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REVIEW

Fruit and vegetables consumption and incident hypertension: dose–response meta-analysis of prospective cohort studies

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The role of dietary factors on chronic diseases seems essential in the potentially adverse or preventive effects. However, no evidence of dose–response meta-analysis of prospective cohort studies has verified the association between the intake of fruit and/or vegetables and the risk of developing hypertension. The PubMed and Embase were searched for prospective cohort studies. A generic inverse-variance method with random effects model was used to calculate the pooled relative risks (RRs) and 95% confidence intervals (CIs). Generalized least squares trend estimation model was used to calculate the study-specific slopes for the dose–response analyses. Seven articles comprised nine cohorts involving 185 676 participants were assessed. The highest intake of fruit or vegetables separately, and total fruit and vegetables were inversely associated with the incident risk of hypertension compared with the lowest level, and the pooled RRs and 95% CIs were 0.87 (0.79, 0.95), 0.88 (0.79, 0.99) and 0.90 (0.84, 0.98), respectively. We also found an inverse dose–response relation between the risk of developing hypertension and fruit intake, and total fruit and vegetables consumption. The incident risk of hypertension was decreased by 1.9% for each serving per day of fruit consumption, and decreased by 1.2% for each serving per day of total fruit and vegetables consumption. Our results support the recommendation to increase the consumption of fruit and vegetables with respect to preventing the risk of developing hypertension. However, further large prospective studies and long-term high-quality randomized controlled trials are still needed to confirm the observed association.

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INTRODUCTION

As a concomitant risk factor of cardiovascular disease and kidney disease, hypertension remains one of the most common causes of morbidity and mortality in many populations.[1–3] Overall, more than a quarter of adults were hypertensive patients in 2000, and this proportion had been estimated to increase by about 60% in 2025.[1] Therefore, primary prevention of hypertension has been considered an important public health issue around the world.

The role of dietary factors in chronic disease seems essential in the potentially adverse or preventive effects.[4–10] Among these dietary factors, previous meta-analyses have demonstrated that fruit and vegetables consumption has a specific powerful association with the incident risk of many chronic diseases, such as type 2 diabetes, stroke and coronary heart disease.[11–14] However, no evidence of dose–response meta-analysis of prospective cohort studies has verified the association between the intake of fruit and/or vegetables and the risk of developing hypertension. Previous cross-sectional studies and case–control studies have ascertained the effect of increasing fruit and vegetables consumption on the decreased risk of hypertension and on the blood pressure reduction; however, these studies leave much uncertainty regarding the causal mechanism of the association.[15–18] Moreover, due to the heterogeneous geographical ethnic backgrounds and various methods of diet assessment and outcome ascertainment among studies, the magnitude of associations were inconsistent in the above findings.[15–18] To date, a number of clinical trials have shown that a diet high in fruit and vegetables has beneficial effect on blood pressure reduction. The duration of intervention ranged from 6 weeks to 8 months among these trials, and thus the long-term effect of fruit and vegetables intake was still unclear.[19–21]

In the present study, we performed a systematic review and meta-analysis to summarise the evidences from prospective cohort studies on fruit and vegetables consumption, fruit consumption or vegetables consumption separately, and the risk of incident hypertension. Furthermore, we also quantified the dose–response patterns of fruit and/or vegetables intake on the risk of developing hypertension.

MATERIALS AND METHODS

Literature search

The present study was carried out in adherence with the Cochrane Handbook for Systematic Reviews[22] and the meta-analysis of observational studies in epidemiology guidelines.[23] We searched the PubMed (1950 to 4 November 2015) and the Embase (1974 to 4 November 2015) databases for records to report fruit and/or vegetables consumption and the risk of incident hypertension with no language restriction. Our search included both free-text and Medical Subject Headings (MeSH) terms, such as 'fruit*', 'vegetable*', 'fruit[MeSH Terms]', 'vegetables[MeSH Terms]', 'hypertension[MeSH Terms]', 'hypertensi*', 'HBP' and 'high blood

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pressure*'. Details of the search strategy are shown in Supplementary Table 1. When multiple publications from the same study were identified, we included the one with the longest duration of follow-up. Furthermore, the reference lists of relevant articles were manually searched to identify more potentially eligible articles.

Selection criteria and data extraction

The initial search was conducted by Wu and Sun independently. Duplicate records were deleted, and the titles and abstracts of each article were screened. We independently identified each article as excluded or requiring further assessment. Any disagreements were resolved by consensus.

