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Fruit and vegetable consumption and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of prospective studies

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Preface

This last year has been exciting, interesting and educational. The process of completing this master's thesis was intense and comprehensive, however we have learned a lot and would not be without this experience. We experienced it as a strength to conduct this master's thesis in pair as we could support each other, and discuss and solve possible challenges together.

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Abstract

Background: The high prevalence of type 2 diabetes has a considerable impact on global health. Several modifiable risk factors for type 2 diabetes have been established, such as dietary factors. The association between intake of fruit and vegetables and their subtypes, and the risk of type 2 diabetes has been investigated in several studies, but the results have not been consistent.

Objective: The aim of this master's thesis is to conduct a systematic review and dose-response meta-analysis of prospective studies on the association between intake of fruit and vegetables and risk of type 2 diabetes, with particular focus on identifying specific types of fruits and vegetables that may be beneficial, and to clarify the strength and shape of the dose-response relationship.

Design: PubMed and Embase databases were searched up to 26th of June 2018. Prospective cohort studies of fruit and vegetable consumption and type 2 diabetes mellitus were included. Summary relative risks and 95% confidence intervals were estimated using a random effects model.

Results: Our results indicated an inverse association between intake of fruits, and fruit and vegetables combined and the risk of type 2 diabetes. No significant associations were found for intake of vegetables. Of subtypes of fruit and vegetables, especially apples, blueberries, grapefruit and grapes and raisins were strongly associated with a reduced risk, while cabbage, cauliflower, kale, mustard and chard greens and potatoes were strongly associated with an increased risk of type 2 diabetes.

Conclusions: This meta-analysis suggests that there is a weak inverse association between fruit and vegetable intake and type 2 diabetes risk. There is some indication of both inverse and positive associations between intake of several fruit and vegetables subtypes and type 2 diabetes risk, however, because of the limited number of studies, further studies are needed before firm conclusions can be made.

Introductory chapter

This master's thesis consists of an introductory chapter and an article. In the introductory chapter we will provide a detailed description of the background of the study and theoretical aspects, before presenting our research questions. Further, we will introduce the methods and the statistical analyses that were used, elaborate on methodological considerations and choices, and discuss some advantages and limitations with our systematic review and meta-analysis. In addition, we will briefly discuss nutrient content in fruit and vegetables. Finally, we will provide an overall conclusion of this master's thesis. In the article we will present the background and aim of our systematic review and meta-analysis, give a short description of the statistical analyses that were used, and present our results followed by a discussion and conclusion.

1.0 Introduction

In this chapter we will introduce the epidemiology, disease classification, risk factors, complications and the global burden of type 2 diabetes. In addition, we will present the nutrient contents and recommendations of fruit and vegetables, dietary assessment methods, and summarize findings from previous studies on the association between fruit and vegetable intake and the risk of type 2 diabetes. Lastly, the research questions will be presented.

1.1 Diabetes

1.1.1 Epidemiology and disease classification of diabetes

The number of people living with type 2 diabetes has increased rapidly over the past two decades from 108 million in 1980 to 422 million in 2014 worldwide (World Health Organization (WHO), 2016). If current trends continue, the prevalence is estimated to pass 700 million by 2025 (NCD Risk Factor Collaboration, 2016). The prevalence is rising faster in low- and middle-income countries compared to high-income (WHO, 2016).

Diabetes mellitus, commonly referred to as diabetes, is a metabolic disease where the pancreas does not produce enough insulin, or where the insulin produced is not used effectively (WHO, 2016). Insulin, which is a peptide hormone produced by beta cells in the islets of Langerhans in the pancreas (Voet & Voet, 2011), is important in regulating the circulating blood glucose concentrations. Diabetes is characterized by elevated levels of blood glucose, known as hyperglycemia (Boland, Rhodes, & Grimsby, 2017). The World Health Organization (WHO) has developed recommendations on diagnostic values for blood glucose concentrations. Diabetes may be diagnosed based on glycated hemoglobin (HbA1c) $\geq 6.5\%$, or fasting plasma glucose ≥ 7.0 mmol/L or plasma glucose ≥ 11.1 mmol/L two hours after a 75 grams oral glucose tolerance test (WHO, 2016).

There are two principal forms of diabetes, type 1 diabetes (T1D) and type 2 diabetes (T2D). Type 1 diabetes, formerly known as insulin-dependent, occurs when the pancreas does not produce enough insulin. Almost all cases of type 1 diabetes occur among children and adolescents. Type 2 diabetes, formerly known as non-insulin-dependent, occurs when the body fails to respond properly to the insulin produced (WHO, 2016). Type 2 diabetes accounts for approximately 90% of diabetes cases worldwide (WHO, 2019). Impaired glucose tolerance (IGT) and impaired fasting glycaemia (IFG) represents intermediate states of

abnormal glucose regulations in the transition between normal blood glucose levels and diabetes. Subjects with IGT and/or IFG are at high risk of developing type 2 diabetes, with relative risks (RRs) of 6.35 (95% CI: 4.87-7.82) in people with IGT, 4.66 (95% CI: 2.47-6.85) in people with IFG, and 12.13 (95% CI: 4.27-20.00) in people with both IFG and IGT (Gerstein et al., 2007). Gestational diabetes (GDM) represents a temporary condition that occurs during pregnancy, with blood glucose values above normal, but below the threshold for the diagnosis of diabetes (WHO, 2016). Women with gestational diabetes are at increased risk of developing type 2 diabetes in the future, with reported risks between 9.5% and 37% (Hopmans et al., 2015).

1.1.2 Risk factors for diabetes

Genetic and environmental influences play a key role in the development of both types of diabetes (Wu, Ding, Tanaka, & Zhang, 2014), although less is known about the causes of type 1 diabetes. Several modifiable risk factors for type 2 diabetes have been established, including overweight and obesity, physical inactivity, unhealthy diet and smoking. Non-modifiable risk factors include ethnicity, family history of diabetes and older age (WHO, 2016). Overweight and obesity are the strongest risk factors for type 2 diabetes with reported relative risks between 10-40% for severe obesity compared to lean individuals (Carlsson, Ahlbom, Lichtenstein, & Andersson, 2013; Field et al., 2001; Njolstad, Arnesen, & Lund-Larsen, 1998; Reeves, Balkwill, Cairns, Green, & Beral, 2014).

Dietary factors are important modifiable risk factors for type 2 diabetes and several previous studies have found increased risk of type 2 diabetes with a high intake of red and processed meat, sugar- sweetened beverages, and low intake of whole grains, fiber, dairy products, fruits and vegetables (Aune, Norat, Romundstad, & Vatten, 2013a, 2013b; Bazzano, Li, Joshipura, & Hu, 2008; Cooper et al., 2012; Du et al., 2017; Imamura et al., 2016; InterAct Consortium, 2015; Montonen et al., 2005; Pan et al., 2011; Villegas et al., 2008). Recent studies have questioned the role of dairy products in reducing diabetes risk (M. Chen et al., 2014; Vissers et al., 2019). A diet rich in fruit and vegetables may indirectly influence the risk of type 2 diabetes by preventing overweight and obesity, which are the main risk factors for developing the disease (Lukas. Schwingshackl et al., 2015), but may also have a benefit independently of adiposity (Cooper et al., 2012).

1.1.3 Complications of diabetes

All types of diabetes can lead to complications in many organ systems such as blindness, neuropathies, nephropathies, cardiovascular disease, cancer and increase the risk of premature mortality (Campbell, Newton, Patel, Jacobs, & Gapstur, 2012; Rao Kondapally Seshasai et al., 2011). Poorly controlled diabetes in pregnancy increases the risk of fetal death as well as other complications (WHO, 2016).

1.1.4 Global burden of diabetes

In 2017, it was estimated that 4 million deaths were directly attributable to diabetes (International Diabetes Federation, 2017). Diabetes imposes a great economic burden on the global health system and national economies through direct medical costs, and indirect costs associated with productivity loss and premature mortality. People with diabetes and their families suffer economic losses due to the disease and its complications. Diabetes is one of the non-communicable diseases (NCDs) prioritized by world leaders (WHO, 2016). In 2013, WHO developed the Global Action Plan for the Prevention and Control of Non-communicable Diseases with nine voluntary targets to reach by 2025. Several of these targets reflect diabetes and its key risk factors (World Health Organization (WHO), 2013). In 2015, these commitments were further deepened by the United Nations General Assembly's adoption of the 2030 Agenda for Sustainable Development (United Nations, 2015).

Public health policy has the potential to reduce the occurrence of type 2 diabetes. A combination of policies, legislation, supportive environments and raising awareness of health risks can be effective approaches to promote healthier diet and physical activity (WHO, 2016).

1.2 Fruit and vegetables

1.2.1 Nutrient content of fruit and vegetables

Fruit and vegetables are important sources of nutrients, dietary fibers, antioxidants, vitamins, minerals and phytochemicals (Slavin & Lloyd, 2012). These components have the potential to influence biological functions in the human body through different mechanisms. Antioxidants may prevent or reduce damage caused by oxidative stress, while phytochemicals such as polyphenols, carotenoids, anthocyanins, quercetin and glucosinolates may reduce insulin resistance and increase insulin sensitivity by influencing signalling pathways. Both

antioxidants and phytochemicals have anti-inflammatory properties (Pisoschi & Pop, 2015; Vinayagam, Xiao, & Xu, 2017). It is likely that the synergistic effects of different phytochemicals, antioxidants and other components are responsible for the health effects associated with fruit and vegetable intake (NNR, 2014). Adequate fruit and vegetable intake should be a part of a healthy diet as it may lower the risk of several chronic diseases, such as cardiovascular disease, cancer and type 2 diabetes, as well as all-cause mortality (Aune et al., 2017).

1.2.2 Recommendations for fruit and vegetable intake

Most countries have national recommendations for the daily amount of fruit and vegetables needed to maintain optimal health, but these recommendations vary globally (Nasjonalt råd for ernæring, 2011). The recommendations are often based on both national and international systematic reviews, and reports by international expert groups such as WHO, European Food Safety Authority (EFSA) and World Cancer Research Fund (WCRF). Most countries recommend three or more servings per day of vegetables and two or more servings per day of fruits; one serving ranging from 80 to 150 gram (**Table 1**) (Nasjonalt råd for ernæring, 2011). Fruit juice contributes with important nutrients, but contains high amounts of naturally occurring sugar, and little or no fiber, which causes them to have moderately high glycemic index (GI). Most countries therefore recommend fruit juice to be consumed in moderation (Brandon J Auerbach, Kratz, Dibey, Krieger, & Vallila-Buchman, 2018). Potatoes are not counted as part of the five recommended servings of fruit and vegetables per day, because of their large amounts of rapidly absorbed starch and high GI and high glycemic load (GL) (Halton et al., 2006; World Cancer Research Fund/American Institute for Cancer Research, 2018).

In most high-income countries where data are available, in particular daily consumption of vegetables is falling short of national targets, while fruit consumption is mostly closer to these targets (World Cancer Research Fund/American Institute for Cancer Research, 2018). A recent meta-analysis on fruit and vegetable intake and cardiovascular disease, cancer and mortality, suggested that intakes beyond the five recommended servings per day may provide additional health benefits (Aune et al., 2017), but whether very high intakes can reduce the risk of type 2 diabetes further is not clear.

Table 1. Examples of official recommendations for fruit, berries and vegetables

Recommendations	Norway	Sweden	Denmark	England	USA	WHO	World Cancer Research Fund
Total intake (gram/day)	≥ 650-750	≥500	≥600	≥400	640-800	≥400	≥400
Fruit (gram/day)	300	300			360		
Vegetable (gram/day)	300-450	200			450		

Source: Nasjonalt råd for ernæring (2011)

1.2.3 Dietary assessment methods

Dietary assessment of individuals' fruit and vegetable intake is often assessed using subjective methods like 24-hour dietary recall (24HR), dietary record (DR), dietary history, and food frequency questionnaire (FFQ). The data collection is either administered by an interviewer or self-administered (Shim, Oh, & Kim, 2014). Both retrospective and prospective dietary assessment methods are prone to sources of error. Retrospective methods like 24HR and FFQ are prone to recall-bias, as it depends on the memory of the participants, while prospective methods such as DR measures the current diet and thereby avoid recall-bias. Misreporting, where individuals may underestimate or overestimate their food intake, might be a source of error in both prospective and retrospective methods (Gibney, 2004).

In large epidemiological studies, FFQs are commonly used and provides information on how often an individual consumes certain foods. This method can provide a relatively good estimate of the intake of the most common foods over time and thus estimate an average intake. Other benefits of FFQ are that the method is relatively inexpensive, simple, and little time consuming for the participants to conduct. Disadvantages of using FFQ are that it does not necessarily cover the entire diet, the measures for portion size can be imprecise, and the questionnaire must be adapted depending on which group you want to study. Further, it might be difficult to capture a changing or varying diet, unless repeated dietary assessments are made. There are several methods that are used to validate FFQs which includes multiple 24-hour recalls, food records and biomarkers (FAO, 2018; Shim et al., 2014).

1.3 Fruit and vegetables and the risk of type 2 diabetes

1.3.1 Findings from prospective observational studies

A high intake of fruit and vegetables has been associated with a reduced risk of type 2 diabetes in several (Bazzano et al., 2008; Cooper et al., 2012; Du et al., 2017; Montonen et al., 2005; Villegas et al., 2008), but not all previous prospective observational studies (Alperet, Butler, Koh, Yuan, & van Dam, 2017; Auerbach et al., 2017; Chen, Koh, Yuan, Qin, & van Dam, 2018; Hodge, English, O'Dea, & Giles, 2004; Kurotani et al., 2013; Liu et al., 2004; Meyer et al., 2000). Studies on the association between fruit juice and type 2 diabetes have shown no association for 100% fruit juice (Auerbach et al., 2017; Eshak et al., 2012; Fagherazzi et al., 2013), while increased risk has been observed for sweetened berry juice (Montonen et al., 2007) and fruit drinks (Palmer et al., 2008). Mixed results have been observed between a high potato intake and the risk of type 2 diabetes, where some studies showed positive associations (Halton et al., 2006; Montonen et al., 2005), while others showed no association (Chen et al., 2018; Hodge et al., 2004) or inverse associations (Villegas et al., 2007).

For subtypes of fruit and vegetables, inverse associations have been observed between the intake of apples/pears (Alperet et al., 2017; Knekt et al., 2002; Song, Manson, Buring, Sesso, & Liu, 2005; Wedick et al., 2012), berries (Bazzano et al., 2008; Cooper et al., 2012; Knekt et al., 2002; Montonen et al., 2005; Wedick et al., 2012), green leafy vegetables (G. C. Chen et al., 2018; Montonen et al., 2005; Villegas et al., 2008), yellow vegetables (Liu et al., 2004; Villegas et al., 2008), root vegetables (Cooper et al., 2012) and tomatoes (Villegas et al., 2008) and the risk of type 2 diabetes, but the available data have not been entirely consistent.

1.3.2 Findings from meta-analyses

A few previous meta-analyses have studied the association between fruit and vegetables and risk of type 2 diabetes. Cooper et al. (2012) found that total fruit and vegetable intake was associated with an 7% reduction in the relative risk of type 2 diabetes, but when examining fruit and vegetables separately, there was no significant reduction in risk for developing type 2 diabetes with summary RRs of 0.92 (95% CI: 0.81-1.02) for fruits and 0.89 (95% CI: 0.75-1.03) for vegetables. Of specific types of vegetables, root vegetables and green leafy vegetables were associated with a significant reduction in the risk of type 2 diabetes (Cooper

et al., 2012). Another meta-analysis from 2014 by Li et al. showed that a higher intake of fruit or vegetables, especially green leafy vegetables, was associated with a significantly reduced risk of type 2 diabetes. They also conducted dose-response analyses, which indicated a 6% lower risk of developing type 2 diabetes per 1 serving/day increment of fruit intake and a 13% lower risk of type 2 diabetes per 0.2 serving/day increment of green leafy vegetables intake (Li, Fan, Zhang, Hou, & Tang, 2014).

A meta-analysis by Imamura et al. found a RR of 1.07 (95% CI: 1.01-1.14) per 1 serving/day of fruit juice (Imamura et al., 2016). Another meta-analysis by Xi et al. investigated the association between 100% fruit juice intake and risk of type 2 diabetes and found no significant association with a RR of 1.03 (95% CI: 0.91-1.18), but found a significant association between sugar sweetened fruit juice intake and the risk of type 2 diabetes with a RR of 1.28 (95% CI: 1.04-1.59) (Xi et al., 2014).

The most recent meta-analysis on fruit and vegetable intake and risk of type 2 diabetes, published in 2017 by Schwingshackl et al. found a borderline inverse association between intake of fruits and vegetables and risk of type 2 diabetes with RRs of 0.96 (95% CI: 0.93-1.00) and 0.98 (95% CI: 0.96-1.00), respectively. There was evidence of a nonlinear dose-response association for both fruit and vegetables, with a decreased risk of T2D by 10% with increasing intakes of fruits up to 200-300 g/day, and a 9% decreased risk with increasing intakes of vegetables up to 300 g/day (Schwingshackl et al., 2017). This meta-analysis did not conduct analyses on subtypes of fruit or vegetables.

Since the most recent meta-analysis, ten prospective studies exploring the association between fruit and vegetable intake and risk of type 2 diabetes have been published (Alperet et al., 2017; Auerbach et al., 2017; Bahadoran, Mirmiran, Momenan, & Azizi, 2017; Chen et al., 2018; Du et al., 2017; Farhadnejad, Teymoori, Asghari, Mirmiran, & Azizi, 2018; Huang et al., 2017; Lv et al., 2017; Ma et al., 2018; Muraki et al., 2013). Previous meta-analyses have only analysed a few specific fruit and vegetable subtypes such as root vegetables, green leafy vegetables or cruciferous vegetables (Chen et al., 2018; Cooper et al., 2012). This meta-analysis could further contribute to the existing evidence and allow further investigation of any association between fruit and vegetable consumption, including subtypes, and the risk of type 2 diabetes.

1.4 Research questions

The aim of this master's thesis is to conduct a systematic review and dose-response meta-analysis of prospective studies on the association between intake of fruit and vegetables and risk of type 2 diabetes, with particular focus on identifying specific types of fruits and vegetables that may be beneficial, and to clarify the strength and shape of the dose-response relationship. The aim of this master's thesis is reflected in the more detailed research questions and will be answered in the article:

- *Is a high intake of fruit and/or vegetables associated with risk of type 2 diabetes?*
 - *How strong is the association between fruit and/or vegetable intake and risk of type 2 diabetes, and what is the shape of the dose-response relationship?*
 - *Are specific types of fruits and vegetables more strongly associated with type 2 diabetes risk than others?*

The article was written using American Journal of Clinical Nutrition's guidelines, link:
https://academic.oup.com/DocumentLibrary/ACN/Information_for_Authors.pdf

2.0 Method

This master's thesis takes a quantitative approach and consists of a systematic review and dose-response meta-analysis of prospective studies. In this chapter we will elaborate on the two methods. Further, our search strategy, selection of studies, data extraction, validity assessment of the included studies and statistical methods will be presented.

2.1 Systematic review and meta-analysis

Amongst all research designs, systematic reviews and meta-analyses provide the highest level of evidence in terms of assessing associations between risk factors and different disease outcomes. Systematic reviews are used to summarize existing literature in a systematic way by including a detailed and comprehensive plan and search strategy made in advance. A systematic review often, but not always, includes a meta-analysis (Egger, Smith, & Schneider, 2001; Guyatt, 2015). A meta-analysis is a statistical method for quantitatively combining the results of multiple studies that measure the same exposure and outcome into a single pooled estimate (Borenstein, Hedges, Higgins, & Rothstein, 2009; Guyatt, 2015). Dose-response analyses can be included in a meta-analysis to quantify the strength and shape of the association between an exposure and an outcome (Egger, Smith, & Schneider, 2001). If an increasing level of an exposure is associated with either an increased or decreased risk of the outcome, there is a dose-response relationship between the exposure and the outcome (Nicola Orsini, Bellocco, & Greenland, 2006). The associations may not always be linear, and J-shaped, U-shaped and other nonlinear associations may also occur (Salkind, 2010). Meta-analysis is often criticized for “mixing apples and oranges”. This metaphor describes the problem of pooling results from heterogenic studies as this may lead to invalid results (Esteves, Majzoub, & Agarwal, 2017). The meta-analytic approach can be used to investigate discrepancies and heterogeneity between studies through subgroup and meta-regression analyses, and makes it possible to explore how the study result varies among subgroups such as men and women, different geographical locations, confounding factors and so on (Egger, Smith, & O'Rourke, 2001).

