	**4,103**	**100.0%**	**0.90 [0.83, 0.97]**

Heterogeneity: Chi² = 8.00, df = 4 (P = 0.09); I² = 50%

Test for overall effect: Z = 2.83 (P = 0.005)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Health Food Shoppers - Appleby 2002	10,741	356	15.3%	1.11 [0.87, 1.40]
JACC - Iso 2007 - M	43,850	1,229	23.7%	0.87 [0.74, 1.02]
JACC - Iso 2007 - F	60,169	1,032	23.7%	0.84 [0.72, 0.99]
Migrant Study - Hjartaker 2015	9,766	1,034	29.4%	0.93 [0.83, 1.05]
Linxian Nutrition - Wang 2016	2,445	452	7.8%	0.62 [0.43, 0.90]
Total (95% CI) [Random Effects]	**126,971**	**4,103**	**100.0%**	**0.89 [0.79, 1.00]**

Heterogeneity: Tau² = 0.01; Chi² = 8.00, df = 4 (P = 0.09); I² = 50%

Test for overall effect: Z = 1.98 (P = 0.05)

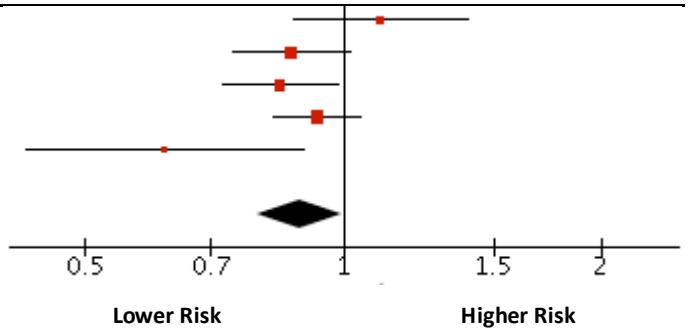


Figure S143. Relation between intake of green leafy vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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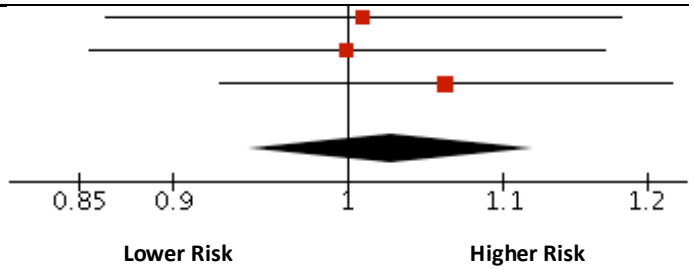
TOMATOES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	56,947	947	30.20%	1.01 [0.86, 1.18]
JACC - Iso 2007 - M	41,547	1,126	30.20%	1.00 [0.85, 1.17]
Migrant Study - Hjartaker 2015	9,766	1,034	39.50%	1.06 [0.93, 1.22]
Total (95% CI)	**108,260**	**3,107**	**100.00%**	**1.03 [0.94, 1.12]**

Heterogeneity: Chi² = 0.38, df = 2 (P = 0.83); I² = 0%

Test for overall effect: Z = 0.61 (P = 0.54)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - F	56,947	947	30.20%	1.01 [0.86, 1.18]
JACC - Iso 2007 - M	41,547	1,126	30.20%	1.00 [0.85, 1.17]
Migrant Study - Hjartaker 2015	9,766	1,034	39.50%	1.06 [0.93, 1.22]
Total (95% CI) [Random Effects]	**108,260**	**3,107**	**100.00%**	**1.03 [0.94, 1.12]**

Heterogeneity: Tau² = 0.00; Chi² = 0.38, df = 2 (P = 0.83); I² = 0%

Test for overall effect: Z = 0.61 (P = 0.54)

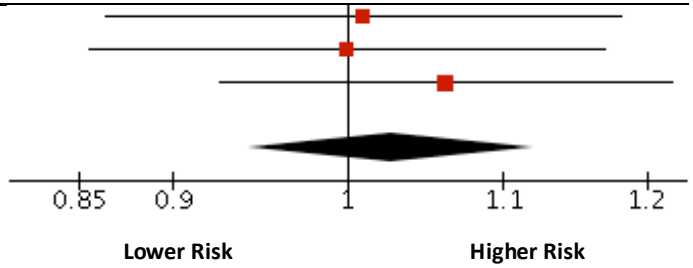
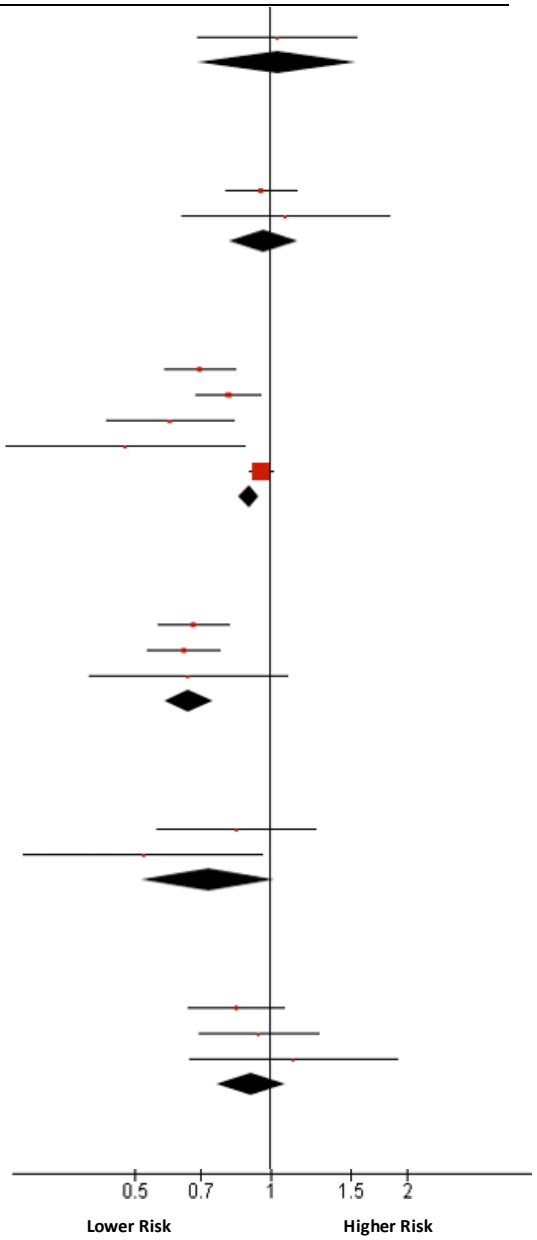


Figure S144. Relation between intake of tomatoes and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Bananas				
Migrant Study - Hjartaker 2015 - banana	9,766	1,034	1.2%	1.04 [0.70, 1.54]
Subtotal (95% CI)	**9,766**	**1,034**	**1.2%**	**1.04 [0.70, 1.54]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 0.20 (P = 0.84)				
Berries				
Migrant Study - Hjartaker 2015	9,766	1,034	6.1%	0.96 [0.81, 1.15]
UK Women's Cohort - Lai 2015	30,458	148	0.7%	1.08 [0.65, 1.80]
Subtotal (95% CI)	**40,224**	**1,182**	**6.8%**	**0.97 [0.82, 1.15]**
Heterogeneity: Tau² = 0.00; Chi² = 0.19, df = 1 (P = 0.66); I² = 0%				
Test for overall effect: Z = 0.32 (P = 0.75)				
Citrus				
JACC - Iso 2007 - F	59,504	1,028	6.1%	0.71 [0.60, 0.85]
JACC - Iso 2007 - M	43,031	1,207	7.7%	0.82 [0.70, 0.96]
Migrant Study - Hjartaker 2015	9,766	1,034	1.9%	0.61 [0.45, 0.84]
UK Women's Cohort - Lai 2015	30,458	148	0.5%	0.49 [0.27, 0.89]
Linxian Nutrition - Wang 2016	2,445	452	54.7%	0.96 [0.91, 1.02]
Subtotal (95% CI)	**145,204**	**3,869**	**70.9%**	**0.90 [0.86, 0.95]**
Heterogeneity: Tau² = 0.03; Chi² = 22.75, df = 4 (P = 0.0001); I² = 82%				
Test for overall effect: Z = 2.79 (P = 0.005)				
Fruit Juice				
JACC - Iso 2007 - M	41,330	1,131	6.1%	0.69 [0.58, 0.82]
JACC - Iso 2007 - F	56,482	953	6.1%	0.66 [0.55, 0.78]
UK Women's Cohort - Lai 2015	30,458	148	0.8%	0.67 [0.41, 1.09]
Subtotal (95% CI)	**128,270**	**2,232**	**12.9%**	**0.67 [0.60, 0.76]**
Heterogeneity: Tau² = 0.00; Chi² = 0.15, df = 2 (P = 0.93); I² = 0%				
Test for overall effect: Z = 6.41 (P < 0.00001)				
Grapes				
Migrant Study - Hjartaker 2015 - grapes	9,766	1,034	1.2%	0.85 [0.58, 1.26]
UK Women's Cohort - Lai 2015 - grapes	30,458	148	0.5%	0.54 [0.30, 0.97]
Subtotal (95% CI)	**40,224**	**1,182**	**1.8%**	**0.74 [0.53, 1.02]**
Heterogeneity: Tau² = 0.04; Chi² = 1.63, df = 1 (P = 0.20); I² = 39%				
Test for overall effect: Z = 1.50 (P = 0.13)				
Pommes				
Iowa WHS - Mink 2007	34,492	469	3.4%	0.85 [0.67, 1.08]
Migrant Study - Hjartaker 2015	9,766	1,034	2.2%	0.95 [0.71, 1.28]
UK Women's Cohort - Lai 2015	30,458	148	0.7%	1.13 [0.68, 1.88]
Subtotal (95% CI)	**74,716**	**1,651**	**6.3%**	**0.91 [0.77, 1.09]**
Heterogeneity: Tau² = 0.00; Chi² = 1.06, df = 2 (P = 0.59); I² = 0%				
Test for overall effect: Z = 1.02 (P = 0.31)				
Test for subgroup differences: Chi² = 23.18, df = 5 (P = 0.0003), I² = 78.4%				



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B. Random Effects

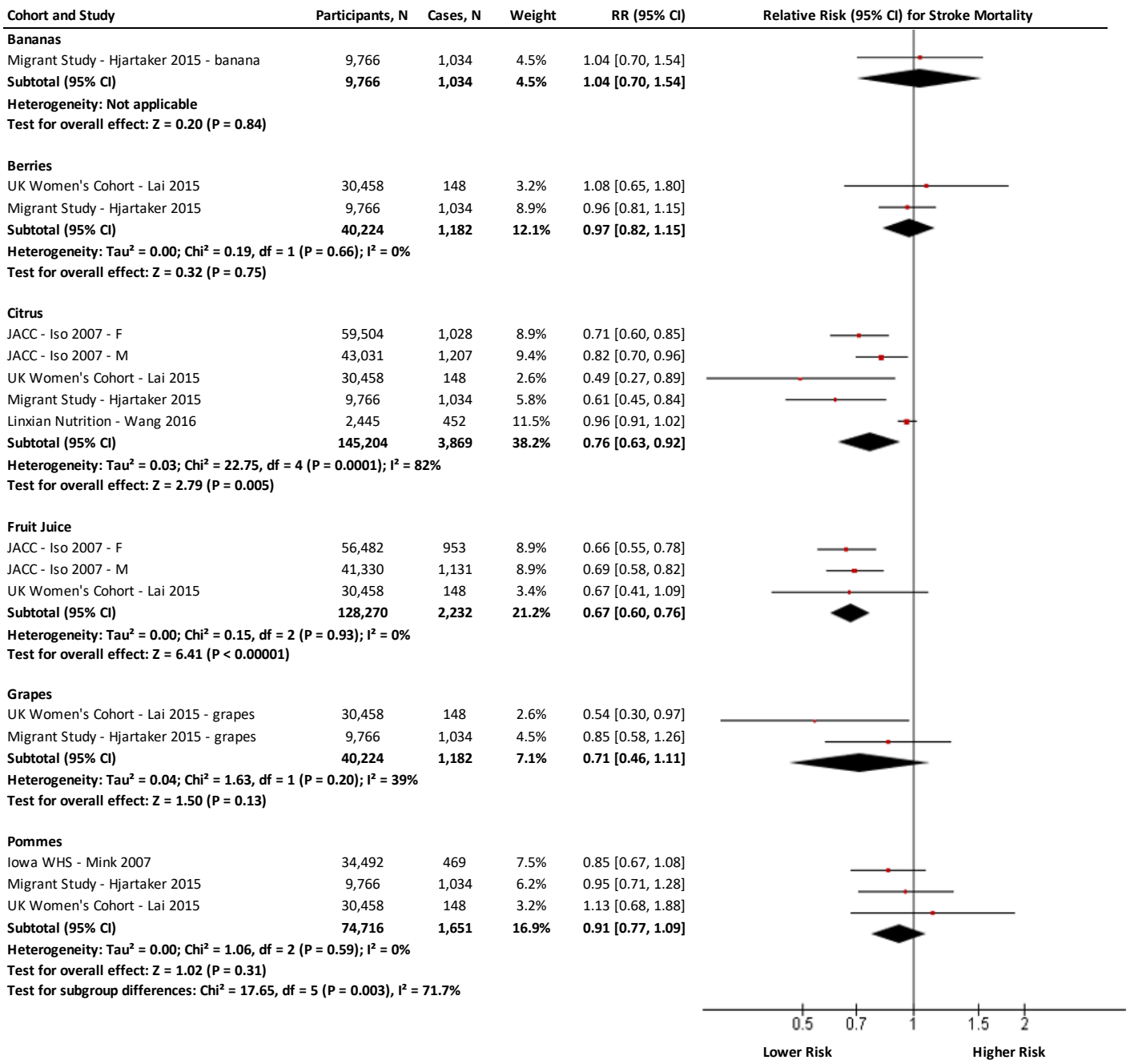
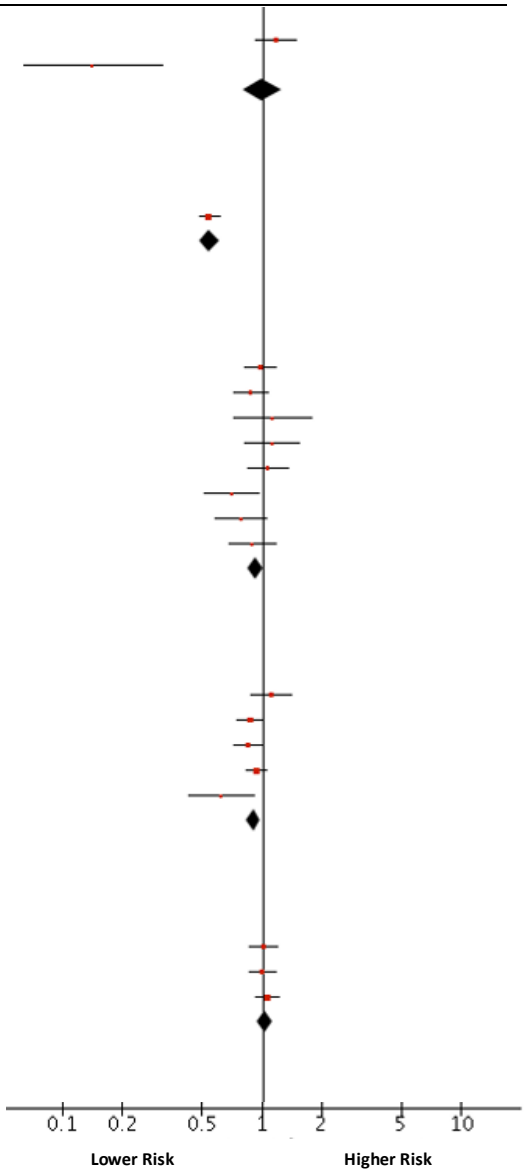


Figure S145. Relation between sources of fruit and stoke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values ≥ 50% indicating substantial heterogeneity.

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A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Allium				
Linxian Nutrition - Wang 2016	2,445	452	3.4%	1.17 [0.93, 1.48]
PLSAW - Blekkenhorst 2017	1,226	92	0.3%	0.14 [0.06, 0.31]
Subtotal (95% CI)	**3,671**	**544**	**3.7%**	**0.99 [0.79, 1.24]**
Heterogeneity: Chi² = 24.86, df = 1 (P < 0.00001); I² = 96%				
Test for overall effect: Z = 0.07 (P = 0.94)				
Carrots				
Miigrant Study - Hjartaker 2015 - carrots	9,766	1,034	13.6%	0.54 [0.48, 0.61]
Subtotal (95% CI)	**9,766**	**1,034**	**13.6%**	**0.54 [0.48, 0.61]**
Heterogeneity: Not applicable				
Test for overall effect: Z = 10.33 (P < 0.00001)				
Cruciferous				
JACC - Iso 2007 - M	39,486	1,098	6.0%	0.97 [0.81, 1.16]
JACC - Iso 2007 - F	54,325	919	4.9%	0.87 [0.71, 1.06]
Migrant Study - Hjartaker 2015 - cabbage	9,766	1,034	0.9%	1.12 [0.71, 1.75]
Migrant Study - Hjartaker 2015 - cauliflower	-	-	1.9%	1.12 [0.82, 1.53]
Linxian Nutrition - Wang 2016	2,445	452	3.4%	1.06 [0.84, 1.34]
PLSAW - Blekkenhorst 2017	1,226	92	1.9%	0.70 [0.51, 0.95]
Japan Public Health Center - Mori 2018 - F	47,562	614	2.2%	0.78 [0.58, 1.04]
Japan Public Health Center - Mori 2018 - M	40,642	856	2.5%	0.89 [0.67, 1.17]
Subtotal (95% CI)	**195,452**	**5,065**	**23.8%**	**0.92 [0.85, 1.01]**
Heterogeneity: Chi² = 8.55, df = 7 (P = 0.29); I² = 18%				
Test for overall effect: Z = 1.75 (P = 0.08)				
Green Leafy				
Health Food Shoppers - Appleby 2002	10,741	356	3.4%	1.11 [0.87, 1.40]
JACC - Iso 2007 - M	43,850	1,229	7.6%	0.87 [0.74, 1.02]
JACC - Iso 2007 - F	60,169	1,032	7.6%	0.84 [0.72, 0.99]
Migrant Study - Hjartaker 2015	9,766	1,034	13.6%	0.93 [0.83, 1.05]
Linxian Nutrition - Wang 2016	2,445	452	1.4%	0.62 [0.43, 0.90]
Subtotal (95% CI)	**126,971**	**4,103**	**33.7%**	**0.90 [0.83, 0.97]**
Heterogeneity: Chi² = 8.00, df = 4 (P = 0.09); I² = 50%				
Test for overall effect: Z = 2.83 (P = 0.005)				
Tomatoes				
JACC - Iso 2007 - F	56,947	947	7.6%	1.01 [0.86, 1.18]
JACC - Iso 2007 - M	41,547	1,126	7.6%	1.00 [0.85, 1.17]
Migrant Study - Hjartaker 2015	9,766	1,034	10.0%	1.06 [0.93, 1.22]
Subtotal (95% CI)	**108,260**	**3,107**	**25.3%**	**1.03 [0.94, 1.12]**
Heterogeneity: Chi² = 0.38, df = 2 (P = 0.83); I² = 0%				
Test for overall effect: Z = 0.61 (P = 0.54)				
Test for subgroup differences: Chi² = 82.07, df = 4 (P < 0.00001), I² = 95.1%				



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B. Random Effects

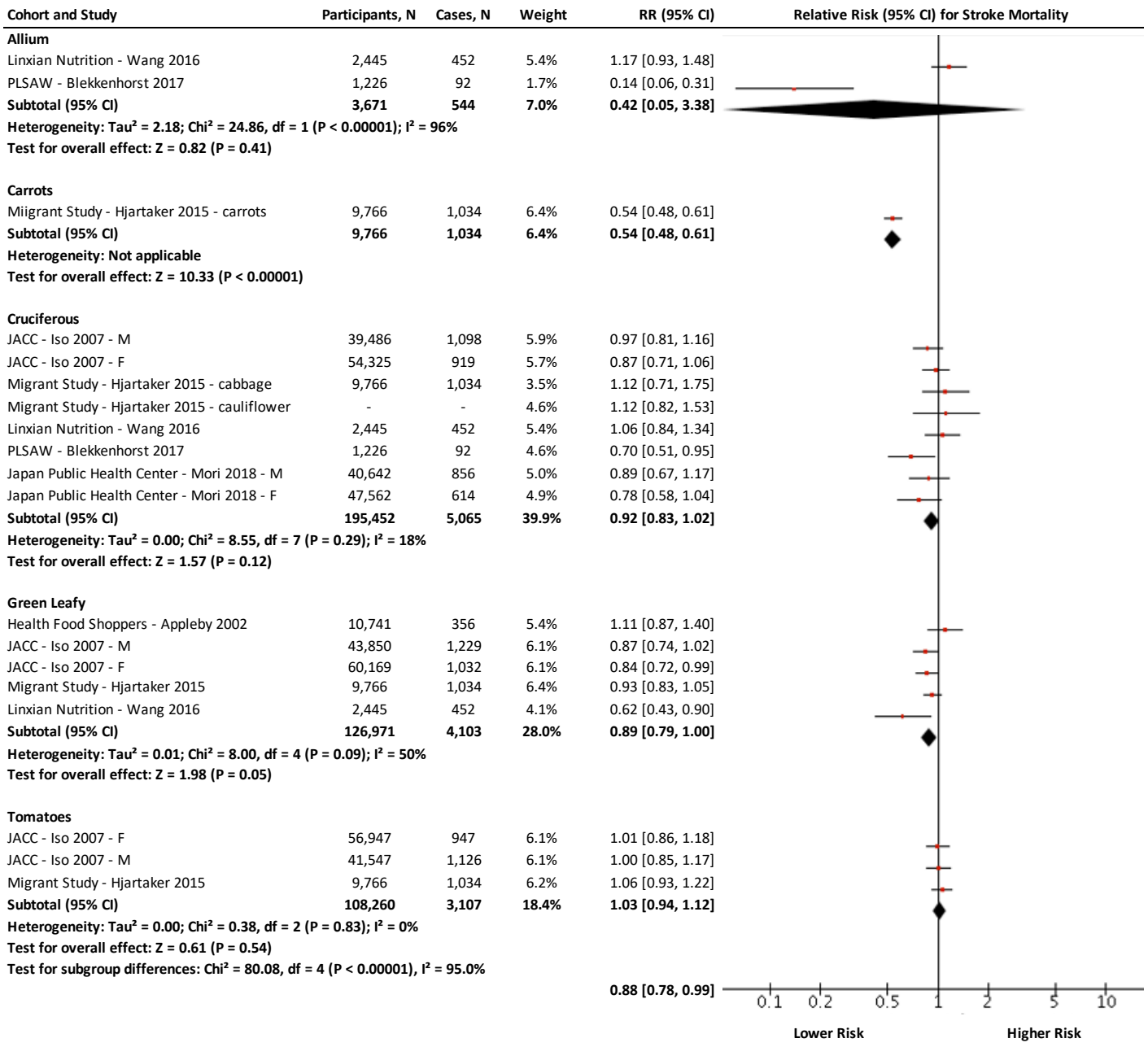


Figure S146. Relation between sources of vegetables and stoke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p<0.10$, and quantified by I^2, with values $\geq 50\%$ indicating substantial heterogeneity.