We included articles that met the following criteria: (1) the studies reported relative risks (RRs) or hazard ratios and their corresponding 95% confidence intervals (CIs) of incident hypertension in relation to total fruit and vegetables intake, and fruit or vegetables intake separately; (2) the study design was based on prospective cohort; (3) the participants were adults aged 18 years or above. Studies were excluded if: (1) the data described the effect of individual fruit and vegetables, such as garlic, beans and soybeans; (2) the data described the surrogate nutrients of fruit and vegetables, such as fruit juice, vegetable protein and fibre; (3) the participants were pregnant or lactating females.

Data extraction was independently performed by Wu and Sun. Disagreements were resolved by discussing with He. The following data were extracted from each article: the first author, the year of publication, the study location, the number of participants, the characteristics of participants, the number of cases, baseline blood pressure, method of exposure and outcome measurements, the duration of follow-up, the RRs or hazard ratios with corresponding 95% CIs of the incident hypertension for all categories of fruit and/or vegetables consumption (the largest number of covariates in the adjusted model). Risks were estimated from the published beta-coefficients if possible.

Quality assessment

We used the Newcastle-Ottawa quality scale to assess the quality of the included cohorts. A quality of 'high' (6–9 points) or 'low' (0–6 points) were assigned according to the following domains: the basis of the cohort selection (0–4 points), the comparability of the cohort design and analysis (0–2 points), and the adequacy of the exposure and outcome measurements (0–3 points). Disagreements were resolved by consensus with the third author (He).

Statistical analysis

We calculated the pooled RRs (95% CIs) for the highest compared with the lowest category of fruit and/or vegetables consumption. A generic inverse-variance method with random effects model was used to pool the outcome data. Between-study heterogeneity was examined by the *Q*-test and I^2 statistic; an I^2 statistic $>50\%$ indicated significant heterogeneity.[24] Subgroup analyses and meta-regression were conducted to explore potential sources of heterogeneity by pre-specified characteristics (study location, duration of follow-up, gender, sample size, assessment method of exposure and outcome ascertainment). Sensitivity analysis was performed to estimate the influence of a single study on the overall pooled results by omitting one study at every turn. Begg's and Egger's tests were used to evaluate the presence of publication bias.[25,26]

Generalized least squares trend estimation model was used to calculate the study-specific slopes for the dose–response analysis based on method reported by Orsini *et al.*[27,28] We extracted data on all (at least three) categories of total fruit and vegetables intake and fruit or vegetables intake separately, the distribution of cases and person-years, and hazard ratios or RRs with 95% CIs of incident hypertension. Doses reported as gram per day were converted to serving per day, using a standard portion size of 106 g.[29] For categories of the fruit and vegetables consumption that were open (for example, 1–2 servings per day), we assigned the median value as the corresponding dose of consumption. If the highest category was open-ended (for example, >2 servings per day), we assumed that the boundary had the same amplitude as the preceding category.[30] The dose–response results in the forest plots were presented for every one serving per day increment in fruit and vegetables consumption. We used a four-knot-restricted cubic spline model to test for nonlinearity hypothesis in the association between fruit and vegetables consumption and the incident risk of hypertension. We verified that the regression coefficients of the last two spline transformations both equal to zero (the joint null hypothesis).[27,28] Simple generalized least squares trend estimation model (without the restricted cubic spline model) was used to test the linear hypothesis, if the test for the non-linear association was not statistically significant.

Stata software, version 12.0 (StataCorp LP, College Station, TX, USA) and Review Manager software, version 5.2 (The Nordic Cochrane Centre, Copenhagen, Denmark) were used for the statistical analyses. *P*-values <0.05 were considered statistically significant.

RESULTS

Study identification and selection

Figure 1 presents the detailed flow diagram of articles included in the present meta-analysis. A total of 1570 articles were identified from the initial database search (Pubmed: 824 articles, Embase: 746 articles). Of those studies, 304 articles were excluded for duplicates. After reading the titles and abstracts, 1233 articles were excluded. The remaining 33 articles were reviewed in full for eligibility. No additional records were identified from the reference