The goal of systematic reviews and meta-analyses is to limit bias by the use of a reproducible scientific process to search the literature, evaluate the quality of the individual studies and provide an overall summery estimate of the association between an exposure and an outcome by the use of statistical analyses (Crowther, Lim, & Crowther, 2010). The quality of a systematic review and meta-analysis relies on the quality of the included studies, and can

be influenced by unsatisfying methodological quality of the primary data (Gopalakrishnan & Ganeshkumar, 2013). Both systematic reviews and meta-analyses can potentially identify important knowledge gaps where further research is needed, and are important in guiding public policies and recommendations with regard to both prevention and treatment of various diseases (Egger, Smith, & O'Rourke, 2001).

Efforts have been made to standardize the reporting of meta-analyses, such as the MOOSE (Meta-analyses of Observational Studies in Epidemiology) criteria and the PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses) statement. These guidelines include items on title, background, search strategy, methods, results, discussion, conclusion and funding. The aim of the guidelines is to improve the usefulness of meta-analyses for authors, reviewers, editors, readers, and decision makers (Moher, Liberati, Tetzlaff, & Altman, 2009; Stroup et al., 2000).

2.1.1 Biases in research

Bias may be defined as “any trend in the collection, analysis, interpretation, publication or review of data that can lead to conclusions that are systematically different from the truth” (Porta, Greenland, Burón, & International Epidemiological, 2014). In epidemiologic research, bias is hard to eliminate, as it unlike chance and confounding, cannot be quantified or controlled for after the data is collected. Bias can influence the study validity and reliability, and may lead to erroneous conclusions (Henderson & Page, 2007). In brief, validity concerns whether a measuring instrument measures what it is meant to measure, and reliability concerns how consistent the measurement are (Porta et al., 2014).

There are several different types of biases. Traditional narrative reviews, which often focus on a subset of studies based on availability or author selection, are prone to selection bias, and provides an unsystematic assessment of the evidence (Uman, 2011). Reporting bias includes several different types of biases, such as language bias, publication bias and duplicate publication bias. All these types of biases affect which studies are disseminated and which are not. Language bias occurs if the included articles are based solely on articles published in one language, often English. Publication bias will be introduced under “2.6.8 *Publication bias*”, in the statistical methods section of this introductory chapter. Duplicate publication bias is present if results from the same study are included more than once and can lead to overestimation of the effects (Egger, Dickersin, & Smith, 2001; Institute of Medicine, 1990; Sterne, Egger, & Moher, 2011). Regression dilution bias occurs when random

measurement errors biases the association between an exposure and an outcome. This may attenuate the regression slope describing the association towards the null (Hutcheon, Chiolero, & Hanley, 2010).

2.2 Search strategy

Our main supervisor for this master's thesis, Dagfinn Aune, searched the PubMed and Embase databases up to 26th of June 2018 for eligible prospective studies of fruit and vegetable intake and type 2 diabetes risk. A more detailed description of the search terms used in the PubMed database is available in the article and in **Supplementary Table 1**. Similar search terms were used in the Embase database.

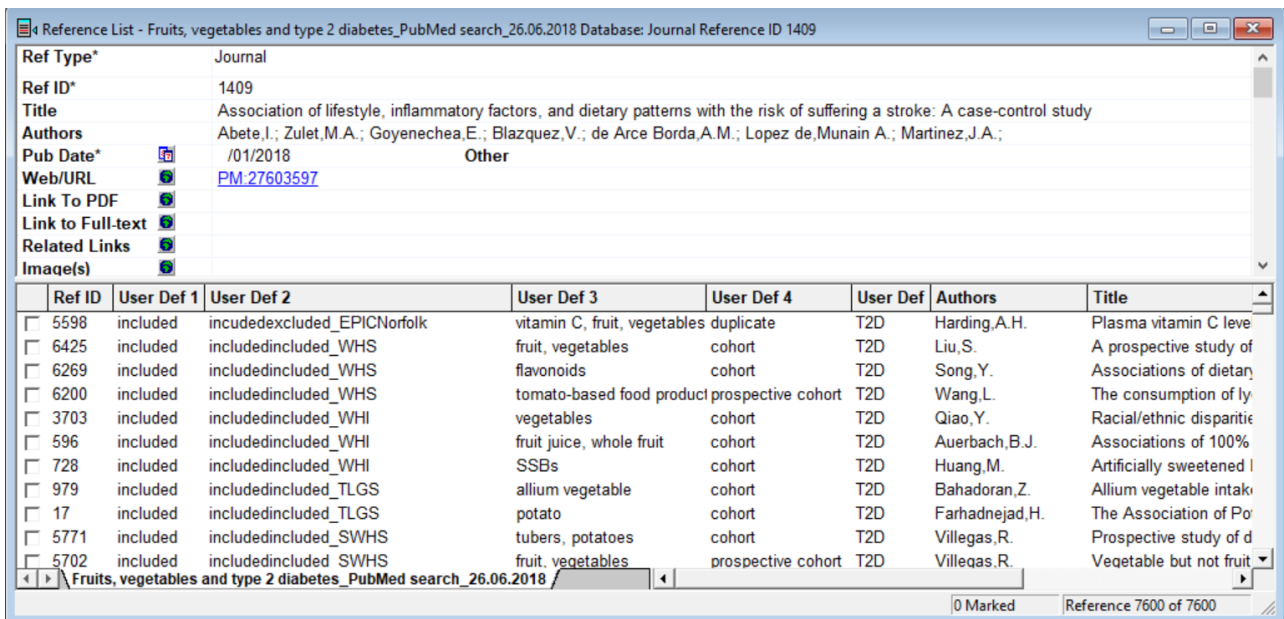
2.3 Study selection

The references from the literature search were downloaded to the program Reference Manager version 11 which were used to screen the relevant studies. Reference Manager is an online search tool and reference database, which specializes in storing, managing, and searching for bibliographic references in a personal reference database (Thomson ISI ReaseachSoft, 2004). Both candidates screened the references in both databases.

In Reference Manager, all studies are listed in a table which consists of eight columns; ID reference number, user definitions (User Def) 1-5, authors and title (**Picture 1**). User Def 1 was preset to "excludedabti", which means that the studies are excluded on the basis of title or abstract. In the first study selection step, all studies from the literature search were inspected for relevance by title and abstract. Studies without relevant information in title or abstract remained unchanged ("excludedabti"). Studies with potentially relevant data on the exposure (fruit and vegetables), and outcome (type 2 diabetes) were included for further investigation, and User Def 1 was modified to "included". To make sure no relevant studies were missed, studies reporting on terms such as risk factors, diet, food groups, lifestyle, dietary patterns, metabolic syndrome, diabetes, pre-diabetes and hyperglycemia etc. were included in the first step of the screening. All study designs with relevant data were also included in the first step to get an overview of the available data.

In the second study selection step we retrieved a pdf of the included studies from the first inspection. If no relevant data could be obtained, User Def 2 was set to "excluded", with reason for exclusion in User Def 4. Examples of exclusion were case-control- or cross-sectional study design, not relevant exposure, not relevant outcome, not relevant data (neither

the exposure or outcome were relevant), no reported risk estimates, meta-analyses, pooled analyses and reviews. Exposure was filled out in User Def 3. When there was relevant data that could be included in the analysis, User Def 2 was modified to “included”, and study design (cohort) was filled out in User Def 4. If duplicate reports from the same study cohort were identified, the study with most cases was included, changing User Def 2 to “includedincluded”, and “includedexcluded” for the duplicate reports. An abbreviation of the study name for the included studies was also added, for example: “includedincluded_NHS” for The Nurses’ Health Study.



Picture 1. Study selection in Reference Manager

2.3.1 Inclusion criteria

To be included the studies had to satisfy several criteria. The studies had to have a cohort, a case-cohort, or a nested case-control (within a cohort) design. Cohort studies have a prospective observational design, where a group of healthy participants (a cohort) are followed for a certain time to see who develop the outcome of interest, and how they differ from those who do not develop the outcome. This ensures that data on the exposure are collected before the outcome occur (Rothman, Greenland, Poole, & Lash, 2008). In a case-cohort study, a single sub-cohort from an initial cohort is selected randomly or by the use of stratified random sampling at the start of the study, and later all the other cases from the cohort outside the sub-cohort are added (Cologne et al., 2012). A nested case-control study is

based on a large cohort where all the identified cases are selected, and then matched with controls that are randomly selected from those in the cohort who have not developed the disease at that time (Ernster, 1994; Langholz & Richardson, 2009). Other inclusion criteria are described in more detail in the article under “2.3 Study Selection”.

2.3.2 Exclusion criteria

A list of excluded studies and exclusions reasons is provided in **Supplementary Table 2**.

2.4 Data extraction

After the study selection process, relevant data were extracted from each study. The extracted data can be found in **Supplementary Table 3**, with an example shown in **Picture 2**. More details about the data extraction are provided in the article.

Supplementary Table 3. Cohort studies of fruit and vegetables and type 2 diabetes

Author, publication year, country	Study name or description	Follow-up period	Study size, gender, age, number of cases	Dietary assessment	Outcome assessment	Exposure	Quantity	RR (95% CI)	Adjustment for confounders
Ford ES et al, 2000, USA	NHANES I Epidemiologic Follow-Up Study	1971-1975 to 1992-1993, 15.8 years follow-up	9665 participants, age 25-74 years, 1018 cases	Single 24-hour dietary recall	Self-report, hospitalization record, death certificate	Fruit and vegetable (total)	0 serv/d	1.00	Age, sex, smoking, systolic blood pressure, cholesterol concentration, use of antihypertensive medication, recreational exercise, nonrecreational activity, alcohol use, BMI, education
							1-4	1.01 (0.78, 1.29)	
							≥5	0.79 (0.59, 1.06)	
						Fruit and vegetable (men)	0 serv/d	1.00	
							1-4	1.23 (0.76, 1.99)	
							≥5	1.14 (0.67, 1.93)	
						Fruit and vegetable (women)	0 serv/d	1.00	
							1-4	0.85 (0.62, 1.16)	
							≥5	0.61 (0.42, 0.88)	

Picture 2. Example of data extracted from the included prospective cohort studies of fruit and vegetables and type 2 diabetes risk

Data from each exposure, in total 31 exposures, was saved in separate excel files “xlsx”, and had to be converted to “csv (comma-delimited)” files to be suited for analyses. Every exposure had their own customized “do-file” which contained the commands necessary for all the different analyses.

Exposures of fruit juices/drinks were divided into two. Fruit juice included studies that specified that the juice contained 100% fruit juice, without added sugar and studies that reported on juice, without specifying the content. Fruit drinks included studies with exposures

that contained added sugar such as sweetened berry juice, Tang, Kool-Aid, Hi-C, sweetened fruit drinks, and juices and nectars in combination where the distinction between the two could not be established.

2.4.1 Serving sizes

The desired unit was gram per day, and for the studies that reported intake by frequency, for example servings per day/week, the serving sizes was used to recalculate the intake in grams per day (g/day). In studies where serving size was specified, this was used. Otherwise, we used a serving size of 80 gram for fruits, vegetables and fruit and vegetables combined, as this has been used in previous meta-analyses (Aune et al., 2017; Cooper et al., 2012; L. Schwingshackl et al., 2017). In accordance with one meta-analysis, a serving size of 250 mL was used for fruit juice and fruit drinks (Imamura et al., 2016). The serving size of potatoes and other subtypes, were taken from an article by Lee et al., which based their estimates on Bowes & Church's Food Values of Portions Commonly Used (Lee et al., 2009). When serving sizes were not mentioned estimates were calculated for "groups" like cruciferous or green leafy vegetables by adding the serving sizes of the individual vegetables in each group and dividing it by the total number of vegetables contributing to that group. The serving sizes used in this meta-analysis are listed in **Supplementary Table 4**.

2.4.2 Converting increment units

Many studies presented the quantity for the different exposures as both categorical and continuous data. The continuous data were often represented as an increment of three servings per week. We used the continuous variable for the analyses, however, in a few cases where either the risk estimates or confidence intervals were deemed unreliable (e.g. continuous risk estimates were inconsistent with the categorical data provided or the confidence intervals for the continuous risk estimates were not symmetrical), we made an exception and used the categorical results. Risk estimates on a continuous scale were recalculated to the increments used in the meta-analysis by taking the natural logarithm of the RR (95% CI) on a continuous scale, then dividing by the increment reported in the original paper and finally multiplied by the increment used for the analysis and back-transformed to non-logarithmic scale by taking the exponential of the betas (regression coefficients) and CIs of the betas. This was done using an excel file which made these calculations directly.

2.5 Validity assessment

The Newcastle–Ottawa quality assessment Scale (NOS) was used to assess the quality of the included observational studies (Wells et al., 2013). We independently assessed all studies and discrepancies were resolved by discussion. For cohort studies, the NOS consisted of three dimensions of quality: selection (4 points), comparability (2 points) and outcome (3 points). It allowed a total score from 0 to 9 points, and we considered a total score of 0-3, 4-6, and 7-9 indicating low, medium and high study quality, respectively (Wells et al., 2013).

2.6 Statistical methods

The statistical software Stata, version 15.1 (StataCorp LLC, College Station, Texas, USA), was used for the statistical analyses. All figures presented in this section are made as illustrative examples, and should not be interpreted as results.

2.6.1 Fixed effect model and random effects model

This meta-analysis is based on studies that differed in terms of design, conduct, participants, and methods for assessing exposure and outcome. Such factors may lead to greater variability in the results of the different studies than what is expected, and is known as heterogeneity. Assumption of heterogeneity plays a critical role in choosing between the two conceptually different approaches to meta-analysis, the fixed effect model or the random effects model (Higgins & Thompson, 2002).

The fixed effect model assumes that the effect size will be identical in every study. If there is observed variation in effect size, this is assumed to be due to random error within each study, implying no heterogeneity. In this model larger studies are thought to give more precise estimates of the common effect and are assigned more weight than smaller studies, which are thought to give less precise estimates (Borenstein et al., 2009).

The random effects model assumes that the effect size will vary in the different studies and the goal is to estimate the effects in a range of populations. The variation is assumed to be due to random error within studies (within-studies variance) plus true variation in effect size from one study to the next (between-studies variance). In this model a small study might include information about a population that no other study has captured, and is given more weight than it would under the fixed effect model, even if the estimated effect is thought to be

imprecise. In the same manner, a large study with high accuracy is not given too much weight, to ensure that the pooled estimate is not overly influenced by one population. In general, the random effects models give more similar weight to the studies than a fixed effects model does (Borenstein et al., 2009).

As we do not assume a common effect size, the random effects model, which takes into account heterogeneity within and between studies, was used to calculate summary relative risks (RR) for the association between fruit and vegetable intake and type 2 diabetes (Borenstein et al., 2009; DerSimonian & Laird, 1986).

In epidemiological research, effect estimates such as relative risks (RRs) and its associated 95% confidence intervals (CIs), are often used to quantify an association between an exposure and an outcome (Hennekens, Buring, Mayrent, & Doll, 1987; Ressing, Blettner, & Klug, 2010). Confidence intervals provide a range of values, which with 95% certainty reflects the true value (du Prel, Hommel, Röhrig, & Blettner, 2009). In this meta-analysis, the hypotheses were two-sided which means that the associations may be positive or negative, and a 2-tailed *P* value of <0.05 was considered statistically significant.

2.6.2 High vs. low analysis

In the high vs. low analyses we calculated summary relative risks (95% confidence intervals) for the association between an exposure and type 2 diabetes, using the extreme exposure categories (Yu, Schmid, Lichtenstein, Lau, & Trikalinos, 2013).

2.6.3 Linear dose-response analysis

The method of Greenland and Longnecker was used for the linear dose-response analysis and study specific slopes (linear trends) and 95% confidence intervals were computed from the natural logarithm of the relative risks across categories of fruit and vegetable intake (Greenland & Longnecker, 1992). For fruit, vegetables, and fruit and vegetables combined, 200 gram per day was used as dose and for most fruit and vegetable subtypes we used 100 gram per day. For total berries, strawberries and blueberries, 50 gram per day was used, and 10 gram per day was used for brussel sprouts and kale, mustard and chard greens, because these increments were within the range of consumption reported in the original studies.

The dose-response analysis requires that the median/mean intake levels for the different exposures, the distribution of cases, and person-years are available for each category. For studies that did not report the distribution of cases, participants or person-years

per category, this was estimated by dividing the total number of cases and the total number of participants by the number of categories. The number of participants per category was then multiplied by the average follow-up time to get person-years per category. The number of cases per category was subtracted from the number of participants per category to find the number of non-cases per category. For one study, Auerbach et al, 2017, the number of participants varied substantially between categories. In order to find cases per category we had to multiply the RR by the number of participants per category, then summarize these RRs. Then each of these individual RRs were divided by the total RR and then multiplied by the total number of cases.

2.6.4 Nonlinear dose-response analysis

Nonlinear dose-response analyses were used to examine the shape of the associations and to see which intake level provided the greatest risk reductions (**Figure 1, 2**). Risk estimates are given for different intake levels in grams per day and are provided in tables, which supplements the figures. Risk estimates are given for different intake levels in grams per day. Nonlinear dose-response analysis was conducted using restricted cubic splines with three knots at 10%, 50%, and 90% centiles of the distribution, which were then combined using multivariable meta-analysis (Jackson, White, & Thompson, 2010; N. Orsini, Li, Wolk, Khudyakov, & Spiegelman, 2012).

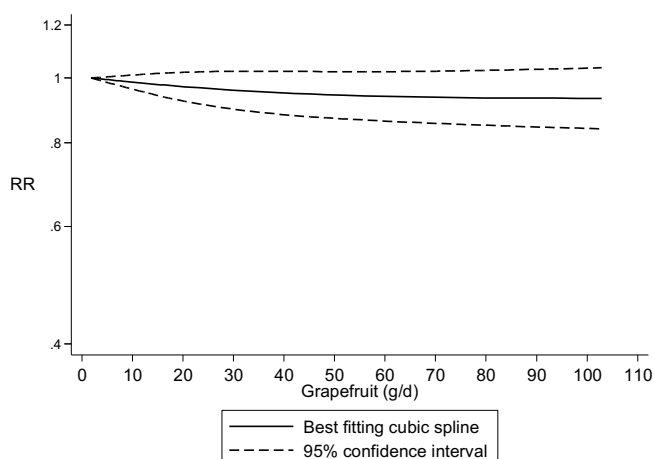


Figure 1. Example of nonlinear dose-response analysis, linear trend
($P_{\text{nonlinearity}} > 0.05$)

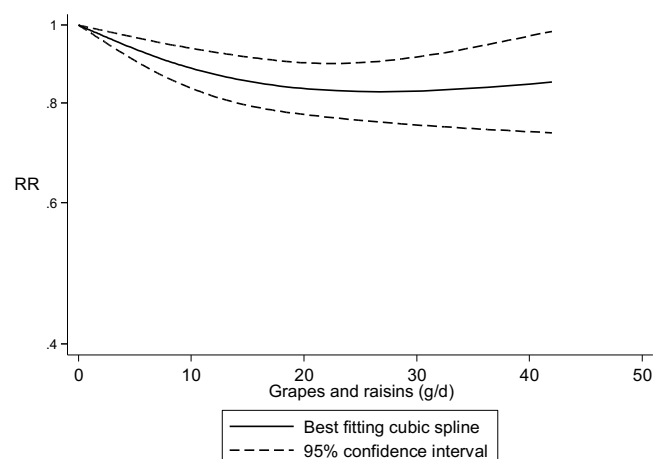


Figure 2. Example of nonlinear dose-response analysis, nonlinear trend
($P_{\text{nonlinearity}} = < 0.05$)

For the nonlinear dose-response analysis only studies with three or more categories were included. In order to conduct the analyses, the reference category had to represent the lowest intake present in the dataset for each exposure. When the second lowest category was used as the reference category, we recalculated the relative risks and confidence intervals so that the lowest category became the reference category using the method by Hamling and colleagues (Hamling, Lee, Weitkunat, & Ambuhl, 2008).

2.6.5 Heterogeneity

Heterogeneity between studies was evaluated with Q and I² statistics (Higgins & Thompson, 2002).

2.6.5.1 Q-test

The Cochran's Q-test is a statistical test of indicating the presence of heterogeneity, which captures the sum of the between-studies variance relative to within-studies variance (Borenstein et al., 2009). This variance, Q, is defined as:

$$Q = \sum w_i (\theta_{\tau} - \theta_{IV})^2$$

The Q-test depends on the number of included studies in the meta-analysis. With few studies, Q has low power, and with many studies Q has inappropriately high power. This makes it difficult to detect the presence of heterogeneity or whether it is clinically important (Gavaghan, Moore, & McQuay, 2000; Higgins & Thompson, 2002).