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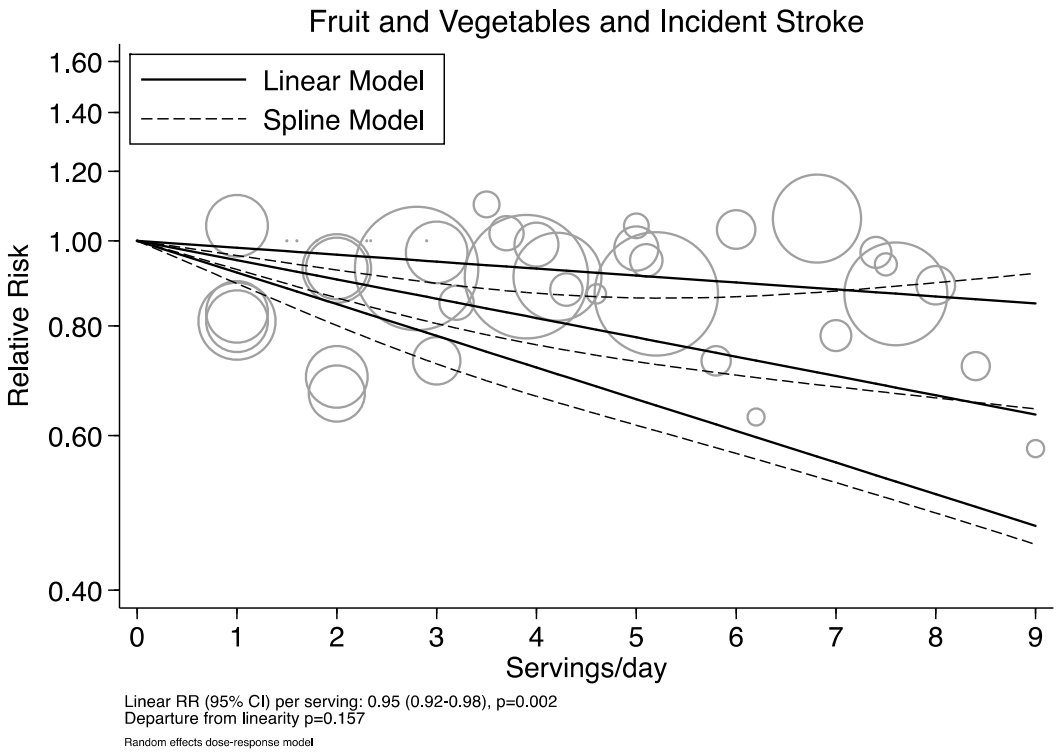


Figure S147. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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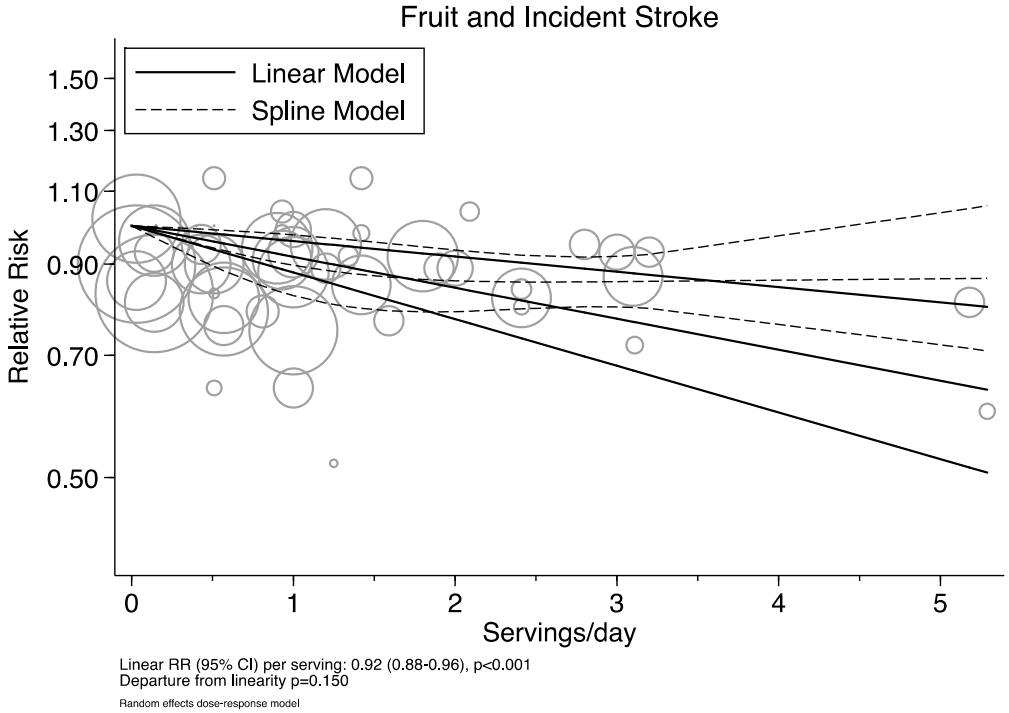


Figure S148. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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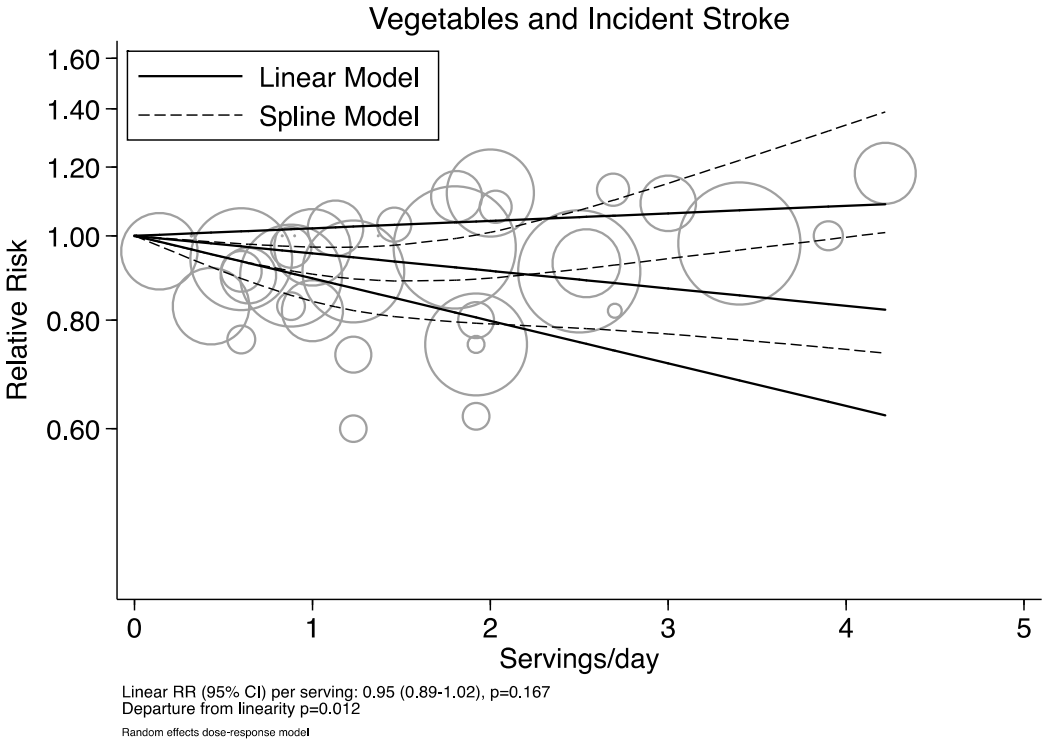


Figure S149. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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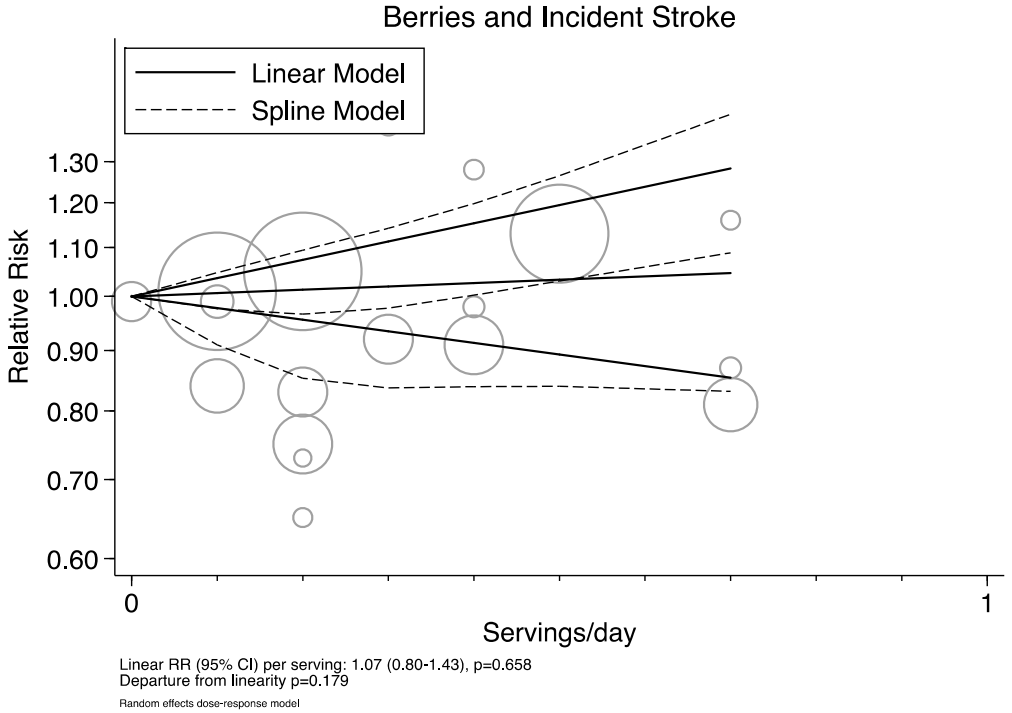


Figure S150. Linear and cubic-spline dose-response relation between increasing berries intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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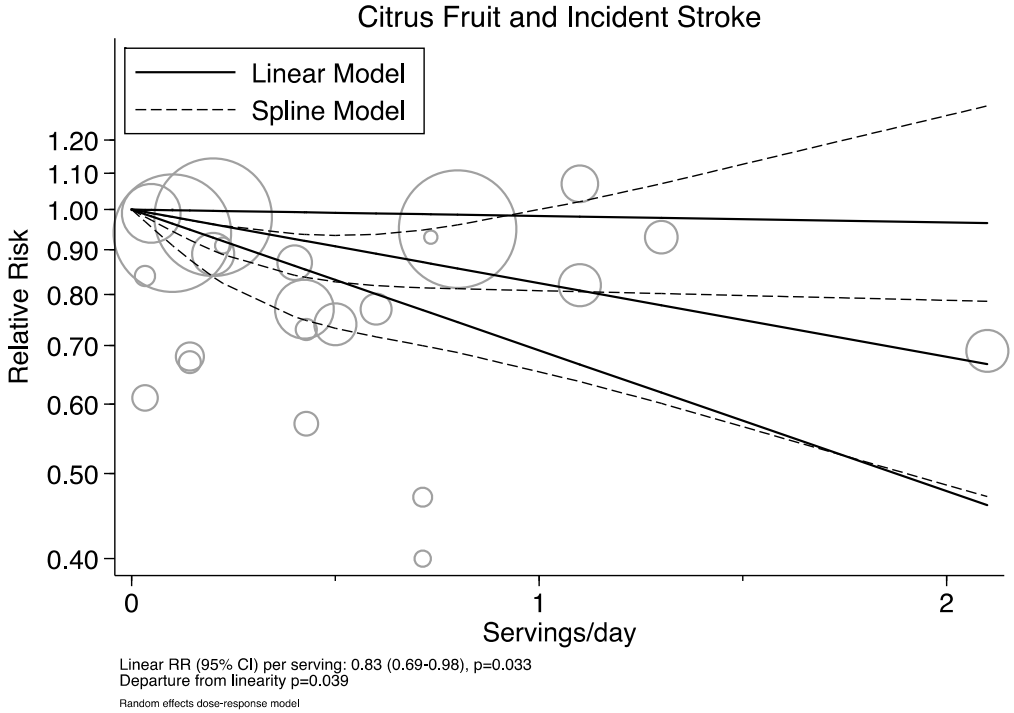


Figure S151. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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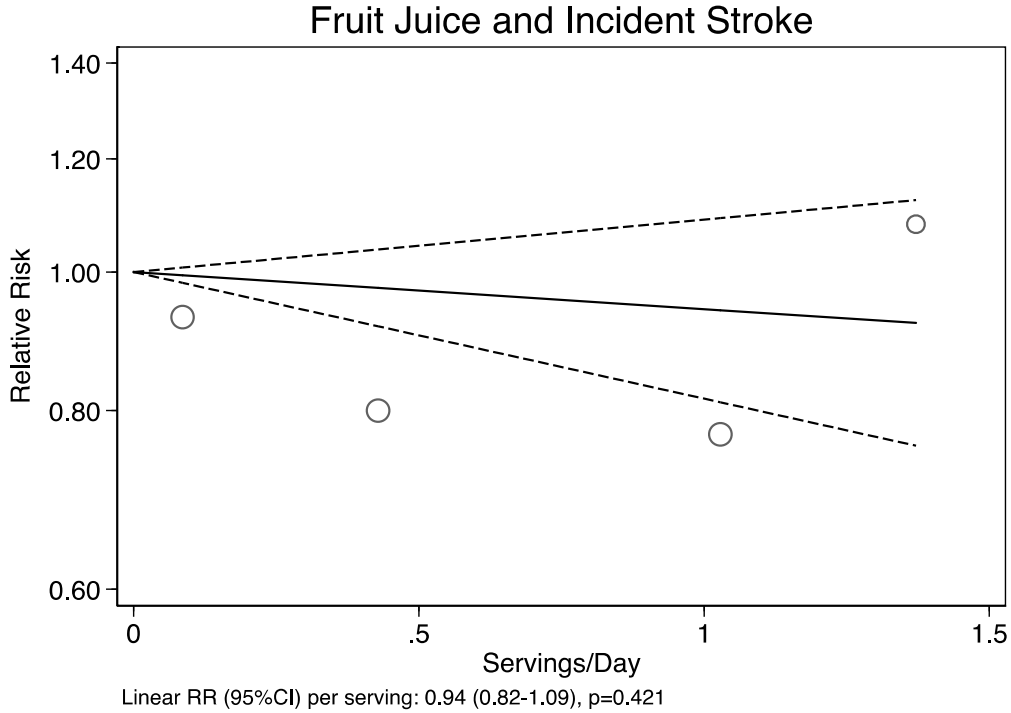


Figure S152. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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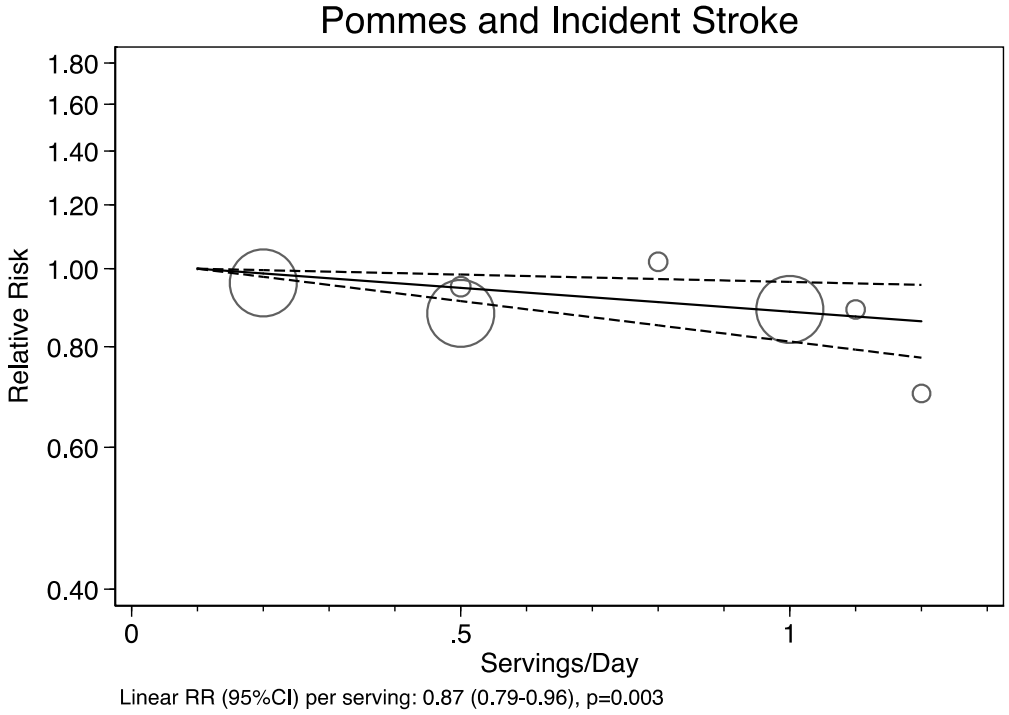


Figure S153. Linear dose-response relation between increasing pommes intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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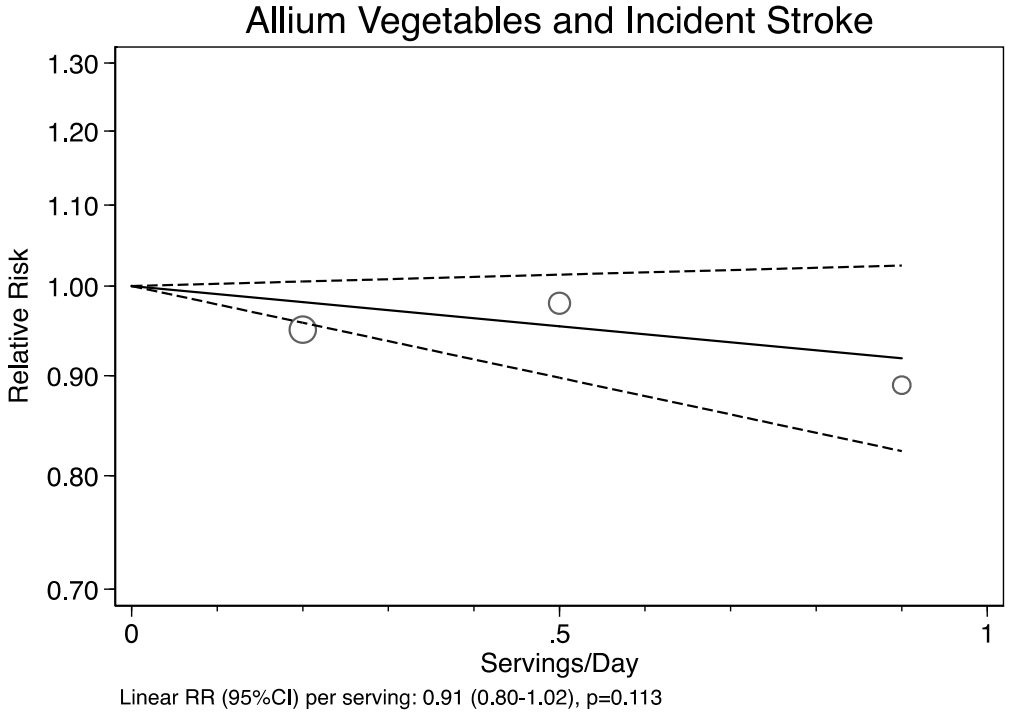


Figure S154 Linear dose-response relation between increasing intake of allium vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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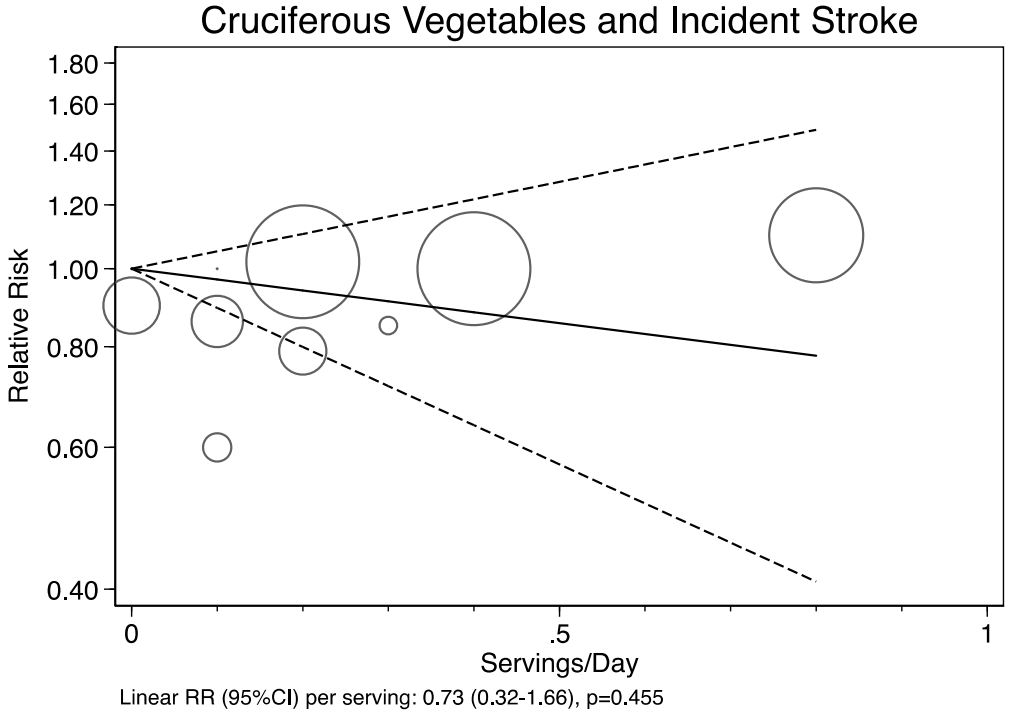


Figure S155. Linear dose-response relation between increasing intake of cruciferous vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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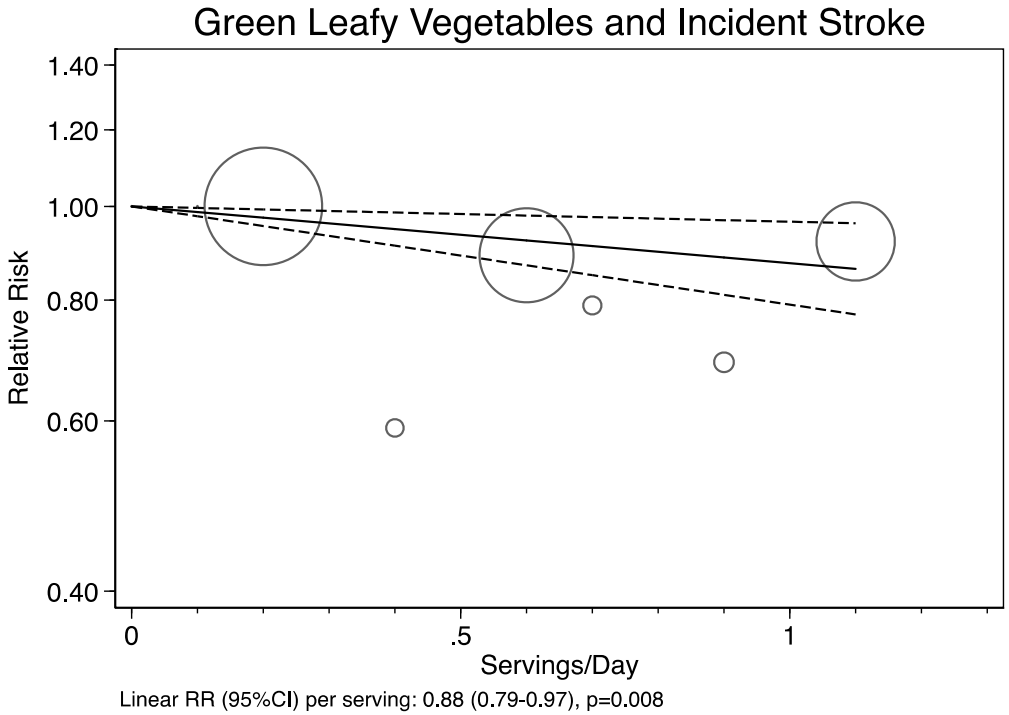


Figure S156. Linear dose-response relation between increasing intake of green leafy vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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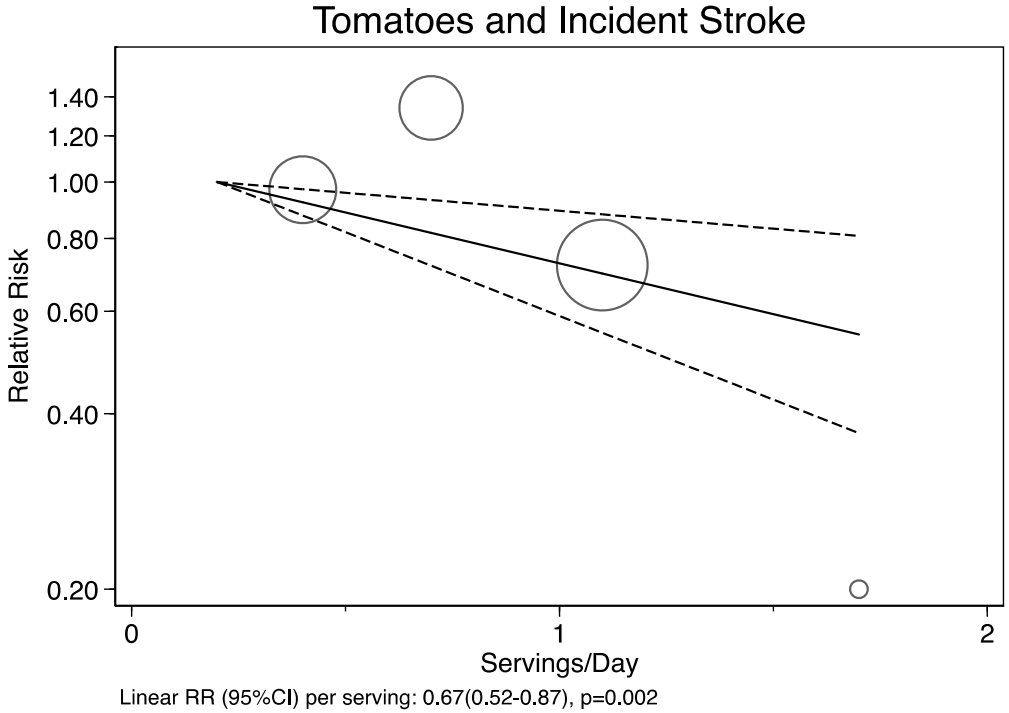


Figure S157. Linear dose-response relation between increasing tomato intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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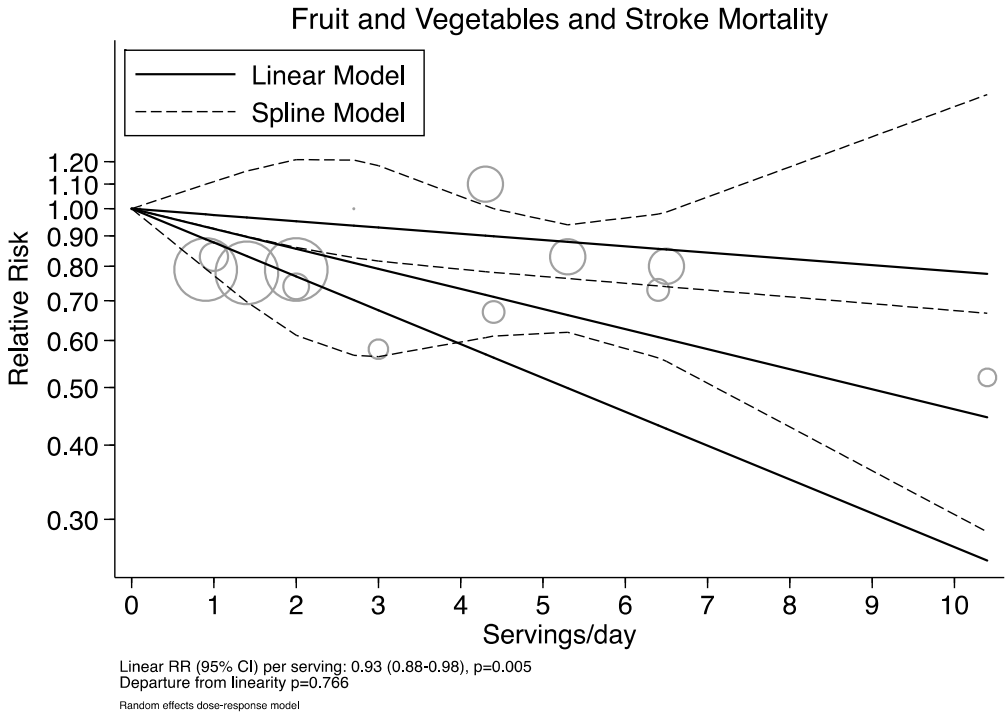


Figure S158. Linear dose-response relation between increasing fruit and vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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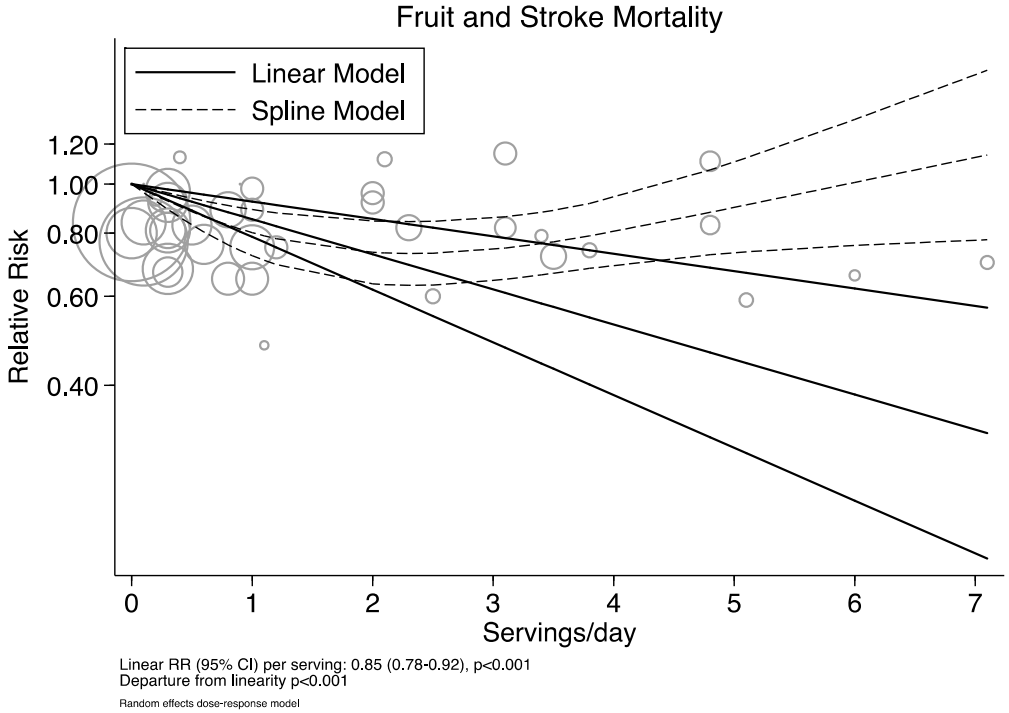