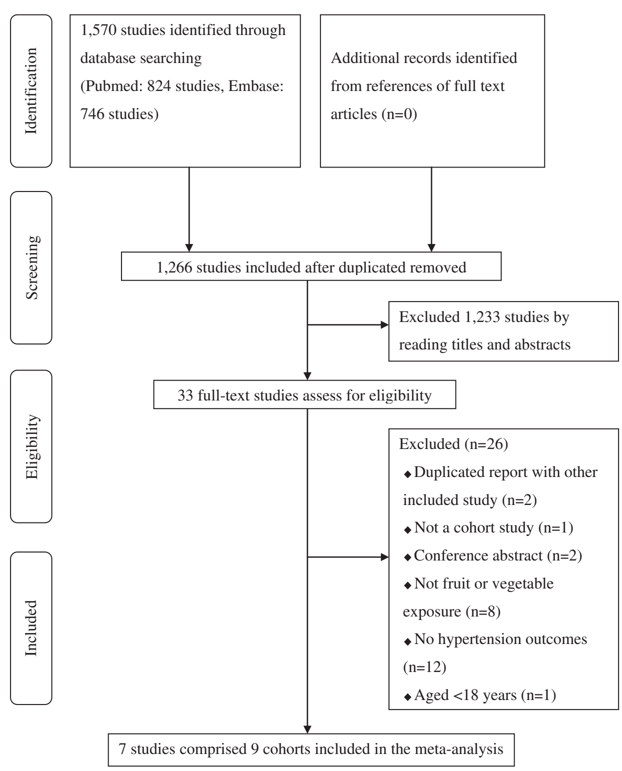

Figure 1. Flow diagram of articles included in the meta-analysis.

Table 1. Characteristics of the included studies

First author, published year	*Country, cohort*	*Baseline year*	*Follow-up (years)*	*Male (%)*	*Age (years) at baseline (min-max)*	*Participants no.*	*Exposure*		*Incident hypertension*		*Adjustment for covariates*
							Method of assessment	*Servings per day (highest vs lowest)*	*Method of assessment*	*Case no.*	
Borgi, 2016[31]	USA, NHS cohort	1976	26.4	0.0	42–58	39 164	FFQ, interviewer-administered	F: $\geqslant 4$ vs <1, V: $\geqslant 4$ vs <1, FV: $\geqslant 6$ vs $\leqslant 1$	Self-reported	35 375	Age, race/ethnicity, BMI, current smoking status, physical activity, weight change per FFQ cycle, menopausal status, alcohol intake, current oral contraceptive use, analgesic, use (nonsteroidal anti-inflammatory drugs, acetaminophen, aspirin), family history of hypertension, total energy intake, animal flesh intake, whole grains, sugar-sweetened and artificially sweetened diet beverage intake
	USA, NHSII cohort	1989	21.0	0.0	37–48	63 885				25 246	
	USA, HPFS cohort	1986	28.0	100.0	43–64	20 010				16 752	
Camões, 2010[32]	Portugal, EPIPorto cohort	1999	3.8	38.3	40	549	FFQ, interviewer-administered	FV: >5 vs <4	Measured	160	Age, gender, education, BMI, physical activity level and total energy intake
Nunez-Cordoba, 2009[33]	Spain, SUN cohort	1999	4.1	37.9	20–95	8594	FFQ, interviewer-administered	F: >4 vs <1.1, V: >4 vs <1.1, FV: >5 vs <2.1	Self-reported	426	Age, gender, total energy intake, BMI, physical activity, alcohol, family history of hypertension, sodium intake, low-fat dairy intake, whole grains intake, fish intake and smoking
Psaltopoulou, 2004[34]	Greece, EPIC cohort	1994	5.0	42.7	20–86	20 343	FFQ, interviewer-administered	F: >3 vs <1, V: >5 vs <1	Measured	2146	Age, gender, residence, and interactions between age and sex, age and residence, and sex and residence
Steffen, 2005[35]	USA, CARDIA cohort	1985	15.0	43.5	18–30	4304	FFQ, interviewer-administered	F: >1.5 vs <0.2, V: >3.3 vs <1.2	Measured	997	Age, sex, race, centre, energy intake, education, physical activity, alcohol intake, smoking, vitamin supplement use
Tsubota-Utsugi, 2011[36]	Japan	1998	4.1	36.8	35	745	FFQ, self-administered	F: >0.94 vs <0.36, V: >2.67 vs <1.35	Home measured	222	Age, gender, BMI, frequency of exercise, smoking status, alcohol consumption, energy-adjusted fat and sodium consumption, baseline SBP, a past history of diabetes, hypercholesterolaemia, cardiovascular disease
Wang, 2012[37]	USA, WHS cohort	1992	12.9	0.0	39	28 082	FFQ, self-administered	F: $\geqslant 3$ vs <0.5, V: $\geqslant 5$ vs <1.5, FV: $\geqslant 8$ vs <2	Self-reported	13 633	Age, race, total energy intake, randomized treatment assignment, smoking status, alcohol use, physical activity, postmenopausal status, postmenopausal hormone use, multivitamin supplement use, history of diabetes and hypercholesterolaemia, intake of whole grains, red meats, low-fat dairy products, nuts, BMI