2.6.5.2 I-squared

When reporting a combined effect size, it is important to be able not only to state the existence of heterogeneity, but also to quantify the extent, as this impact the interpretation of the conclusion. Higgins et al. proposed an index to quantify the variance as a proportion of the total variance, called I-squared (I²), defined as:

$$\text{Variance (Between-studies) / Variance (Total)}$$

This index is multiplied by 100 and reported on a scale of 0 to 100. I^2 describes the percentage of total variation in point estimates that is due to heterogeneity rather than sampling error (Higgins & Thompson, 2002). Higgins suggest that the values of 25%, 50% and 75% represent low, moderate and high heterogeneity, respectively (Higgins, Thompson, Deeks, & Altman, 2003). I^2 statistics are not directly affected by the number of studies in the analysis, and may therefore be used in meta-analyses of different sizes. In addition to Q and I^2 statistics, the forest plot should be investigated to consider the range of effects and the implications of this range. The interpretation of the heterogeneity also depends on the direction of the observed effects. It is more problematic if high heterogeneity is caused by studies showing different directions of effects with inverse, null and positive associations observed, than if all studies show effects in the same direction, but with differing effect sizes, and where the heterogeneity is caused by differences in the effect sizes (Borenstein et al., 2009).

2.6.6 Forest plot

The forest plot serves as a visual representation of the data in a meta-analysis. Further, the forest plot provides a simple way to visually explore the amount of study heterogeneity. The plot can help to ensure that the data are interpreted properly, and may help to highlight outliers that require attention (Borenstein et al., 2009).

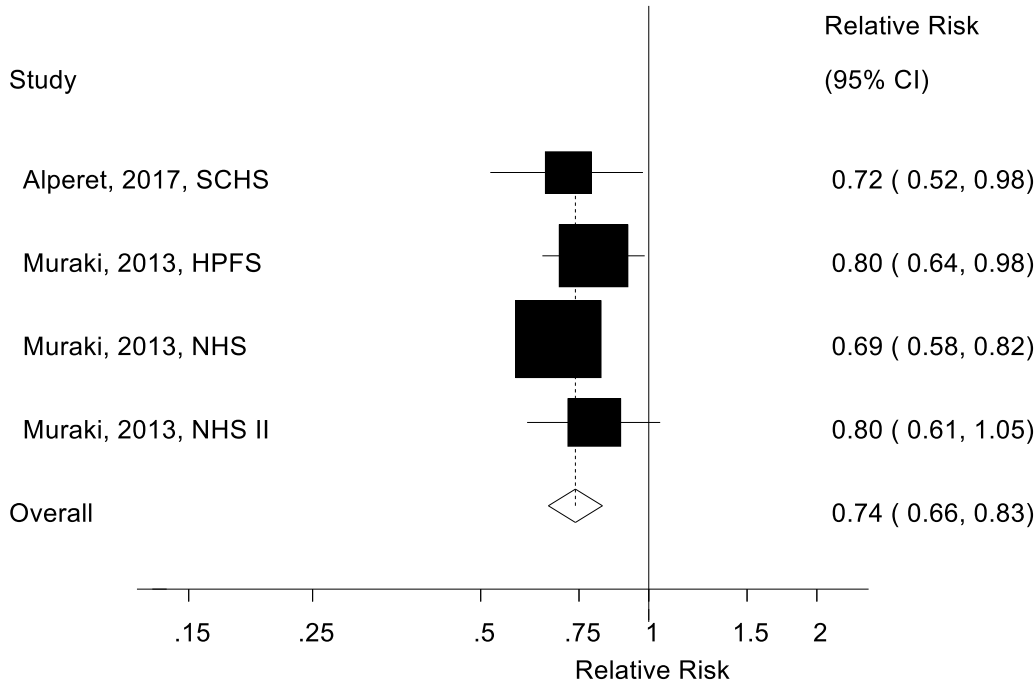


Figure 3. Example of forest plot, indicating a significant inverse association

In the forest plot, the authors and publication year of the studies are listed on the left, and sorted chronologically by publication year, from newest to oldest, with an abbreviation of the study name (**Figure 3**). On the right side, the effect size is expressed as RR with the 95% CI. The plot shows the point estimates (RR) of the individual studies in the meta-analysis, represented as squares proportional to the weight that the study contributed to the meta-analysis, and with horizontal lines showing the CI for each study. A solid vertical line represents no significant effect (RR = 1.0). If the 95% CI for the individual studies overlap with this line, the results would be non-significant. At the bottom of the plot, the summary estimate is represented as a diamond, with its' widths indicating the CI, and a dotted vertical line drawn out of the center. If the diamond is clear of the line of no effect, the observed effect is significant (Egger, Smith, & O'Rourke, 2001; Lewis & Clarke, 2001).

2.6.7 Subgroup and meta-regression analyses

In the subgroup analyses participant data was split into subgroups to examine if study characteristic were associated with the observed effects in the meta-analysis. Significance level was set to $P = <0.05$. Meta-regression analyses were used to test for differences in the outcome variable when analyses were stratified by subgroups (Baker, White, Cappelleri, Kluger, & Coleman, 2009). The subgroup and meta-regression analyses stratified by study characteristics including duration of follow-up, gender, geographical location, number of cases, and adjustment for confounding factors were conducted to investigate sources of heterogeneity. The duration of follow-up was divided into <10 years and ≥ 10 years. The subgroup analyses were stratified by sex (men, women, and men and women combined). Geographic locations were Europe, America, Asia and Australia. Number of cases were divided into three categories, <1.000 , $1.000- <2.000$ and ≥ 2.000 . Study quality based on NOS score, were also divided into three categories, 0-3, 4-6 and 7-9. The confounding factors included age, education, ethnicity, family history of diabetes, BMI, waist circumference/WHR, hypertension, alcohol, smoking, physical activity, consumption of meat, soft drinks, whole grains, coffee and total energy intake.

2.6.8 Publication bias

Publication bias was assessed by inspection of funnel plots and with Egger's test. When there was evidence of publication bias, we used the trim and fill method to assess its potential influence on the results. We explored whether this was driven by one or a few outlying

studies and conducted sensitivity analyses excluding such studies to see if the test for publication bias was attenuated, and also whether the summary estimate was altered. We also considered using the trim and fill method (Duval & Tweedie, 2000), however, no studies were added to the analyses when using this method and thus we only report results from the previously mentioned sensitivity analyses. Publication bias occurs when results of published studies are systematically different from results of unpublished studies. The direction and statistical significance of the results often has a big impact (Rothstein, Sutton, & Borenstein, 2005), as studies with “positive” and statistically significant results are more likely to be published than those with statistically non-significant or null results (Dickersin, 2005). This gives an unrepresentative picture of the body of evidence, and wrong conclusions may be drawn. If the sample of studies included in a meta-analysis is biased, the validity of the results of a meta-analysis is threatened (Rothstein et al., 2005). Publication bias tests have been developed to assess the likely extent of the bias, and to determine what conclusions can be drawn despite the potential for bias (Borenstein et al., 2009). In addition, funnel plot asymmetry may not always be due to publication bias. For example, smaller studies may have lower methodological quality, which may exaggerate treatment effects. In some circumstances smaller studies may also allow for more comprehensive or intensive interventions, higher compliance and thereby greater treatment effects, than in large studies (Sterne, Becker, & Egger, 2005). The term “small-study effects” is therefore often preferred to publication bias because it does not imply the cause of the asymmetry (Sterne, Gavaghan, & Egger, 2000).

2.6.8.1 Funnel plots

Funnel plots were used to investigate the presence of small-study effects in this meta-analysis for exposures with eight or more studies. The funnel plot gives a visual representation of the effect size (logarithm of the relative risk) estimated from individual studies against a measure of study size (standard error of the logarithm of the relative risk) (Sterne et al., 2005).

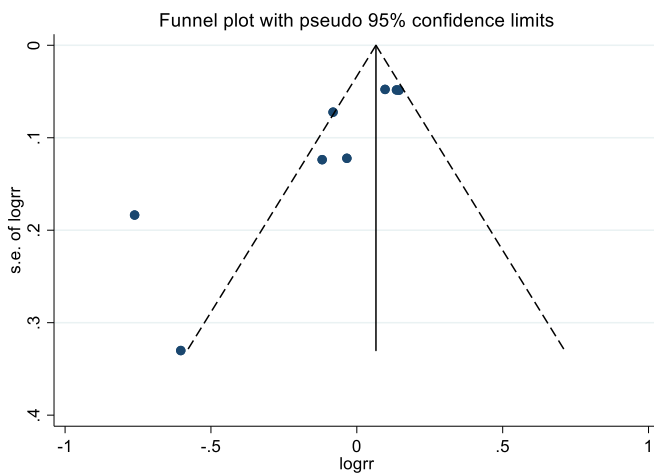


Figure 4. Example of funnel plot, indicating publication bias

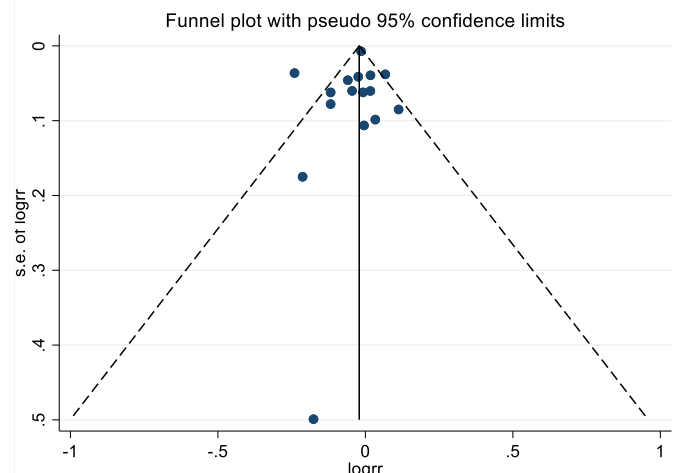


Figure 5. Example of funnel plot, indicating no publication bias

Funnel plot asymmetry might indicate publication bias, and is shown by a higher concentration of studies on one side of the mean (the vertical line) than the other (**Figure 4**). The asymmetry is usually driven by smaller studies (with larger standard errors) missing towards the bottom on one of the sides of the funnel plot. In contrast, if publication bias is absent, the studies will be distributed symmetrically about the mean (**Figure 5**). Visual inspections of the funnel plot are subjective and statistical tests are therefore needed to quantify the amount of bias captured (Borenstein et al., 2009).

2.6.8.2 Egger's test

Egger et al. (1997) introduced a linear regression approach, called Egger's test, which uses the values of the effect sizes and their precision to quantify the bias captured by the funnel plot. A threshold of $P < 0.1$ indicates presence of publication bias (Egger, Smith, Schneider, & Minder, 1997). The power of this test is low unless there is severe bias, or a substantial number of studies (Sterne et al., 2000). Asymmetry in the funnel plots or a statistically significant Egger's test does not prove that there is publication bias in the analysis.

Asymmetry might be a result of selection bias, true heterogeneity, data irregularities, chance and so on (Egger et al., 1997).

2.6.8.3 Trim and Fill method

The concern with publication bias is that the potentially missing studies affects the combined estimate, and therefore the number of missing studies should be estimated, and the effect that these studies might have on the outcome should be investigated. The Trim and Fill method developed by Duval and Tweedie was used to adjust the meta-analysis for the impact of missing studies (Duval & Tweedie, 2000), where the funnel plot indicated publication bias. This method is used both to identify and correct for asymmetry in the funnel plot, and makes it possible to estimate where the missing studies are likely to fall (Borenstein et al., 2009). The smaller studies thought to cause asymmetry is removed, and the trimmed plot is then used to re-estimate the mean effect size. Finally, the excluded studies and their missing counterparts are filled in and the meta-analysis is conducted again with the inclusion of the filled studies (Duval & Tweedie, 2000).

2.6.8.4 Sensitivity analyses

Sensitivity analyses were used to explore the impact different statistical decisions have on the results in our meta-analysis. The robustness of the findings was tested in sensitivity analyses excluding one study at a time from the meta-analysis to clarify whether the results were driven by one very large study or a study with an extreme result (Russo, 2007).

3.0 Results

In this chapter we will shortly summarize the main findings from the statistical analyses of fruit and vegetables, and their subtypes, in order to avoid duplicate reporting of our results presented in the article.

We found an inverse association between intake of fruits, and fruit and vegetables combined and the risk of type 2 diabetes. No significant associations were found for intake of vegetables. Of subtypes of fruit and vegetables, especially apples, blueberries, and grapes and raisins were strongly associated with a reduced risk, while cabbage, cauliflower, kale, mustard and chard greens and potatoes were strongly associated with an increased risk of type 2 diabetes. For the remaining exposures there was no significant association was observed.

We chose to present figures and tables from the main findings throughout the article to make the results section more accessible as the supplementary materials are extensive. More detailed information and additional results from this meta-analysis can be found in **Attachment 1**.

4.0 Discussion

In this chapter we will discuss some advantages and limitations with systematic reviews and meta-analyses as methods, as well as experienced challenges. Further, the nutrient content in fruit and vegetables will be discussed briefly. Before continuing reading this section, we recommend reading the article.

4.1 Discussion of the methods

4.1.1 Advantages

An advantage of this systematic review on fruit and vegetable intake and risk of type 2 diabetes, is that by developing a comprehensive plan for identifying, appraising, and synthesizing all relevant studies, selection bias and the risk of relevant studies not being detected was reduced (Uman, 2011). In this way, a systematic review allows a more objective appraisal of the evidence, and are not subject to personal opinions that may affect the more traditional narrative reviews (Egger, Smith, & O'Rourke, 2001).

Few individual studies are large enough to detect statistically significant differences in effect estimates. This may produce false negative results, which indicates no significant effect, even when such effect in reality is present. Therefore, a major advantage of this meta-analysis is that by combining studies we increased the sample size and the precision of the effect estimates (Borenstein et al., 2009; Egger, Smith, & O'Rourke, 2001).

The increasing volume of new research makes it difficult for policy makers and health professionals to evaluate and synthesize current knowledge. By accumulating evidence from individual prospective studies on the association of fruit and vegetable intake and the risk of type 2 diabetes into a systematic review and meta-analysis, we can provide an important tool for practitioners to keep up with the evidence. In addition, a meta-analysis can reduce erroneous findings due to chance, and may identify potential areas where further research are needed (Egger, Smith, & O'Rourke, 2001).

4.1.2 Limitations

The limitations of our systematic review and meta-analysis could be caused by different types of biases, which may threaten the validity of the results (Egger, Dickersin, et al., 2001; Institute of Medicine, 1990). To reduce the risk of language bias we had no restrictions on

language in the search for relevant studies. Despite that, we cannot exclude the possibility that language bias was already present in the databases. Most studies were published in English, and it can therefore be difficult to predict if studies published in other languages than English could have had an impact on the results of this meta-analysis. However, in a study of several meta-analysis the findings indicated that exclusion of non-English language trials did not have an impact the summary treatment effect estimates (Juni, Holenstein, Sterne, Bartlett, & Egger, 2002). In several meta-analysis we have seen cases of duplicate publication bias, which may lead to overestimation of the effects. However, in our meta-analysis, we were aware of this problem prior to the analyses and duplicate studies were excluded. Most studies reported all information needed to conduct a meta-analysis in their publications. However, in some eligible cohorts we lacked necessary information to conduct analyses, and had to try to obtain this information by correspondence with the authors. For all but one study we got the needed information, and therefore we cannot rule out the possibility that this study could have affected our results.

In recent years, there has been a rush to publish first on a topic. This have resulted in many poorly conducted meta-analyses with methodological flaws, such as incomplete literature searches and data collection, loose definitions of inclusion or exclusion criteria, and duplicate data, which may lead to wrong conclusions. If public policies and recommendations are based on poorly conducted meta-analysis this may negatively impact public health (Satija et al., 2015).

4.1.3 Experienced challenges

We experienced several challenges in the process of conducting this systematic review and meta-analysis. The screening process involved a large number of studies and was therefore time-consuming as we both independently screened all the potentially relevant studies from the search of both the PubMed and Embase databases. However, it was important that two investigators screened all studies to ensure that eligible studies were not missed, and that the inclusion and/or exclusion criteria of studies were not too loose, or too strict, which can be problematic with only one investigator (Singh, 2017). We experienced it as a strength to conduct this master's thesis in pair as we could plan, discuss, double check the data extracted, as well as conduct analyses and solve possible challenges together.

4.2 Discussion of nutrient content in fruit and vegetables

The observed protective effect of fruit and vegetable intake on the risk of type 2 diabetes may partially be explained by their high content of dietary fiber, antioxidants, vitamins, minerals and phytochemicals. Intake of dietary fiber has been suggested to reduce postprandial glucose responses by delaying gastric emptying, reduce the rate of glucose absorption and reduce blood sugar concentrations (Jenkins et al., 1978). Especially diets high in insoluble fiber have shown reduced diabetes risk. However, fruit and vegetables contain more soluble fiber and a protective effect has been less clear observed for fruit or vegetable fiber, compared to cereal fiber (InterAct Consortium, 2015). It is also possible that other substances in fruit and vegetables than the fiber content may be responsible for the protective effect (Russell et al., 2016).

Although fruit juice may contain nutrients and polyphenols, such as those that are present in whole fruits, healthy compounds in fruit juice may decrease during the processing (Crowe & Murray, 2013). Consistent with other meta-analyses we found an increased risk association per 250 mL/day for fruit juice (Imamura et al., 2016), and fruit drink intake (Xi et al., 2014), and type 2 diabetes. As fruit juice is fluid and have a moderately high glycemic index (Atkinson, Foster-Powell, & Brand-Miller, 2008), intake may lead to a rapid increase in blood glucose levels (Radulian, Rusu, Dragomir, & Posea, 2009). In most countries fruit juice is therefore recommended to consume in moderation (B. J. Auerbach et al., 2017). Potatoes contain large amounts of rapidly absorbed starch and high GI and GL (Halton et al., 2006), which lead to a rapid increase in blood glucose and insulin concentrations and is associated with an increased risk for T2D (Augustin, Franceschi, Jenkins, Kendall, & La Vecchia, 2002). Both intake of fruit juices and potatoes has been associated with excess weight gain over time and this could also contribute to an increased risk of type 2 diabetes (Mozaffarian, Hao, Rimm, Willett, & Hu, 2011).

Although many compounds in fruit and vegetables seem to have a protective effect on type 2 diabetes, there is a possibility that other compounds also have an effect. Fruit and vegetables are not consumed in isolation, but are a part of a wider diet which consist of other nutrients (NNR, 2014). It is also likely that the different types of fruit and vegetables consumed varies between different populations. For example, in Asia cruciferous vegetables may include vegetables that are not commonly eaten in the U.S. or Europe, such as Chinese cabbage (bok choy). Further, the nutrient content in different sorts of fruits and vegetables may differ. Blueberries consumed in America may have a different content than the European

blueberries (bilberries). In addition, the same type of fruit or vegetables may differ in nutrient content through season and by different growing methods and conditions (Burdulis et al., 2009; Chu W, Cheung SCM, & Lau RAW, 2011). Further, food preparation methods and degree of processing may influence the nutrient content (Fabbri & Crosby, 2016; Slavin & Lloyd, 2012). Altogether, these factors can make the study of diet in relation to health challenging. Further, research on the underlying mechanisms affecting the protective effect of fruit and vegetable intake on the risk of type 2 diabetes are needed.

5. Overall conclusion and implications for public health

The prevalence of type 2 diabetes has increased rapidly worldwide over the past two decades and had a considerable impact on global health. Several modifiable risk factors for type 2 diabetes have been established, such as dietary factors. Fruit and vegetables and their subtypes are important sources of nutrients, dietary fibers, antioxidants, vitamins, minerals and phytochemicals, and have the potential to influence biological functions in the human body through different mechanisms. The different compounds and their underlying mechanisms have been linked to the protective effect of fruit and vegetable intake. However, the findings from several meta-analysis have been inconsistent in establishing a robust association between fruit and vegetables and risk of type 2 diabetes, and more studies are needed to clarify the association.

This meta-analysis provides the most comprehensive and up-to-date summary of the available evidence to date and have important public health implications given the current epidemic of adiposity and diabetes globally (GBD 2018 DALYs and HALE Collaborators, 2018). Policy makers and health professionals may find it difficult to keep up with the increasing volume of research, and our systematic reviews and meta-analyses can therefore serve as an important tool for keeping updated. Our findings support existing recommendations to increase the intake of fruit and vegetables, but suggest certain subtypes of fruits including apples, blueberries, grapefruit, grapes and raisins may be particularly beneficial, while potatoes, and fruit juice and fruit drinks may increase the risk. In addition, some venues for further areas that need clarification have been identified. Any further studies should report in more detail associations between subtypes of fruits and vegetables and type 2 diabetes, adjust for more dietary confounders, and report analyses stratified by other risk factors to better be able to rule out residual confounding. In addition, because most of the available studies have been conducted in Europe, North America and Asia further studies are needed from other geographic regions.

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Article

Fruit and vegetable consumption and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of prospective studies

Word Count: 6073

ABSTRACT

Background: The association between intake of fruit and vegetables and their subtypes, and the risk of type 2 diabetes has been investigated in several studies, but the results have not been consistent.

Objective: We conducted an updated systematic review and dose-response meta-analysis of prospective studies of the association between fruit and vegetable consumption and subtypes of fruit and vegetables and the risk of type 2 diabetes.