Figure S159. Linear and cubic-spline dose-response relation between increasing fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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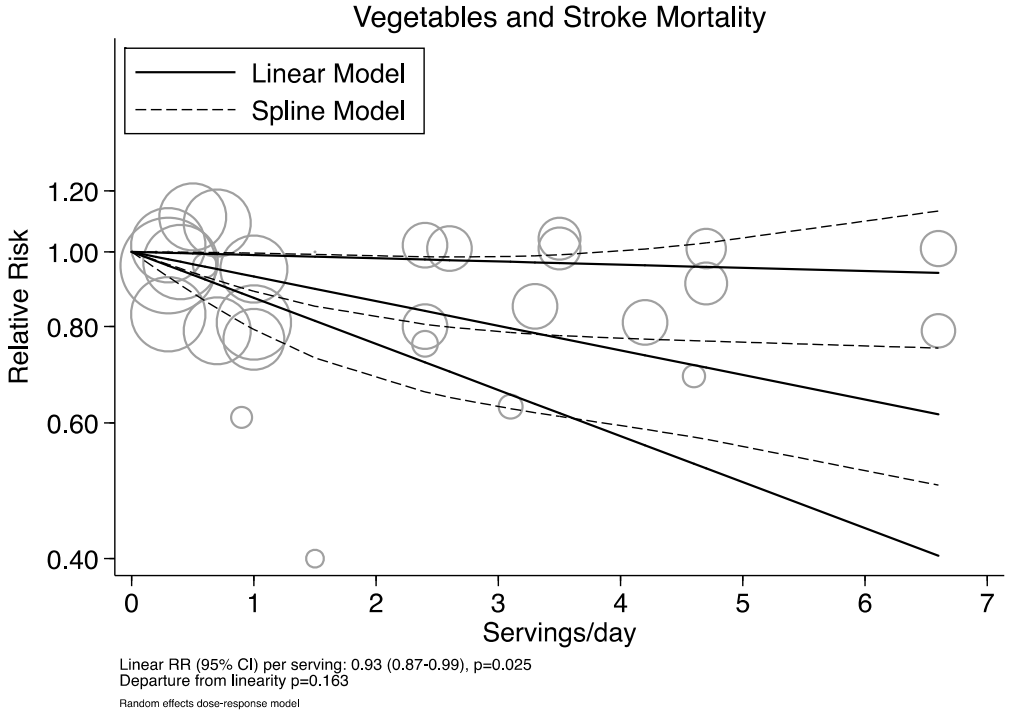


Figure S160. Linear and cubic-spline dose-response relation between increasing intake of vegetables and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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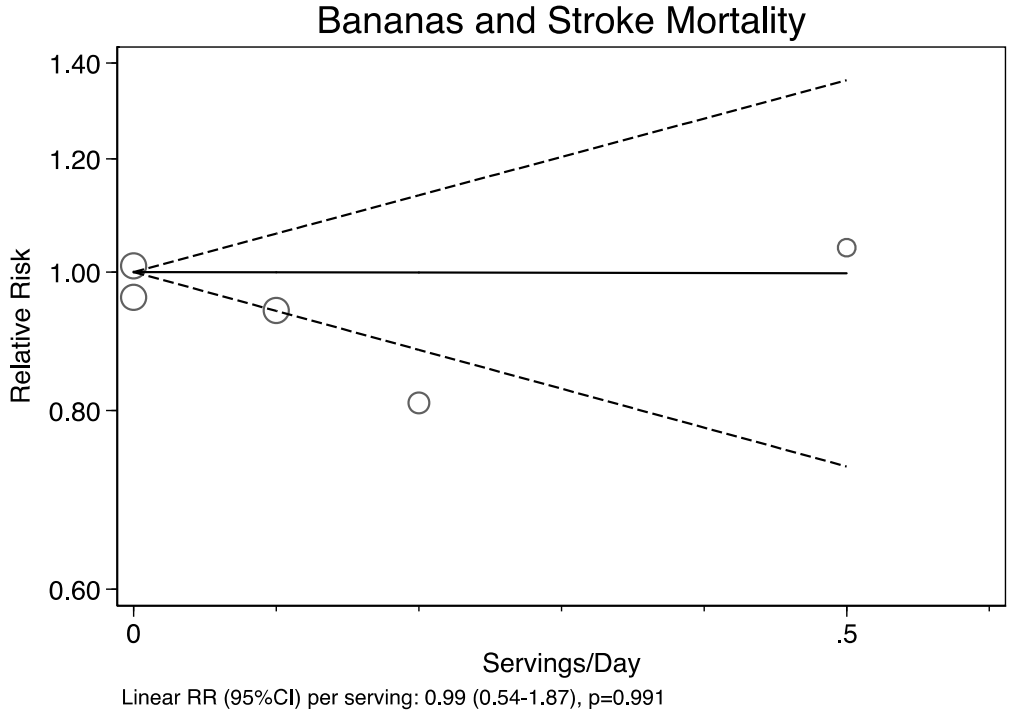


Figure S161 Linear dose-response relation between increasing banana intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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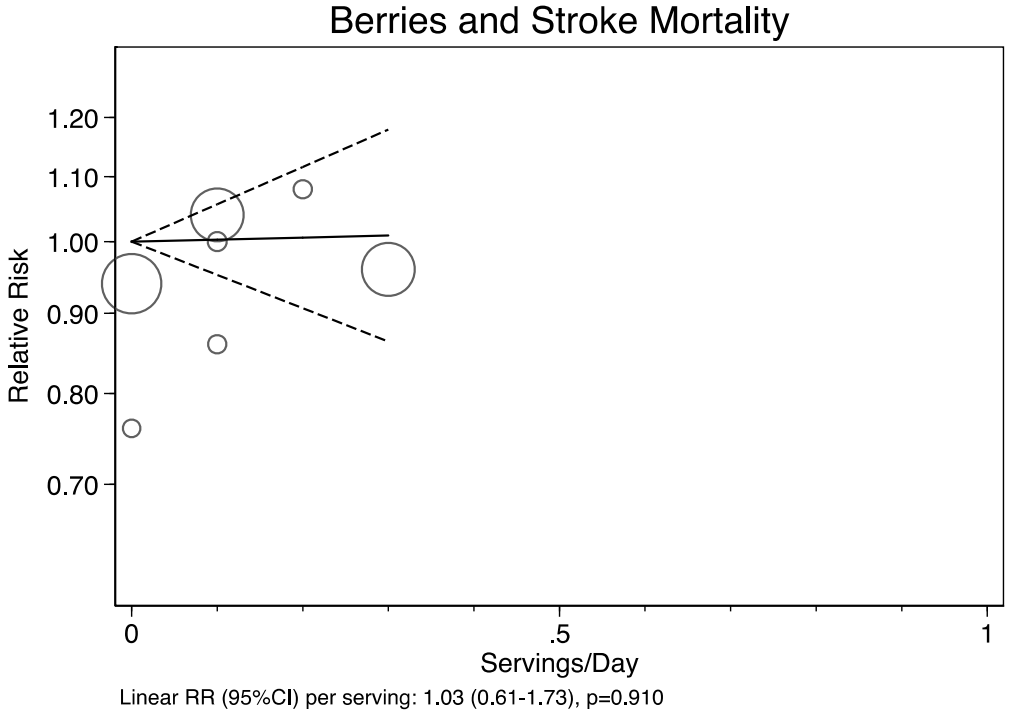


Figure S162. Linear dose-response relation between increasing berries intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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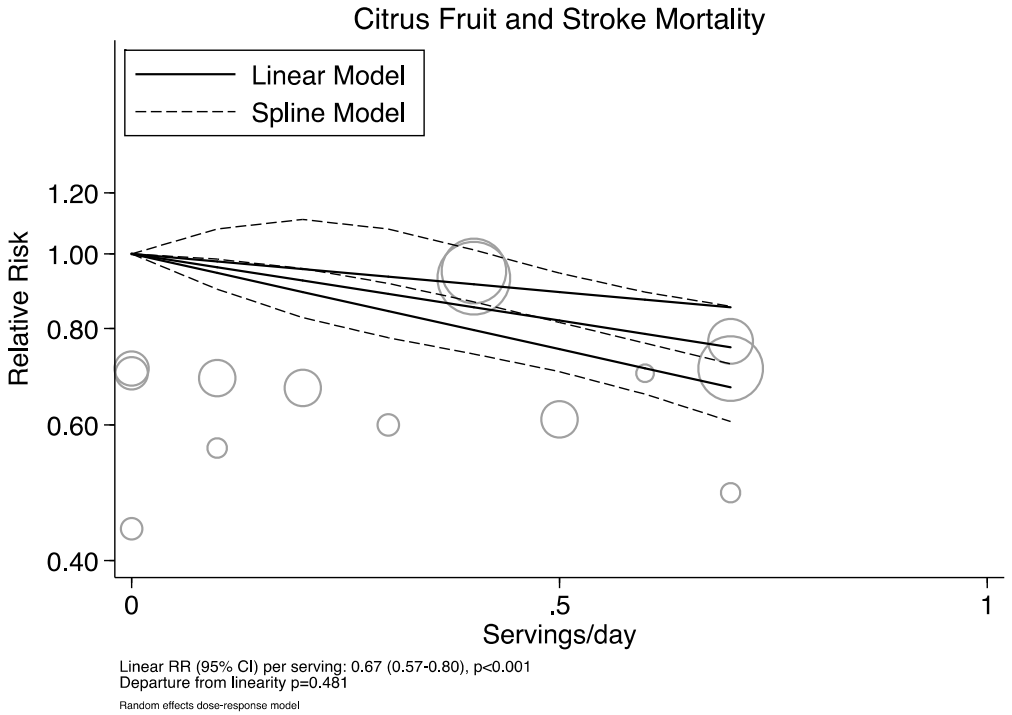


Figure S163. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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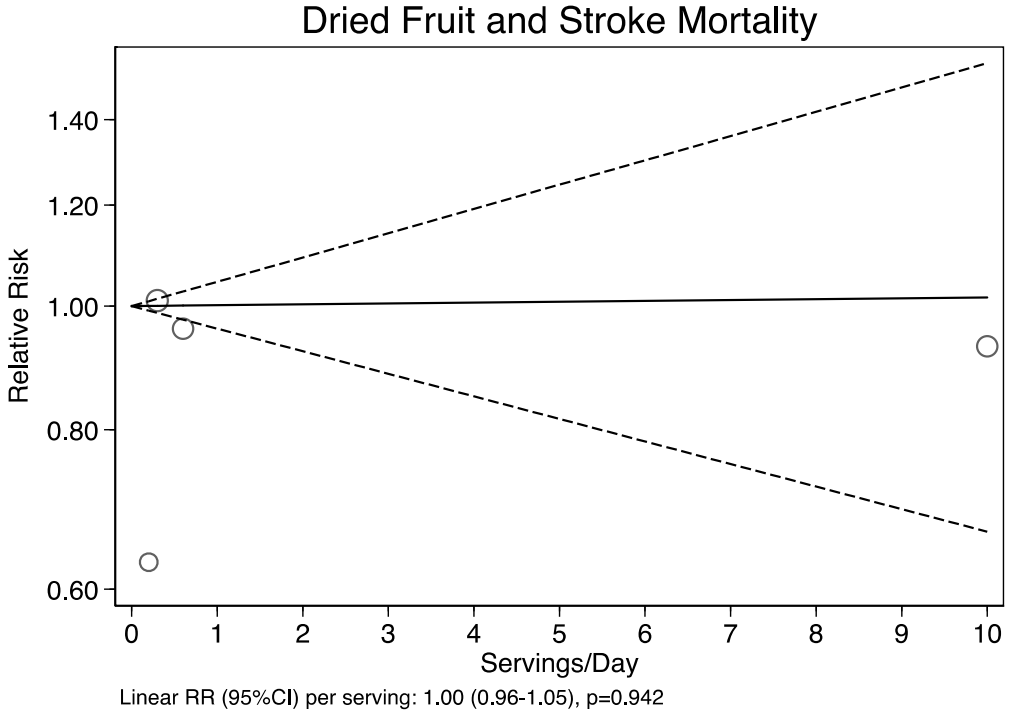


Figure S164. Linear and cubic-spline dose-response relation between increasing dried fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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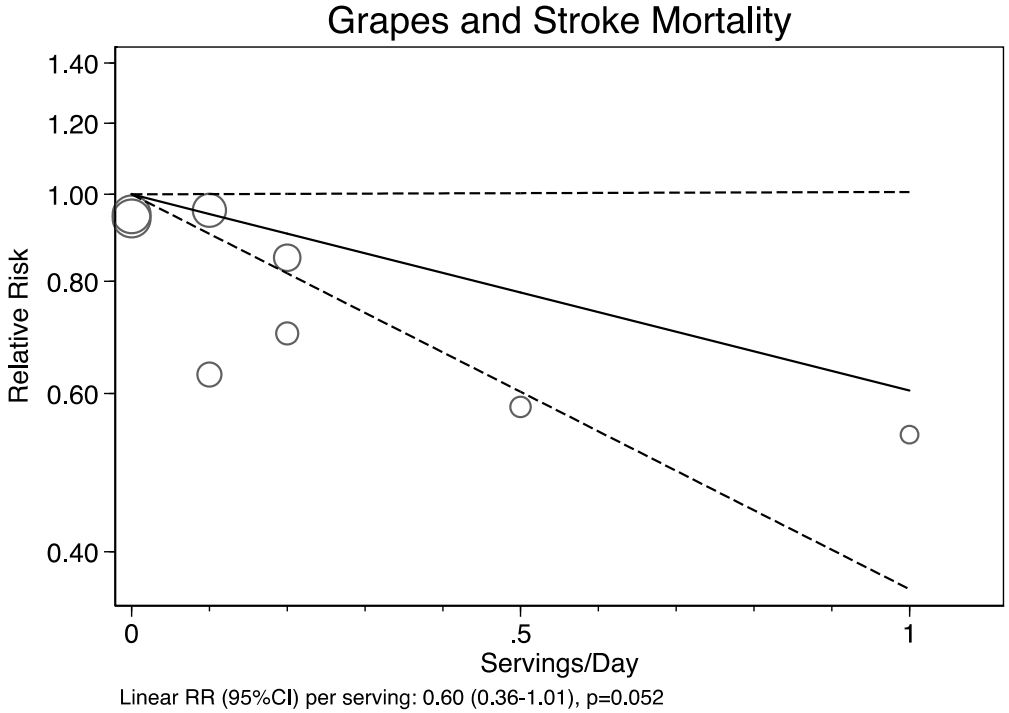


Figure S165. Linear dose-response relation between increasing grapes intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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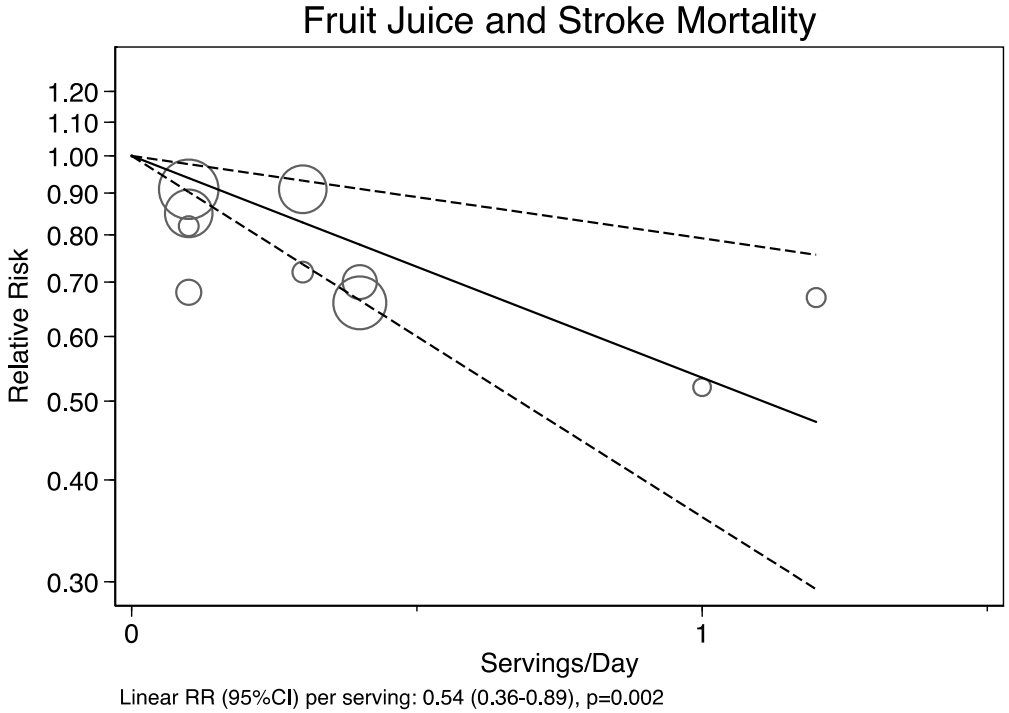


Figure S166. Linear dose-response relation between increasing fruit juice intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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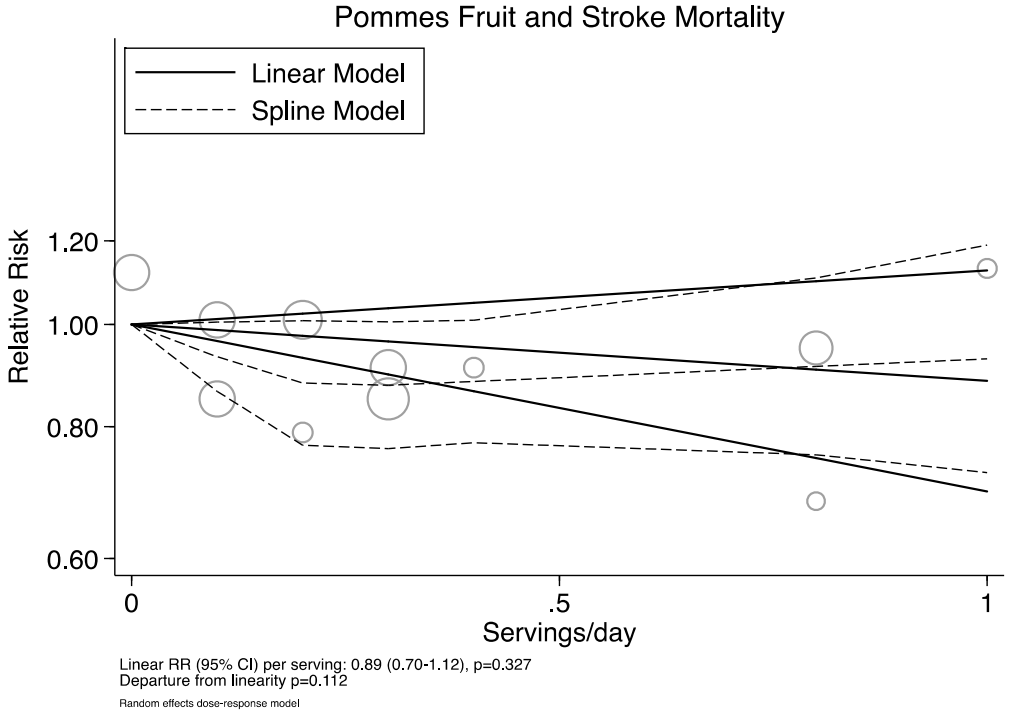


Figure S167. Linear and cubic-spline dose-response relation between increasing pomme fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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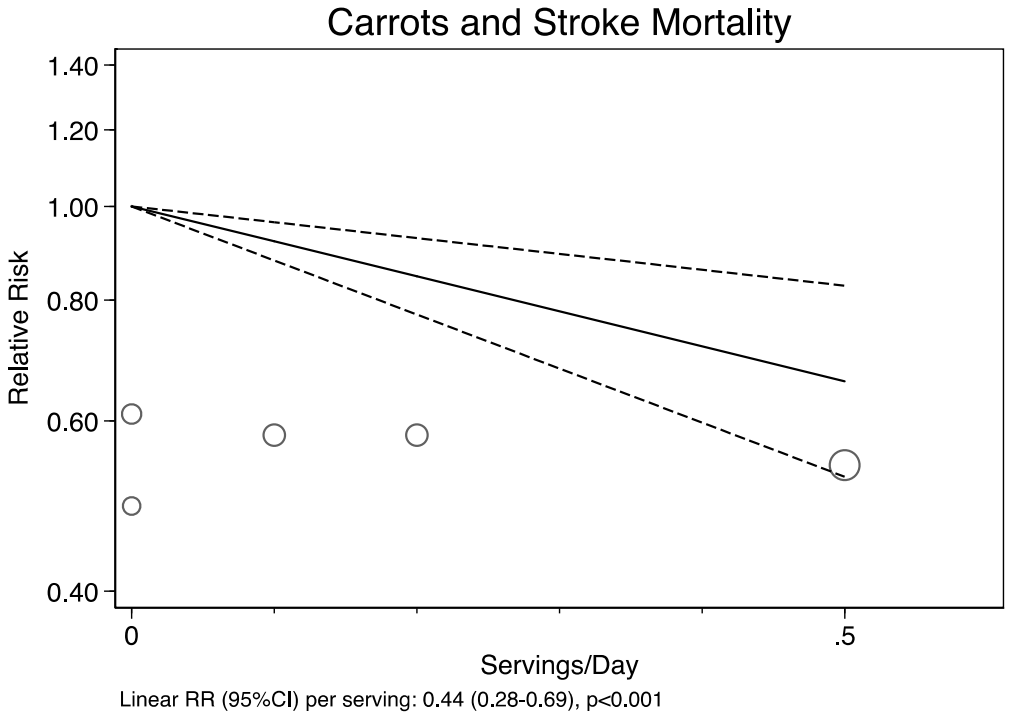


Figure S168. Linear dose-response relation between increasing intake of carrots and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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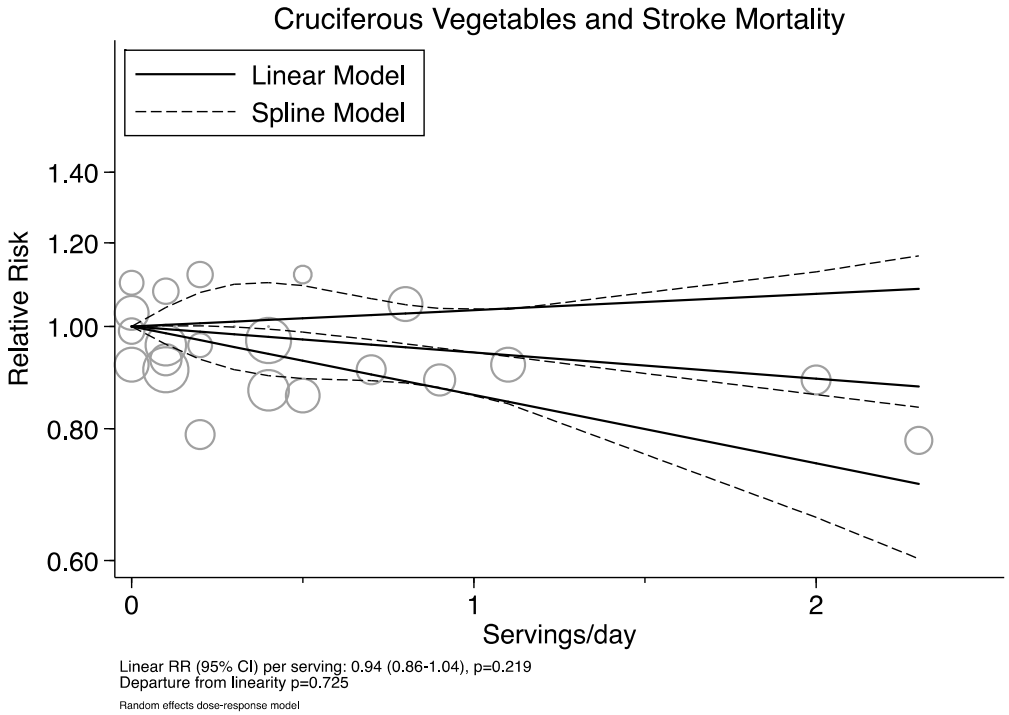


Figure S169. Linear and cubic-spline dose-response relation between increasing cruciferous vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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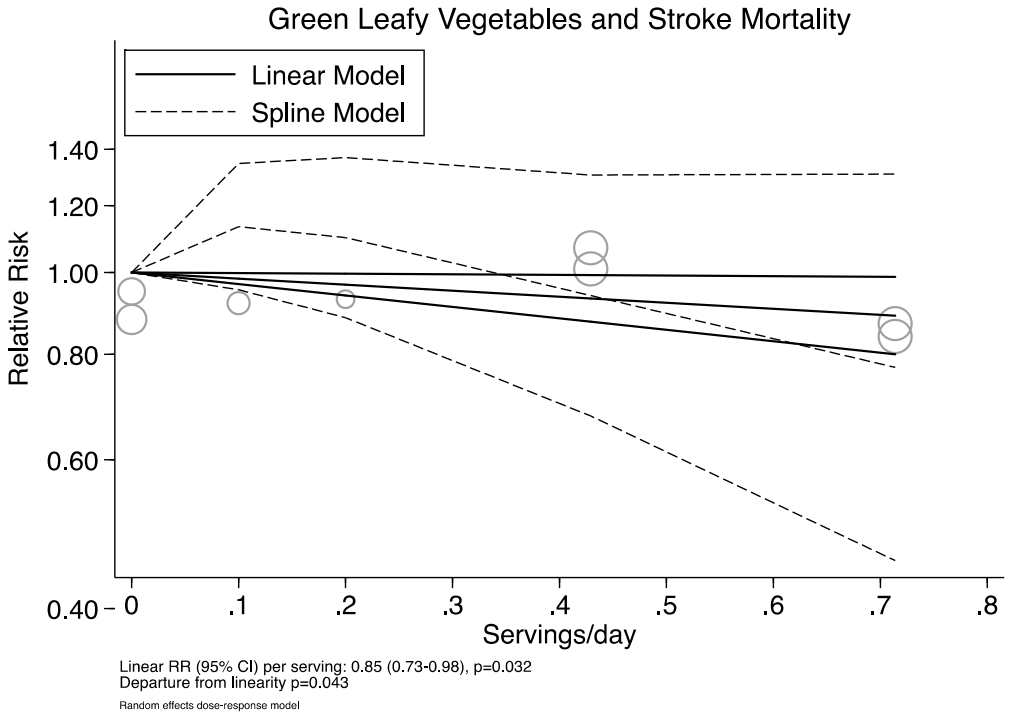