Abbreviations: BMI, body mass index; CARDIA, coronary artery risk development in young adults; EPIC, European prospective investigation into cancer and nutrition; F, fruit; FFQ, food-frequency questionnaire; FV, total fruit and vegetables; HPFS, health professionals follow-up study; NHS, nurses' health study; SUN, Seguimiento University of Navarra; V, vegetables; WHS, women's health study.

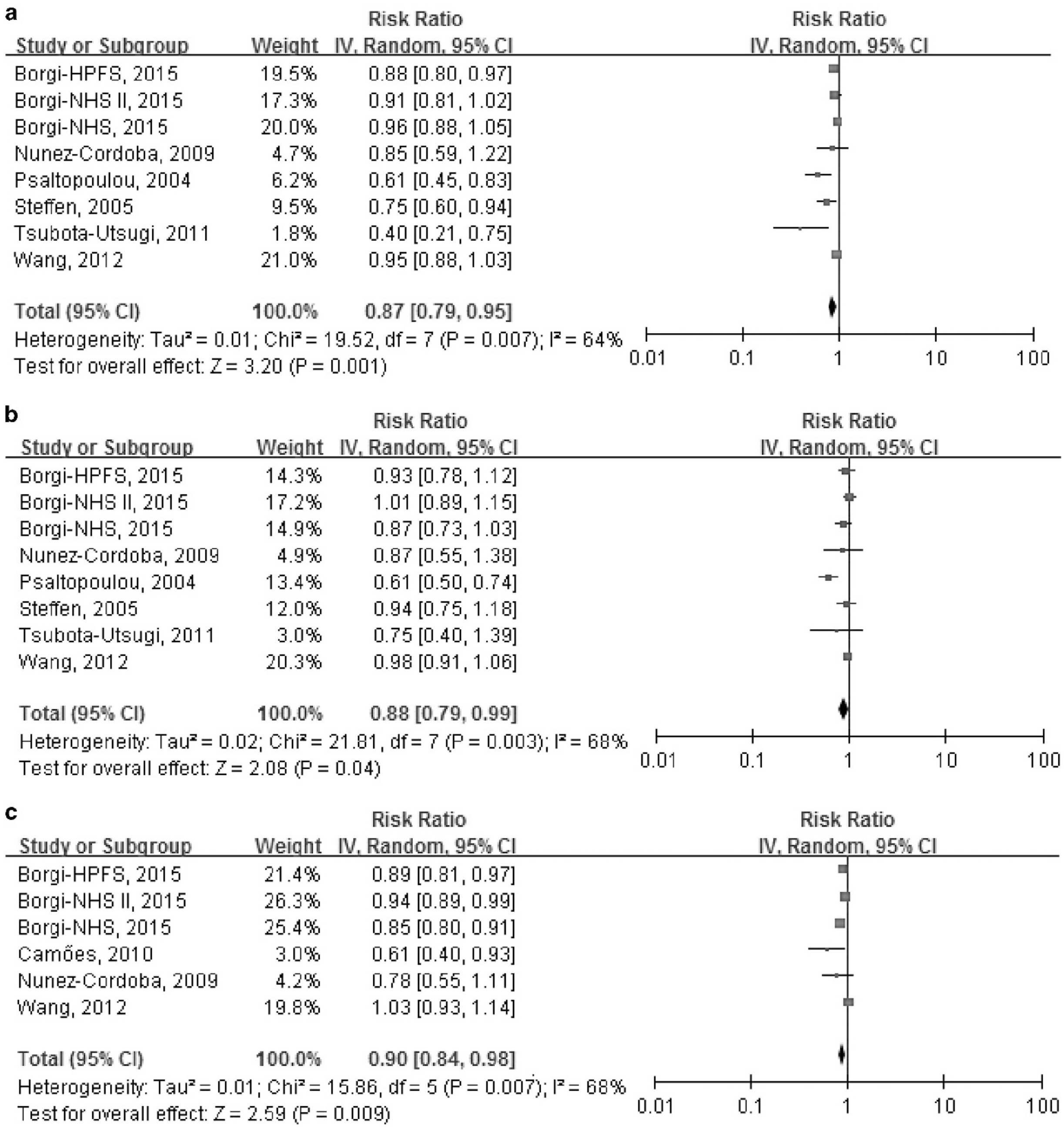

Figure 2. Meta-analysis of the association between (**a**) fruit, (**b**) vegetables and (**c**) total fruit and vegetables consumption (highest vs lowest) and the risk of incident hypertension.

lists of included articles. Finally, seven prospective cohort studies which comprised nine independent cohorts were selected for the present meta-analysis.[31–37] One study[31] consisted of three separate cohorts, that is, the NHS (Nurses' Health Study) cohort, the NHSII cohort, and the HPFS (Health Professionals Follow-up Study) cohort, and they were entered as three independent cohorts.

Study characteristics

Table 1 shows the main characteristics of the included studies. These studies were published between 2004 and 2015. Three of the included studies were performed in the United States,[31,35,37] three studies were performed in the European countries (Spain, Greece and Portugal),[32–34] and the other study was conducted in Japan.[36] The range of follow-up duration ranged from 3.8 to 28 years. Five articles included both male and female participants,[32–36] and one article included only female participants.[37] In an article by Borgi *et al.*,[31] participants in the NHS cohort and the NHSII cohort were women, and the HPFS cohort included only men. The sample size ranged from 549 to 63 885 for a total number of 185 676. The fruit and/or vegetables intake was assessed by food-frequency questionnaire (FFQ) in all articles. The incident of hypertension was diagnosed from self-reported[31,33,37] or measurement.[32,34–36] Participants who reported a diagnosis of hypertension at baseline were excluded from the analysis in all included studies. Six of the seven studies included studies reported fruit or vegetables intake separately,[31,33–37] and four of seven studies reported intake of total fruit and vegetables.[31–33,37]