Design: PubMed and Embase databases were searched up to 26th of June 2018. Prospective cohort studies of fruit and vegetable consumption and type 2 diabetes mellitus were included. Summary relative risks (RRs) and 95% confidence intervals (CIs) were estimated using a random effects model.

Results: We included 43 cohort studies. In the dose-response analysis, the summary RR per 200 g/day were 0.98 (95% CI: 0.95-1.01, $I^2 = 37.8\%$, $n = 7$) of fruit and vegetables, 0.96 (95% CI: 0.92-1.01, $I^2 = 71.6\%$, $n = 16$) of fruits, and 0.98 (95% CI: 0.94-1.02, $I^2 = 48.3\%$, $n = 12$) of vegetables. For 250 g/day of 100% fruit juice, the summary RR was 0.97 (95% CI: 0.91-1.03, $I^2 = 0\%$, $n = 3$), and for 100 g/day of potatoes the summary RR was 1.08 (95% CI: 1.02-1.15, $I^2 = 55.4\%$, $n = 8$). Inverse associations were observed for apples, apples and pears, blueberries, grapefruit and grapes and raisins, while positive associations were observed for intakes of cantaloupe, brussel sprouts, cauliflower and kale, mustard and chard greens, however, most of these associations were based on few studies and need further confirmation in additional studies. Nonlinear inverse associations were observed for fruits, vegetables, bananas, blueberries, grapes and raisins, and allium vegetables, which in general were steeper at low to moderate intakes than at higher intakes.

Conclusions: This meta-analysis suggests that there is a weak inverse association between fruit and vegetable intake and type 2 diabetes risk. There is some indication of both inverse and positive associations between intake of several fruit and vegetables subtypes and type 2 diabetes risk, however, because of the limited number of studies, further studies are needed before firm conclusions can be made.

Key terms: fruit and vegetables, nutrition, type 2 diabetes, cohort, systematic review, meta-analysis.

INTRODUCTION

The prevalence of type 2 diabetes has increased rapidly over the past two decades from 108 million in 1980 to 422 million in 2014 worldwide (1). If current trends continue the prevalence is estimated to pass 700 million by 2025 (2). As type 2 diabetes contributes to blindness, neuropathies, nephropathies, cardiovascular disease, cancer and premature mortality (3, 4), the increasing prevalence has a considerable impact on public health globally (1).

There have been established several modifiable risk factors for type 2 diabetes, including overweight and obesity, physical inactivity, unhealthy diet and smoking. Non-modifiable risk factors include ethnicity, family history of diabetes, previous gestational diabetes, and older age (1). Overweight and obesity are the strongest risk factors for type 2 diabetes with relative risks reported of between 10-40 for severe obesity compared to lean individuals (5-8). Dietary factors are important modifiable risk factors for type 2 diabetes and several previous studies have found increased risk of type 2 diabetes with a high intake of red and processed meat, sugar- sweetened beverages, and low intake of whole grains, fiber, dairy products, fruits and vegetables (9-18), although more recent studies have questioned the role of dairy products in reducing diabetes risk (19, 20).

Most countries have national recommendations for the daily amount of fruit and vegetables that is needed to maintain optimal health, but these recommendations vary globally. Often three or more servings per day of vegetables and two or more servings per day of fruits are recommended with one serving often standardized to 80 grams (21).

A high intake of fruit and vegetables has been associated with a reduced risk of type 2 diabetes in several (11-13, 16, 18), but not all previous prospective observational studies (22-28). The evidence has been slightly more consistent in showing an inverse association between fruit intake and type 2 diabetes, than for vegetables. However, most of the available studies have been too small to detect a statistically significant reduction in risk, and in general the observed associations have been weak. In addition, some studies have suggested that specific types of fruits and vegetables may be more strongly associated with reduced risk of type 2 diabetes than overall fruit and vegetable intake. Inverse associations have been observed between the intake of apples/pears (22, 29-31), berries (11, 12, 16, 29, 31), green leafy vegetables (16, 18, 24), yellow vegetables (18, 27), root vegetables (12) and tomatoes (18) and the risk of type 2 diabetes, however, the available data have not been entirely

consistent. Although potatoes are not counted as part of the five recommended servings of fruits and vegetables per day, clarifying whether there is an association with type 2 diabetes would be important. Studies to date have found mixed results with some showing positive associations (16, 32), while others show no association (24, 25), or inverse associations with a high potato intake (33).

Previous meta-analyses have only analyzed a few specific fruit and vegetable subtypes. For example, Cooper et al. only considered green leafy vegetables and root vegetables (12), Jia et al. only considered citrus fruits and cruciferous vegetables (34), Chen et al. only considered green leafy and cruciferous vegetables (24) and Guo et al. only considered apples and pears (35).

The most recent meta-analysis only investigated total fruit and total vegetable intake (36). Ten additional studies (13, 22-24, 37-42) have been published since these meta-analyses came out, thus we conducted a comprehensive and up-to-date meta-analysis of prospective studies on intakes of fruit and vegetable and subtypes of fruit and vegetables and the risk of developing type 2 diabetes.

METHODS

Search strategy

We conducted a systematic search of PubMed (<https://www.ncbi.nlm.nih.gov/pubmed/>) and Embase (<https://www.elsevier.com/solutions/embase-biomedical-research>) databases up to 26th of June 2018 for eligible prospective cohort studies examining the association between the intake of fruit and vegetables and risk of T2D. We used the following search terms: (fruits OR vegetables OR fruit OR vegetable OR berry OR berries OR strawberries OR blueberries OR citrus OR “citrus fruits” OR orange OR apples OR pears OR banana OR cruciferae OR “cruciferous vegetables” OR broccoli OR cauliflower OR cabbages OR “allium vegetables” OR onion OR garlic OR tomato OR tomatoes OR potato OR "french fries" OR juice OR food OR "food groups") AND diabetes (**Supplementary Table 1**). The reference lists of retrieved articles were also scrutinized. There were no language restrictions.

Study selection

Studies were included if they had a prospective cohort, a case-cohort, or a nested case-control design and investigated the association between the intake of fruit and/or vegetables, subtypes of fruit and vegetables, fruit juices and/or fruit drinks, and risk of type 2 diabetes. The participants had to be free from type 2 diabetes at baseline. Multivariable adjusted risk estimates (relative risks (RRs), or odds ratios (ORs), hazard ratios (HRs)) with their corresponding 95% confidence intervals (CIs) had to be available in the publication. Intake levels for the different exposures, in addition to total number of cases and person-years, had to be available for the dose-response analyses. In addition, the reference lists of these articles were scrutinized for potentially relevant studies.

If duplicate reports from the same study cohort were identified, the study with most cases was included. Both the EPIC-InterAct Study (12) and the EPIC-Elderly Greece Study (43) were included as Greece is not a part of the EPIC-InterAct Study. ME and REH conducted the literature screening and study selection in duplicate.

Data extraction

The following data were extracted from each study: the first author’s last name, publication year, geographic location, name of the study, recruitment and follow-up period, sample size, age, sex, number of cases, dietary assessment method including number of food items

assessed and whether it has been validated, exposure, quantity of the exposure, relative risks and 95% CIs for the association, and confounders adjusted for in the analysis. We used the RR that reflected the greatest degree of adjustment for confounding variables. Data were extracted by two reviewers, ME and REH. Three publications (41, 42, 44) included data from multiple cohorts and results from each cohort were used rather than the pooled results. Standard criteria for the reporting of meta-analyses of observational studies, the MOOSE criteria and the PRISMA statement, were followed in this meta-analysis (45, 46).

Validity assessment

We independently assessed all included studies using The Newcastle–Ottawa Scale (NOS) to evaluate study quality, which allows a total score from 0 to 9 points, and we considered a total score of 0-3, 4-6, and 7-9 indicating low, medium and high study quality, respectively (47).

Statistical methods

The random-effects model by DerSimonian and Laird, which take into account heterogeneity within and between studies, were used to calculate summary relative risks for the association between fruit and vegetable intake and type 2 diabetes (48). A 2-tailed *P* value <0.05 was considered statistically significant.

The method of Greenland and Longnecker were used for the linear dose-response analysis and study specific slopes (linear trends) and 95% confidence intervals were computed from the natural logarithm of the relative risks across categories of fruit and vegetable intake (49). For studies that did not report the distribution of cases or person-years, this was estimated using the total number of cases or person-years. If studies had missing data on median or mean intake, we calculated the midpoint of the upper and lower boundaries of each category group to determine mean fruit or vegetable intake levels. In studies where the highest or lowest category was open ended, the open-ended interval length was assumed to be the same as the adjacent interval. Three studies (26, 50, 51), expressed data separately for men and women, and a fixed effects model was used to pool the results in order to obtain an overall risk estimate for men and women combined in these studies. For the China Kadoorie Biobank Study by Du H. et al (13), we had to estimate missing 95% CIs by using the formula $\log(\text{RR}) \pm 1.96 \times \text{SE}$ before transforming the numbers back to logarithmic scale. In studies where serving size was specified, this was used. Otherwise, in accordance with other meta-

analyses, we used a serving size of 80 g for fruit and vegetable intake (12, 36), and 250 mL for fruit juice and fruit drinks (14). For subtypes of fruit and vegetables we used serving sizes based on an article by Lee et al. (52) (**Supplementary Table 4**). We contacted the authors of two studies (33, 53) to get missing information on cut-off values or median intake on different exposures, confidence intervals and adjustments, and received detailed information from one author (53). The other study (33) was excluded.

Nonlinear dose-response analyses were conducted using restricted cubic splines with three knots at 10%, 50% and 90% centiles of the distribution, which were then combined using multivariable meta-analysis (54, 55). Heterogeneity between studies was evaluated with Q and I^2 statistics (56). I^2 values of 25%, 50% and 75% represents low, moderate and high heterogeneity, respectively. Subgroup and meta-regression analyses stratified by study characteristics were conducted for fruit and vegetable exposures with at least eight studies in the analysis. Subgroup analyses were conducted stratified by duration of follow-up, sex, geographical location, number of cases, study quality and adjustment for confounding factors were conducted to investigate sources of heterogeneity. Publication bias was assessed using Egger's test (57) and funnel plots were inspected for asymmetry when there were at least 8 studies in the analysis. A $P = <0.1$ indicated presence of publication bias. When there was evidence of publication bias, we explored whether this was driven by one or a few outlying studies and conducted sensitivity analyses excluding such studies to see if the test for publication bias was attenuated, and also whether the summary estimate was altered. We also considered using the trim and fill method (58), however, no studies were added to the analyses when using this method and thus we only report results from the previously mentioned sensitivity analyses.

The robustness of the findings was tested in sensitivity analyses excluding one study at a time from the meta-analysis to clarify whether the results were driven by one very large study or a study with an extreme result. The statistical analyses were conducted using the software package Stata, version 15.1 (StataCorp LLC, College Station, Texas, USA).

RESULTS

Literature search

The literature search conducted 26th of June 2018 resulted in 7600 records in PubMed, and 5937 records in Embase. The inclusion of MeSH terms for diabetes and fruit and vegetables did not change the number of records. The process of the study selection is shown in **Figure 1**. A total of 13 538 studies were identified, 13008 of those were excluded because they were irrelevant. 530 potentially eligible studies reported on fruit and vegetable intake and type 2 diabetes. The excluded studies are listed in **Supplementary Table 2**.

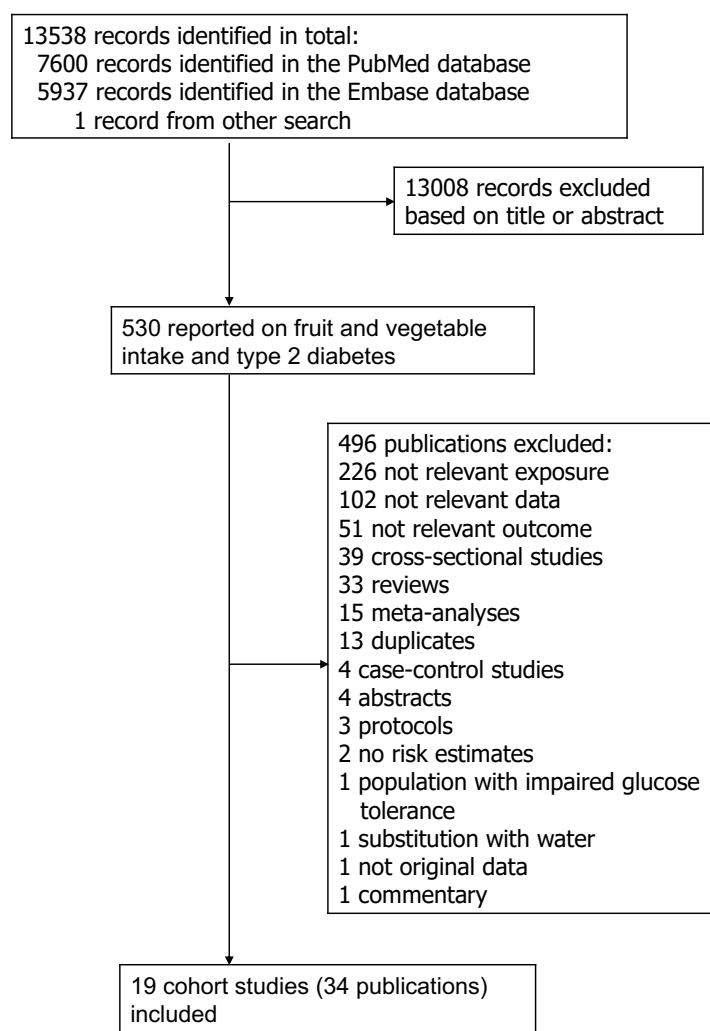


Figure 1. Flow-chart of study selection

Study characteristics

We included 41 studies in total. The follow-up periods ranged from 4 to 28 years. 22 of the studies were from America, 9 from Europe, 9 from Asia and 1 from Australia.

Supplementary table 3 shows the characteristics extracted from the included studies.

Validity assessment

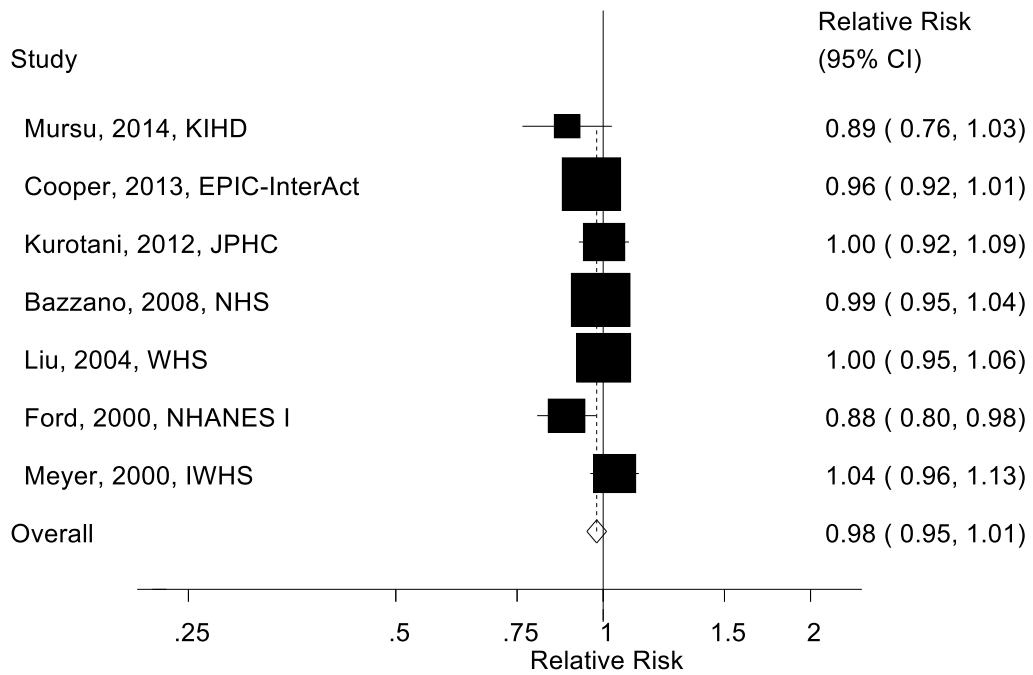
The quality scores ranged from 7 to 9 when evaluated with the NOS. All included studies had a NOS score of ≥ 7 , with an average score of 7.5, indicating the presence of high methodological quality (**Supplementary Table 5**).

Fruit and vegetables

A total of 8 cohort studies (8 publications) (11, 12, 26-28, 40, 53, 59) investigated the association between total fruit and vegetable intake and type 2 diabetes risk; these included 29 235 cases among 681 797 participants. All the studies were included in the high vs. low analysis. The summary RR for high vs. low intake was 0.94 (95% CI: 0.89-0.99) and the heterogeneity between studies was low ($I^2 = 19.1\%$ and $P_{\text{heterogeneity}} = 0.28$) (**Supplementary Figure 1, Table 1**). For the linear dose-response analysis 7 (11, 12, 26-28, 53, 59) of the 8 studies were included. The summary RR per 200 g/day was 0.98 (95% CI: 0.95-1.01, $I^2 = 37.8\%$, $P_{\text{heterogeneity}} = 0.14$) (**Figure 2a, Table 1**). In sensitivity analysis, the summary RR ranged from 0.97 (95% CI: 0.94-1.01) when excluding the Women's Health Study (WHS) by Liu et al. to 0.99 (95% CI: 0.96-1.01) when excluding the National Health and Nutrition Examination Study I (NHANES I) by Ford et al. (**Supplementary Figure 94**). There was no evidence of publication bias with Egger's test, $P = 0.41$ or by inspection of the funnel plots (Table 1, **Supplementary Figure 88**). Although the test for nonlinearity was not significant, $P_{\text{nonlinearity}} = 0.13$, there was a marginally significant 9-10% reduction in risk at an intake of 600-700g/day compared to 0 g/day (**Figure 2b, Supplementary Table 6**).

A

Fruits and vegetables and type 2 diabetes, per 200 g/d

**B**

Fruits and vegetables and type 2 diabetes, nonlinear dose-response

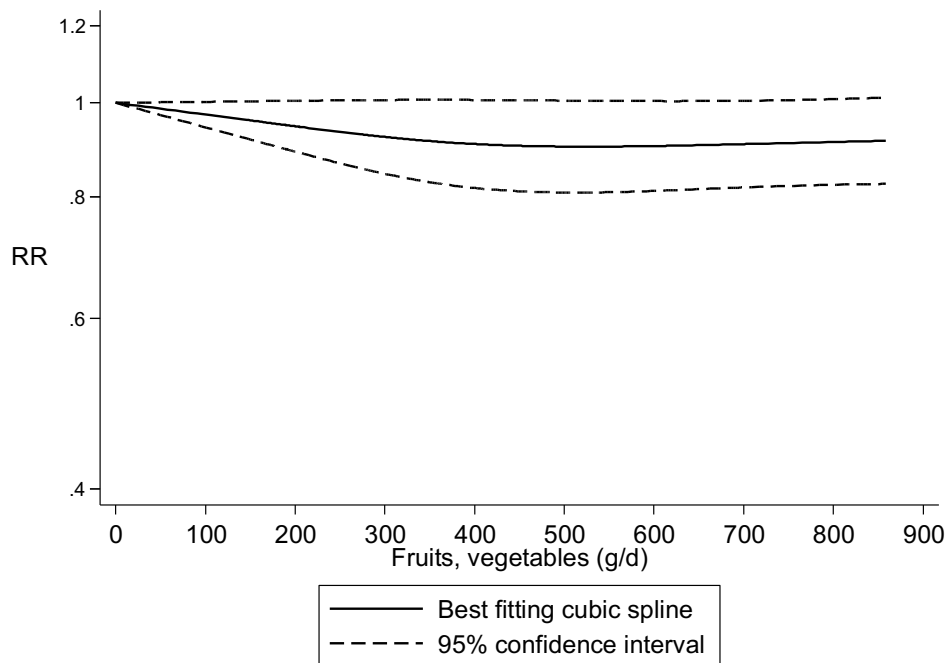


Figure 2. Fruit and vegetables and type 2 diabetes, linear and nonlinear dose-response

Fruits

A total of 16 cohort studies (13 publications) (12, 13, 16, 18, 22, 25-28, 42, 43, 53, 60) investigated the association between total fruit intake and type 2 diabetes risk; these included 79 516 cases among 1 478 790 participants. All of the 16 studies were included in the high vs. low analysis and in the linear dose-response analysis. The summary RR for high vs. low intake was 0.94 (95% CI: 0.90-0.97) and the heterogeneity between studies was low ($I^2 = 12.1\%$ and $P_{\text{heterogeneity}} = 0.31$) (**Supplementary Figure 2**, Table 1). The summary RR per 200 g/day was 0.96 (95% CI: 0.92-1.01, $I^2 = 71.6\%$, $P_{\text{heterogeneity}} = <0.001$) (**Figure 3a**, Table 1). In sensitivity analysis, the summary RR ranged from 0.95 (95% CI: 0.91-1.00) when excluding the Shanghai Women's Health Study (SWHS) by Villegas et al. to 0.99 (95% CI: 0.97-1.01) when excluding the China Kadoorie Biobank Study (CKB) by Du et al. (**Supplementary Figure 95**). There was no evidence of publication bias with Egger's test, $P = 0.48$ or by inspection of the funnel plots (Table 1, **Supplementary Figure 89**). There was evidence of a nonlinear association, $P_{\text{nonlinearity}} = 0.001$, which showed an 8-12% reduction in risk at an intake of 200-500g/day (**Figure 3b**, Supplementary Table 6).