Figure S170. Linear and cubic-spline dose-response relation between increasing green leafy vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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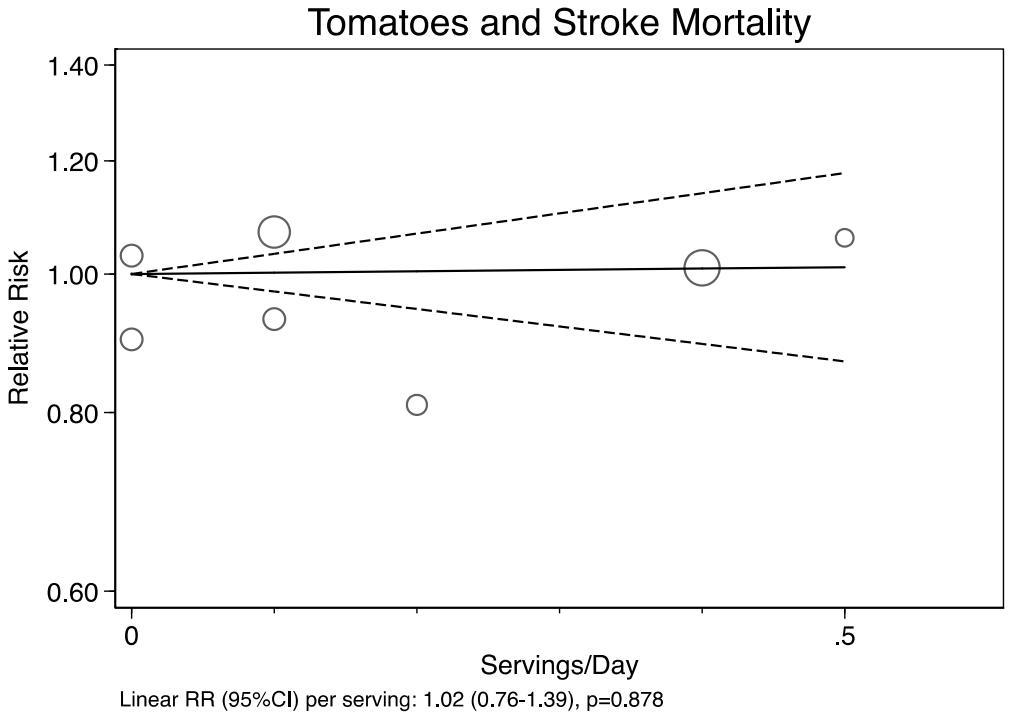


Figure S171. Linear dose-response relation between increasing intake of tomatoes and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker[23] method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

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TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates RR [95% CIs] for Fruit and Vegetables and Incident CVD	Residual I^2	*p* -value	Adjusted Alpha Level
					Within Subgr	Between Subgroups			
Total	-	**12**	**501,744**	**24,310**	**0.92 [0.88, 0**	-	-	-
Sex	Females	3	174,785	6,980	0.91 [0.80, 1	F vs. M: 0.95 [0.79, 1.16]	34.01%	0.88	0.007
	Males	4	49,532	2,776	0.95 [0.83, 1	F vs. Mix: 0.99 [0.85, 1.16]			
	Mxed	7	277,427	14,554	0.92 [0.84, 1	M vs. Mix: 1.03 [0.87, 1.23]			
Age (y)	<60	6	209,845	12,102	0.89 [0.82, 0	0.95 [0.86, 1.04]	22.88%	0.23	0.008
	≥60	6	291,899	291,899	0.94 [0.89, 0				
Follow Up (y)	<10	4	259,569	6,930	0.88 [0.74, 1	0.95 [0.79, 1.13]	28.44%	0.53	0.010
	≥10	10	242,175	17,380	0.93 [0.88, 0				
Statistical Adjustments	<8	2	5,563	1,334	0.98 [0.79, 1	1.06 [0.86, 1.33]	28.70%	0.54	0.013
	≥8	10	496,181	22,976	0.92 [0.87, 0				
NOS	<6	-	-	-	-	-	-	-	0.017
	≥6	12	501,744	24,310	0.92 [0.88, 0				
Exposure Assessment Tool	Validated FFQ	9	474,421	18,022	0.93 [0.89, 0	vFFQ vs. uFFQ: 1.06 [0.89, 1.27]	17.71%	0.19	0.025
	Unvalidated FFQ	2	4,331	1,323	0.99 [0.83, 1	vFFQ vs. record: 0.90 [0.80, 1.03]			
	Food Record	1	22,992	4,965	0.84 [0.75, 0	uFFQ vs. record: 0.85 [0.69, 1.05]			
Location	Asia	1	77,891	1,386	0.93 [0.72, 1	†	39.02%	0.98	0.050
	Europe	5	22,935	2,859	0.95 [0.82, 1				
	North America	5	265,583	15,281	0.92 [0.85, 0				
	Global	1	135,335	4,784	0.93 [0.63, 1				

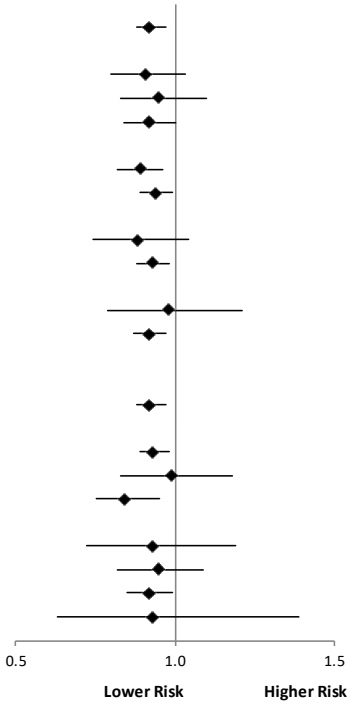


Figure S172. Categorical subgroup analyses of total fruit and vegetable intake and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.98 [0.74, 1.31]; Europe vs. Global 0.99 [0.64, 1.51]; Europe vs. North America 0.97 [0.83, 1.14]; Asia vs. Global 0.99 [0.62, 1.60]; Asia vs. North America 1.00 [0.78, 1.32]; Global vs. North America 1.02 [0.68, 1.53];

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FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% Cis] for Fruit and Incident CVD	Residual I^2	*p* -value	Adjusted Alpha Level
			0.0471227		Within Subgroups	Between Subgroups			
Total	-	**16**	577,323	27,205	0.91 [0.86, 0.97]	-	-	-	-
Sex	Females	5	189,962	9,212	0.90 [0.78, 1.03]	F vs. M: 0.96 [0.79, 1.18]	45.27%	0.91	0.007
	Males	5	58,075	3,944	0.93 [0.80, 1.09]	F vs. Mix: 0.98 [0.83, 1.16]			
	Mxed	8	329,286	14,049	0.92 [0.83, 1.01]	M vs. Mix: 1.02 [0.85, 1.22]			
Age (y)	<59	8	255,899	11,747	0.92 [0.83, 1.01]	1.01 [0.88, 1.15]	42.56%	0.94	0.008
	≥59	8	321,424	15,458	0.91 [0.83, 1.00]				
Follow Up (y)	<10	5	247,053	7,204	0.93 [0.82, 1.06]	1.02 [0.88, 1.19]	40.64%	0.88	0.010
	≥10	11	330,270	20,001	0.91 [0.84, 0.98]				
Statistical Adjustments	<8	4	8,987	1,892	0.87 [0.72, 1.05]	0.94 [0.78, 1.15]	41.21%	0.57	0.013
	≥8	12	568,336	24,953	0.92 [0.86, 0.99]				
NOS	<6	2	37,907	1,423	0.86 [0.66, 1.12]	0.94 [0.72, 1.23]	41.54%	0.63	0.017
	≥6	14	539,416	25,782	0.92 [0.86, 0.98]				
Exposure Assessment Tool	Validated FFQ	13	571,664	25,895	0.86 [0.67, 1.10]	vFFQ vs. uFFQ: 1.07 [0.83, 1.38]	44.25%	0.85	0.025
	Unvalidated FFQ	2	3,810	555	0.92 [0.85, 0.99]	vFFQ vs. record: 1.02 [0.62, 1.67]			
	Food record	1	1,849	755	0.88 [0.58, 1.35]	uFFQ vs. record: 0.96 [0.63, 1.47]			
Location	Asia	1	77,891	1,386	0.80 [0.62, 1.03]	†	43.21%	0.65	0.050
	Europe	10	125,806	10,043	0.94 [0.85, 1.03]				
	North America	4	238,291	10,992	0.91 [0.80, 1.03]				
	Global	1	135,335	4,784	0.89 [0.69, 1.13]				

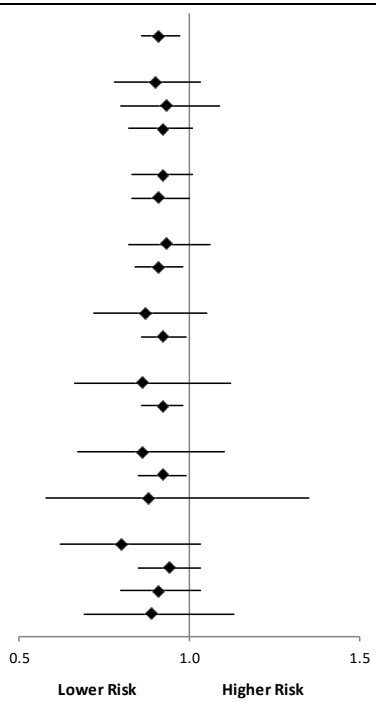
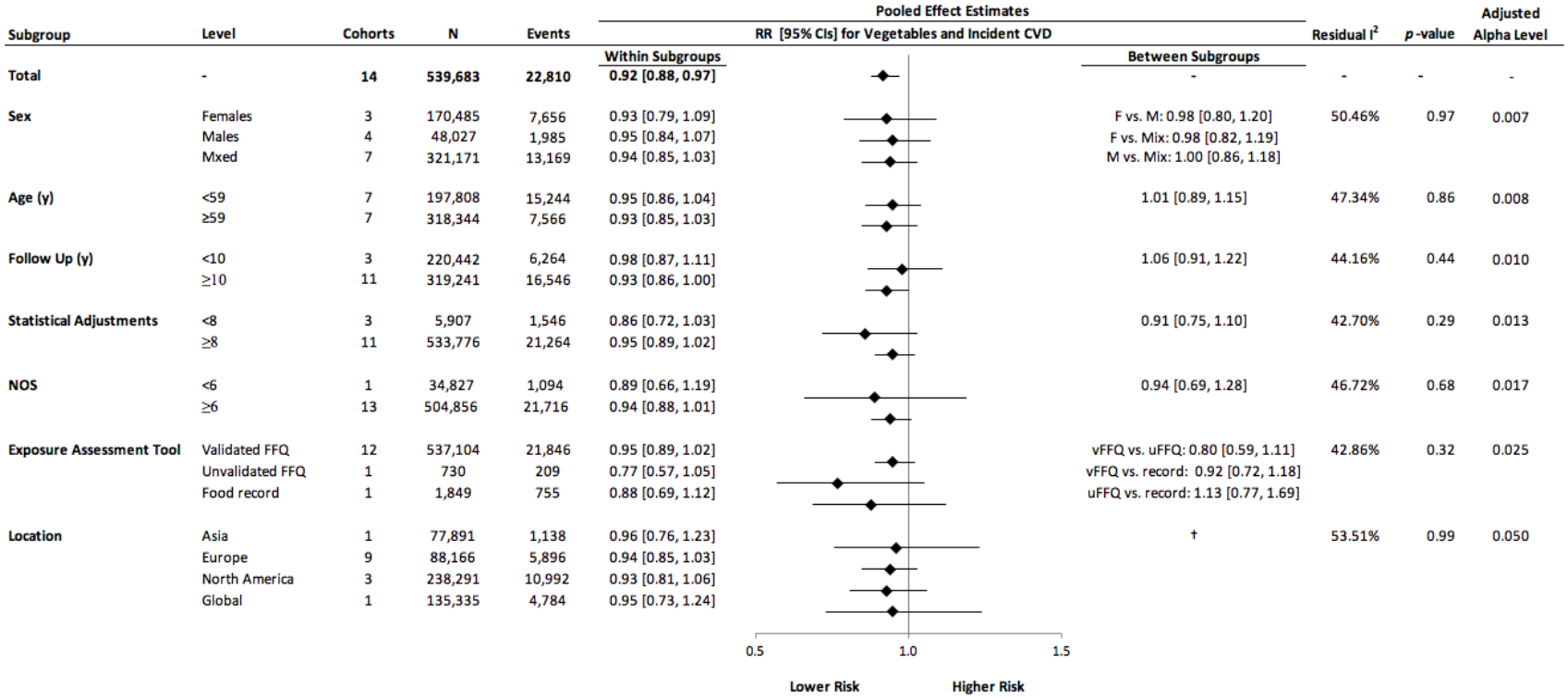


Figure S173. Categorical subgroup analyses of fruit intake and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 0.85 [0.65, 1.12]; Europe vs. Global 0.94 [0.73, 1.23]; Europe vs. North America 0.96 [0.82, 1.13]; Asia vs. Global 0.90 [0.63, 1.29]; Asia vs. North America 0.88 [0.67, 1.18]; Global vs. North America 0.98 [0.74, 1.29]

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VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE



Subgroup	Level	Cohorts	N	Events	Within Subgroups	Between Subgroups	Residual I^2	*p*-value	Adjusted Alpha Level
Total	-	**14**	**539,683**	**22,810**	**0.92 [0.88, 0.97]**	-	-	-	-
Sex	Females	3	170,485	7,656	0.93 [0.79, 1.09]	F vs. M: 0.98 [0.80, 1.20]	50.46%	0.97	0.007
	Males	4	48,027	1,985	0.95 [0.84, 1.07]	F vs. Mix: 0.98 [0.82, 1.19]			
	Mxed	7	321,171	13,169	0.94 [0.85, 1.03]	M vs. Mix: 1.00 [0.86, 1.18]			
Age (y)	<59	7	197,808	15,244	0.95 [0.86, 1.04]	1.01 [0.89, 1.15]	47.34%	0.86	0.008
	≥59	7	318,344	7,566	0.93 [0.85, 1.03]				
Follow Up (y)	<10	3	220,442	6,264	0.98 [0.87, 1.11]	1.06 [0.91, 1.22]	44.16%	0.44	0.010
	≥10	11	319,241	16,546	0.93 [0.86, 1.00]				
Statistical Adjustments	<8	3	5,907	1,546	0.86 [0.72, 1.03]	0.91 [0.75, 1.10]	42.70%	0.29	0.013
	≥8	11	533,776	21,264	0.95 [0.89, 1.02]				
NOS	<6	1	34,827	1,094	0.89 [0.66, 1.19]	0.94 [0.69, 1.28]	46.72%	0.68	0.017
	≥6	13	504,856	21,716	0.94 [0.88, 1.01]				
Exposure Assessment Tool	Validated FFQ	12	537,104	21,846	0.95 [0.89, 1.02]	vFFQ vs. uFFQ: 0.80 [0.59, 1.11]	42.86%	0.32	0.025
	Unvalidated FFQ	1	730	209	0.77 [0.57, 1.05]	vFFQ vs. record: 0.92 [0.72, 1.18]			
	Food record	1	1,849	755	0.88 [0.69, 1.12]	uFFQ vs. record: 1.13 [0.77, 1.69]			
Location	Asia	1	77,891	1,138	0.96 [0.76, 1.23]	†	53.51%	0.99	0.050
	Europe	9	88,166	5,896	0.94 [0.85, 1.03]				
	North America	3	238,291	10,992	0.93 [0.81, 1.06]				
	Global	1	135,335	4,784	0.95 [0.73, 1.24]				

Figure S174. Categorical subgroup analyses of intake of vegetables and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 1.03 [0.79, 1.33]; Europe vs. Global 1.01 [0.76, 1.34]; Europe vs. NA 0.99 [0.84, 1.16]; Asia vs. Global 1.01 [0.71, 1.45]; Asia vs. NA 1.04 [0.79, 1.37]; Global vs. NA 1.03 [0.76, 1.38]

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TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

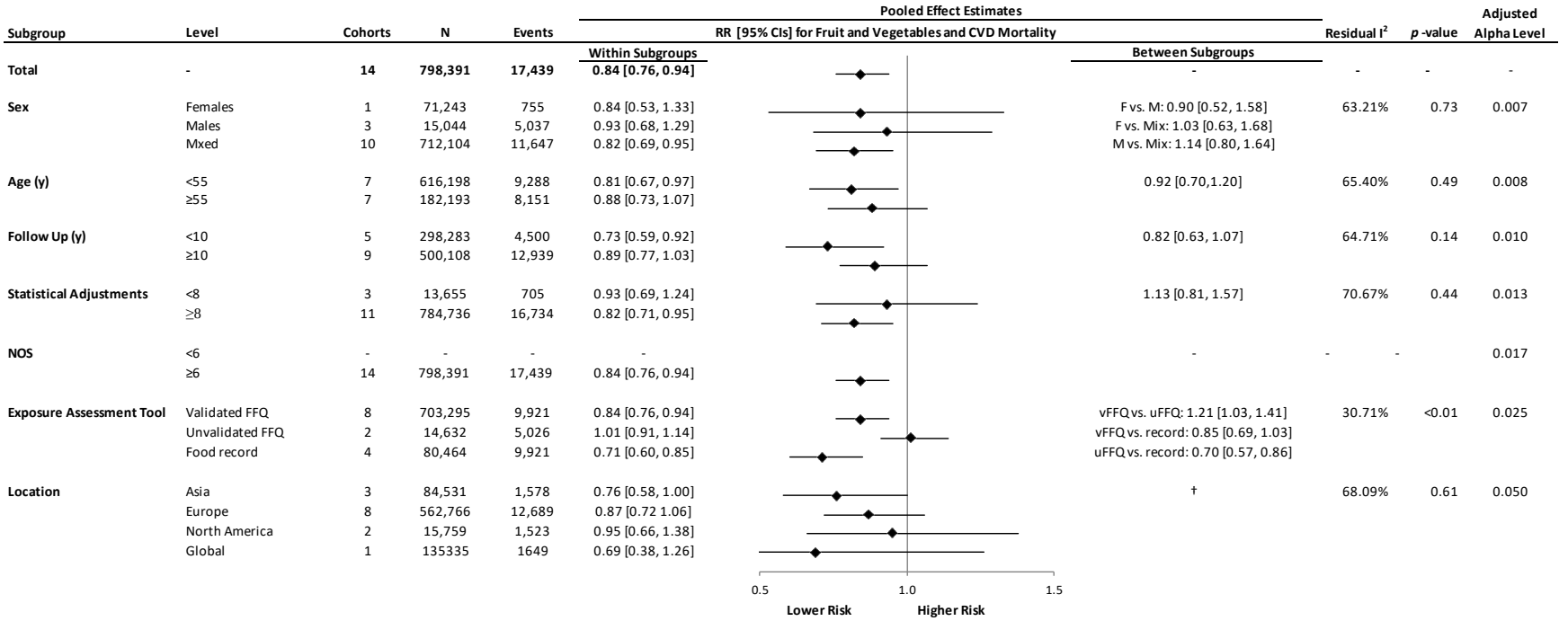


Figure S175. Categorical subgroup analyses of total fruit and vegetable intake and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.87 [0.63, 1.22]; Europe vs. Global 0.79 [0.42, 1.49]; Europe vs. North America 1.09 [0.72, 1.66]; Asia vs. Global 1.10 [0.57, 2.13]; Asia vs. North America 0.80 [0.50, 1.27]; Global vs. North America 0.73 [0.36, 1.47]

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FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% CIs] for Fruit and CVD Mortality — Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**27**	**1,581,506**	**39,623**	**0.83 [0.77, 0.89]**	-	-	-	-
Sex	Females	5	135,154	5,157	0.76 [0.63, 0.91]	F vs. M: 0.84 [0.66, 1.07]	77.74%	0.33	0.007
	Males	7	101,605	8,641	0.92 [0.75, 1.13]	F vs. Mix: 0.92 [0.75, 1.13]			
	Mxed	17	329,286	14,049	0.82 [0.75, 0.90]	M vs. Mix: 1.10 [0.92, 1.31]			
Age (y)	<54	10	1,115,225	15,507	0.77 [0.69, 0.86]	0.88 [0.77, 1.02]	71.00%	0.08	0.008
	≥54	17	466,281	24,116	0.87 [0.80, 0.94]				
Follow Up (y)	<11	13	916,897	17,780	0.80 [0.72, 0.90]	0.95 [0.82, 1.09]	76.29%	0.44	0.010
	≥11	14	664,609	21,843	0.85 [0.77, 0.93]				
Statistical Adjustments	<8	8	57,260	5,303	0.88 [0.78, 0.99]	1.10 [0.95, 1.27]	79.15%	0.22	0.013
	≥8	19	1,524,246	34,320	0.80 [0.73, 0.88]				
NOS	<6	2	20,531	1,717	0.89 [0.73, 1.08]	1.08 [0.88, 1.33]	78.84%	0.45	0.017
	≥6	24	1,560,975	37,906	0.82 [0.76, 0.89]				
Exposure Assessment Tool	Validated FFQ	15	990,789	22,863	0.84 [0.76, 0.93]	vFFQ vs. uFFQ: 0.97 [0.80, 1.17]	78.17%	0.93	0.025
	Unvalidated FFQ	4	483,979	12,238	0.81 [0.69, 0.95]	vFFQ vs. record: 0.99 [0.83, 1.18]			
	Food record	6	106,738	4,522	0.83 [0.72, 0.96]	uFFQ vs. record: 1.02 [0.83, 1.27]			
Location	Asia	6	752,255	20,127	0.80 [0.71, 0.89]	†	78.55%	0.32	0.050
	Australia	1	40,653	697	0.69 [0.46, 1.05]				
	Europe	14	629,562	16,072	0.83 [0.75, 0.91]				
	North America	2	13,944	563	1.02 [0.77, 1.38]				
	Global	2	145,092	2,164	0.94 [0.75 1.19]				

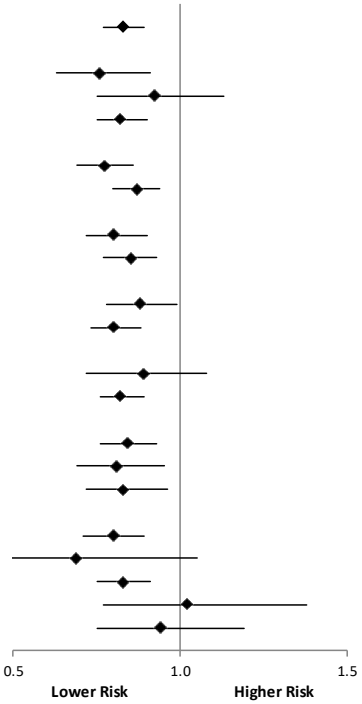
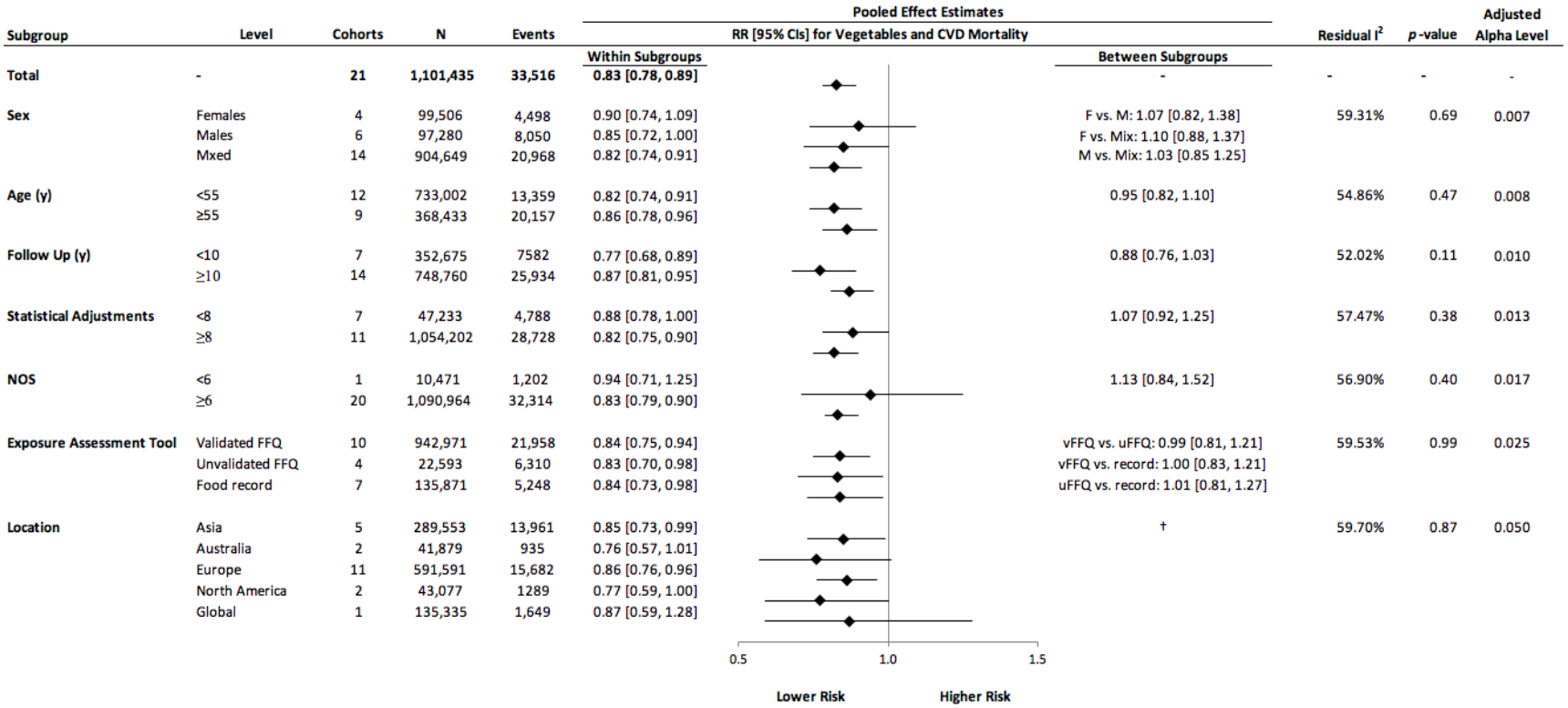


Figure S176. Categorical subgroup analyses of fruit intake and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 0.96 [0.83, 1.13]; Europe vs Australia 0.84 [0.55, 1.28]; Europe vs. Global 1.14 [0.89, 1.47]; Europe vs. North America 1.25 [0.92, 1.70]; Asia vs. Australia 1.15 [0.75, 1.77]; Asia vs. Global 0.85 [0.65, 1.10]; Asia vs. North America 0.77 [0.56, 1.06]; Australia vs. Global 0.73 [0.46, 1.18]; Australia vs. North America 0.67 [0.41, 1.12]; Global vs. North America 0.92 [0.63, 1.33]

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VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY



Subgroup	Level	Cohorts	N	Events	Within Subgroups	Between Subgroups	Residual I^2	*p*-value	Adjusted Alpha Level
Total	-	**21**	**1,101,435**	**33,516**	**0.83 [0.78, 0.89]**	-	-	-	-
Sex	Females	4	99,506	4,498	0.90 [0.74, 1.09]	F vs. M: 1.07 [0.82, 1.38]	59.31%	0.69	0.007
	Males	6	97,280	8,050	0.85 [0.72, 1.00]	F vs. Mix: 1.10 [0.88, 1.37]			
	Mxed	14	904,649	20,968	0.82 [0.74, 0.91]	M vs. Mix: 1.03 [0.85 1.25]			
Age (y)	<55	12	733,002	13,359	0.82 [0.74, 0.91]	0.95 [0.82, 1.10]	54.86%	0.47	0.008
	≥55	9	368,433	20,157	0.86 [0.78, 0.96]				
Follow Up (y)	<10	7	352,675	7582	0.77 [0.68, 0.89]	0.88 [0.76, 1.03]	52.02%	0.11	0.010
	≥10	14	748,760	25,934	0.87 [0.81, 0.95]				
Statistical Adjustments	<8	7	47,233	4,788	0.88 [0.78, 1.00]	1.07 [0.92, 1.25]	57.47%	0.38	0.013
	≥8	11	1,054,202	28,728	0.82 [0.75, 0.90]				
NOS	<6	1	10,471	1,202	0.94 [0.71, 1.25]	1.13 [0.84, 1.52]	56.90%	0.40	0.017
	≥6	20	1,090,964	32,314	0.83 [0.79, 0.90]				
Exposure Assessment Tool	Validated FFQ	10	942,971	21,958	0.84 [0.75, 0.94]	vFFQ vs. uFFQ: 0.99 [0.81, 1.21]	59.53%	0.99	0.025
	Unvalidated FFQ	4	22,593	6,310	0.83 [0.70, 0.98]	vFFQ vs. record: 1.00 [0.83, 1.21]			
	Food record	7	135,871	5,248	0.84 [0.73, 0.98]	uFFQ vs. record: 1.01 [0.81, 1.27]			
Location	Asia	5	289,553	13,961	0.85 [0.73, 0.99]	†	59.70%	0.87	0.050
	Australia	2	41,879	935	0.76 [0.57, 1.01]				
	Europe	11	591,591	15,682	0.86 [0.76, 0.96]				
	North America	2	43,077	1289	0.77 [0.59, 1.00]				
	Global	1	135,335	1,649	0.87 [0.59, 1.28]				

Figure S177. Categorical subgroup analyses of intake of vegetables and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 0.99 [0.82, 1.19]; Europe vs Australia 0.88 [0.65, 1.20]; Europe vs. Global 1.01 [0.68, 1.52]; Europe vs. North America 0.90 [0.67, 1.20]; Asia vs. Australia 1.12 [0.81, 1.56]; Asia vs. Global 0.98 [0.64, 1.48]; Asia vs. North America 1.11 [0.81, 1.50]; Australia vs. Global 0.87 [0.54, 1.41]; Australia vs. North America 0.98 [0.66, 1.46]; Global vs. North America 1.13 [0.71, 1.81]

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TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

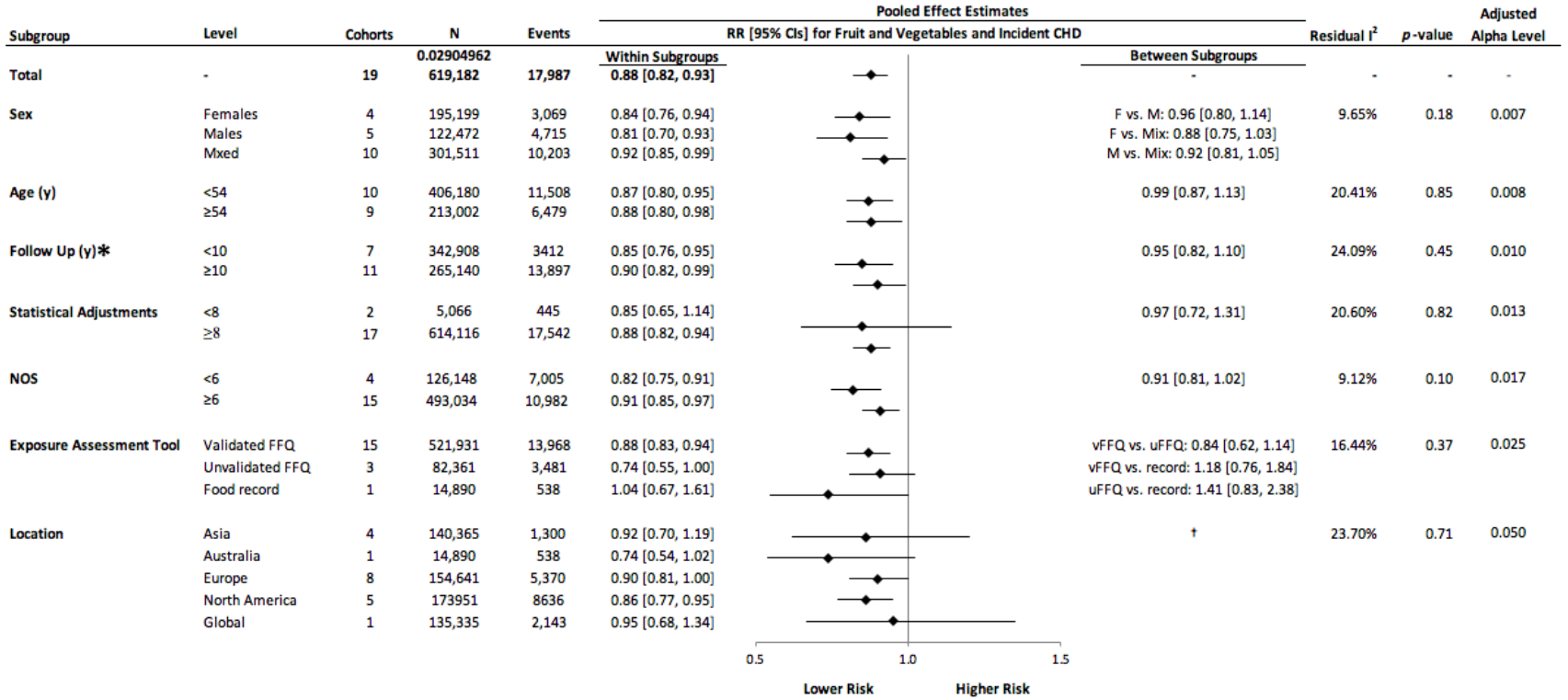


Figure S178. Categorical subgroup analyses of total fruit and vegetable intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. * Follow-up years incudes 17 cohorts as Bingham et al. 2008 (EPIC Norfolk) did not report follow-up time. † Europe vs. Asia 1.02 [0.77, 1.35]; Europe vs. Australia 0.82 [0.59, 1.14]; Europe vs. Global 1.06 [0.74, 1.51]; Europe vs. North America 0.95 [0.83, 1.10]; Asia vs. Australia 1.24 [0.82, 1.87]; Asia vs. Global 0.96 [0.63, 1.48]; Asia vs. North America 1.07 [0.81, 1.42]; Australia vs. Global 0.78 [0.49, 1.24]; Australia vs. North America 0.86 [0.62, 1.21]; Global vs. North America 1.11 [0.78, 1.58]

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FRUIT AND CORONARY HEART DISEASE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% CIs] for Fruit and Incident CHD — Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**20**	**1,170,021**	**23,856**	**0.88 [0.84, 0.93]**	-	-	-	-
Sex	Females	6	251,883	3,255	0.86 [0.76, 1.00]	F vs. M: 0.97 [0.82, 1.15]	11.32%	0.88	0.007
	Males	6	166,015	9,697	0.90 [0.82, 0.99]	F vs. Mix: 1.00 [0.85, 1.19]			
	Mxed	10	752,123	10,904	0.87 [0.79, 0.96]	M vs. Mix: 1.03 [0.90, 1.18]			
Age (y)	<55	10	899,185	13,731	0.86 [0.80, 0.93]	0.94 [0.84, 1.06]	3.79%	0.31	0.008
	≥55	10	270,836	10,125	0.91 [0.83, 0.99]				
Follow Up (y)	<10	9	865,523	6,656	0.82 [0.74, 0.92]	0.92 [0.82, 1.04]	0.00%	0.16	0.010
	≥10	11	304,498	17,200	0.90 [0.85, 0.95]				
Statistical Adjustments	<8	4	206,008	11,296	0.91 [0.77, 1.09]	1.04 [0.87, 1.25]	8.05%	0.63	0.013
	≥8	16	964,013	12,560	0.88 [0.82, 0.93]				
NOS	<6	2	113,276	6,189	0.87 [0.77, 0.99]	0.99 [0.86, 1.14]	8.97%	0.89	0.017
	≥6	18	1,056,745	17,667	0.88 [0.82, 0.95]				
Exposure Assessment Tool	Validated FFQ	17	637,980	18,321	0.90 [0.85, 0.95]	vFFQ vs. uFFQ: 0.87 [0.76, 0.99]	0.00%	0.07	0.025
	Unvalidated FFQ	2	530,192	5,374	0.78 [0.69, 0.88]	vFFQ vs. record: 1.12 [0.80, 1.57]			
	Food record	1	1,849	161	1.01 [0.72, 1.41]	uFFQ vs. record: 1.29 [0.91, 1.84]			
Location	Asia	4	590,798	3,755	0.76 [0.65, 0.89]	†	0.00%	0.22	0.050
	Europe	11	265,012	11,509	0.90 [0.84, 0.97]				
	North America	4	178876	6449	0.87 [0.80, 0.96]				
	Global	1	135,335	2,143	0.91 [0.73, 1.15]				

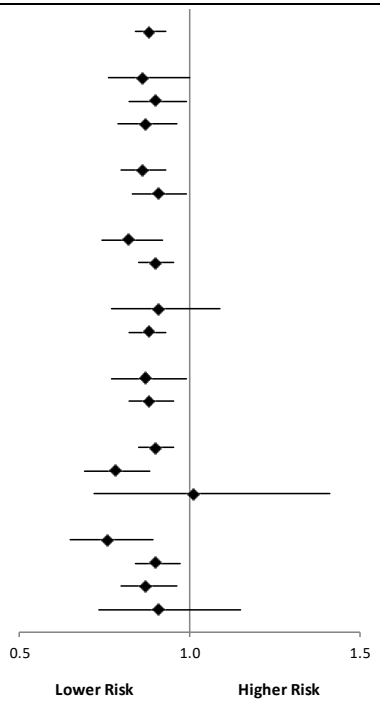


Figure S179. Categorical subgroup analyses of fruit intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.84 [0.71, 0.99]; Europe vs. Global 1.01 [0.79, 1.29]; Europe vs. North America 0.96 [0.85, 1.08]; Asia vs. Global 0.83 [0.63, 1.10]; Asia vs. North America 0.87 [0.73, 1.04]; Global vs. North America 1.05 [0.82, 1.34]

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VEGETABLE AND CORONARY HEART DISEASE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% CIs] for Vegetables and Incident CHD — Within Subgroups	Between Subgroups	Residual I^2	*p*-value	Adjusted Alpha Level
			0.0246607						
Total	-	**18**	**696,330**	**17,172**	**0.92 [0.85, 0.99]**	-	-	-	-
Sex	Females	6	251,883	3,820	0.87 [0.77, 0.98]	F vs. M: 0.99 [0.86, 1.14]	0.00%	0.86	0.007
	Males	8	184,953	7,023	0.88 [0.81, 0.95]	F vs. Mix: 0.96 [0.82, 1.13]			
	Mxed	6	259,494	6,329	0.91 [0.81, 1.01]	M vs. Mix: 0.97 [0.85, 1.11]			
Age (y)	<55	11	422,076	10,304	0.88 [0.81, 0.94]	0.98 [0.87, 1.09]	0.00%	0.67	0.008
	≥55	7	274,254	6,868	0.90 [0.82, 0.97]				
Follow Up (y)	<10	9	403,213	5986	0.83 [0.74, 0.94]	0.95 [0.83, 1.09]	0.00%	0.43	0.010
	≥10	9	252,241	10,712	0.88 [0.82, 0.95]				
Statistical Adjustments	<8	2	5,177	468	0.96 [0.73, 1.26]	1.09 [0.82, 1.44]	0.00%	0.53	0.013
	≥8	16	691,153	16,704	0.88 [0.83, 0.93]				
NOS	<6	2	113,276	6,189	0.87 [0.80, 0.96]	0.99 [0.88, 1.11]	0.00%	0.78	0.017
	≥6	16	583,054	10,983	0.89 [0.83, 0.95]				
Exposure Assessment Tool	Validated FFQ	16	615,954	14,188	0.88 [0.83, 0.94]	vFFQ vs. uFFQ: 0.99 [0.84, 1.16]	0.00%	0.69	0.025
	Unvalidated FFQ	1	78,527	2,823	0.87 [0.75, 1.01]	vFFQ vs. record: 1.15 [0.82, 1.61]			
	Food record	1	1,849	161	1.01 [0.72, 1.41]	uFFQ vs. record: 1.16 [0.81, 1.68]			
Location	Asia	3	139,133	1,204	0.96 [0.73, 1.25]	†	0.00%	0.79	0.050
	Europe	10	253,939	6,362	0.89 [0.83, 0.97]				
	North America	4	167,923	7,463	0.86 [0.79, 0.94]				
	Global	1	135,335	2,143	0.91 [0.73, 1.15]				

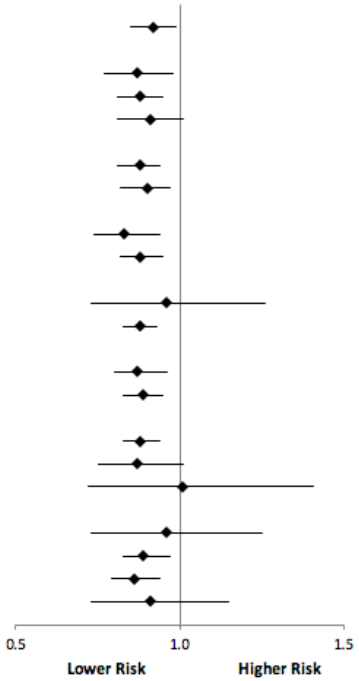


Figure S180. Categorical subgroup analyses of intake of vegetables and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 1.07 [0.81, 1.41]; Europe vs. Global 1.02 [0.80, 1.30]; Europe vs. NA 0.96 [0.85, 1.08]; Asia vs. Global 1.05 [0.74, 1.49]; Asia vs. NA 1.11 [0.84, 1.48]; Global vs. NA 1.07 [0.83, 1.37]

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CITRUS FRUIT AND CORONARY HEART DISEASE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% CIs] for Citrus Fruit and Incident CHD — Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**10**	**364,978**	**8,333**	**0.91 [0.85, 0.98]**	-	-	-	-
Sex	Females	5	202,835	3,152	0.90 [0.80, 1.02]	F vs. M: 0.99 [0.84, 1.17]	0.00%	0.76	0.007
	Males	5	134,858	4,830	0.91 [0.82, 1.02]	F vs. Mix: 0.88 [0.60, 1.29]			
	Mxed	2	27,285	351	1.02 [0.72, 1.47]	M vs. Mix: 0.89 [0.61, 1.29]			
Age (y)	<55	7	293,756	7,064	0.90 [0.83, 0.98]	0.91 [0.74, 1.14]	0.00%	0.38	0.008
	≥55	3	71,222	1,269	0.99 [0.81, 1.20]				
Follow Up (y)	<10	5	212,923	1702	0.97 [0.82, 1.15]	1.08 [0.90, 1.31]	0.00%	0.38	0.010
	≥10	5	152,055	6,631	0.90 [0.82, 0.98]				
Statistical Adjustments	<8	-	-	-	-	-	-	-	0.013
	≥8	10	364,978	8,333	0.91 [0.85, 0.98]				
NOS	<6	2	113,276	6,189	0.91 [0.82, 1.00]	0.97 [0.82, 1.15]	0.00%	0.73	0.017
	≥6	8	251,702	2,144	0.93 [0.81, 1.07]				
Exposure Assessment Tool	Validated FFQ	10	364,978	8,333	0.91 [0.85, 0.98]	-	-	-	0.025
	Unvalidated FFQ	-	-	-	-				
	Food record	-	-	-	-		-		
Location	Asia	3	133,258	441	0.81 [0.58, 1.12]	Europe vs. Asia: 0.84 [0.58, 1.20]	0.00%	0.54	0.050
	Europe	5	118,444	1,703	0.96 [0.82, 1.13]	Europe vs. NA: 0.94 [0.78, 1.13]			
	North America	2	113,276	6,189	0.91 [0.82, 1.00]	Asia vs. NA: 0.89 [0.64, 1.25]			

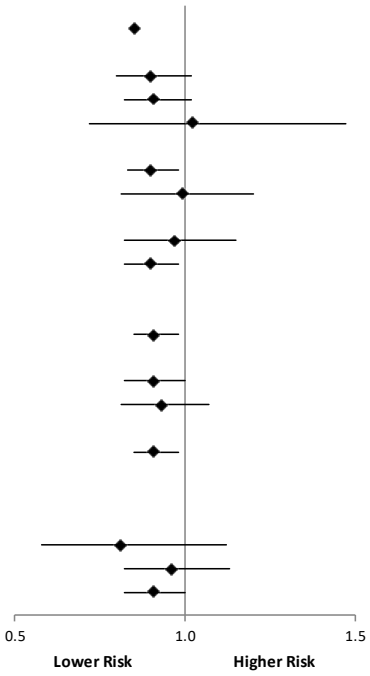
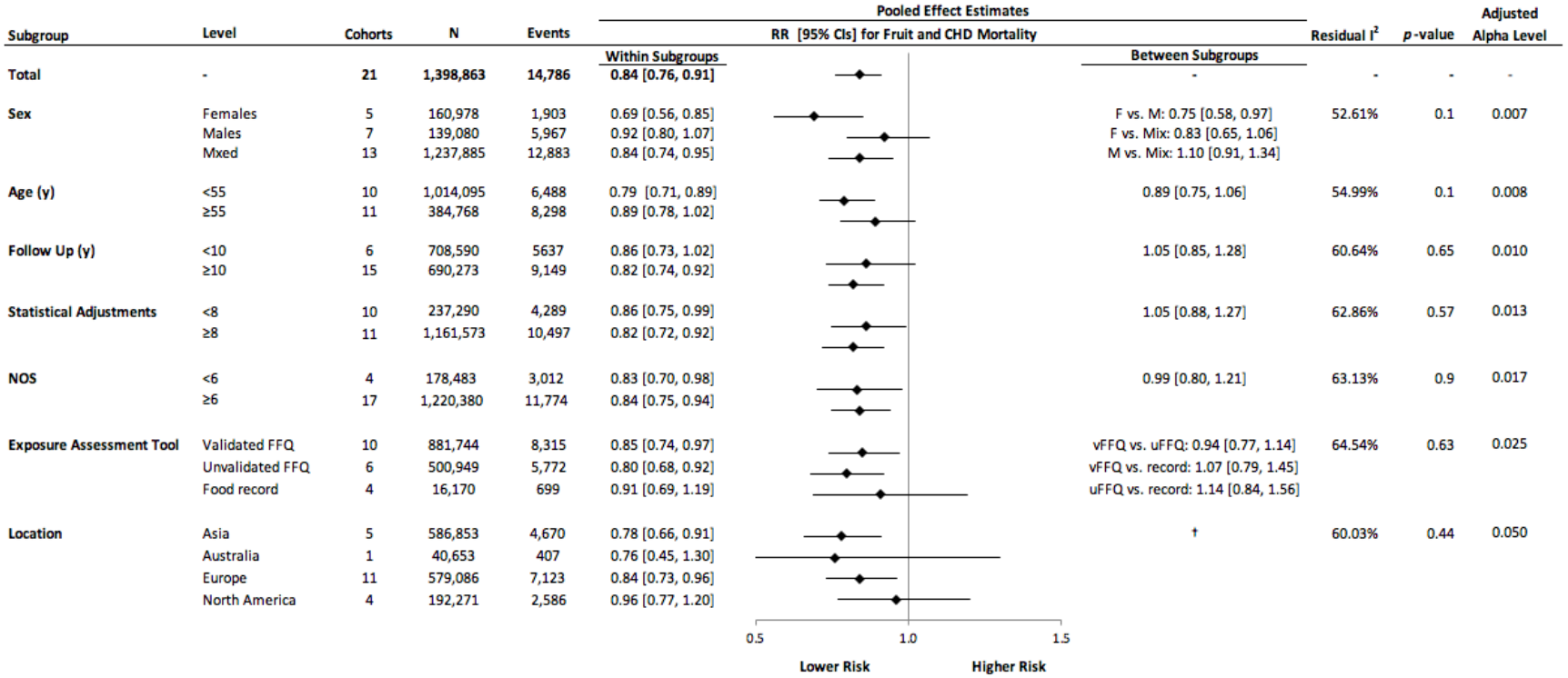


Figure S181. Categorical subgroup analyses of citrus fruit intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

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FRUIT AND CORONARY HEART DISEASE MORTALITY



Subgroup	Level	Cohorts	N	Events	Within Subgroups	Between Subgroups	Residual I^2	*p*-value	Adjusted Alpha Level
Total	-	**21**	**1,398,863**	**14,786**	**0.84 [0.76, 0.91]**	-	-	-	-
Sex	Females	5	160,978	1,903	0.69 [0.56, 0.85]	F vs. M: 0.75 [0.58, 0.97]	52.61%	0.1	0.007
	Males	7	139,080	5,967	0.92 [0.80, 1.07]	F vs. Mix: 0.83 [0.65, 1.06]			
	Mxed	13	1,237,885	12,883	0.84 [0.74, 0.95]	M vs. Mix: 1.10 [0.91, 1.34]			
Age (y)	<55	10	1,014,095	6,488	0.79 [0.71, 0.89]	0.89 [0.75, 1.06]	54.99%	0.1	0.008
	≥55	11	384,768	8,298	0.89 [0.78, 1.02]				
Follow Up (y)	<10	6	708,590	5637	0.86 [0.73, 1.02]	1.05 [0.85, 1.28]	60.64%	0.65	0.010
	≥10	15	690,273	9,149	0.82 [0.74, 0.92]				
Statistical Adjustments	<8	10	237,290	4,289	0.86 [0.75, 0.99]	1.05 [0.88, 1.27]	62.86%	0.57	0.013
	≥8	11	1,161,573	10,497	0.82 [0.72, 0.92]				
NOS	<6	4	178,483	3,012	0.83 [0.70, 0.98]	0.99 [0.80, 1.21]	63.13%	0.9	0.017
	≥6	17	1,220,380	11,774	0.84 [0.75, 0.94]				
Exposure Assessment Tool	Validated FFQ	10	881,744	8,315	0.85 [0.74, 0.97]	vFFQ vs. uFFQ: 0.94 [0.77, 1.14]	64.54%	0.63	0.025
	Unvalidated FFQ	6	500,949	5,772	0.80 [0.68, 0.92]	vFFQ vs. record: 1.07 [0.79, 1.45]			
	Food record	4	16,170	699	0.91 [0.69, 1.19]	uFFQ vs. record: 1.14 [0.84, 1.56]			
Location	Asia	5	586,853	4,670	0.78 [0.66, 0.91]	†	60.03%	0.44	0.050
	Australia	1	40,653	407	0.76 [0.45, 1.30]				
	Europe	11	579,086	7,123	0.84 [0.73, 0.96]				
	North America	4	192,271	2,586	0.96 [0.77, 1.20]				

Figure S182. Categorical subgroup analyses of fruit intake and coronary heart disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs. Asia 0.93 [0.76, 1.14]; Europe vs. Australia 0.91 [0.53, 1.57]; Europe vs. North America 1.15 [0.89, 1.47]; Asia vs. Australia 1.01 [0.59, 1.77]; Asia vs. North America 0.81 [0.62, 1.06]; Australia vs. North America 0.80 [0.45, 1.41]

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VEGETABLES AND CORONARY HEART DISEASE MORTALITY

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: Within Subgroup	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**18**	**1,968,325**	**26,007**	**0.84 [0.80, 0.8**	-	-	-	-
Sex	Females	4	704,423	5,693	0.85 [0.76, 0.9	F vs. M: 0.96 [0.88, 1.06]	26.01%	0.73	0.007
	Males	6	592,634	13,892	0.86 [0.78, 0.9	F vs. Mix: 0.98 [0.89, 1.09]			
	Mxed	12	671,268	6,422	0.83 [0.76, 0.9	M vs. Mix: 1.02 [0.93 1.11]			
Age (y)	<56	10	1,054,654	14,251	0.87 [0.83, 0.9	1.04 [0.96, 1.13]	20.98%	0.31	0.008
	≥56	10	913,671	11,756	0.84 [0.78, 0.8				
Follow Up (y)	<13	9	1,404,076	18332	0.84 [0.78, 0.9	0.98 [0.89, 1.10]	25.06%	0.73	0.010
	≥13	9	564,249	7,675	0.85 [0.79, 0.9				
Statistical Adjustments	<8	5	205,972	3,242	0.86 [0.78, 0.9	1.01 [0.91, 1.14]	24.96%	0.81	0.013
	≥8	13	1,762,353	22,765	0.84 [0.80, 0.9				
NOS	<6	3	167,742	2,407	0.86 [0.77, 0.9	1.02 [0.90, 1.15]	24.72%	0.73	0.017
	≥6	15	1,800,583	23,600	0.84 [0.80, 0.8				
Exposure Assessment Tool	Validated FFQ	7	814,011	7,649	0.82 [0.75, 0.9	vFFQ vs. uFFQ: 1.07 [0.96, 1.18]	0.00%	0.02	0.025
	Unvalidated FFQ	5	1,109,011	17,103	0.88 [0.84, 0.9	vFFQ vs. record: 0.77 [0.62, 0.96]			
	Food record	6	45,303	1,255	0.64 [0.52, 0.7	uFFQ vs. record: 0.72 {0.59, 0.89]			
Location	Asia	4	124,511	2,632	0.85 [0.74, 0.9	†	32.13%	0.98	0.050
	Australia	2	41,879	535	0.83 [0.66, 1.0				
	Europe	7	543,981	6,400	0.85 [0.75, 0.9				
	North America	5	1,257,954	16,440	0.82 [0.75, 0.9				