Table 2. Pooled measure on fruit and/or vegetables consumption and risk of incident hypertension (highest vs lowest)

Outcome	*Fruit consumption*		*Vegetables consumption*		*Total fruit and vegetables consumption*	
	Comparisons, no.	*RR (95% CI)*	*Comparisons, no.*	*RR (95% CI)*	*Comparisons, no.*	*RR (95% CI)*
All included comparisons	8	0.87 (0.79, 0.95)	8	0.88 (0.79, 0.99)	6	0.90 (0.84, 0.98)
Study location						
Western (USA and Spain)	7	0.88 (0.81, 0.95)	7	0.89 (0.80, 0.99)	6	0.90 (0.84, 0.98)
Eastern (Japan)	1	0.40 (0.21, 0.75)	1	0.75 (0.40, 1.40)	—	—
P-value for meta-regression		0.084		0.629		—
Duration of follow-up (years)						
<10	3	0.63 (0.45, 0.89)	3	0.67 (0.55, 0.82)	2	0.71 (0.54, 0.92)
$\geqslant 10$	5	0.92 (0.87, 0.97)	5	0.97 (0.91, 1.02)	4	0.92 (0.86, 0.99)
P-value for meta-regression		0.037		0.015		0.149
Gender						
Female	3	0.95 (0.90, 1.00)	3	0.97 (0.91, 1.03)	3	0.93 (0.84, 1.03)
Male	1	0.88 (0.80, 0.96)	1	0.93 (0.78, 1.11)	1	0.89 (0.81, 0.97)
Both sexes	4	0.68 (0.54, 0.84)	4	0.77 (0.60, 0.99)	2	0.71 (0.54, 0.92)
P-value for meta-regression		0.017		0.104		0.181
Sample size						
$<10\,000$	3	0.70 (0.51, 0.96)	3	0.91 (0.75, 1.10)	2	0.71 (0.54, 0.92)
$\geqslant 10\,000$	5	0.89 (0.82, 0.97)	5	0.88 (0.77, 0.99)	4	0.92 (0.86, 0.99)
P-value for meta-regression		0.266		0.995		0.149
Assessment method of exposure						
Self-administrated	2	0.65 (0.28, 1.51)	2	0.98 (0.91, 1.05)	1	0.88 (0.82, 0.95)
Interviewer administrated	6	0.85 (0.77, 0.94)	6	0.87 (0.76, 0.99)	5	1.03 (0.93, 1.14)
P-value for difference		0.878		0.620		0.147
Outcome ascertainment						
Self-reported	5	0.93 (0.89, 0.97)	5	0.97 (0.91, 0.99)	5	0.92 (0.85, 0.98)
Measured	3	0.63 (0.49, 0.82)	3	0.76 (0.55, 1.04)	1	0.61 (0.40, 0.93)
P-value for meta-regression		0.009		0.086		0.151

Abbreviations: CI, confidential interval; RR, relative risk.