Vegetables

A total of 13 cohort studies (12 publications) (11, 12, 16, 18, 24-28, 43, 53, 61) investigated the association between total vegetable intake and type 2 diabetes risk; these included 51 162 cases among 920 437 participants. All 13 studies were included in the high vs. low analysis. The summary RR for high vs. low intake was 0.96 (95% CI: 0.89-1.03) with moderate heterogeneity between studies ($I^2 = 66.8\%$ and $P_{\text{heterogeneity}} = <0.0001$) (**Supplementary Figure 3**, Table 1). For the linear dose-response analysis 12 (11, 12, 16, 18, 24-28, 43, 53) of the 13 studies were included. The summary RR per 200 g/day was 0.98 (95% CI: 0.94-1.02, $I^2 = 48.3\%$, $P_{\text{heterogeneity}} = 0.03$) (**Figure 4a**, Table 1). In sensitivity analysis, the summary RR ranged from 0.96 (95% CI: 0.92-1.01) when excluding the Nurses' Health Study (NHS) by Bazzano et al to 1.00 (95% CI: 0.98-1.02) when excluding the Shanghai Women's Health Study (SWHS) by Villegas et al. (**Supplementary Figure 96**). There was evidence of publication bias with Egger's test, $P = 0.08$, and by inspection of the funnel plots (Table 1, **Supplementary Figure 90**). However, exclusion of the study by Hodge et al, which appeared to be an outlier, attenuated Egger's test to $P = 0.12$, but did not materially alter the association, summary RR = 0.98 (95% CI: 0.94-1.02, $I^2 = 51\%$). There was evidence of a nonlinear association, $P_{\text{nonlinearity}} = 0.01$, and the risk reduction appeared to be steeper for

lower intakes. The strongest risk reduction was observed at an intake of 300 g/day, with no further risk reduction at intake above this level (**Figure 4b**, Supplementary Table 6).

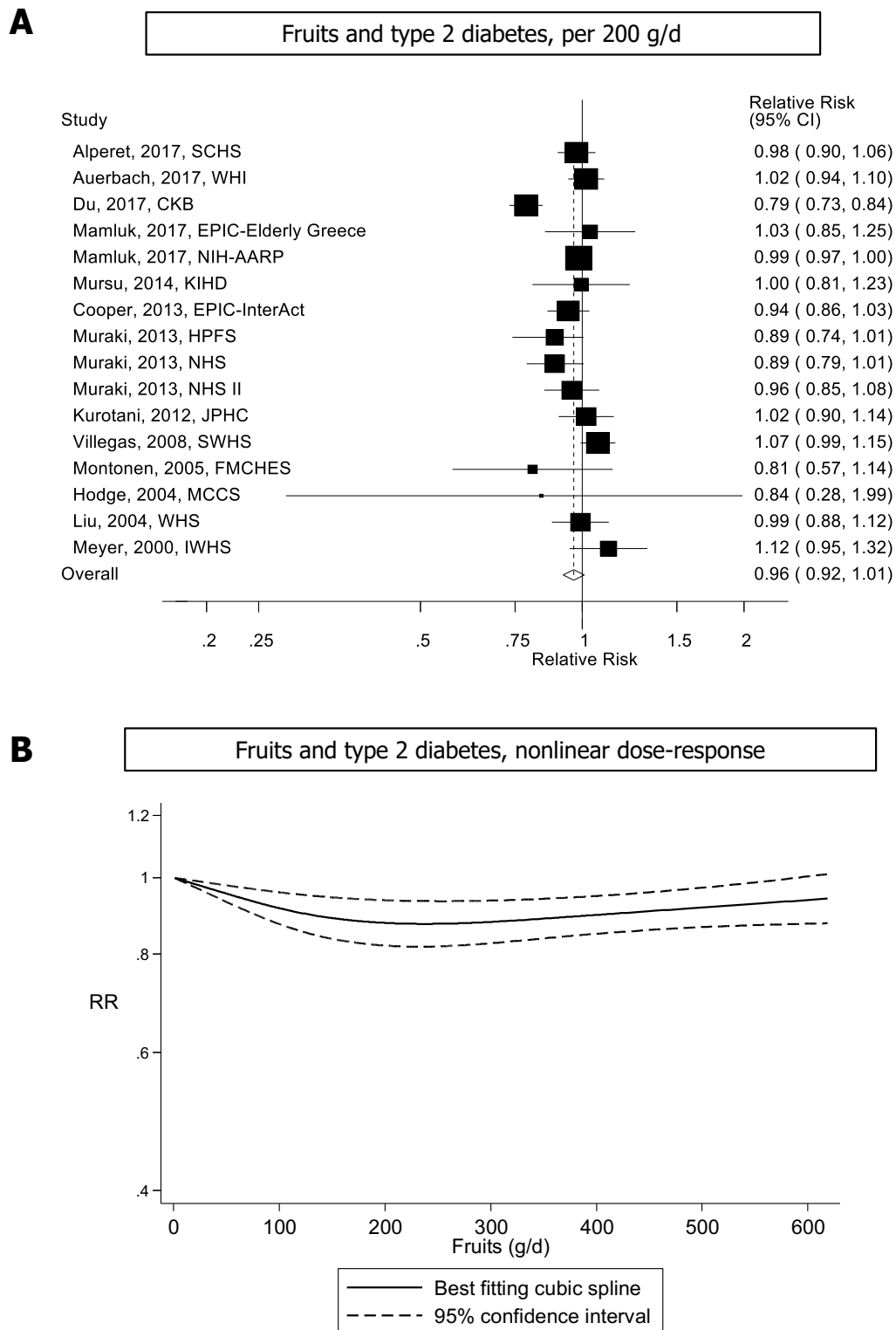


Figure 3. Fruits and type 2 diabetes, linear and nonlinear dose-response

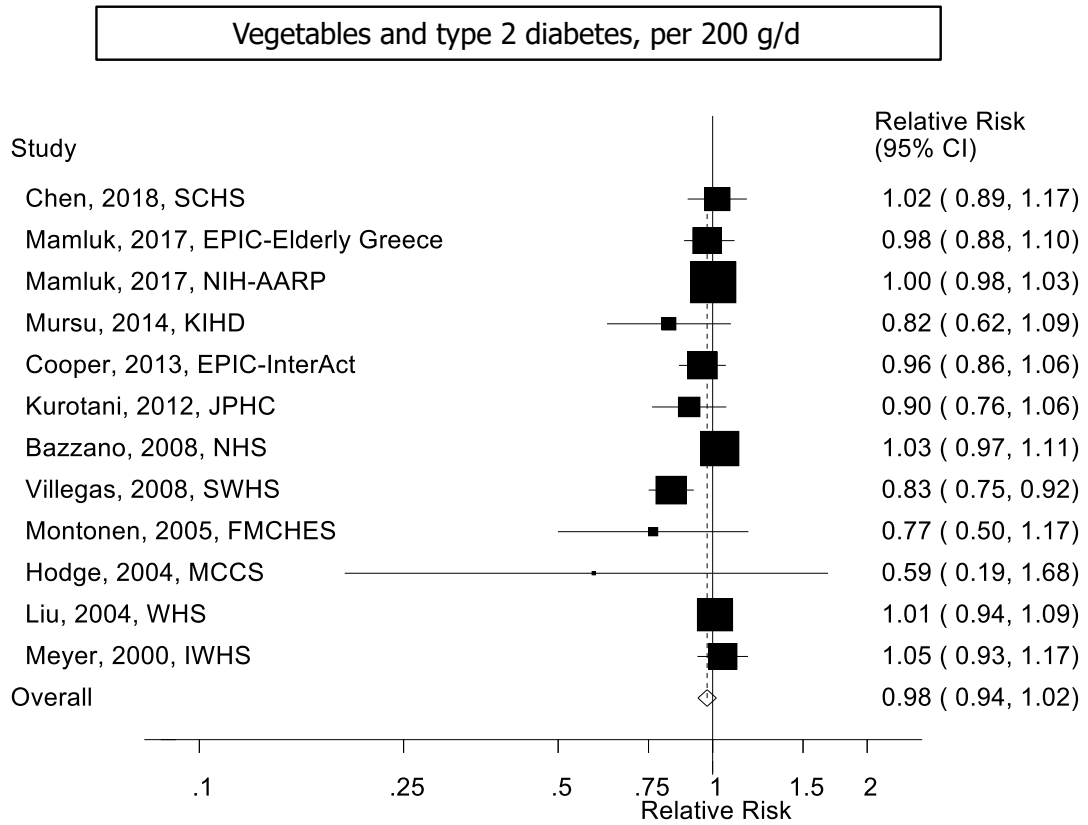
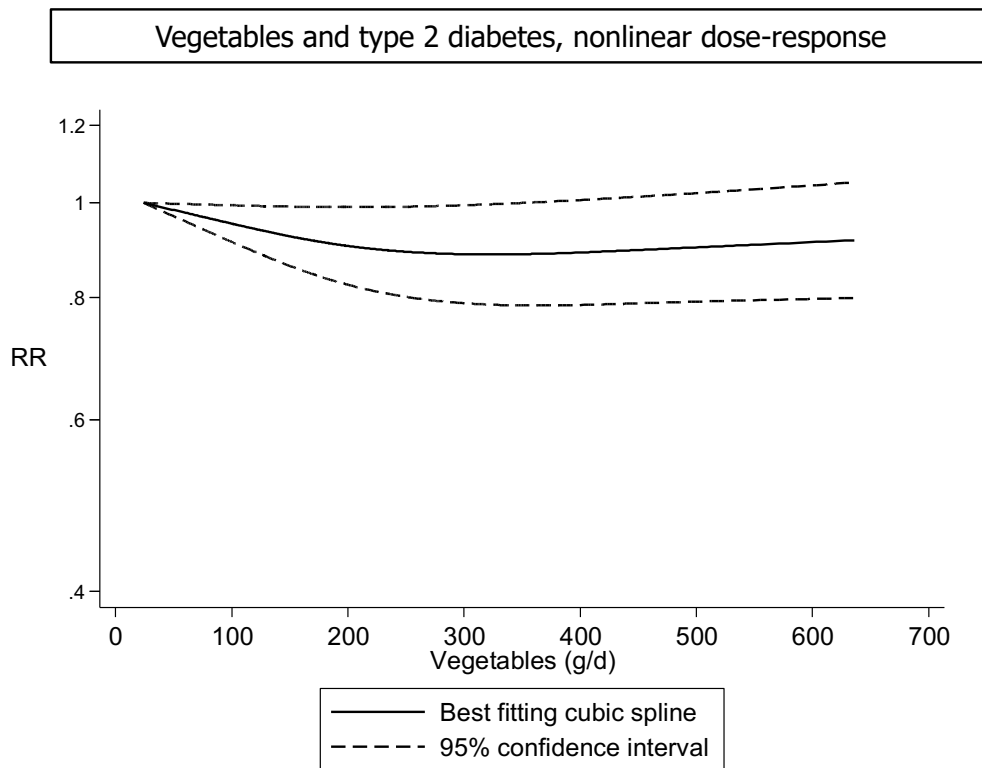
A**B**

Figure 4. Vegetables and type 2 diabetes, linear and nonlinear dose-response

Subtypes of fruits

Several studies investigated the association between subtypes of fruits and type 2 diabetes. Significant inverse associations were observed in the dose-response analyses, were the summary RRs per 100 g/day was 0.91 (95% CI: 0.88-0.95, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.87$) for apples, 0.90 (95% CI: 0.83-0.97, $I^2 = 38.4\%$, $P_{\text{heterogeneity}} = 0.18$) for apples and pears, 0.90 (95% CI: 0.82-0.99, $I^2 = 4.6\%$, $P_{\text{heterogeneity}} = 0.35$) for grapefruit, while significant positive associations were observed with summary RRs of 1.18 (95% CI: 1.04-1.34, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.82$) for cantaloupe, 1.07 (95% CI: 1.03-1.34, $I^2 = 69.0\%$, $P_{\text{heterogeneity}} = 0.007$) for fruit drinks, 1.09 (95% CI: 1.01-1.18, $I^2 = 71.4\%$, $P_{\text{heterogeneity}} = 0.002$) for fruit juice, and 1.05 (95% CI: 1.00-1.11, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.72$) for watermelon. The summary RR per 50 g/day was 0.60 (95% CI: 0.49-0.73, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.59$) for blueberries and 0.74 (95% CI: 0.66-0.83, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.69$) for grapes and raisins. No significant associations were observed for bananas, berries, citrus fruits, fruit drinks, oranges, peaches, plums and apricots, prunes, strawberries. Nonlinear inverse associations were observed for bananas $P_{\text{nonlinearity}} = 0.04$, blueberries $P_{\text{nonlinearity}} = 0.003$, and grapes and raisins $P_{\text{nonlinearity}} = 0.01$, with steeper reductions in risk at lower levels of intake, while a nonlinear positive association was observed for cantaloupe $P_{\text{nonlinearity}} = 0.04$, with steeper increase in risk at lower levels of intake. The association between apples, apples and pears, grapefruit, prunes and type 2 diabetes appeared to be linear (**Table 2, Supplementary Table 7-9, Supplementary Figures 4-51**).

Table 2. Summary relative risks for subtypes of fruits and type 2 diabetes, high vs. low-and dose-response analyses

Fruit subtype	High vs. low analysis						Dose-response analysis						
	<i>n</i>	RR (95% CI)	I ²	<i>P</i> _h	Egger	References	<i>n</i>	Increment	RR (95% CI)	I ²	<i>P</i> _h	Egger	References
Apples	3	0.79 (0.72-0.87)	0	0.52	0.03	(22, 29, 30)	2	Per. 100 g/d	0.91 (0.88-0.95)	0	0.87	-	(22, 62)
Apples and pears	5	0.88 (0.77-1.00)	74.7	0.003	0.21	(42, 51, 63)	4	Per. 100 g/d	0.90 (0.83-0.97)	38.4	0.18	0.98	
Bananas	5	0.97 (0.84-1.13)	70.5	0.009	0.80		5	Per. 100 g/d	0.93 (0.80-1.08)	84.4	<0.0001	0.72	
Berries	5	0.89 (0.72-1.10)	79.6	0.001	0.27		5	Per. 50 g/d	0.94 (0.77-1.14)	86.7	<0.0001	0.76	
Blueberries	3	0.76 (0.67-0.87)	0	0.49	0.54		3	Per. 50 g/d	0.60 (0.49-0.73)	0	0.59	0.70	
Cantaloupe	3	1.11 (1.02-1.20)	0	0.57	0.37		3	Per. 100 g/d	1.18 (1.04-1.34)	0	0.82	0.18	
Citrus fruits	6	1.04 (0.98-1.11)	0	0.92	0.79		6	Per. 100 g/d	1.02 (0.96-1.08)	46.9	0.09	0.29	
Fruit drinks	5	1.34 (1.11-1.62)	62.7	0.03	0.22		6	Per. 250 g/d	1.17 (1.03-1.34)	69.0	0.007	0.12	
Fruit juice	7	1.12 (1.04-1.20)	41.0	0.12	0.17		7	Per. 250 g/d	1.09 (1.01-1.18)	71.4	0.002	0.58	
Grapefruit	3	0.94 (0.81-1.10)	48.4	0.14	0.75		3	Per. 100 g/d	0.90 (0.82-0.99)	4.6	0.35	0.44	
Grapes and raisins	4	0.83 (0.76-0.91)	0	0.78	0.98		4	Per. 50 g/d	0.74 (0.66-0.83)	0	0.69	0.45	
Oranges	4	0.99 (0.93-1.06)	0	0.51	0.35		4	Per. 100 g/d	0.97 (0.92-1.04)	0	0.89	0.53	
Peaches, plums and apricots	3	0.91 (0.80-1.04)	2.6	0.36	0.72		3	Per. 100 g/d	0.89 (0.73-1.09)	44.9	0.16	0.33	
Prunes	3	0.94 (0.80-1.11)	34.0	0.22	0.63		3	Per. 100 g/d	0.73 (0.52-1.02)	0	0.45	0.78	
Strawberries	3	1.11 (0.90-1.38)	35.2	0.21	0.08		3	Per. 50 g/d	1.10 (0.88-1.38)	76.5	0.01	0.24	
Watermelon	2	1.06 (0.95-1.20)	0	0.64	-		2	Per. 100 g/d	1.05 (1.00-1.11)	0	0.72	-	

n = number of studies*P*_h = *P*-value for heterogeneity

Subtypes of vegetables

Significant positive associations were observed in the dose-response analyses, where the summary RR per 100 g/day was 1.31 (95% CI: 1.08-1.58, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.98$) for cauliflower, and the summary RRs per 10 g/day was 1.07 (95% CI: 1.03-1.12, $I^2 = 63.9\%$, $P_{\text{heterogeneity}} = 0.06$) for brussel sprouts, and 1.03 (95% CI: 1.00-1.06, $I^2 = 0\%$, $P_{\text{heterogeneity}} = 0.93$) for kale, mustard and chard greens. No associations were observed for allium vegetables, boiled potato, broccoli, cabbage, cruciferous vegetables, green leafy vegetables, tomatoes and yellow vegetables. Nonlinear inverse associations were observed for allium vegetables $P_{\text{nonlinearity}} = 0.045$, and the risk reduction appeared to be steeper for lower intakes, with a flattening of the curve by increasing intakes. Nonlinear positive associations were observed for cabbage $P_{\text{nonlinearity}} = 0.04$, with steeper increase in risk at lower levels of intake, and cauliflower $P_{\text{nonlinearity}} = 0.03$ with a slightly increase in risk at increasing levels of intake. The association between brussel sprouts and type 2 diabetes appeared to be linear (Table 2, **Supplementary Table 9-11, Supplementary Figure 52-87**). There was indication of publication bias in the analysis of total potatoes and type 2 diabetes ($P = 0.06$), however, the asymmetry in the funnel plot indicated missing positive studies (**Supplementary Figure 91**). Exclusion of one outlying study by Farhadnejad et al (38) attenuated Egger's test to 0.23, but did not substantially alter the results, summary RR = 1.09 (95% CI: 1.04-1.14, $I^2 = 40.2\%$). Although Egger's test was not significant in the analysis of green leafy vegetables and type 2 diabetes ($P = 0.46$), there was some indication of asymmetry in the funnel plot (**Supplementary Figure 92**), which appeared to be driven by the studies by Cooper et al. (12) and Kurotani et al. (26). However, the results were not materially altered by exclusion of these two studies, summary RR = 0.96 (95% CI: 0.92-1.01, $I^2 = 78.4\%$). There was evidence of publication bias in the analysis of cruciferous vegetables and type 2 diabetes ($P = 0.006$), which remained significant after exclusion of two apparently outlying studies (18, 53). The association remained non-significant when these two studies were excluded, summary RR = 1.06 (95% CI: 0.98-1.15, $I^2 = 57\%$), although the direction of the association changed.