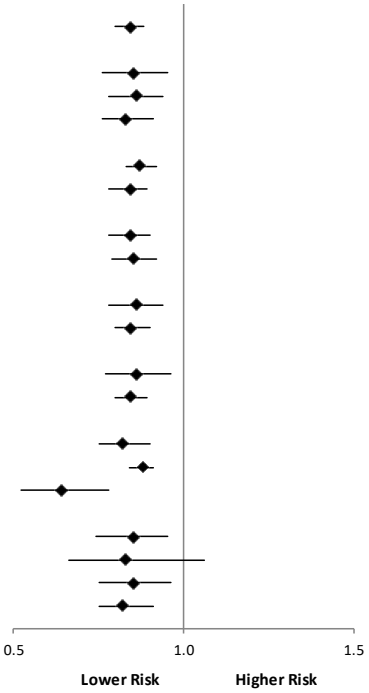
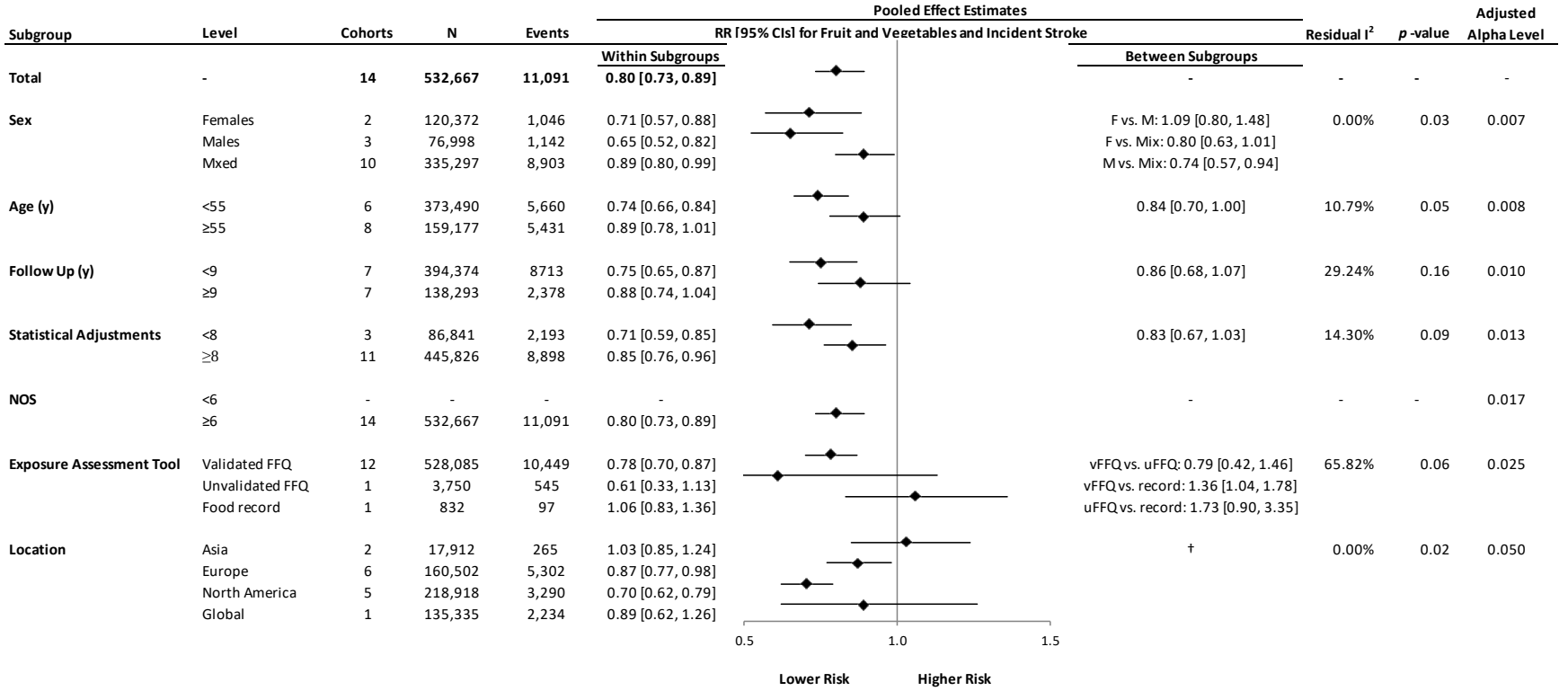


Figure S183. Categorical subgroup analyses of intake of vegetables and coronary heart disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 0.98 [0.83, 1.17]; Europe vs. Australia 0.98 [0.75, 1.28]; Europe vs. North America 0.97 [0.83, 1.14]; Asia vs. Australia 1.01 [0.77, 1.32]; Asia vs. North America 1.02 [0.87, 1.19]; Australia vs. North America 1.01 [0.78, 1.30]

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TOTAL FRUIT AND VEGETABLES AND STROKE INCIDENCE

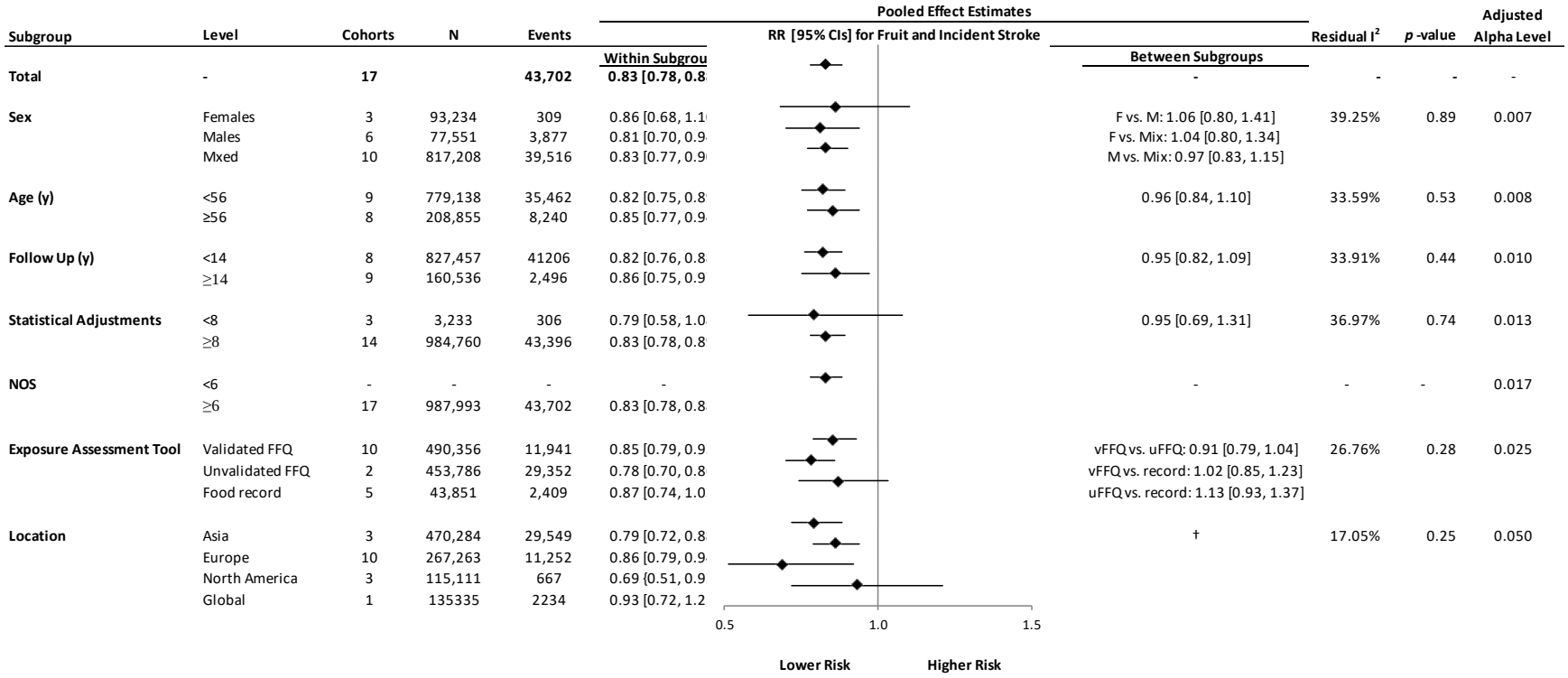


Subgroup	Level	Cohorts	N	Events	Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**14**	**532,667**	**11,091**	**0.80 [0.73, 0.89]**	-	-	-	-
Sex	Females	2	120,372	1,046	0.71 [0.57, 0.88]	F vs. M: 1.09 [0.80, 1.48]	0.00%	0.03	0.007
	Males	3	76,998	1,142	0.65 [0.52, 0.82]	F vs. Mix: 0.80 [0.63, 1.01]			
	Mxed	10	335,297	8,903	0.89 [0.80, 0.99]	M vs. Mix: 0.74 [0.57, 0.94]			
Age (y)	<55	6	373,490	5,660	0.74 [0.66, 0.84]	0.84 [0.70, 1.00]	10.79%	0.05	0.008
	≥55	8	159,177	5,431	0.89 [0.78, 1.01]				
Follow Up (y)	<9	7	394,374	8713	0.75 [0.65, 0.87]	0.86 [0.68, 1.07]	29.24%	0.16	0.010
	≥9	7	138,293	2,378	0.88 [0.74, 1.04]				
Statistical Adjustments	<8	3	86,841	2,193	0.71 [0.59, 0.85]	0.83 [0.67, 1.03]	14.30%	0.09	0.013
	≥8	11	445,826	8,898	0.85 [0.76, 0.96]				
NOS	<6	-	-	-	-	-	-	-	0.017
	≥6	14	532,667	11,091	0.80 [0.73, 0.89]				
Exposure Assessment Tool	Validated FFQ	12	528,085	10,449	0.78 [0.70, 0.87]	vFFQ vs. uFFQ: 0.79 [0.42, 1.46]	65.82%	0.06	0.025
	Unvalidated FFQ	1	3,750	545	0.61 [0.33, 1.13]	vFFQ vs. record: 1.36 [1.04, 1.78]			
	Food record	1	832	97	1.06 [0.83, 1.36]	uFFQ vs. record: 1.73 [0.90, 3.35]			
Location	Asia	2	17,912	265	1.03 [0.85, 1.24]	†	0.00%	0.02	0.050
	Europe	6	160,502	5,302	0.87 [0.77, 0.98]				
	North America	5	218,918	3,290	0.70 [0.62, 0.79]				
	Global	1	135,335	2,234	0.89 [0.62, 1.26]				

Figure S184. Categorical subgroup analyses of total fruit and vegetable intake and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs Asia 1.17 [0.94, 1.47]; Europe vs Global 1.02 [0.70, 1.48]; Europe vs NA 0.81 [0.68, 0.96]; Asia vs Global 1.16 [0.77, 1.72]; Asia vs NA 1.46 [1.16, 1.84]; Global vs NA 1.27 [0.87, 1.84]

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FRUIT AND STROKE INCIDENCE



Subgroup	Level	Cohorts	N	Events	Within Subgroup	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**17**		**43,702**	**0.83 [0.78, 0.8**	-	-	-	-
Sex	Females	3	93,234	309	0.86 [0.68, 1.1	F vs. M: 1.06 [0.80, 1.41]	39.25%	0.89	0.007
	Males	6	77,551	3,877	0.81 [0.70, 0.9	F vs. Mix: 1.04 [0.80, 1.34]			
	Mxed	10	817,208	39,516	0.83 [0.77, 0.9	M vs. Mix: 0.97 [0.83, 1.15]			
Age (y)	<56	9	779,138	35,462	0.82 [0.75, 0.8	0.96 [0.84, 1.10]	33.59%	0.53	0.008
	≥56	8	208,855	8,240	0.85 [0.77, 0.9				
Follow Up (y)	<14	8	827,457	41206	0.82 [0.76, 0.8	0.95 [0.82, 1.09]	33.91%	0.44	0.010
	≥14	9	160,536	2,496	0.86 [0.75, 0.9				
Statistical Adjustments	<8	3	3,233	306	0.79 [0.58, 1.0	0.95 [0.69, 1.31]	36.97%	0.74	0.013
	≥8	14	984,760	43,396	0.83 [0.78, 0.8				
NOS	<6	-	-	-	-	-	-	-	0.017
	≥6	17	987,993	43,702	0.83 [0.78, 0.8				
Exposure Assessment Tool	Validated FFQ	10	490,356	11,941	0.85 [0.79, 0.9	vFFQ vs. uFFQ: 0.91 [0.79, 1.04]	26.76%	0.28	0.025
	Unvalidated FFQ	2	453,786	29,352	0.78 [0.70, 0.8	vFFQ vs. record: 1.02 [0.85, 1.23]			
	Food record	5	43,851	2,409	0.87 [0.74, 1.0	uFFQ vs. record: 1.13 [0.93, 1.37]			
Location	Asia	3	470,284	29,549	0.79 [0.72, 0.8	†	17.05%	0.25	0.050
	Europe	10	267,263	11,252	0.86 [0.79, 0.9				
	North America	3	115,111	667	0.69 [0.51, 0.9				
	Global	1	135335	2234	0.93 [0.72, 1.2				

Figure S185. Categorical subgroup analyses of fruit intake and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.92 [0.81, 1.05]; Europe vs. Global 1.09 [0.82, 1.42]; Europe vs. North America 0.80 [0.59, 1.09]; Asia vs. Global 0.85 [0.65, 1.12]; Asia vs. North America 1.15 [0.85, 1.57]; Global vs. North America 1.36 [0.92, 2.01]

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VEGETABLES AND STROKE INCIDENCE

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: RR [95% CIs] for Fruit and CVD Mortality		Residual I^2	*p*-value	Adjusted Alpha Level
			0.0241		Within Subgroups	Between Subgroups			
Total	-	**16**	**564,531**	**13,607**	**0.89 [0.81, 0.97]**	-	-	-	-
Sex	Females	3	93,234	309	0.82 [0.61, 1.09]	F vs. M: 1.00 [0.72, 1.38]	41.34%	0.18	0.007
	Males	9	134,595	4,472	0.82 [0.70, 0.95]	F vs. Mix: 0.84 [0.62, 1.16]			
	Mxed	8	336,702	8,826	0.97 [0.85, 1.10]	M vs. Mix: 0.85 [0.69, 1.03]			
Age (y)	<58	8	437,979	9,019	0.88 [0.78, 1.00]	0.99 [0.78, 1.22]	50.42%	0.89	0.008
	≥58	9	126,552	4,588	0.90 [0.75, 1.06]				
Follow Up (y)	<14	8	432,836	12,645	0.88 [0.78, 1.00]]	0.97 [0.80, 1.20]	49.72%	0.80	0.010
	≥14	8	131,695	962	0.90 [0.76, 1.07]				
Statistical Adjustments	<8	3	3,233	306	0.82 [0.58, 1.15]	0.92 [0.64, 1.31]	52.43%	0.62	0.013
	≥8	13	561,298	13,301	0.89 [0.81, 0.99]				
NOS	<6	-	-	-	-	-	-	-	0.017
	≥6	16	564,531	13,607	0.87 [0.80, 0.94]				
Exposure Assessment Tool	Validated FFQ	10	512,840	11,401	0.91 [0.81, 1.01]	vFFQ vs. uFFQ: 0.47 [0.21, 1.05]	49.62%	0.17	0.025
	Unvalidated FFQ	1	2,121	196	0.43 [0.19, 0.95]	vFFQ vs. record: 0.95 [0.76, 1.20]			
	Food record	5	49,570	2,010	0.86 [0.70, 1.06]	uFFQ vs. record: 2.02 [0.89, 4.58]			
Location	Asia	3	28,270	619	1.03 [0.82, 1.30]	†	41.17%	0.23	0.050
	Europe	9	285,815	10,117	0.85 [0.77, 0.95]				
	North America	3	115,111	637	0.82 [0.60, 1.12]				
	Global	1	135,335	2,234	1.09 [0.79, 1.52]				

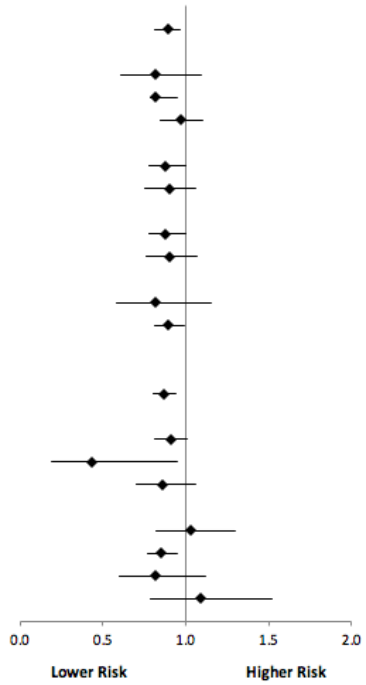


Figure S186. Categorical subgroup analyses of intake of vegetables and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 1.21 [0.94, 1.56]; Europe vs. Global 1.28 [0.91, 1.81]; Europe vs. NA 0.96 [0.69, 1.33]; Asia vs. Global 0.94 [0.63, 1.40]; Asia vs. NA 1.26 [0.86, 1.86]; Global vs. NA 1.34 [0.85, 2.10]

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FRUIT AND STROKE MORTALITY

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates RR [95% CIs] for Fruit and Stroke Mortality: Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**14**	**1,282,756**	**10,899**	**0.79 [0.71, 0.89]**	-	-	-	-
Sex	Females	4	155,963	2,022	0.76 [0.59, 0.99]	F vs. M: 0.90 [0.64, 1.29}	75.38%	0.8	0.007
	Males	4	107,467	2,302	0.85 [0.67, 1.07]	F vs. Mix: 0.98 [0.72, 1.32]			
	Mxed	9	1,126,793	8,877	0.78 [0.67, 0.91]	M vs. Mix: 1.08 [0.81, 1.44]			
Age (y)	<55	8	972,126	5,691	0.80 [0.69, 0.93]	1.02 [0.81, 1.28]	71.67%	0.85	0.008
	≥55	6	310,630	5,208	0.78 [0.66, 0.93]				
Follow Up (y)	<10	3	655,633	3905	0.76 [0.61, 0.95]	0.95 [0.73, 1.22]	69.52%	0.65	0.010
	≥10	14	627,123	6,994	0.81 [0.71, 0.92]				
Statistical Adjustments	<8	5	193,091	1,791	0.88 [0.673, 1.06]	1.15 [0.92, 1.44]	58.65%	0.19	0.013
	≥8	9	1,089,665	9,108	0.76 [0.67, 0.86]				
NOS	<6	3	187,214	1,668	0.92 [0.76, 1.12]	1.23 [0.99, 1.54]	54.86%	0.07	0.017
	≥6	11	1,095,542	9,231	0.75 [0.67, 0.85]				
Exposure Assessment Tool	Validated FFQ	7	772,822	5,387	0.80 [0.68, 0.96]	vFFQ vs. uFFQ: 1.01 [0.80, 1.29]	74.29%	0.49	0.025
	Unvalidated FFQ	5	494,945	5,004	0.82 [0.69, 0.97]	vFFQ vs. record: 0.81 [0.54, 1.21]			
	Food record	2	14,989	508	0.65 [0.45, 0.94]	uFFQ vs. record: 0.79 [0.53, 1.19]			
Location	Asia	6	581,472	6,978	0.74 [0.65, 0.85]	Europe vs. NA: 1.12 [0.75, 1.68]	77.24%	0.25	0.050
	Europe	7	527,256	3,061	0.86 [0.71, 1.03]	Europe vs. Asia: 0.87 [0.69, 1.10]			
	North America	1	174,028	860	0.96 [0.67, 1.37]	Asia vs. NA: 0.77 [0.53, 1.13]			

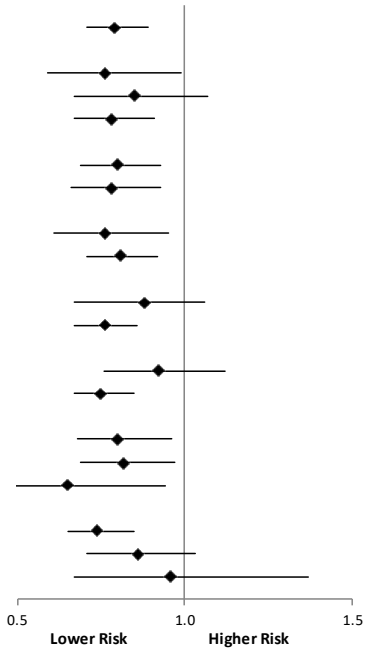


Figure S187. Categorical subgroup analyses of fruit intake and stroke mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

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VEGETABLES AND STROKE MORTALITY

Subgroup	Level	Cohorts	N	Events	Pooled Effect Estimates: Within Subgroups	Between Subgroups	Residual I^2	*p* -value	Adjusted Alpha Level
Total	-	**12**	**780,441**	**7,551**	**0.86 [0.78, 0.96]**	-	-	-	-
Sex	Females	3	120,315	1,752	0.80 [0.63, 1.02]	F vs. M: 0.90 [0.64, 1.27]	59.38%	0.76	0.007
	Males	4	103,142	2,160	0.89 [0.70, 1.13]	F vs. Mix: 0.91 [0.68, 1.22]			
	Mxed	7	556,984	3,639	0.88 [0.75, 1.03]	M vs. Mix: 1.01 [0.76, 1.35]			
Age (y)	<58	7	497,499	258	0.84 [0.71, 1.00]	0.95 [0.75, 1.19]	62.43%	0.63	0.008
	≥58	5	282,942	7,293	0.88 [0.76, 1.03]				
Follow Up (y)	<10	1	193,291	969	0.85 [0.61, 1.18]	0.98 [0.69, 1.38]	64.65%	0.9	0.010
	≥10	9	587,150	6,582	0.87 [0.77, 0.98]				
Statistical Adjustments	<8	3	182,350	1,425	0.92 [0.75, 1.13]	1.08 [0.85, 1.38]	49.01%	0.51	0.013
	≥8	7	598,091	6,126	0.85 [0.75, 0.97]				
NOS	<6	1	174,028	860	0.97 [0.79, 1.19]	1.16 [0.91, 1.47]	44.20%	0.21	0.017
	≥6	11	606,413	6,691	0.84 [0.74, 0.94]				
Exposure Assessment Tool	Validated FFQ	5	742,364	5,239	0.83 [0.72, 0.96]	vFF vs. uFFQ: 1.15 {0.92, 1.43]	45.92%	0.26	0.025
	Unvalidated FFQ	4	23,088	1,804	0.95 [0.81, 1.12]	vFFQ vs. record: 0.90 [0.62, 1.29]			
	Food record	3	14,989	508	0.75 [0.53, 1.04]	uFFQ vs. record: 0.78 [0.54, 1.13]			
Location	Asia	5	116,685	3,590	0.92 [0.79, 1.07]	†	57.00%	0.50	0.050
	Australia	1	1,226	92	0.80 [0.53, 1.21]				
	Europe	5	486,057	2,557	0.76 [0.61, 0.96]				
	North America	1	174,028	860	0.90 [0.62, 1.29]				

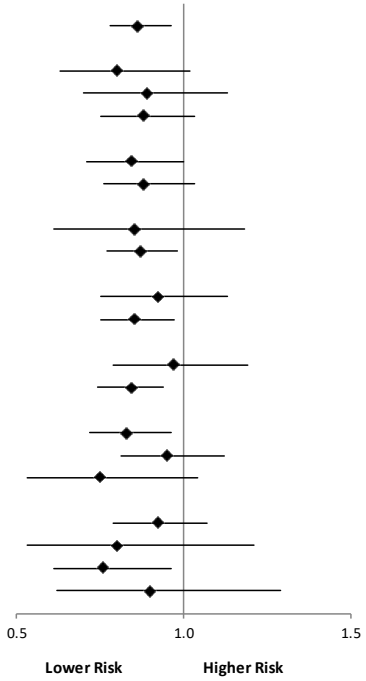


Figure S188. Categorical subgroup analyses of intake of vegetables and stroke mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I^2 value indicates the inter-study heterogeneity unexplained by the subgroup. FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
† Europe vs. Asia 1.20 [0.92, 1.57]; Europe vs. Australia 1.05 [0.66, 1.67]; Europe vs. North America 1.17 [0.76, 1.80]; Asia vs. Australia 1.44 [0.74, 1.77]; Asia vs. North America 1.03 [0.69, 1.53]; Australia vs. North America 0.90 [0.52, 1.55

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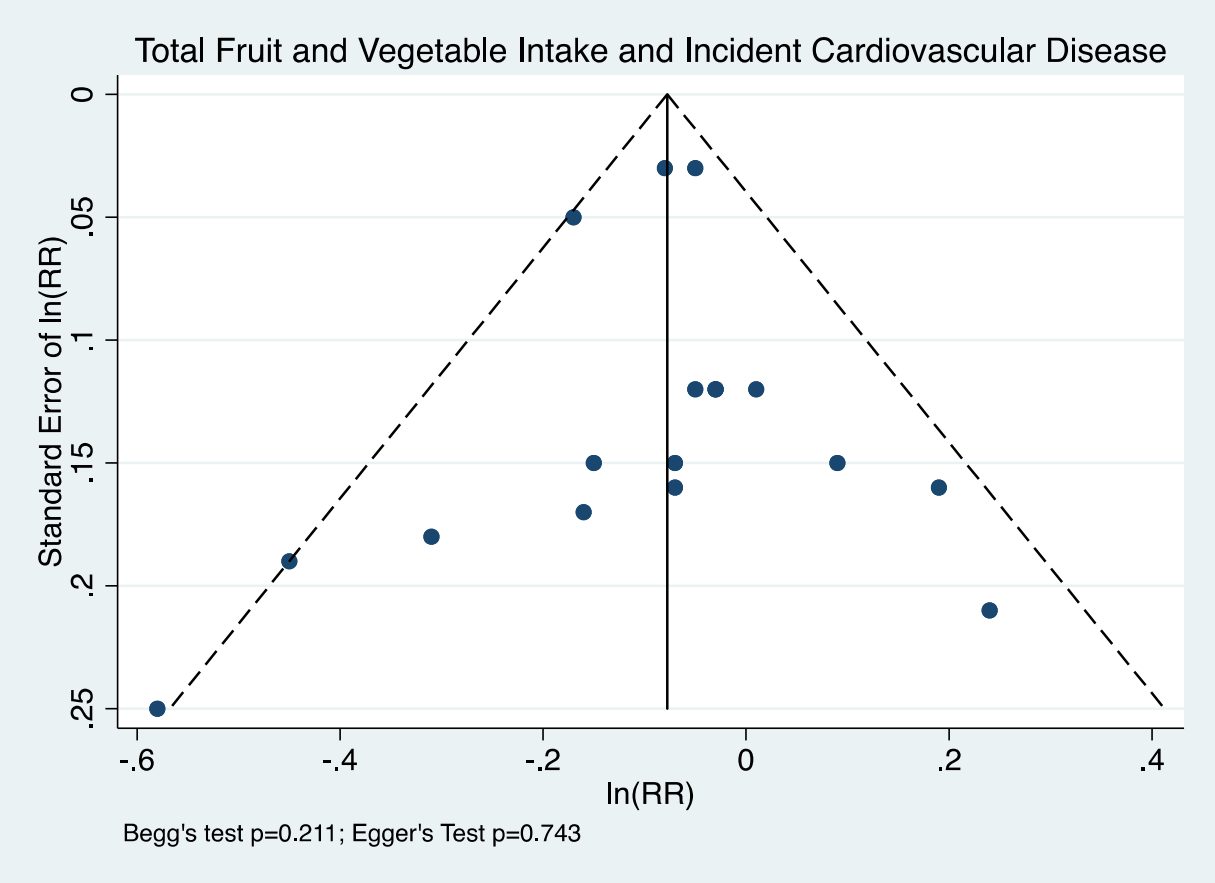