Quality assessment

All studies met the quality score of 7–8 stars (Supplementary Table 2). The main quality issues were listed as follows. Two articles measured the fruit and vegetables intake by self-administered.[36,37] The diagnosis of hypertension was based on self-reported.[31,33,37] The follow-up duration was <10 years.[32–34,36] Two articles did not adjust for potential covariate of BMI.[32,34]

Association between fruit or vegetable intake separately, and total fruit and vegetable intake and incident hypertension

Highest vs lowest

Six studies comprised eight cohorts reported an association between fruit or vegetables intake separately and incident risk of hypertension (Figures 2a and b). The highest intake of fruit or vegetables separately was inversely associated with the incident risk of hypertension compared to the lowest level, and the pooled RR and 95% CI was 0.87 (0.79, 0.95) for fruit consumption and 0.88 (0.79, 0.99) for vegetables consumption, both with an evidence of significant heterogeneity among included articles ($I^2=64\%$ and $I^2=68\%$, respectively).

Four studies comprised six cohorts reported an association between total fruit and vegetables intake and incident risk of hypertension. Figure 2c shows that the highest intake of total fruit and vegetables was inversely associated with the incident risk of hypertension compared with the lowest level, and the pooled RR and 95% CI was 0.90 (0.84, 0.98), with an evidence of heterogeneity ($I^2=68\%$, $P=0.007$).

Subgroup analysis and meta-regression

As shown in Table 2, subgroup analysis and meta-regression were performed to explore the potential sources of heterogeneity between fruit or vegetables intake separately, and total fruit and vegetables intake and incident hypertension. Analyses stratified by duration of follow-up (*P*-value for meta-regression = 0.037), gender (*P*-value for meta-regression = 0.017) and method of outcome ascertainment (*P*-value for meta-regression = 0.009) gave some clues to explain the heterogeneity between fruit intake and incident hypertension. Analysis stratified by duration of follow-up (*P*-value for meta-regression = 0.015) might explain the heterogeneity between vegetables intake and incident hypertension. No statistically significant source of heterogeneity was found for the association between total fruit and vegetables consumption and incident hypertension in the subgroup and meta-regression analysis ($P>0.05$ for each).

Publication bias and sensitivity analysis

As shown in Supplementary Figure 1, visual inspection of the funnel plot suggests an evidence of publication bias among the articles for fruit consumption (Egger's test, $P=0.035$; Begg's test, $P=0.009$), but not for vegetables consumption (Egger's test, $P=0.108$; Begg's test, $P=0.127$), and total fruit and vegetables consumption (Egger's test, $P=0.707$; Begg's test, $P=0.518$). However, the low power with <10 articles limited the interpretability of the finding.[22]