Table 3. Summary relative risks for subtypes of vegetables and type 2 diabetes, high vs. low-and dose-response analyses

Vegetables subtype	High vs. low analysis						Dose-response analysis						
	<i>n</i>	RR (95% CI)	I ²	<i>P</i> _h	Egger	References	<i>n</i>	Increment	RR (95% CI)	I ²	<i>P</i> _h	Egger	References
Allium vegetables	4	0.89 (0.66-1.20)	79.7	0.002	0.55		4	Per. 100 g/d	0.50 (0.19-1.29)	86.5	<0.0001	0.18	
Broccoli	4	1.06 (0.90-1.12)	64.1	0.04	0.85		4	Per. 100 g/d	1.04 (0.93-1.16)	0	0.72	0.14	
Brussel sprouts	3	1.18 (1.07-1.29)	54.9	0.11	0.48		3	Per. 10 g/d	1.07 (1.03-1.12)	63.9	0.06	0.44	
Cabbage	6	1.10 (1.02-1.19)	50.1	0.08	0.29		6	Per. 100 g/d	1.04 (0.98-1.10)	61.9	0.02	0.47	
Cauliflower	3	1.05 (1.00-1.10)	0	0.72	0.55		3	Per. 100 g/d	1.31 (1.08-1.58)	0	0.98	0.03	
Cruciferous vegetables	8	0.98 (0.87-1.11)	81.1	<0.0001	0.49		8	Per. 100 g/d	0.96 (0.84-1.09)	80.9	<0.0001	0.006	
Green leafy vegetables	8	0.93 (0.85-1.02)	76.5	<0.0001	0.31		8	Per. 100 g/d	0.96 (0.91-1.01)	75.0	<0.0001	0.46	
Kale, mustard and chard greens	3	1.10 (0.99-1.22)	0	0.69	0.90		3	Per. 10 g/d	1.03 (1.00-1.06)	0	0.93	0.72	
Potatoes, boiled	2	0.75 (0.34-1.69)	86.6	0.006	-		2	Per. 100 g/d	0.46 (0.07-3.16)	82.1	0.02	-	
Potatoes, total	8	1.11 (0.95-1.31)	75.0	<0.0001	0.44		8	Per. 100 g/d	1.08 (1.02-1.15)	55.4	0.03	0.06	
Tomatoes	3	0.93 (0.75-1.15)	82.7	0.003	0.59		3	Per. 100 g/d	1.13 (0.78-1.63)	86.2	0.001	0.46	
Yellow vegetables	4	0.77 (0.57-1.03)	92.1	<0.0001	0.50		4	Per. 100 g/d	0.56 (0.30-1.04)	91.0	<0.0001	0.37	

n = number of studies*P*_h = *P*-value for heterogeneity

Subgroup, meta-regression and sensitivity analyses

Subgroup and meta-regression analyses were conducted for fruit and vegetables combined, fruits, vegetables, total potatoes, cruciferous vegetables and green leafy vegetables (**Supplementary Tables 12-17**). In the subgroup analysis for fruit and green leafy vegetables, there was no association across most subgroups and there was no heterogeneity between most subgroups (Supplementary Table 13 and 17). There was some evidence of between subgroup heterogeneity when analyses were stratified by adjustment for family history of type 2 diabetes in the analysis of fruit, with a stronger and significant association among studies with such adjustment compared to studies without such adjustment (Supplementary Table 13).

In the subgroup analysis of cruciferous vegetables intake and type 2 diabetes, there was no significant heterogeneity between most subgroups. However, there was suggestion of heterogeneity when studies were stratified by adjustment for ethnicity a significant increased association in studies with this adjustment, $P_{\text{heterogeneity}} = 0.03$, compared to studies without such adjustment. A significant decreased association among studies with adjustment for waist circumference/WHR, $P_{\text{heterogeneity}} = 0.005$, compared to an increased association (not significant) in studies without such adjustment.

The results for fruits, vegetables, fruit and vegetables combined and cruciferous vegetables, appeared to be robust in sensitivity analyses, when excluding one study at a time in the analysis. When excluding Chen et al. or Mamluk et al. (EPIC-Elderly Greece) from the analysis of green leafy vegetable, there was a borderline significant risk reduction. When excluding Muraki et al. (NHS, NHS II and HPFS) or Montonen et al. from the analysis of total potato, the inverse association was no longer significant (**Supplementary Figures 97**).

DISCUSSION

The findings from this meta-analysis suggest that a high intake of fruit and vegetables are associated with a reduced risk of type 2 diabetes. In the high vs. low analyses, we observed a 6% reduction in RR of type 2 diabetes for intake of both fruit and vegetables combined and for total fruit, but there was no significant association with the intake of vegetables. The associations were not significant in the linear dose-response analyses, however, there was evidence of nonlinearity in several analyses and there were significant 8-12% reductions in risk with a fruit intake between 200-500 g/d and 11-13% reduction in risk with a vegetable intake between 200-300 g/d. Several subtypes of fruits were inversely associated with type 2 diabetes including apples, apples and pears combined, blueberries, grapefruit, grapes and raisins, while cantaloupe, fruit juice, total potato, brussel sprouts, cauliflower, and kale, mustard and chard greens were positively associated with type 2 diabetes risk. No association were observed for bananas, berries, citrus fruits, fruit drinks, oranges, peaches, plums and apricots, prunes, strawberries, allium vegetables, boiled potato, broccoli, cabbage, cruciferous vegetables, green leafy vegetables, tomatoes and yellow vegetables and type 2 diabetes. However, the analyses of these subtypes are based on few studies and the observed associations may therefore be biased due to selective reporting. Further studies on specific subtypes of fruit and vegetables are therefore needed before firm conclusions can be drawn with regard to the association between a number of subtypes of fruits and vegetables and risk of type 2 diabetes. Nonlinear associations were observed for fruits, vegetables, bananas, blueberries, grapes and raisins, allium vegetables, and the risk reduction appeared to be steeper for lower intakes, with a flattening of the curve by increasing intakes.

The findings from this meta-analysis are consistent with some, but not all results from previous meta-analyses. In the current meta-analysis, there was a significant inverse association between high vs. low intake of fruit and vegetables combined and risk of type 2 diabetes based on eight studies, while previous meta-analyses (12, 64) found non-significant associations based on five and seven studies, respectively. There was also a weak inverse association between total fruit intake and type 2 diabetes, consistent with some (36, 52), but not all meta-analyses (12), while the association with total vegetables was not significant, consistent with all previous meta-analyses (12, 36, 64). With regard to specific types of fruits and vegetables the current meta-analysis found stronger inverse associations between intake of apples and pears than a previous meta-analysis (35), but no significant association for

green leafy vegetables which is in contrast to previous meta-analyses (12, 24, 64). A meta-analysis from 2018 (24), based on five studies, found a borderline risk reduction of 13%. However, with three additional studies included in the analysis we observed a non-significant association. Xi et al. (65) investigated the association of 100% fruit juice and type 2 diabetes, and found no significant association, while a significantly increased risk for sugar sweetened fruit juice, which is consistent with our findings. However, these results were based on few studies. For fruit juice our results were consistent with those of Imamura et al. (14) with significantly increased risk per one serving of 250 mL/day. However, the definition of fruit juice was rather heterogeneous in the latter meta-analysis ranging from 100% fruit juice, to fruit juice including nectar, and this may have masked differences between types of fruit-based drinks.

Mechanisms

The observed protective effect of fruit and vegetable intake on the risk of type 2 diabetes may partially be explained by their high content of dietary fiber, antioxidants, vitamins, minerals and phytochemicals, such as polyphenol, carotenoids, anthocyanins, quercetin and glucosinolates. Metabolic inflammation is an important factor contributing in the development of type 2 diabetes, and antioxidant phytochemicals have been found to have anti-inflammatory action(66). Anthocyanins improves glucose metabolism and insulin resistance (31), quercetin have hypoglycemic effects and reduce glucose absorption (67), and isothiocyanates (ITC) derived from glucosinolates have been suggested to have antioxidant and anti-inflammatory properties through the activation of enzymes (41).

Blueberries have a high content of a subclass of flavonoids called anthocyanins while apples and blueberries have a high content of quercetin (24, 67). We found that for the intake of apples, each 100 g/day increment was associated with a 9% decreased risk of type 2 diabetes. For the intake of blueberries and grapes and raisins, each 50 g/day increment was associated with a 40% and 26% decreased risk of type 2 diabetes, respectively.

Glucosinolates, a group of phytochemicals, are abundant in cruciferous vegetables. We found no significant association between intake of cruciferous vegetables and type 2 diabetes based on data from eight studies, but for individual items within this group, such as cabbage, cauliflower and kale, mustard and chard greens there was a significantly increased risk of type 2 diabetes, however, these latter results were based on data from only three studies (NHS, NHS II, HPFS - Ma et al., 2018 (41)). Since the same three studies were the only studies that reported increased type 2 diabetes risk with total cruciferous vegetable

intake, which was counter-acted by null or inverse associations in five other studies leading to an overall null association for total cruciferous vegetable intake, it is possible that selective reporting and/or publication bias or chance may explain the positive associations observed for the specific subtypes of cruciferous vegetables.

Although fruit juice may contain nutrients and polyphenols, such as those that are present in whole fruits, healthy compounds in fruit juice may decrease during the processing (68). Consistent with other meta-analyses we found an increased risk association per 250 mL/day for fruit drinks and fruit juice intake and type 2 diabetes (14, 65). As fruit juice is fluid and have a moderately high glycemic index (69), intake may lead to a rapid increase in blood glucose levels (70). In most countries fruit juice is therefore recommended to consume in moderation (23). Potatoes contain large amounts of rapidly absorbed starch and has high GI and GL (32), which lead to rapid increases in blood glucose and insulin concentrations, and is associated with an increased risk for T2D (71). Both intake of fruit juices and potatoes has been associated with excess weight gain over time and this could also contribute to an increased risk of type 2 diabetes (72).

Limitations

This meta-analysis has some limitations that should be considered when interpreting the results. As this meta-analysis is based on studies from different populations with differences in the 1) amount and range of fruit and vegetable intakes, 2) cooking and preparation methods, 3) dietary patterns, 4) prevalence of confounding factors, 5) rates of type 2 diabetes, and 6) in the detail of the dietary assessment used, some heterogeneity is expected between studies. All of the included studies have adjusted for confounding factors that may impact the results, but not all studies have included the same factors. Most of the included studies adjusted for lifestyle factors such as overweight and obesity, physical activity, smoking, that are common risk factors for type 2 diabetes, as well as other possible confounding factors. In the dose-response analysis, the heterogeneity was low in the analyses of 100% fruit juice, moderate for fruit and vegetables, vegetables, and high for fruits and potatoes. However, when exploring the reason for heterogeneity through subgroup and meta-regression analyses, we found little evidence that the results were materially altered whether these confounding factors were adjusted for or not. Nevertheless, relatively few of the available studies adjusted for other dietary factors and residual confounding can therefore not be completely ruled out. We can also not exclude the possibility that other unknown factors or factors not taken into account, could have affected the observed associations.

Most studies used self-reporting methods, such as FFQ to assess fruit and vegetable intake. Although nearly all studies used FFQs that had shown good validity, measurement errors are known to affect results of epidemiologic studies on diet and health and may have biased the observed effect estimates. However, because we only included prospective studies any measurement errors in the assessment of fruit and vegetable intake would most likely have attenuated the observed associations toward the null. None of the studies included in this meta-analysis made any attempts to correct for measurement errors, however, previous studies on fruit and vegetable intake and coronary heart disease and mortality found risk reductions which were twice as strong after correcting for measurement errors compared to the uncorrected risk estimates (68, 73). Most of the included studies only assessed fruit and vegetables intake at baseline, which does not take into account that people may change their intake over time and the results may therefore be prone to regression dilution bias. Several of the included studies based the assessment of outcome on self-reported type 2 diabetes. However, all studies, except for Mamluk et al. (43) included a validation of self-reported diabetes through record linkage, medication use or supplementary questionnaires.

As with any meta-analysis of published studies we cannot rule out the possibility that publication bias may have affected the observed results. In the current analysis there was some indication of publication bias with Egger's test in the analysis of vegetables, potatoes, and cruciferous vegetables, and there was some evidence of asymmetry in the funnel plot for green leafy vegetables, although Egger's test was not significant in the latter analysis. We found that Egger's test and/or the asymmetries in the funnel plots in most of these cases were explained by one or two outlying studies, which when excluded did not materially alter the results. This is as expected as it is typically the smaller studies (or lack of publication of these), towards the bottom of the funnel plot, which cause publication bias, however, because these potentially "missing" studies are smaller in size they also are given less weight in the meta-analysis and therefore have less impact on the summary estimate. Egger's test was also significant in a few other analyses, however, the limited number of studies ($n = 3-5$) makes the interpretation of those results difficult.

Because there was a limited number of studies in the analyses for many subtypes of fruits and vegetables and because of the potential for selective reporting of significant results, further studies are urgently needed to provide firm conclusions on the association between subtypes of fruits and vegetables and risk of type 2 diabetes.

Strengths

Strengths of the present meta-analysis include the comprehensive search strategy with broad search terms, duplicate screening and assessment of the included studies, large number of studies included, and a large sample size which increases the precision of the effect estimate estimates, and the high study quality of the included studies. We conducted high vs. low, linear- and nonlinear dose-response analyses of fruit and vegetables combined, separately, and across subtypes of fruit and vegetables, and in addition we conducted detailed subgroup and sensitivity analyses. The detailed dose-response analyses allowed us to clarify the strength and shape of the dose-response relationship between fruit and vegetable intake and these outcomes. The associations were consistent when stratified by the different confounding factors in the subgroup analyses, suggesting that these factors did not substantially affect the results. The prospective design of the included studies minimized the chance of the results being affected by recall- and selection bias and the study quality was relatively high across studies. The factors that most frequently contributed to a less than full study quality score was adequate follow-up or lack of reporting of participants lost to follow-up as well as studies not being representative of the general population.

Conclusion

This meta-analysis provides the most comprehensive and up-to-date summary of the available evidence to date and have important public health implications given the current epidemic of adiposity and diabetes globally (74). The study supports existing recommendations to increase the intake of fruit and vegetables, but suggest certain subtypes of fruits including apples, blueberries, grapefruit, grapes and raisins may be particularly beneficial, while potatoes and fruit juice may increase the risk. In addition, some venues for further areas that need clarification have been identified. Any further studies should report in more detail associations between subtypes of fruits and vegetables and type 2 diabetes, adjust for more dietary confounders, and report analyses stratified by other risk factors to better be able to rule out residual confounding. In addition, because most of the available studies have been conducted in Europe, North America and Asia further studies are needed from other geographic regions.

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Author Contributions

Mathilde Elvestad and Rine Elise Halvorsen had full access to the data, conducted the statistical analyses and take responsibility for the integrity of the data and the accuracy of the data analysis.

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Supplementary Table 1. Search strategy in PubMed

1. fruits
2. vegetables
3. fruit
4. vegetable
5. berry
6. berries
7. strawberries
8. blueberries
9. citrus
10. "citrus fruits"
11. orange
12. apples
13. pears
14. banana
15. cruciferae
16. "cruciferous vegetables"
17. broccoli
18. cauliflower
19. cabbages
20. "allium vegetables"
21. onion
22. garlic
23. tomato
24. tomatoes
25. potato
26. "french fries"
27. juice
28. food
29. "food groups"
30. (1 OR 2 OR 3 OR 4 OR 5 OR 6 OR 7 OR 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14 OR 15 OR 16 OR 17 OR 18 OR 19 OR 20 OR 21 OR 22 OR 23 OR 24 OR 25 OR 26 OR 27 OR 28 OR 29)
31. diabetes
32. "case-control"
33. cohort
34. cohorts
35. prospective
36. longitudinal
37. retrospective
38. "follow-up"
39. "cross-sectional"
40. "population-based"
41. "relative risk"
42. "odds ratio"
43. "hazard ratio"
44. "incidence rate ratio"
45. (32 OR 33 OR 34 OR 35 OR 36 OR 37 OR 38 OR 39 OR 40 OR 41 OR 42 OR 43 OR 44)
46. (30 AND 31 AND 45)

Supplementary Table 2. List of studies excluded studies and exclusion reason

Exclusion reason	Reference number
Abstract	(1-4)
Case-control study	(5-8)
Commentary	(9)
Cross-sectional study	(10-48)
Duplicate	(49-61)
Impaired glucose tolerance population	(62)
Meta-analysis	(63-77)
No risk estimates	(78;79)
Not original data	(80)
Not relevant data	(81-182)
Not relevant exposure	(183-408)
Not relevant outcome	(409-459)
Protocol	(460-462)
Review	(463-495)
Substitution of juice with water	(496)

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Supplementary Table 3. Cohort studies of fruit and vegetables and type 2 diabetes

Author, publication year, country	Study name or description	Follow-up period	Study size, gender, age, number of cases	Dietary assessment	Outcome assessment	Exposure	Quantity	RR (95% CI)	Adjustment for confounders
Ford ES et al, 2000, USA	NHANES I Epidemiologic Follow-Up Study	1971-1975 to 1992-1993, 15.8 years follow-up	9665 participants, age 25-74 years, 1018 cases	Single 24-hour dietary recall	Self-report, hospitalization record, death certificate	Fruit and vegetable (total) Fruit and vegetable (men) Fruit and vegetable (women)	0 serv/d 1-4 ≥5 0 serv/d 1-4 ≥5 0 serv/d 1-4 ≥5	1.00 1.01 (0.78, 1.29) 0.79 (0.59, 1.06) 1.00 1.23 (0.76, 1.99) 1.14 (0.67, 1.93) 1.00 0.85 (0.62, 1.16) 0.61 (0.42, 0.88)	Age, sex, smoking, systolic blood pressure, cholesterol concentration, use of antihypertensive medication, recreational exercise, nonrecreational activity, alcohol use, BMI, education
Meyer KA et al, 2000, USA	The Iowa Women's Health Study (IWHS)	1986-1992, 6 years follow-up	35 988 women, age 55-69 years, 1141 cases	Validated FFQ, 127 items	Self-reported, validated by physician/medical records	Total fruit and vegetable Total fruit Total vegetable	18.0 serv/wk 27.0 35.0 44.0 62.0 4.0 serv/wk 8.5 12.0 16.0 23.5 11.0 serv/wk 17.0 22.0 28.5 41.5	1.00 1.00 (0.82, 1.22) 1.12 (0.92, 1.36) 1.21 (0.99, 1.49) 1.05 (0.84, 1.31) 1.00 1.05 (0.87, 1.26) 1.00 (0.82, 1.22) 1.08 (0.88, 1.32) 1.14 (0.93, 1.39) 1.00 1.03 (0.85, 1.24) 0.99 (0.82, 1.21) 1.09 (0.90, 1.34) 1.07 (0.86, 1.32)	Age, total energy intake, BMI, WHR, education, smoking, alcohol intake, physical activity

Knekt P et al, 2002, Finland	The Finnish Mobile Clinic Health Examination Survey (FMCHES)	1966-1972 to 1994, 28 years follow-up	9878 participants, age >15 years, 526 cases	Dietary history interview, >100 items	Linkage to the Social Insurance Institution	Apple	>47 vs. 0 g/d	0.73 (0.57, 0.92)	Sex, age, disease-specific nondietary confounding factors, intakes of vegetables and fruit other than apples, ischemic heart disease, energy intake
Hodge AM et al, 2004, Australia	The Melbourne Collaborative Cohort Study (MCCS)	1990-1994, 4 years follow-up	31 641 participants, age 27-75 years, 365 cases	Self-administered FFQ, 121 items	Self-reported/doctor confirmation	Vegetable Potato Fruit	<3.0 times/d 3.0-4.9 5.0-6.9 ≥7.0 Increase of 1 time/wk <2.0 times/wk 2.0-3.9 4.0-6.4 ≥6.5 Increase of 1 time/wk <2.0 times/d 2.0-3.9 4.0-5.9 >6.0 Increase of 1 time/wk	1.00 1.09 (0.78, 1.54) 0.97 (0.68, 1.39) 0.88 (0.60, 1.28) 0.97 (0.91, 1.03) 1.00 0.84 (0.63, 1.12) 0.82 (0.60, 1.12) 0.98 (0.70, 1.37) 0.99 (0.94, 1.04) 1.00 0.81 (0.59, 1.12) 0.82 (0.58, 1.16) 0.85 (0.59, 1.22) 0.99 (0.93, 1.04)	Age, sex, country of birth, physical activity, family history of diabetes, alcohol, education, weight change in the last 5 years, energy intake, BMI, WHR
Liu S et al, 2004, USA	The Women's Health Study (WHS)	1993-2003, 8.8 years follow-up	38 018 women, age ≥45 years, 1614 cases	Validated semi-quantitative FFQ, 131 items	Self-reported/ADA criteria	All fruits and vegetables All fruits All vegetables	2.54 serv/d 4.13 5.49 7.09 10.16 0.62 serv/d 1.32 1.91 2.62 3.91 1.47 serv/d 2.49 3.40	1.00 1.03 (0.88, 1.20) 0.94 (0.79, 1.11) 0.93 (0.78, 1.10) 1.04 (0.87, 1.25) 1.00 0.93 (0.79, 1.09) 0.87 (0.74, 1.03) 0.94 (0.80, 1.11) 0.97 (0.82, 1.16) 1.00 1.01 (0.86, 1.19) 0.98 (0.83, 1.16)	Age, smoking, total calories, alcohol use, BMI, exercise, history of hypertension, history of high cholesterol and family history of diabetes