Figure S189. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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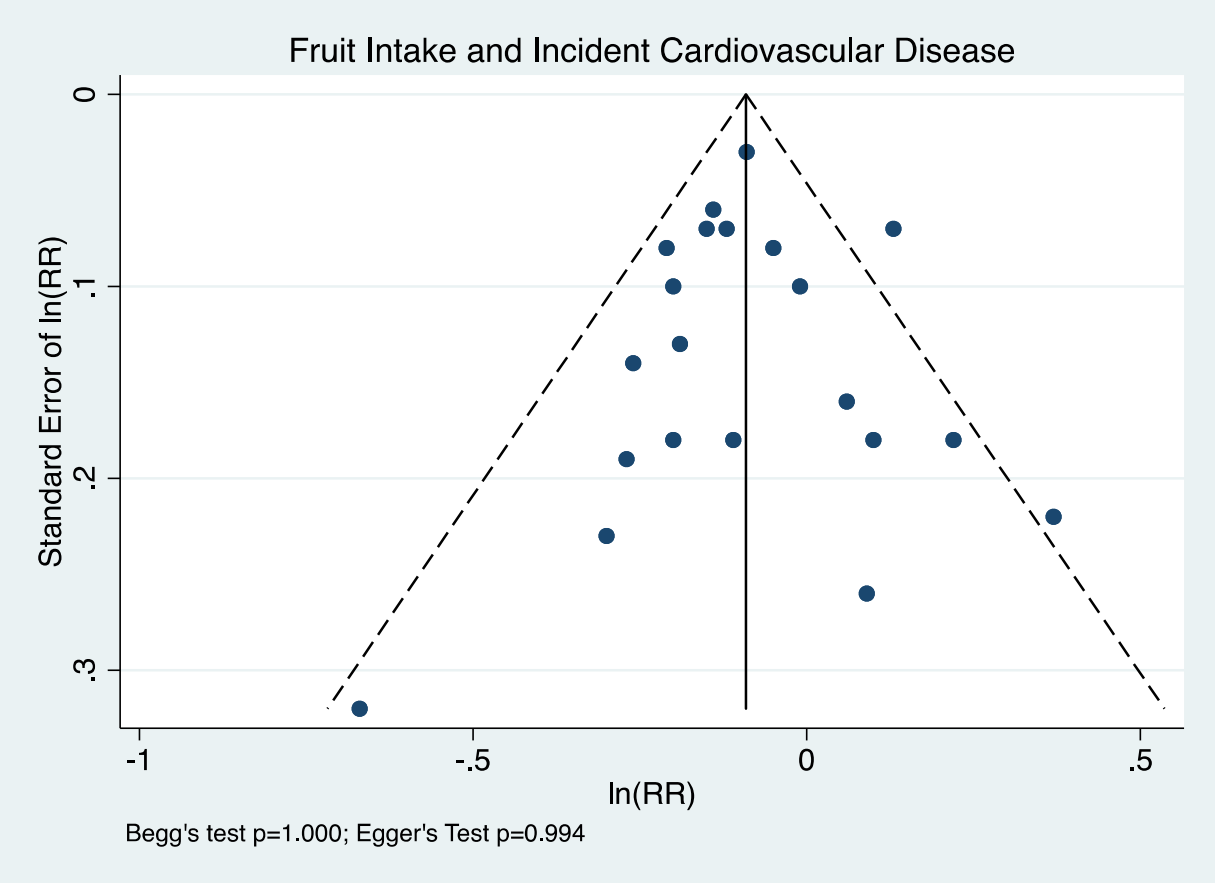


Figure S190. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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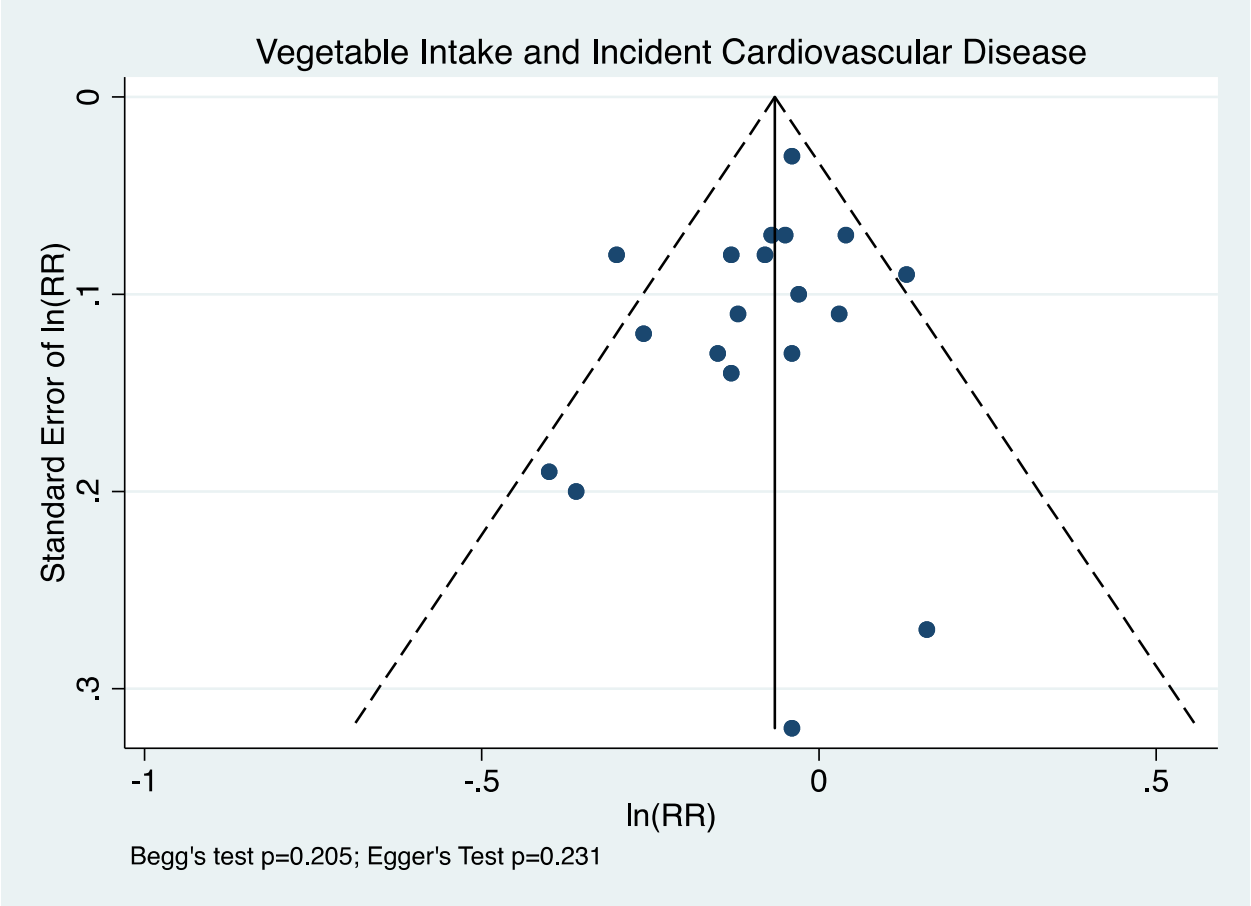


Figure S191. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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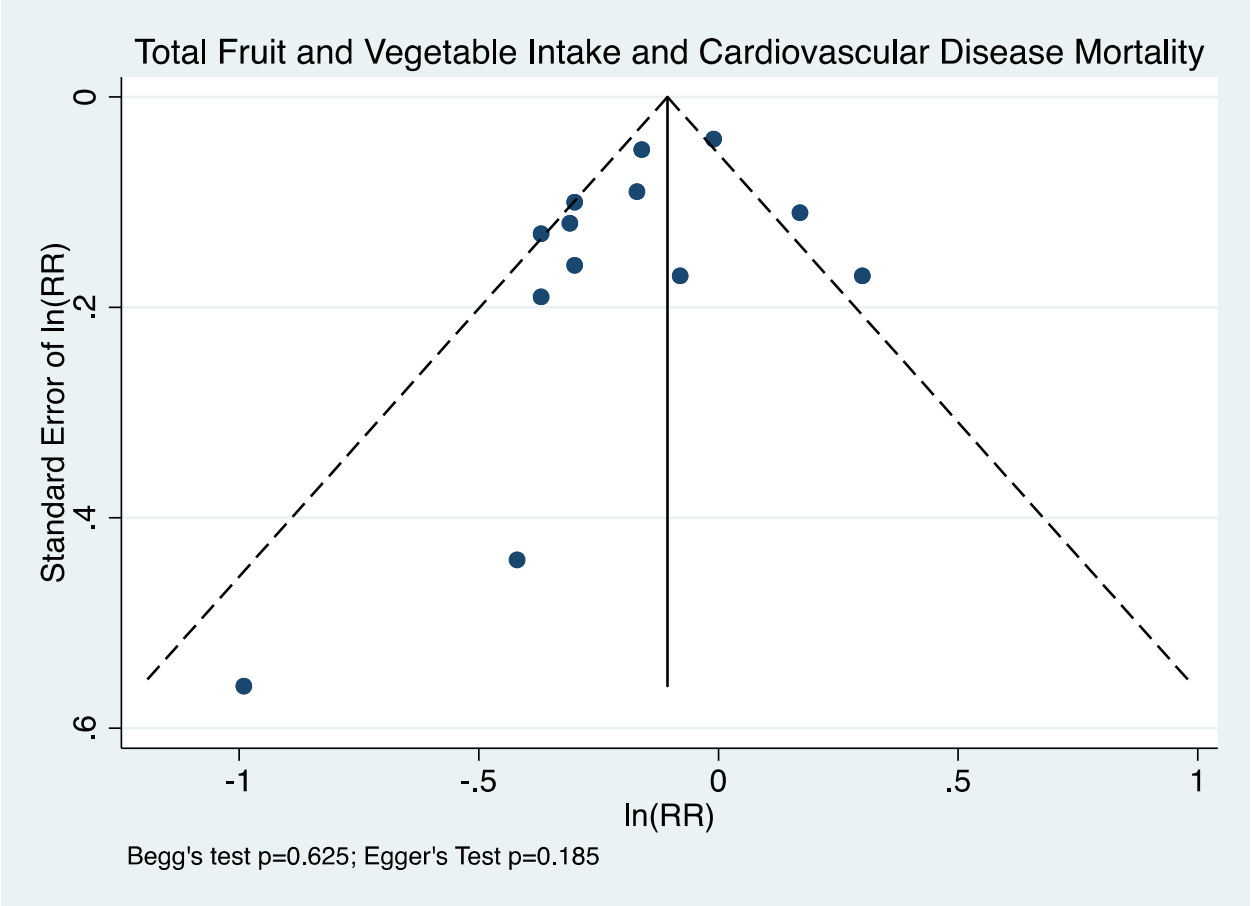


Figure S192. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease mortality comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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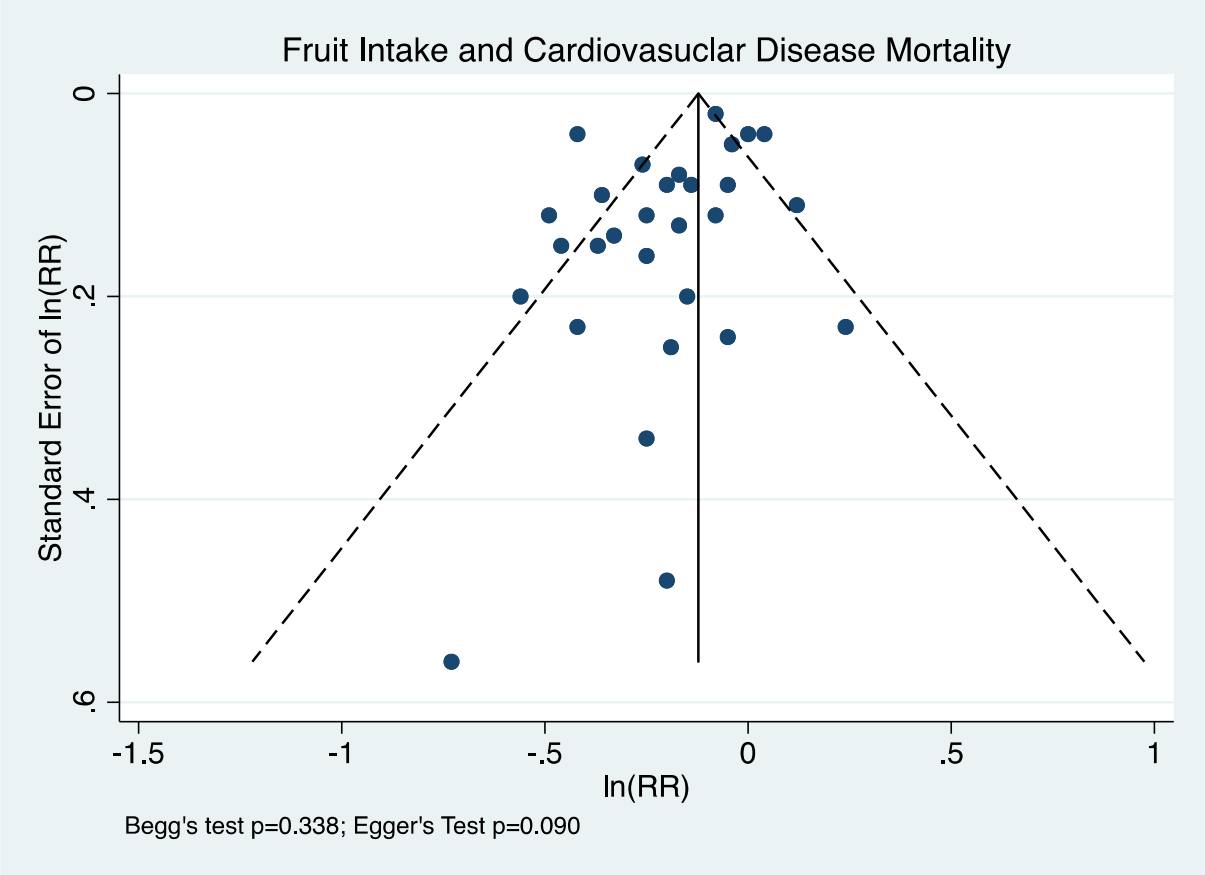


Figure S193. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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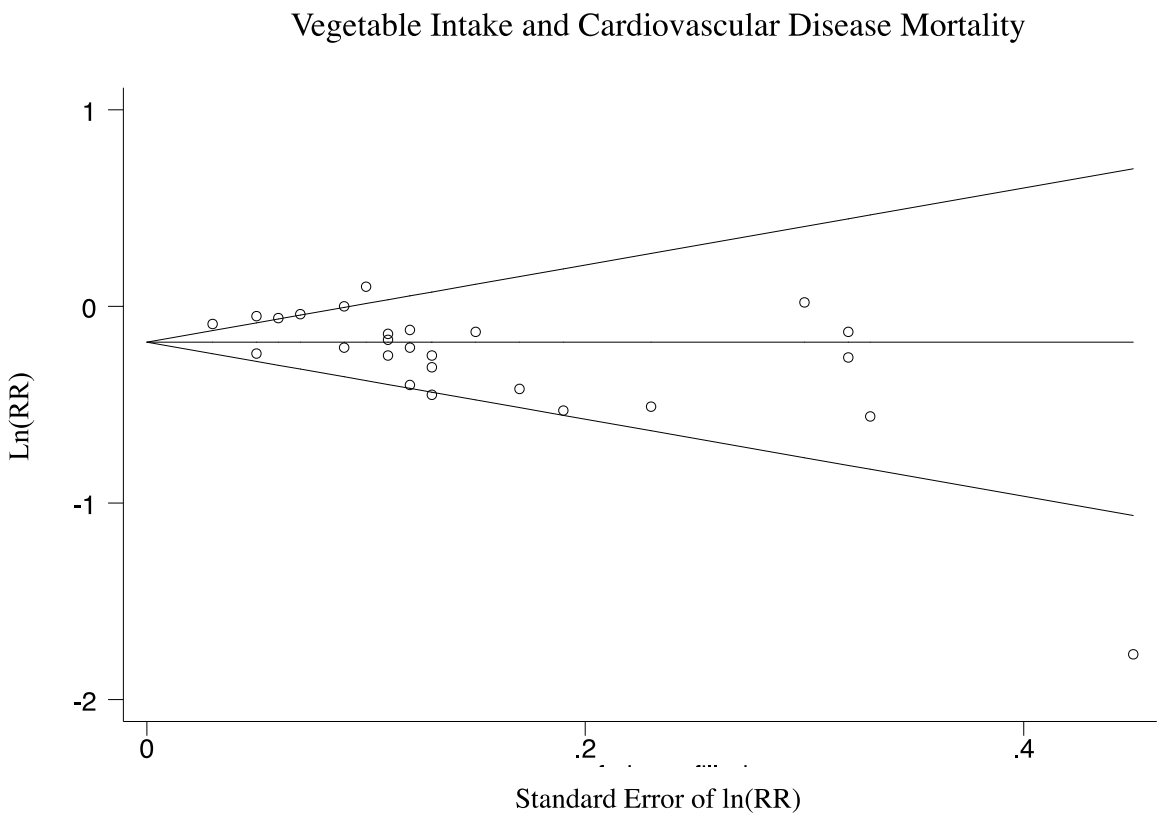


Figure S194. Funnel plot for trim-and-fill analysis for coronary heart disease mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [ln(RR)]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.

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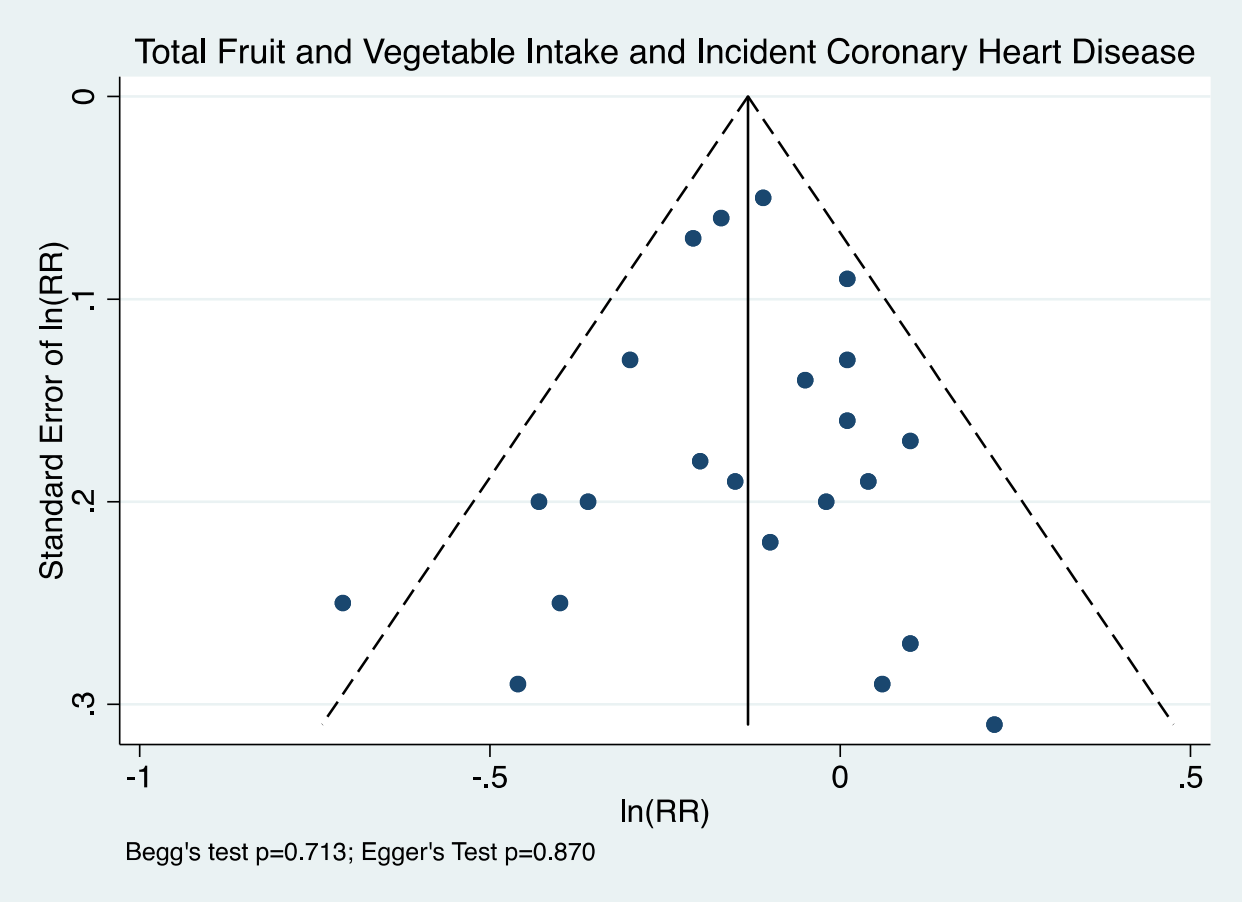


Figure S195. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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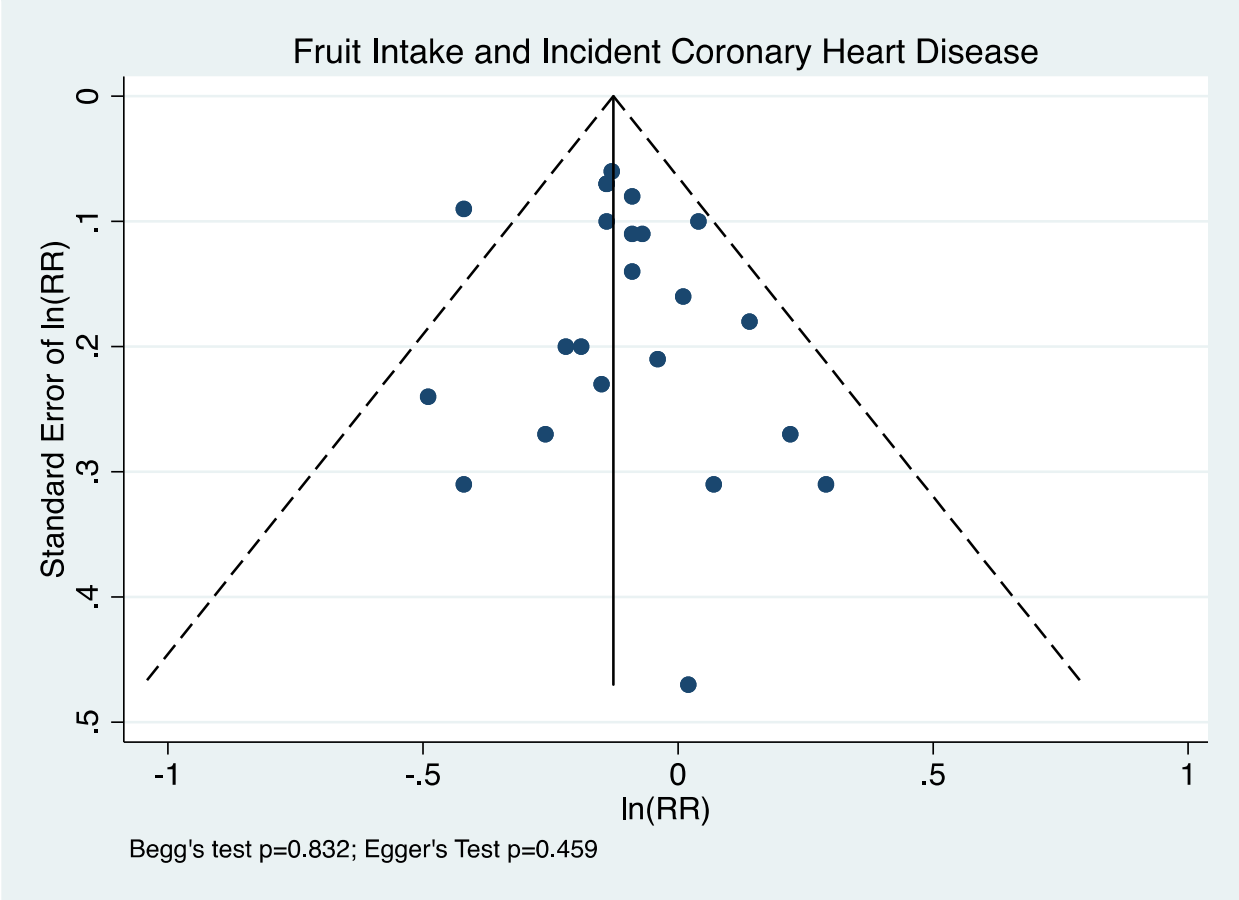


Figure S196. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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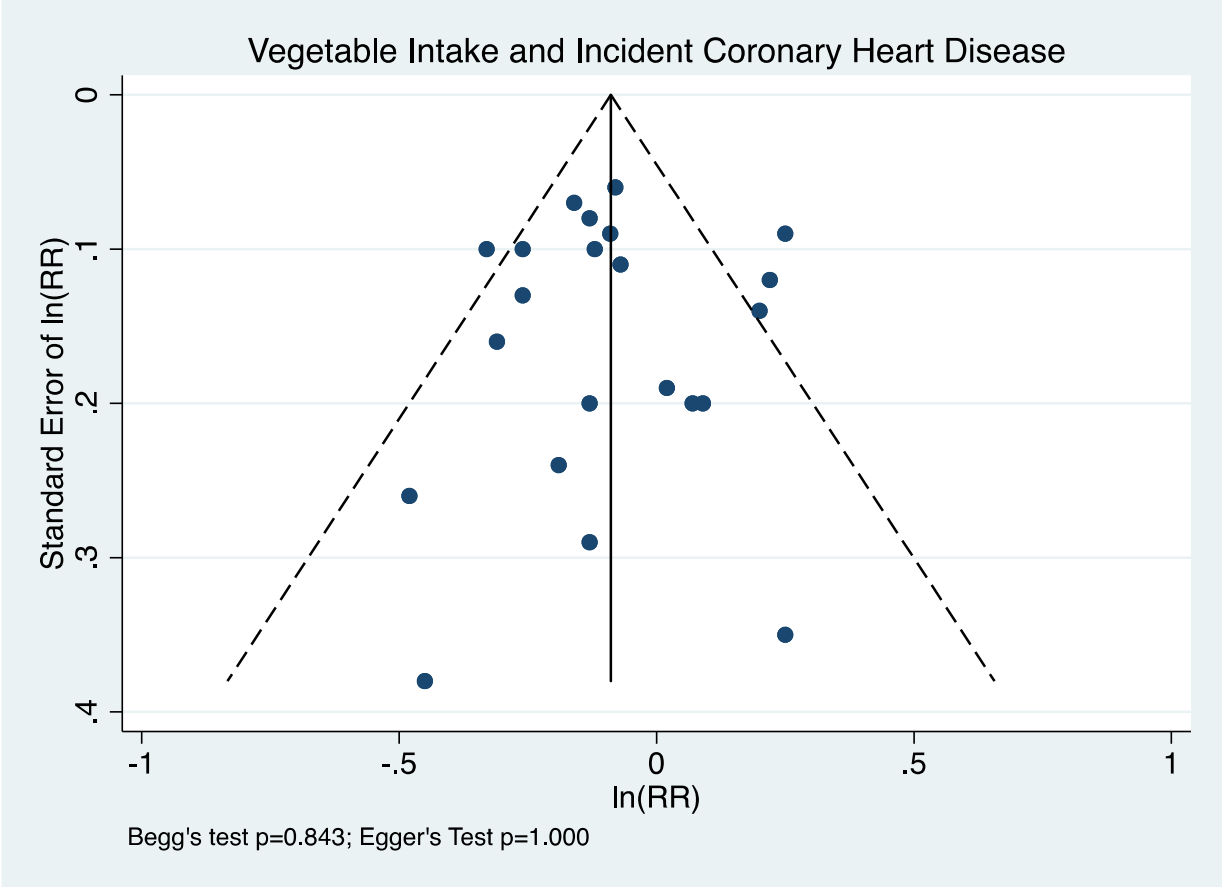