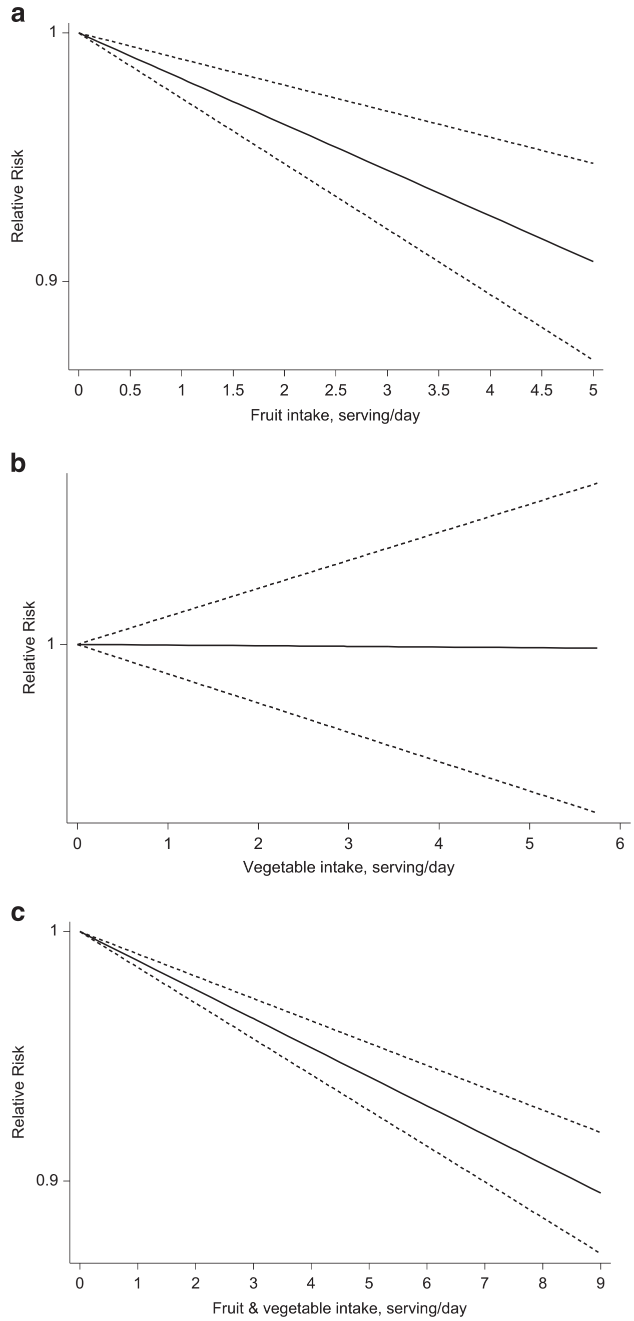

Figure 3. Dose–response association between (**a**) fruit, (**b**) vegetables and (**c**) total fruit and vegetables consumption and the risk of incident hypertension. Solid line, best-fitting restricted cubic spine; dotted line, 95% CI.

Further exclusion of any single article did not significantly alter the overall combined RR, which ranged from 0.84 (0.75, 0.93) to 0.90 (0.83, 0.97) for fruit consumption and 0.89 (0.80, 0.98) to 0.92 (0.85, 1.00) for total fruit and vegetables consumption. However, exclusion of an article by Psaltopoulou *et al.*[34] significantly altered the overall combined RR, which ranged from 0.86 (0.74, 0.99) to 0.96 (0.91, 1.02) for vegetables consumption.

Dose–response analysis

After excluding one study[32] reported less than three categories of fruit or vegetables consumption separately, and one study[34] did not report the detailed data of fruit or vegetables consumption separately, five studies with seven cohorts were included in the dose–response analyses of fruit or vegetables consumption separately and the risk of incident hypertension.[31,33,35–37] Four studies[31–33,37] with six cohorts were included in the dose–response analysis of total fruit and vegetables consumption and the risk of hypertension. The test for the non-linear association between fruit consumption (*P* for nonlinearity = 0.434), vegetables consumption (*P* for nonlinearity = 0.464), and total fruit and vegetables consumption (*P* for nonlinearity = 0.098) and the incident risk of hypertension were not significant. Under the linear hypothesis, a higher consumption of fruit consumption and total fruit and vegetables consumption were significantly associated with a decreased risk of incident hypertension, and the summary RR (95% CI) was 0.981 (0.973, 0.989) and 0.988 (0.985, 0.991), respectively. Vegetables consumption was not associated with incident hypertension, and the summary RR (95% CI) was 1.00 (0.989, 1.011) (Figure 3).

DISCUSSION

This meta-analysis identified nine cohorts involving a total of 185 676 participants. The pooled analysis showed that highest level of fruit or vegetables consumption separately and total fruit and vegetables consumption were inversely associated with the occurrence of hypertension after adjustment for potential confounding factors. We also found an inverse dose–response relation between the risk of developing hypertension and fruit intake, and total fruit and vegetables consumption. The incident risk of hypertension was decreased by 1.9% for each serving per day of fruit consumption, and decreased by 1.2% for each serving per day of total fruit and vegetables consumption.