						Citrus fruits	4.58 6.84 0.07 serv/d 0.28 0.57 1.00 1.57	0.99 (0.84, 1.18) 1.03 (0.86, 1.23) 1.00 1.06 (0.90, 1.24) 0.90 (0.76, 1.07) 1.14 (0.98, 1.34) 1.07 (0.90, 1.26)	
						Green leafy vegetables	0.14 serv/d 0.35 0.56 0.92 1.42	1.00 0.92 (0.79, 1.08) 0.93 (0.79, 1.09) 0.84 (0.72, 0.99) 0.96 (0.81, 1.13)	
						Cruciferous vegetables	0.13 serv/d 0.21 0.35 0.57 1.00	1.00 0.91 (0.76, 1.09) 0.98 (0.84, 1.14) 0.96 (0.81, 1.14) 0.95 (0.80, 1.12)	
						Dark yellow vegetables	0.07 serv/d 0.2 0.34 0.57 1.00	1.00 0.90 (0.76, 1.07) 0.89 (0.75, 1.07) 0.92 (0.76, 1.11) 0.81 (0.67, 0.98)	
						Potatoes	0.13 serv/d 0.28 0.43 0.56 0.93	1.00 1.03 (0.87, 1.22) 0.97 (0.79, 1.19) 0.96 (0.81, 1.13) 1.02 (0.86, 1.22)	
Schulze M et al, 2004, USA	The Nurses' Health Study II (NHS II)	1991-1999, 7.8 years follow-up	91 249 women, age 24-44 years, 741 cases	Validated semi-quantitative FFQ, 133 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes	Fruit punch	<1/mo 1-4/ 2-6/wk ≥1/d	1.00 0.90 (0.68, 1.18) 1.15 (0.79, 1.66) 2.00 (1.33, 3.03)	Alcohol intake, physical activity, family history of diabetes, smoking, postmenopausal hormone use, oral contraceptive use, intake of cereal fiber, magnesium, trans-fat, and ratio

					criteria (after 1998)				of polyunsaturated to saturated fat; and consumption of sugar-sweetened soft drinks, diet soft drinks, fruit juice, and fruit punch (other than the main exposure, depending on model)
Montonen J et al, 2005, Finland	The Finnish Mobile Clinic Health Examination Survey (FMCHES)	1967-1972 to 1995, 23 years follow-up	4304 participants, age 40-69 years, 383 cases	Dietary history interview, > 100 food items	Linkage to the Social Insurance Institution	Potato	<132 g/d 132-196 197-283 >283	1.00 1.09 (0.82, 1.46) 1.27 (0.94, 1.72) 1.42 (1.02, 1.98)	Age, sex, BMI, energy intake, smoking, family history of diabetes, geographic area
						Vegetables	<42 g/d 42-78 79-130 >130	1.00 0.75 (0.56, 1.00) 0.93 (0.70, 1.22) 0.77 (0.57, 1.03)	
						Yellow and red vegetables	<19 g/d 19-41 42-77 >77	1.00 0.78 (0.59, 1.04) 0.90 (0.68, 1.18) 0.80 (0.60, 1.06)	
						Green vegetables	<11 g/d 11-24 25-43 >43	1.00 0.92 (0.71, 1.21) 0.91 (0.69, 1.20) 0.69 (0.50, 0.93)	
						Other vegetables	<1 g/d 1-3 4-10 >10	1.00 0.97 (0.73, 1.30) 0.94 (0.71, 1.24) 0.79 (0.58, 1.07)	
						Fruits and berries	<33 g/d 33-83 84-156 >156	1.00 0.77 (0.58, 1.02) 0.83 (0.63, 1.10) 0.69 (0.51, 0.92)	
						Fruit	<20 g/d 20-66	1.00 0.89 (0.67, 1.18)	

						Berries	67-138 >138 <4 g/d 4-10 11-20 >20	0.88 (0.66, 1.17) 0.82 (0.61, 1.11) 1.00 0.69 (0.53, 0.92) 0.65 (0.49, 0.87) 0.63 (0.47, 0.85)	
Song Y et al, 2005, USA	The Women's Health Study (WHS)	1993-2003, 8.8 years follow-up	38 018 women, age ≥45 years, 1614 cases	Validated semi-quantitative FFQ, 131 items	Self-reported, validated by supplementary questionnaire and ADA criteria	Broccoli Apples Onions	None ≤1 serv/wk 2-4 ≥5 None ≤1 serv/wk 2-6 ≥1/d None ≤1 serv/wk 2-4 ≥5	1.00 0.95 (0.77, 1.16) 0.94 (0.75, 1.18) 0.95 (0.69, 1.31) 1.00 0.83 (0.70, 0.98) 0.73 (0.60, 0.88) 0.72 (0.55, 0.94) 1.00 1.09 (0.97, 1.22) 1.10 (0.92, 1.33) 1.18 (0.94, 1.48)	Age, BMI, total energy intake, smoking, exercise, alcohol use, history of hypertension, history of high cholesterol, family history of diabetes, fiber intake, glycemic load, magnesium, total fat
Wang L et al, 2006, USA	The Women's Health Study (WHS)	1992-2003, 10.2 years follow-up	35 783 women, age ≥45 years, 1544 cases	Validated semi-quantitative FFQ, 131 food items	Self-reported, validated by supplementary questionnaire and ADA criteria	Tomatoes Tomato juice	None 1-3 serv/mo 1-4 serv/wk ≥5 None 1-3 serv/mo 1 serv/wk ≥2	1.00 0.81 (0.64, 1.03) 0.94 (0.76, 1.17) 0.95 (0.74, 1.22) 1.00 1.00 (0.88, 1.13) 1.11 (0.94, 1.31) 0.93 (0.74, 1.15)	Age, energy, randomized treatment assignment, smoking, alcohol, exercise, family history of diabetes, post-menopause, postmenopausal hormone use, multivitamin use, BMI, history of hypertension, history of hypercholesterolemia
Montonen J et al, 2007, Finland	The Finnish Mobile Clinic Health Examination	1967-1972 to 1994-1995, 12 years follow-up	4284 participants, age 40-69	Dietary history interview, >100 food items	Linkage to the Social Insurance Institution	Sweetened berry juice	0 g/d 7.5 21 51	1.00 0.68 (0.41, 1.14) 0.95 (0.60, 1.49) 1.56 (1.08, 2.26)	Age, sex, BMI, energy intake, smoking, geographic area,

	Survey (FMCHES)		years, 177 cases						physical activity, family history of diabetes, prudent dietary pattern score, conservative pattern score, serum cholesterol, blood pressure, history of infarction, history of angina pectoris, history of cardiac failure
Bazzano LA et al, 2008, USA	The Nurses' Health Study (NHS)	1984-2002, 18 years follow-up	71 346 women, age 38-63 years, 4529 cases	Validated semi-quantitative FFQ, 116 items	Self-reported/ supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998)	Vegetables Fruit and vegetables (fruit juice excluded) Green leafy vegetables Apple, orange, grapefruit/other fruit juices	1.61 serv/d 2.35 3.09 4.25 5.40 3 serv/d increase 2.35 serv/d 3.41 4.47 6.07 7.66 3 serv/d increase 0.25 serv/d 0.49 0.72 1.10 1.48 1 serv/d increase 0.04 serv/d 0.29 0.54 0.94 1.33 1 serv/d increase	1.00 1.00 (0.91, 1.10) 1.02 (0.93, 1.12) 1.08 (0.98, 1.19) 1.05 (0.94, 1.16) 1.04 (0.97, 1.13) 1.00 1.01 (0.92, 1.11) 1.00 (0.91, 1.10) 0.99 (0.89, 1.09) 1.01 (0.90, 1.12) 0.99 (0.94, 1.05) 1.00 1.00 (0.91, 1.10) 1.02 (0.93, 1.11) 0.93 (0.85, 1.03) 0.90 (0.82, 1.00) 0.91 (0.84, 0.98) 1.00 1.21 (1.10, 1.33) 1.29 (1.17, 1.42) 1.25 (1.14, 1.38) 1.35 (1.22, 1.50) 1.18 (1.10, 1.26)	Age, BMI, physical activity, family history of diabetes, postmenopausal hormone use, alcohol, smoking, total energy intake, whole grains, nuts, processed meats, coffee, potatoes, and sugar-sweetened soft drinks

Palmer JR et al, 2008, USA	Black Women's Health Study (BWHS)	1995-2005, 10 years follow-up	43 960 women, age 21-69 years, 2713 cases	Validated FFQ, 68-items	Self-reported, validated by physician	Sweetened fruit drink Orange or grapefruit juice	<1 drink/mo 1-7 2-6 drinks/wk 1 drink/d ≥2 <1 drink/mo 1-7 2-6 drinks/wk 1 drink/d ≥2	1.00 1.08 (0.96, 1.22) 1.08 (0.96, 1.21) 1.17 (1.02, 1.33) 1.31 (1.13, 1.52) 1 0.93 (0.83, 1.05) 0.99 (0.88, 1.11) 0.99 (0.87, 1.14) 1.11 (0.92, 1.35)	Age, family history of diabetes, physical activity, cigarette smoking, years of education, and each of the 2 other types of drinks, intake of red meat, processed meats, cereal fiber, and coffee, and glycemic index
Villegas R et al, 2008, China	The Shanghai Women's Health Study (SWHS)	2000-2002 and 2002-2004, 4.6 years follow-up	64 191 women, age 40-70 years, 1608 cases	In-person interview with FFQ, 77 items	Self-reported/ validated by fasting glucose level (ADA criteria) and/or an oral glucose tolerance test (OGTT) and/or use of hypoglycaemic medication	All vegetables Cruciferous vegetables Green leafy vegetables Yellow vegetables Allium vegetables Tomatoes	121.5 g/d 181.6 236.0 302.6 428.0 5.0 g/d 10.9 17.0 25.8 45.2 28.0 g/d 51.3 70.7 94.1 136.1 0.04 g/d 0.62 2.0 5.6 17.3 2.2 g/d 4.2 6.5 9.8 17.9 6.8 g/d	1.00 0.74 (0.64, 0.87) 0.68 (0.58, 0.80) 0.72 (0.61, 0.84) 0.72 (0.61, 0.85) 1.00 0.79 (0.68, 0.91) 0.69 (0.60, 0.81) 0.60 (0.51, 0.71) 0.72 (0.61, 0.83) 1.00 0.78 (0.68, 0.91) 0.61 (0.52, 0.71) 0.58 (0.49, 0.68) 0.82 (0.71, 0.95) 1.00 0.69 (0.60, 0.80) 0.63 (0.54, 0.73) 0.51 (0.43, 0.60) 0.55 (0.47, 0.64) 1.00 0.79 (0.68, 0.92) 0.70 (0.60, 0.81) 0.70 (0.60, 0.82) 0.69 (0.59, 0.81) 1.00	Age, daily energy intake, meat intake, BMI, WHR, smoking, alcohol consumption, physical activity, income level, education level, occupational status, and hypertension

							17.0 30.3 49.2 88.5 40.7 g/d 66.8 90.9 121.4 181.0 87.0 g/d 170.4 239.4 315.0 483 2.5 g/d 10.0 16.7 25.2 44.4 29.6 g/d 71.3 109.7 149.1 221.0 27.6 g/d 67.2 102.2 142.7 217.6	0.68 (0.59, 0.79) 0.73 (0.63, 0.85) 0.61 (0.52, 0.71) 0.78 (0.67, 0.91) 1.00 0.76 (0.65, 0.88) 0.84 (0.72, 0.98) 0.76 (0.64, 0.89) 0.76 (0.64, 0.89) 1.00 0.76 (0.65, 0.88) 0.79 (0.67, 0.92) 0.87 (0.74, 1.02) 1.05 (0.90, 1.23) 1.00 0.84 (0.72, 0.98) 0.84 (0.72, 0.98) 0.81 (0.69, 0.95) 1.11 (0.95, 1.29) 1.00 0.84 (0.72, 0.98) 0.83 (0.71, 0.97) 0.90 (0.77, 1.05) 1.04 (0.89, 1.21) 1.00 0.77 (0.66, 0.90) 0.68 (0.58, 0.80) 0.85 (0.73, 0.99) 0.90 (0.77, 1.05)	
de Koning L et al, 2011, USA	The Health Professionals Follow-up Study (HPFS)	1986-2006, 20 years follow-up	51 529 men, age 40-75 years,	Validated semi-quantitative FFQ, 131 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American	Fruit punches, lemonades, other noncarbonated fruit drinks	<1 serv/d ≥1 serv/d	1.00	Age, smoking, physical activity, alcohol intake, multivitamin use, family history of type 2 diabetes, high triglycerides (in 1986), high

					Diabetes criteria (after 1998). Questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review				blood pressure, and use of diuretics
Eshak ES et al, 2012, Japan	Japan Public Health Center-based Prospective Study (JPHC)	1990-1995 to 1990-2000, 10 year follow-up	27 585 participants, (12 137 men, 15 448 women), age 40-59 years, 824 cases (484 men, 340 women)	Validated FFQ, 44 items	Self-reported, validated by medical records	100% fruit juice 100% fruit juice Vegetable juice Vegetable juice	Rarely ≤2 times/wk 3-4 times/wk Almost every day Rarely ≤2 times/wk 3-4 times/wk Almost every day Rarely ≤2 times/wk 3-4 times/wk Almost every day Rarely ≤2 times/wk 3-4 times/wk Almost every day	1.00 0.81 (0.65, 1.01) 0.93 (0.65, 1.35) 1.17 (0.69, 2.00) 1.00 0.94 (0.73, 1.21) 0.90 (0.58, 1.40) 1.37 (0.79, 2.37) 1.00 0.84 (0.65, 1.09) 0.81 (0.49, 1.39) 1.27 (0.65, 2.51) 1.00 0.97 (0.69, 1.35) 0.92 (0.47, 1.79) 0.71 (0.28, 1.82)	Age, BMI, family history of diabetes, education, occupation, smoking status, alcohol, history of hypertension, physical activity, coffee, green tea, energy-adjusted intakes of dietary magnesium, calcium, vitamin D, rice and total dietary fiber, and total energy intake
Kurotani K et al, 2012, Japan	Japan Public Health Center-based Prospective Study (JPHC)	1995-1998 to 2000-2003, 5 years follow-up	48 437 men and women (21 269 men, 27 168 women), age 45-75 years, 896 cases (530 men, 366 women)	Validated self-administered FFQ, 147 items	Self-reported, validated by medical records	Total vegetable and fruit intake (men) Total vegetable and fruit intake (women) Total vegetable intake (men)	146 g/d 273.1 414.1 686.8 209.7 g/d 365.7 532.9 858.7 75.2 g/d 141.7 213.1	1.00 0.85 (0.66, 1.10) 1.08 (0.83, 1.40) 0.93 (0.67, 1.29) 1.00 0.94 (0.69, 1.28) 0.79 (0.56, 1.11) 1.04 (0.69, 1.55) 1.00 0.93 (0.73, 1.19) 0.92 (0.70, 1.20)	Age, public health centre area, BMI, smoking alcohol consumption, leisure-time activity, history of hypertension, coffee consumption, family history of diabetes, magnesium intake,

						Total vegetable intake (women)	355.4 99.5 g/d 172.7 252.5 406.9	0.81 (0.59, 1.13) 1.00 1.04 (0.77, 1.41) 0.76 (0.54, 1.08) 0.99 (0.66, 1.47)	calcium intake, energy intake
						Total fruit intake (men)	36.4 g/d 113.1 191.6 362.4	1.00 0.94 (0.73, 1.19) 0.91 (0.70, 1.18) 0.94 (0.71, 1.26)	
						Total fruit intake (women)	74.4 g/d 166.3 272.2 487.1	1.00 0.73 (0.53, 1.00) 0.96 (0.70, 1.32) 1.04 (0.73, 1.48)	
						Total green and yellow vegetables (men)	24.7 g/d 58.8 94.6 172.4	1.00 0.82 (0.64, 1.06) 1.05 (0.82, 1.36) 0.90 (0.66, 1.22)	
						Total green and yellow vegetables (women)	35.4 g/d 70.9 113.2 197.5	1.00 1.06 (0.79, 1.42) 0.84 (0.61, 1.17) 0.89 (0.61, 1.29)	
						Green leafy vegetables (men)	4.5 g/d 11.8 22.7 47.2	1.00 0.92 (0.72, 1.17) 0.88 (0.68, 1.14) 0.83 (0.62, 1.12)	
						Green leafy vegetables (women)	7.4 g/d 16.7 29.5 57.5	1.00 0.81 (0.60, 1.10) 0.88 (0.65, 1.20) 0.81 (0.57, 1.16)	
						Cruciferous vegetables (men)	17.6 g/d 37.3 60.8 103.9	1.00 1.02 (0.80, 1.30) 0.94 (0.73, 1.22) 0.78 (0.58, 1.06)	
						Cruciferous vegetables (women)	24.0 g/d 47.6 72.5 119.8	1.00 1.09 (0.80, 1.48) 1.13 (0.82, 1.55) 1.10 (0.77, 1.57)	

						Citrus fruits (men)	7.2 g/d 46.5 79.3 165.4	1.00 1.00 (0.79, 1.28) 0.85 (0.65, 1.10) 1.04 (0.79, 1.36)	
						Citrus fruits (women)	19.1 g/d 66.0 114.8 248.9	1.00 0.91 (0.67, 1.23) 0.92 (0.67, 1.27) 1.14 (0.82, 1.58)	
Cooper AJ et al, 2013, UK	The EPIC-InterAct Study	1991-2007, 11 years follow-up	Sub-cohort: 14 800 participants, age 40-79 years, 10 821 cases	Country-specific, validated dietary questionnaires	Self-reported/registers/drug registers/hospital admissions/mortality data	Total fruit and vegetables	<235.7 g/d ≥235.7 - <369.1 ≥369.1 - <544.8 ≥544.8	1.00 0.92 (0.83, 1.03) 0.93 (0.84, 1.03) 0.90 (0.80, 1.01)	Country, age, centre, sex, education, BMI, physical activity, smoking, total energy intake and alcohol intake Total fruit: additionally adjusted for total vegetable intake Citrus-and non-citrus fruit: adjusted for other fruit sub-types Non-citrus fruit: Umea (Sweden) excluded (no info) Total vegetables: additionally adjusted for total fruit intake Green leafy vegetables,
						Total fruit	<103.7 g/d ≥103.7 - <193.4 ≥193.4 - <315.9 ≥315.9	1.00 0.92 (0.83, 1.03) 0.94 (0.83, 1.05) 0.89 (0.76, 1.04)	
						Citrus fruit	<10.1 g/d ≥10.1 - <35.9 ≥35.9 - <79.4 ≥79.4	1.00 0.96 (0.86, 1.07) 1.00 (0.90, 1.10) 1.01 (0.86, 1.19)	
						Non-citrus fruit	<53.0 g/d ≥53.0 - <120.9 ≥120.9 - <213.5 ≥213.5	1.00 1.02 (0.92, 1.13) 0.97 (0.87, 1.08) 0.94 (0.79, 1.13)	
						Total vegetable	<100.5 g/d ≥100.5 - <154.8 ≥154.8 - <237.6 ≥237.6	1.00 0.92 (0.84, 1.01) 0.93 (0.83, 1.05) 0.94 (0.84, 1.05)	
						Green leafy vegetables	<3.2 g/d ≥3.2 - <14.1 ≥14.1 - <37.7 ≥37.7	1.00 0.74 (0.65, 0.84) 0.75 (0.65, 0.86) 0.84 (0.65, 1.07)	
						Fruiting vegetables	<28.6 g/d ≥28.6 - <50.5 ≥50.5 - <87.1 ≥87.1	1.00 0.94 (0.86, 1.04) 0.96 (0.86, 1.06) 0.97 (0.85, 1.12)	
						Root vegetables	<3.9 g/d	1.00	