Figure S197. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease incidence comparing the highest and lowest quantiles of vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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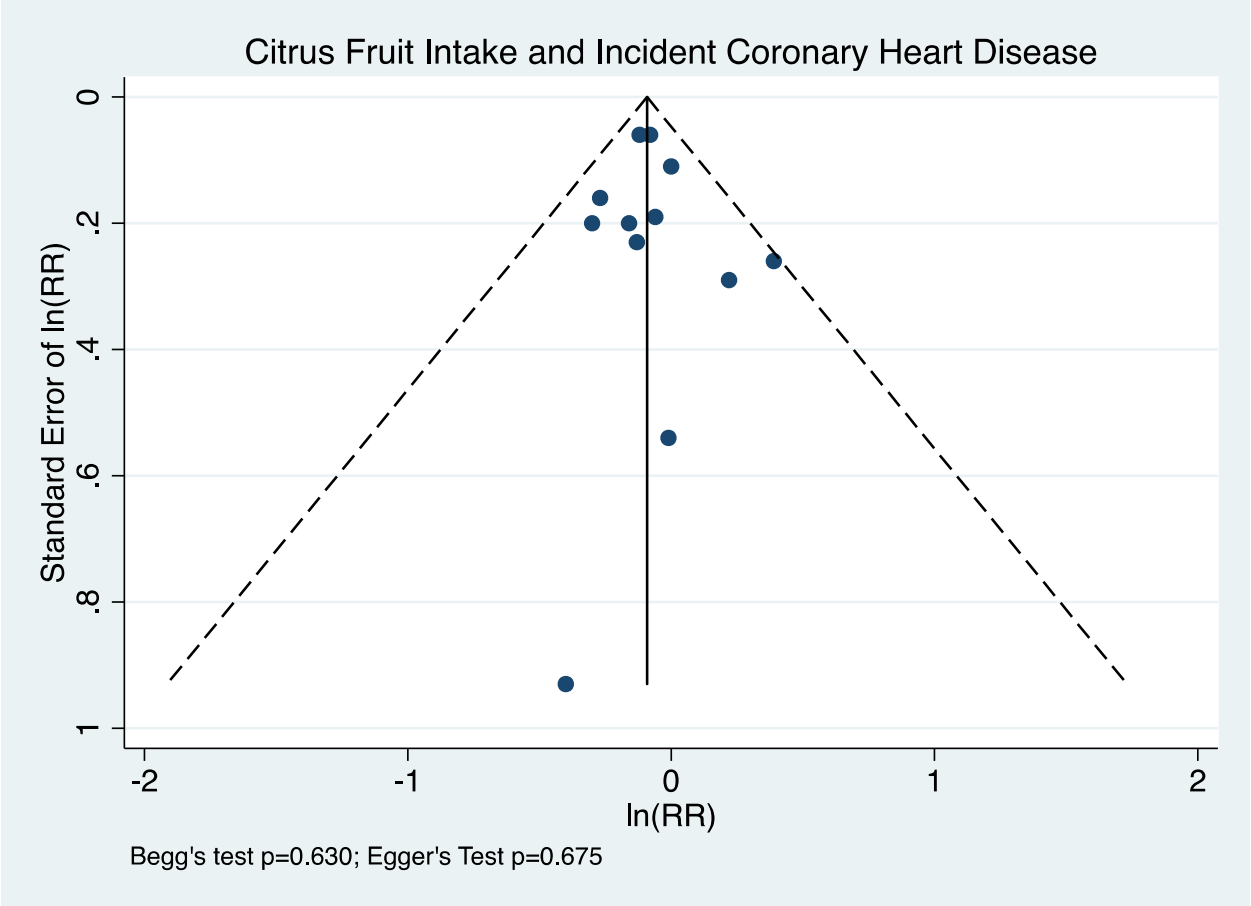


Figure S198. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease incidence comparing the highest and lowest quantiles of citrus fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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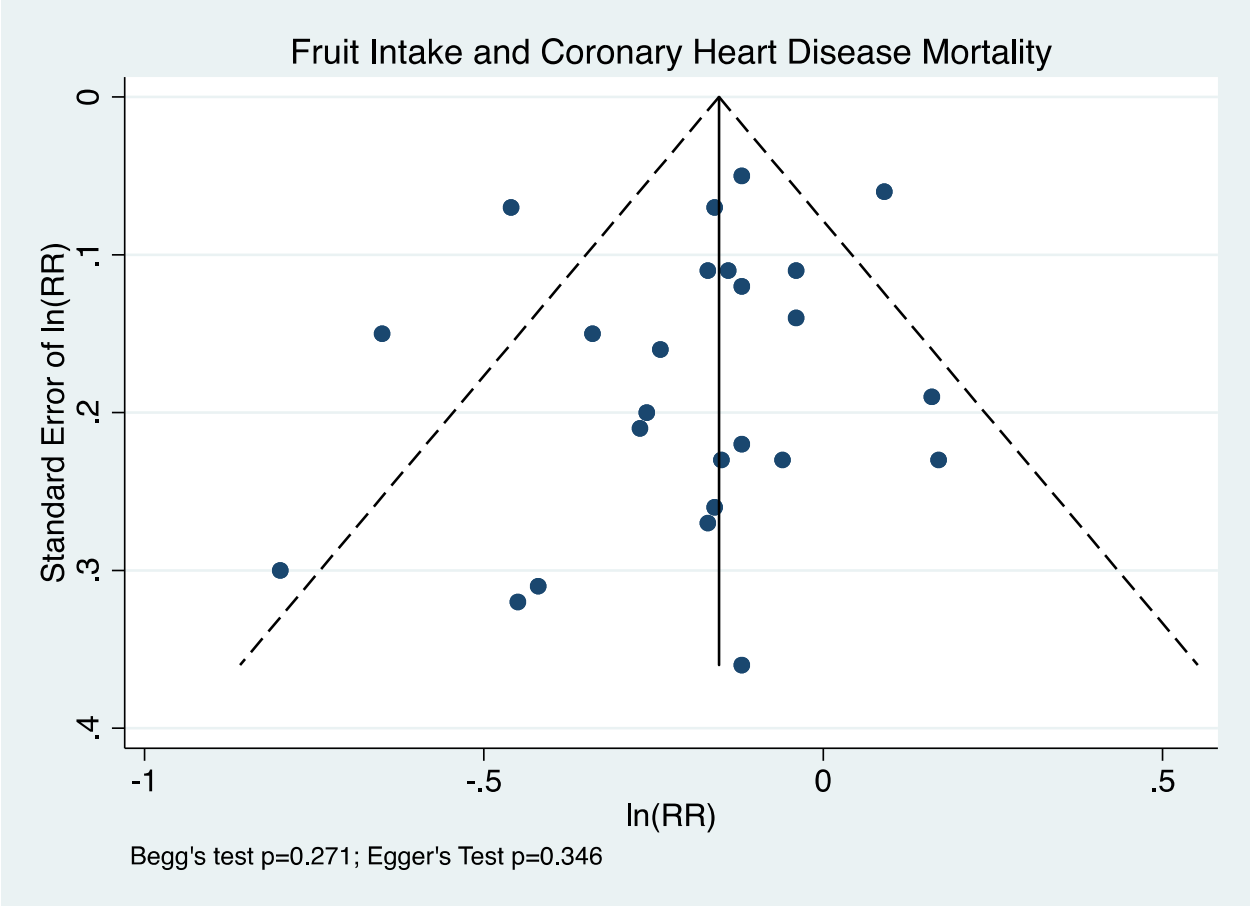


Figure S199. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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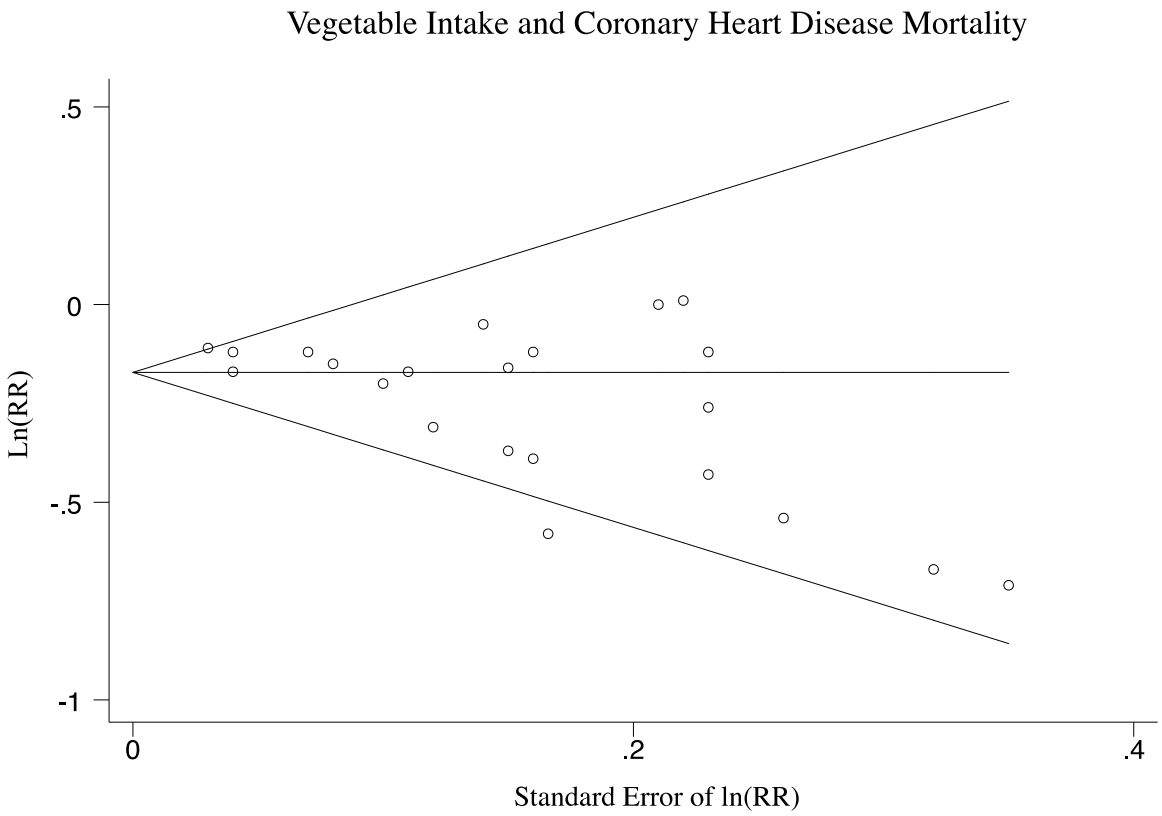


Figure S200. Funnel plot for trim-and-fill analysis for coronary heart disease mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [ln(RR)]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.

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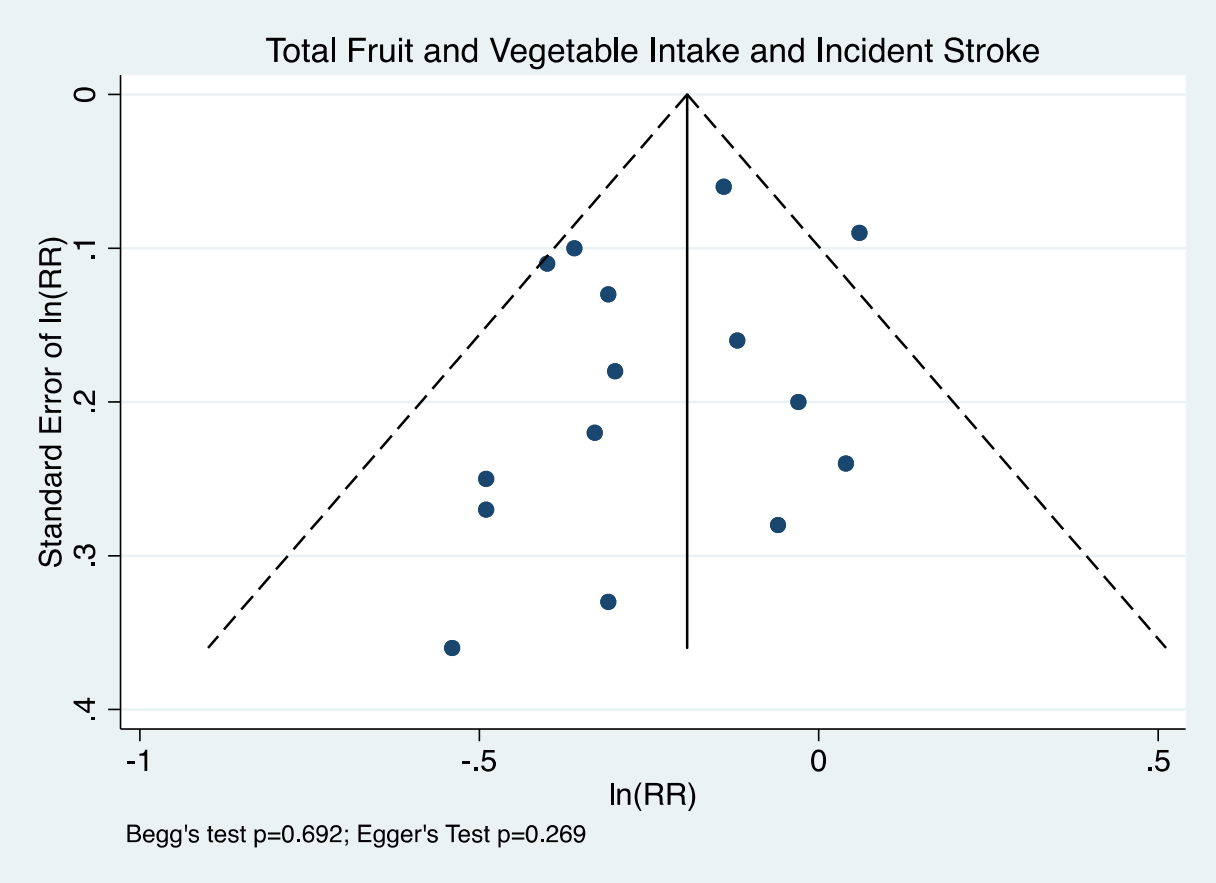


Figure S201. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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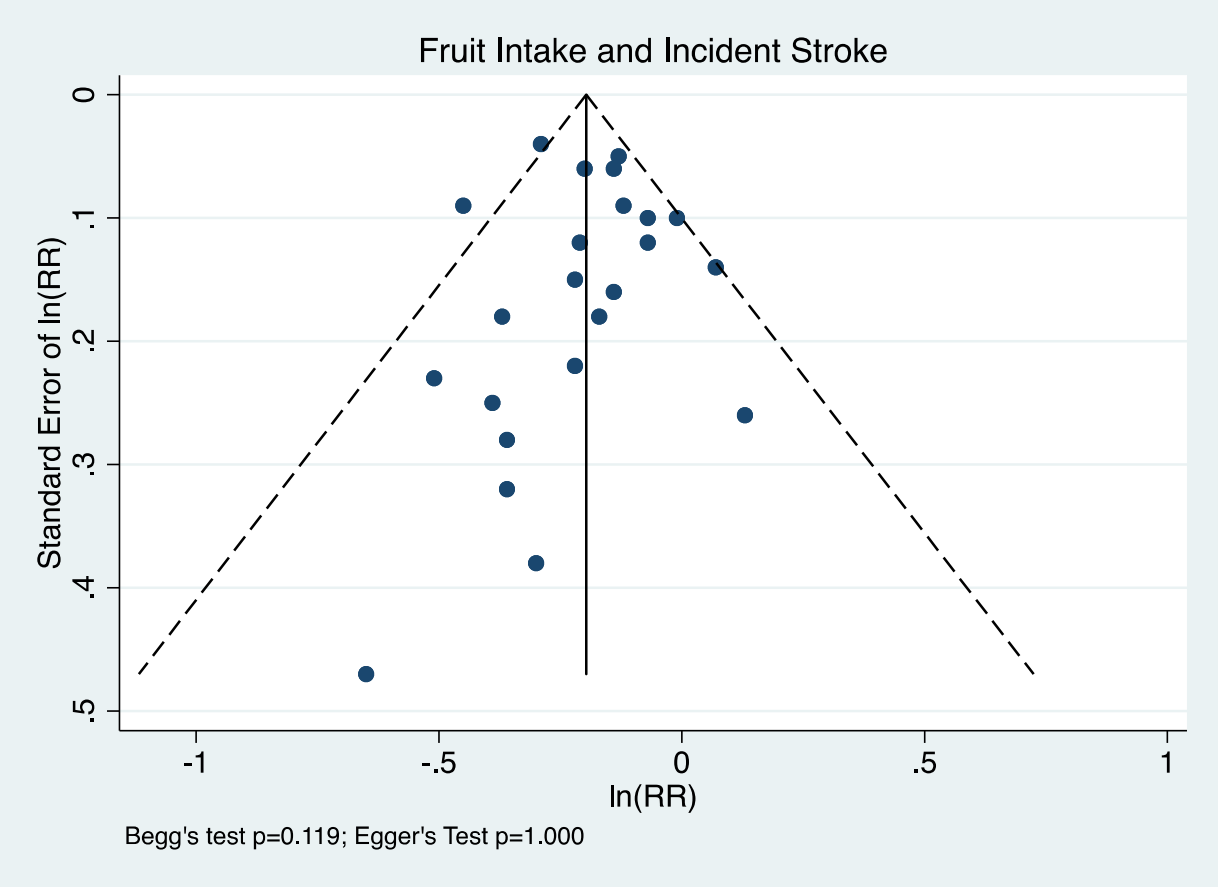


Figure S202. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke incidence comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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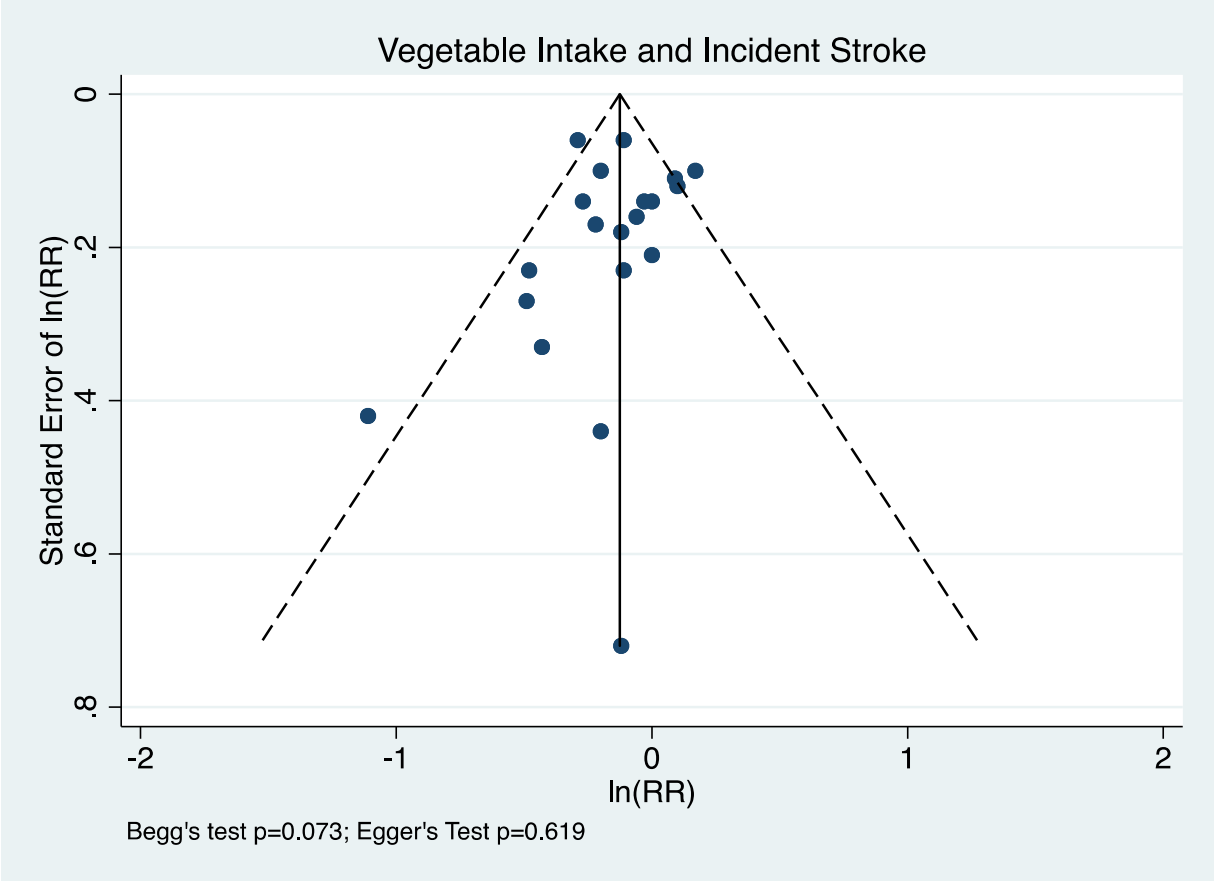
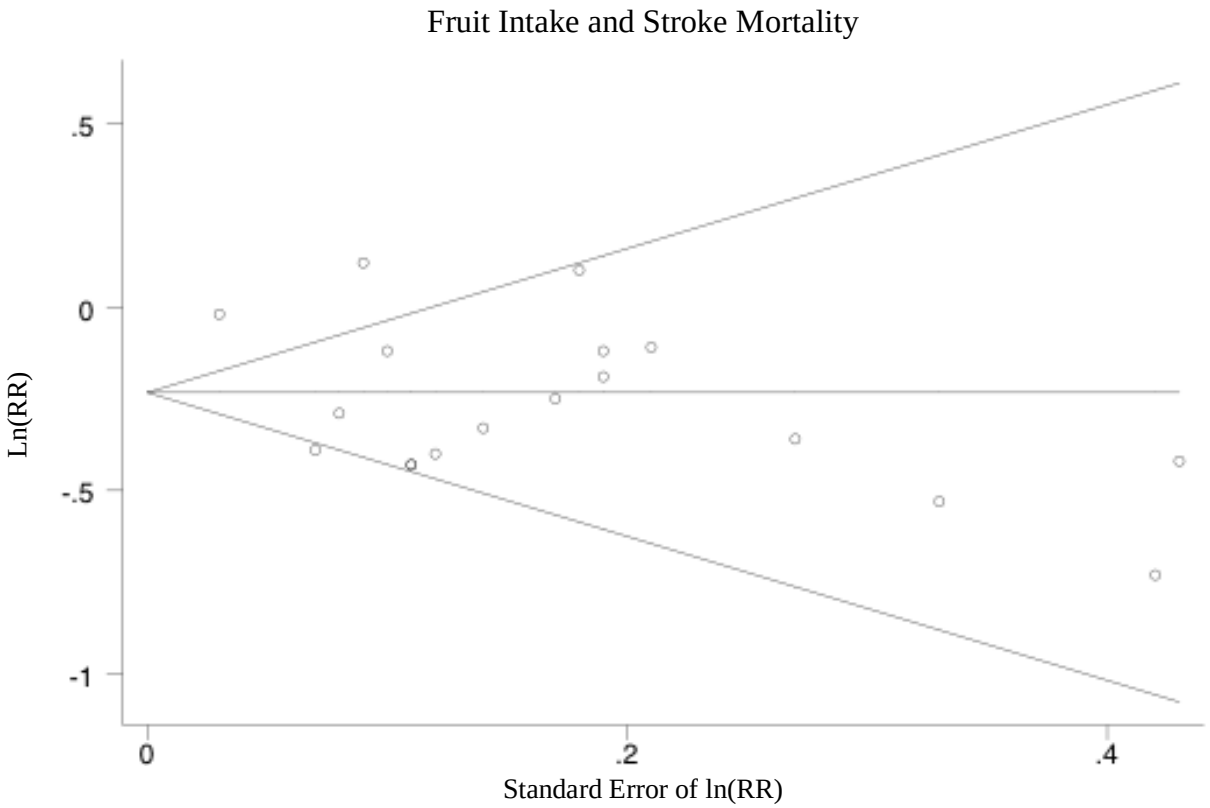


Figure S203. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke incidence comparing the highest and lowest quantiles of vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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Imputed RR accounting for publication bias: N/A
P-value: N/A

Figure S204. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

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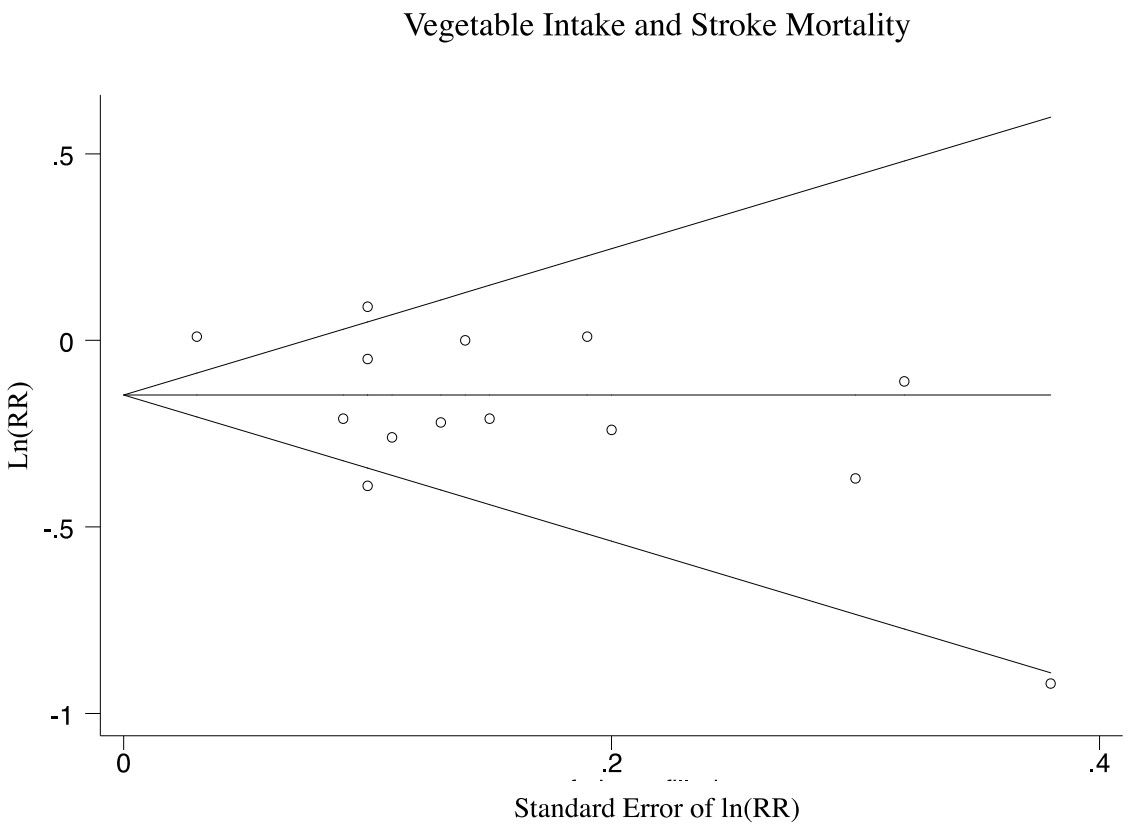


Figure S205. Funnel plot for trim-and-fill analysis for stroke mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [ln(RR)]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.

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