The protective effect of fruit and/or vegetables consumption against hypertension is biologically plausible. Clinical and biological investigations have reported that the micro- and macro-constituents of fruit and vegetables, such as vitamin C, vitamin E, fibre and potassium are effective in lowering blood pressure.[38–40] In consistence with the results of our study, diets rich in plant foods have been demonstrated to lower blood pressure and to reduce the prevalence of hypertension in both normotensive and hypertensive patients.[41–47] No evidence from long-term randomized controlled trials has investigated the effect of fruit consumption and/or vegetables consumption on the prevention of hypertension occurrence, but the Dietary Approaches to Stop Hypertension (DASH) trial has shown that a diet high in fruit and vegetables, low in sodium, and low in saturated and total fat significantly reduced blood pressure.[19]

The pooled results of our study found that the highest level of vegetables intake was significantly associated with hypertension occurrence compared with the lowest level, but the dose–response analysis showed non-significant result. One possible explanation for the absence of the beneficial effect might be attributed to the added of fats and seasonings (sauce and salt), and the method of cooking (such as deep frying).[36,48] These unhealthy dietary styles might dilute the beneficial effect of vegetables consumption. In addition, the small number of included cohorts might also cause non-significant finding.

A substantial heterogeneity across studies was apparent in our study. Stratified analysis and meta-regression for fruit or vegetables intake separately revealed that the heterogeneity was associated with duration of follow-up, gender and method of outcome ascertainment. The non-significant source of heterogeneity for total fruit and vegetables consumption and incident hypertension may result from other unreported factors, such as different types of fruit and vegetables, various therapy methods of hypertensive patients and statistical model. More studies are warranted to investigate the potential difference in various subgroups.

To the best of our knowledge, this is the first dose–response meta-analysis of prospective cohort studies evaluated the association between fruit and/or vegetables intake and the occurrence of hypertension. We obtained an important finding of the significantly inverse association between fruit or vegetables intake separately, total fruit and vegetables intake and incident hypertension. In addition, we also detected a linear association of hypertension risk with fruit intake and total fruit and vegetables intake, which help to quantify the association and to examine the shape of the possible association. Epidemiological studies have consistently reported that hypertension is one of the important risk factors for various negative health outcomes. Minor changes in blood pressure can have a substantial impact on cardiovascular events.[49] Previous meta-analyses have demonstrated the beneficial effects of fruit and/or vegetables on diabetes, cancer, and cardiovascular outcomes[12,13,50–52], and these findings were in accordance with our results. Wu *et al.*[12] reported that vegetables and fruit intake linked with a lower risk of type 2 diabetes mellitus. Gan *et al.*[13] demonstrated that fruit or vegetables consumption separately, and total fruit and vegetables consumption were significantly associated with the lower incident of coronary heart disease. Zhan *et al.*[50] provided strong support for the recommendation to consume a higher amount of fruit and vegetables to reduce the risk of cardiovascular disease.

Admittedly, the present meta-analysis has several limitations. First, the finding of a possible publication bias suggested that the pooled RR was probably overestimated. One possible explanation is that several smaller studies showing non-significant association may be underreported in the literature. In addition, the low power with only seven articles limits the interpretability of the finding. Second, most of the included articles were conducted in the western developed countries, and thus our findings seemed difficult to generalise to a broader range of population. Third, the majority of the included studies used the food-frequency questionnaire (FFQ) to assess levels of dietary consumption. Although previous studies have showed that FFQ was a reasonable tool to assess the fruit and vegetables intake, measurement bias might also exist in the present meta-analysis.[53] Fourth, the dietary factor is dynamic, changes in fruit and/or vegetables intake may have taken place during the long follow-up periods. Lastly, all of the included studies adjusted for multiple potential confounding variables; however, the possibility of other unmeasured covariates might have affected the results of the present analysis. For example, participants who consumed more fruit and vegetables tend to follow other healthier lifestyles, and thus it is difficult to identify the independent effects of fruit consumption or vegetables consumption separately from other lifestyle and dietary factors.

CONCLUSIONS

In conclusion, the results from the present meta-analysis of seven studies comprised nine independent cohorts showed an inverse association between fruit or vegetables consumption separately, and total fruit and vegetable consumption and the incident risk of hypertension. Although the association was statistically significant, the present study is limited by its possible publication bias and its small number of included articles. Our results support the recommendation to increase the consumption of fruit and vegetables with respect to preventing the risk of developing hypertension. However, further large prospective studies and long-term high-quality randomized controlled trials are still needed to confirm the observed association.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

LW and YH were responsible for the conception and design. LW, DS and YH took part in the acquisition, analysis and interpretation of data. All authors have read and approved the final manuscript.

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