							≥ 3.9 - < 11.1 ≥ 11.1 - < 27.3 ≥ 27.3 < 1.5 g/d ≥ 1.5 - < 8.5 ≥ 8.5 - < 21.4 ≥ 21.4 < 2.6 g/d ≥ 2.6 - < 7.0 ≥ 7.0 - < 17.7 ≥ 17.7 < 0.2 g/d ≥ 0.2 - < 3.8 ≥ 3.8 - < 9.8 ≥ 9.8 < 3.4 g/d ≥ 3.4 - < 10.2 ≥ 10.2 - < 23.0 ≥ 23.0	0.98 (0.88, 1.08) 0.85 (0.76, 0.95) 0.87 (0.77, 0.99) 1.00 0.94 (0.74, 1.19) 0.93 (0.80, 1.07) 0.90 (0.75, 1.09) 1.00 0.94 (0.75, 1.18) 0.88 (0.71, 1.10) 0.92 (0.63, 1.33) 1.00 0.91 (0.70, 1.18) 0.78 (0.68, 0.91) 0.82 (0.63, 1.07) 1.00 1.01 (0.87, 1.19) 0.90 (0.78, 1.04) 0.96 (0.76, 1.22)	cabbages, onion and garlic, stalk vegetables and sprouts, other vegetables: Umea (Sweden) excluded (no info) Green leafy vegetable: Denmark excluded from analysis as there was not enough information to calculate HRs and 95% CIs Onion and garlic: France excluded (no info)
Fagherazzi G et al, 2013, France	Etude Epidémiologique auprès des femmes de la Mutuelle Générale de l'Éducation Nationale—European Prospective Investigation into Cancer and Nutrition cohort (E3N)	1993-2007, 14 years follow-up	66 118 women, age 40-65 years, 1369 cases	Validated diet-history questionnaire, 208 items	Self-reported/ a diabetes diet plan/ the use of diabetic drugs/ a hospitalization for diabetes, validated by drug registries or supplementary questionnaire	100% fruit juice	Non-consumers < 180 180–447 mL/wk 448–967 mL/wk > 967 mL/wk	1.00 0.90 (0.76, 1.07) 0.95 (0.81, 1.12) 1.18 (1.01, 1.38) 0.93 (0.78, 1.10)	Age, years of education, smoking, physical activity, hypertension, hypercholesterolemia, use of hormone replacement therapy, family history of diabetes, self-reported use of antidiabetic drugs, alcohol, omega-3 fatty acid intake, carbohydrate, coffee, fruit and vegetables, and processed-meat

									consumption, dietary pattern, total energy intake and BMI
Jacques PF et al, 2013, USA	Framingham Heart Study Offspring (FHSO)	1991-2008, 11.9 years follow-up	2 915 participants, age 10-70 years, 308 cases	Semi-quantitative FFQ, 145 items	Fasting glucose concentrations and/or a medical and medication use history	Apples and pears Banana	<138 g/wk 138-620 621-896 ≥897 <114 g/wk 114-512 513-740 ≥741	1.00 0.99 (0.67, 1.46) 0.63 (0.31, 1.26) 0.73 (0.35, 1.56) 1.00 1.16 (0.78, 1.73) 1.06 (0.59, 1.89) 1.36 (0.76, 2.43)	Sex, time-dependent variables age, cardiovascular disease, current smoker, BMI, cumulative mean energy intake
Muraki I et al, 2013, USA	The Nurses' Health Study (NHS)	1984-2008, 21 years follow-up	66 105 women, age 30-55 years, 6358 cases	Validated semi-quantitative FFQ, 116 items	Self-reported/supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). Questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review	Total whole fruit consumption Grapes and raisins Peaches, plums and apricots Prunes Bananas	<4 serv/wk 5-6 1 serv/d 2 ≥3 Every 3 serv/wk <1 serv/mo 1-3 1 serv/wk 2-4 ≥5 Every 3 serv/wk <1 serv/mo 1-3 1 serv/wk 2-4 ≥5 Every 3 serv/wk <1 serv/mo 1-3 1 serv/wk 2-≥5 Every 3 serv/wk <1 serv/mo 1-3	1.00 0.92 (0.85, 0.99) 0.96 (0.80, 0.93) 0.86 (0.79, 0.93) 0.90 (0.81, 0.99) 0.98, 0.96, 1.00) 1.00 0.91 (0.86, 0.97) 0.88 (0.80, 0.95) 0.80 (0.72, 0.88) 0.77 (0.64, 0.92) 0.84 (0.78, 0.91) 1.00 0.99 (0.93, 1.07) 1.00 (0.92, 1.08) 1.04 (0.94, 1.14) 0.92 (0.78, 1.09) 1.00 (0.93, 1.07) 1.00 0.99 (0.92, 1.07) 0.86 (0.73, 1.02) 0.89 (0.75, 1.06) 0.87 (0.74, 1.03) 1.00 1.08 (0.98, 1.19)	Age, ethnicity, BMI, smoking, multivitamin use, physical activity, family history of diabetes, menopausal status and post-menopausal hormone use, oral contraceptive use, total energy intake, fruit juice consumption and modified alternate healthy eating index score. Individual fruit consumption was mutually adjusted

						1 serv/wk	1.05 (0.95, 1.17)	
						2-4	1.04 (0.94, 1.15)	
						≥5	1.08 (0.96, 1.21)	
						Every 3 serv/wk	1.01 (0.96, 1.06)	
					Cantaloupe	<1 serv/mo	1.00	
						1-3	1.00 (0.93, 1.08)	
						1 serv/wk	1.06 (0.98, 1.15)	
						2-≥5	1.07 (0.96, 1.19)	
						Every 3 serv/wk	1.08 (0.98, 1.18)	
					Apples and pears	<1 serv/mo	1.00	
						1-3	0.94 (0.84, 1.04)	
						1 serv/wk	0.94 (0.84, 1.05)	
						2-4	0.85 (0.77, 0.95)	
						≥5	0.82 (0.73, 0.92)	
						Every 3 serv/wk	0.91 (0.87, 0.95)	
					Oranges	<1 serv/mo	1.00	
						1-3	0.96 (0.89, 1.04)	
						1 serv/wk	1.03 (0.94, 1.13)	
						2-4	0.96 (0.87, 1.05)	
						≥5	1.03 (0.92, 1.15)	
						Every 3 serv/wk	1.00 (0.95, 1.06)	
					Grapefruit	<1 serv/mo	1.00	
						1-3	0.91 (0.85, 0.97)	
						1 serv/wk	0.95 (0.88, 1.03)	
						2-4	0.88 (0.80, 0.96)	
						≥5	0.86 (0.75, 0.98)	
						Every 3 serv/wk	0.92 (0.87, 0.98)	
					Total berries	<1 serv/mo	1.00	
						1-3	0.93 (0.86, 1.01)	
						1 serv/wk	0.95 (0.87, 1.03)	
						2-4	0.91 (0.82, 0.99)	
						≥5	0.96 (0.83, 1.11)	
						Every 3 serv/wk	0.97 (0.91, 1.03)	
					Strawberries	<1 serv/mo	1.00	
						1-3	0.94 (0.87, 1.01)	
						1 serv/wk	0.98 (0.90, 1.07)	
						2-4	0.87 (0.77, 0.98)	

						Blueberries	≥ 5 Every 3 serv/wk <1 serv/mo 1-3 1 serv/wk 2- ≥ 5	0.99 (0.79, 1.25) 0.94 (0.85, 1.03) 1.00 0.90 (0.85, 0.96) 0.89 (0.82, 0.98) 0.82 (0.69, 0.98)	
						Fruit juice	Every 3 serv/wk <1 serv/wk 1 2-4 5-6 ≥ 1 serv/d Per 3 serv/wk	0.77 (0.66, 0.91) 1.00 1.09 (0.98, 1.21) 1.13 (1.03, 1.23) 1.13 (1.03, 1.24) 1.21 (1.12, 1.31) 1.07 (1.04, 1.11)	
Muraki I et al, 2013, USA	The Nurses' Health Study II (NHS II)	1991-2009, 20 years follow-up	85 104 women, age 25-42 years, 3153 cases	Validated semi-quantitative FFQ, 131 items	Self-reported/ supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998)	Total whole fruit consumption	<4 serv/wk 5-6 1 serv/d 2 ≥ 3 Every 3 serv/wk	1.00 0.86 (0.77, 0.95) 0.84 (0.76, 0.94) 0.88 (0.78, 0.98) 0.92 (0.78, 1.08) 0.99 (0.96, 1.00)	Age, ethnicity, BMI, smoking, multivitamin use, physical activity, family history of diabetes, menopausal status and post-menopausal hormone use, oral contraceptive use, total energy intake, fruit juice consumption and modified alternate healthy eating index score. Individual fruit consumption was mutually adjusted
					Grapes and raisins	<1 serv/mo 1-3 1 serv/wk 2-4 ≥ 5 Every 3 serv/wk	1.00 0.81 (0.74, 0.88) 0.85 (0.75, 0.96) 0.83 (0.72, 0.97) 0.88 (0.66, 1.16) 0.91 (0.81, 1.02)		
					Peaches, plums and apricots	<1 serv/mo 1-3 1 serv/wk 2-4 ≥ 5 Every 3 serv/wk	1.00 1.07 (0.97, 1.18) 1.03 (0.91, 1.16) 0.99 (0.86, 1.14) 1.01 (0.78, 1.31) 0.97 (0.87, 1.08)		
					Prunes	<1 serv/mo 1-3 1 serv/wk 2- ≥ 5 Every 3 serv/wk	1.00 0.85 (0.75, 0.96) 1.00 (0.77, 1.31) 1.16 (0.88, 1.53) 1.03 (0.79, 1.34)		
					Bananas	<1 serv/mo	1.00		

						1-3	0.95 (0.84, 1.07)	
						1 serv/wk	0.95 (0.83, 1.08)	
						2-4	0.82 (0.72, 0.94)	
						≥5	0.80 (0.67, 0.94)	
						Every 3 serv/wk	0.87 (0.81, 0.94)	
					Cantaloupe	<1 serv/mo	1.00	
						1-3	0.99 (0.90, 1.09)	
						1 serv/wk	1.05 (0.94, 1.17)	
						2-≥5	1.11 (0.94, 1.30)	
						Every 3 serv/wk	1.12 (0.96, 1.32)	
					Apples and pears	<1 serv/mo	1.00	
						1-3	0.83 (0.72, 0.95)	
						1 serv/wk	0.83 (0.72, 0.96)	
						2-4	0.79 (0.68, 0.91)	
						≥5	0.76 (0.64, 0.90)	
						Every 3 serv/wk	0.92 (0.86, 0.99)	
					Oranges	<1 serv/mo	1.00	
						1-3	0.94 (0.85, 1.04)	
						1 serv/wk	0.93 (0.82, 1.05)	
						2-4	0.93 (0.81, 1.07)	
						≥5	0.97 (0.78, 1.21)	
						Every 3 serv/wk	0.99 (0.89, 1.09)	
					Grapefruit	<1 serv/mo	1.00	
						1-3	1.00 (0.91, 1.09)	
						1 serv/wk	1.06 (0.94, 1.20)	
						2-4	0.97 (0.83, 1.14)	
						≥5	0.91 (0.69, 1.21)	
						Every 3 serv/wk	0.97 (0.86, 1.09)	
					Total berries	<1 serv/mo	1.00	
						1-3	0.93 (0.84, 1.05)	
						1 serv/wk	0.93 (0.82, 1.05)	
						2-4	0.92 (0.80, 1.05)	
						≥5	1.03 (0.86, 1.24)	
						Every 3 serv/wk	1.02 (0.94, 1.11)	
					Strawberries	<1 serv/mo	1.00	
						1-3	0.97 (0.87, 1.08)	
						1 serv/wk	1.01 (0.89, 1.15)	

						Blueberries	2-4 ≥5 Every 3 serv/wk <1 serv/mo 1-3 1 serv/wk 2-≥5	1.09 (0.93, 1.27) 1.08 (0.81, 1.43) 1.09 (0.97, 1.22) 1.00 0.83 (0.76, 0.91) 0.90 (0.79, 1.04) 0.69 (0.55, 0.87)	
						Fruit juice	Every 3 serv/wk <1 serv/wk 1 2-4 5-6 ≥1 serv/d Per 3 serv/wk	0.67 (0.54, 0.83) 1.00 0.92 (0.81, 1.05) 0.97 (0.87, 1.00) 0.97 (0.86, 1.09) 1.14 (1.02, 1.27) 1.07 (1.02, 1.11)	
Muraki I et al, 2013, USA	The Health Professionals Follow-up Study (HPFS)	1986-2008, 22 years follow-up	36 173 men, age 40-75 years, 2687 cases	Validated semi-quantitative FFQ, 131 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). Questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review	Total whole fruit consumption	<4 serv/wk 5-6 1 serv/d 2 ≥3 Every 3 serv/wk <1 serv/mo	1.00 1.00 (0.88, 1.12) 0.92 (0.82, 1.03) 0.89 (0.79, 1.01) 0.90 (0.78, 1.04) 0.98 (0.95, 1.00) 1.00	Age, ethnicity, BMI, smoking, multivitamin use, physical activity, family history of diabetes, total energy intake, fruit juice consumption and modified alternate healthy eating index score. Individual fruit consumption was mutually adjusted
					Grapes and raisins	1-3 1 serv/wk 2-4 ≥5 Every 3 serv/wk <1 serv/mo	0.95 (0.87, 1.05) 0.95 (0.84, 1.08) 0.87 (0.76, 1.01) 0.84 (0.69, 1.04) 0.91 (0.82, 0.99) 1.00		
					Peaches, plums and apricots	1-3 1 serv/wk 2-4 ≥5 Every 3 serv/wk <1 serv/mo	0.98 (0.88, 1.08) 1.03 (0.90, 1.18) 0.88 (0.75, 1.04) 0.75 (0.55, 1.04) 0.87 (0.77, 0.99) 1.00		
					Prunes	1-3 1 serv/wk 2-≥5 Every 3 serv/wk	0.92 (0.80, 1.06) 0.83 (0.63, 1.10) 0.86 (0.66, 1.12) 0.82 (0.63, 1.07)		

						Bananas	<1 serv/mo	1.00	
							1-3	1.09 (0.95, 1.25)	
							1 serv/wk	1.01 (0.87, 1.18)	
							2-4	0.93 (0.80, 1.07)	
							≥5	0.86 (0.73, 1.01)	
							Every 3 serv/wk	0.89 (0.83, 0.95)	
						Cantaloupe	<1 serv/mo	1.00	
							1-3	1.15 (1.03, 1.27)	
							1 serv/wk	1.17 (1.03, 1.34)	
							2-≥5	1.19 (1.01, 1.40)	
							Every 3 serv/wk	1.14 (0.98, 1.34)	
						Apples and pears	<1 serv/mo	1.00	
							1-3	0.91 (0.78, 1.06)	
							1 serv/wk	0.98 (0.83, 1.16)	
							2-4	0.91 (0.77, 1.07)	
							≥5	0.93 (0.78, 1.11)	
							Every 3 serv/wk	0.98 (0.92, 1.06)	
						Oranges	<1 serv/mo	1.00	
							1-3	0.89 (0.79, 1.01)	
							1 serv/wk	0.91 (0.79, 1.04)	
							2-4	0.89 (0.78, 1.03)	
							≥5	0.89 (0.76, 1.05)	
							Every 3 serv/wk	0.97 (0.90, 1.05)	
						Grapefruit	<1 serv/mo	1.00	
							1-3	1.03 (0.93, 1.14)	
							1 serv/wk	1.09 (0.96, 1.24)	
							2-4	0.93 (0.81, 1.06)	
							≥5	1.08 (0.90, 1.30)	
							Every 3 serv/wk	0.99 (0.91, 1.08)	
						Total berries	<1 serv/mo	1.00	
							1-3	0.93 (0.83, 1.03)	
							1 serv/wk	0.95 (0.84, 1.07)	
							2-4	0.94 (0.81, 1.09)	
							≥5	1.22 (0.98, 1.52)	
							Per 3 serv/wk	1.24 (1.08, 1.42)	
						Strawberries	<1 serv/mo	1.00	
							1-3	0.95 (0.85, 1.05)	

						Blueberries	1 serv/wk 2-4 ≥5 Every 3 serv/wk <1 serv/mo 1-3	0.98 (0.85, 1.13) 1.16 (0.95, 1.42) 1.51 (1.00, 2.28) 1.22 (1.03, 1.43) 1.00 0.94 (0.85, 1.03)	
						Fruit juice	1 serv/wk 2-≥5 Every 3 serv/wk <1 serv/wk 1 2-4 5-6 ≥1 serv/d Per 3 serv/wk	0.96 (0.80, 1.15) 0.74 (0.55, 1.00) 0.75 (0.58, 0.98) 1.00 1.07 (0.91, 1.26) 0.99 (0.86, 1.13) 1.05 (0.92, 1.20) 1.13 (1.01, 1.27) 1.06 (1.01, 1.11)	
Romaguera D et al, 2013, UK	The EPIC-InterAct Study	1991-2007, 11.72 years follow-up	Sub-cohort: 15 374 participants, age 40-79, 11 684 cases	Country-specific validated dietary questionnaires	Self-report, validated by linkage to primary-care registers, secondary-care registers, medication use (drug registers), hospital admissions and mortality data	Juices and nectar	0.0 g/d 17.1 100.0 338.3	1.00 0.97 (0.86, 1.10) 1.04 (0.96, 1.13) 1.06 (0.90, 1.25)	Sex, educational level, physical activity, smoking status, alcohol consumption; juices and total soft drinks were mutually adjusted; sugar-sweetened and artificially sweetened soft drinks were also mutually adjusted plus adjustment for juice consumption, energy intake and BMI
Mursu J et al, 2014, Finland	Kuopio Ischaemic Heart Disease Risk Factor Study (KIHD)	1984-1989 to 2006-2008, 19.3 years follow-up	2332 men, age 42-60 years, 432 cases	Instructed 4-day food recording	Self-reported, diabetes register, blood glucose	Total fruit and vegetables Fruit	90.94 g/d 192.50 284.80 469.26 0.71 g/d	1.00 0.79 (0.60, 1.03) 0.89 (0.68, 1.16) 0.76 (0.57, 1.02) 1.00	Age, examination years, BMI, WHR, smoking, education, leisure time physical activity,

					measurements and OGTT		33.82 99.13 241.25 0.00 g/d 14.68 41.04 108.55 0.00 g/d 39.03 128.21 387.25 36.29 g/d 82.73 128.18 231.92 0.00 g/d 3.76 14.28 43.95	0.95 (0.72, 1.25) 0.87 (0.66, 1.15) 0.98 (0.75, 1.29) 1.00 1.15 (0.90, 1.47) 0.89 (0.68, 1.17) 0.65 (0.49, 0.88) 1.00 1.07 (0.82, 1.39) 1.03 (0.78, 1.34) 0.99 (0.74, 1.31) 1.00 0.90 (0.69, 1.17) 0.92 (0.70, 1.20) 0.81 (0.61, 1.07) 1.00 1.15 (0.89, 1.49) 0.89 (0.67, 1.76) 0.79 (0.59, 1.05)	family history of diabetes, intake of energy, alcohol
Qiao Y et al, 2014, USA	The Women's Health Initiative (WHI)	1993-2005, 7.6 years follow-up	154 493 participants, age 50-79 years, 10 307 cases	Validated FFQ, 122 items	Self-reported, validated by medication and laboratory data	Vegetables	<3.01 serv/d ≥3.01	1.00 1.10 (0.96, 1.26)	Age, education, cigarette smoking, BMI, WHR, physical activity, log (daily energy intake), family history of diabetes, study arms and hormone therapy use
Lacoppidan SA et al, 2015, Denmark	The Diet, Cancer, and Health cohort (DCH)	1993-2011, 15.3 years follow-up	55 060 participants (28 953 women, 26 107 men), age 50-64 years, 7366 cases (3269	Validated FFQ, 192-items	Linkage to National Diabetes Registry	Apples and pears (women) Apples and pears (men)	<70.99 g/d ≥71 <55.99 g/d ≥56	1.00 1.03 (0.96, 1.11) 1.00 0.97 (0.91, 1.04)	Age, schooling level, participation in sports, smoking status, alcohol intake, red and processed meat, total energy intake,

			women, 4097 men)						BMI and waist circumference
Muraki I et al, 2016, USA	The Nurses' Health Study (NHS)	1984-2010, 22.389 years follow-up	70 773 women, age 30-55 years, 7436 cases	Validated semi- quantitative FFQ, 116 items	Self-reported/ supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). Questionnaire -confirmed diagnosis of T2D was reconfirmed by medical record review	Potatoes	<1 serv/wk 1 2-4 5-6 ≥7 Every 3 serv/wk	1.00 1.08 (0.93, 1.26) 1.15 (1.00, 1.32) 1.22 (1.05, 1.40) 1.27 (1.04, 1.56) 1.08 (1.04, 1.13)	Age, ethnicity, smoking status, alcohol intake, multivitamin use, physical activity, a family history of diabetes, menopausal status and postmenopausal hormone use, oral contraceptive use, total energy intake, modified aHEI score and baseline BMI
Muraki I et al, 2016, USA	The Nurses' Health Study II (NHS II)	1991-2011, 18.353 years follow-up	87 739 women, age 25-42 years, 4621 cases	Validated semi- quantitative FFQ, 131 items	Self-reported/ supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998).	Potatoes	<1 serv/wk 1 2-4 5-6 ≥7 Every 3 serv/wk	1.00 0.95 (0.78, 1.16) 0.99 (0.82, 1.19) 1.09 (0.90, 1.31) 1.38 (1.08, 1.76) 1.12 (1.05, 1.18)	Age, ethnicity, smoking status, alcohol intake, multivitamin use, physical activity, family history of diabetes, menopausal status and postmenopausal hormone use, oral contraceptive use, total energy intake, modified aHEI score and baseline BMI

Muraki I et al, 2016, USA	The Health Professionals Follow-up Study (HPFS)	1986-2010, 19.501 years follow-up	40 669 men, age 40-75 years, 3305 cases	Validated semi-quantitative FFQ, 131 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). Questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review	Potatoes	<1 serv/wk 1 2-4 5-6 ≥7 Every 3 serv/wk	1.00 0.94 (0.76, 1.17) 1.03 (0.85, 1.24) 1.09 (0.89, 1.32) 1.38 (1.07, 1.78) 1.10 (1.03, 1.17)	Age, ethnicity, smoking status, alcohol intake, multivitamin use, physical activity, a family history of diabetes, total energy intake, modified aHEI score and baseline BMI
Muraki et al, 2016, USA	The Nurses' Health Study (NHS) The Nurses' Health Study II (NHS II) The Health Professionals Follow-up Study (HPFS)	1984-2010, 21 years follow-up 1991-2011, 18.353 years follow-up 1986-2010, 19.501 years follow-up	70 773 women, age 30-55 years, 7436 cases 87 739 women, age 25-42 years, 4621 cases 40 669 men, age 40-75 years, 3305 cases	Validated semi-quantitative FFQ, 116 items Validated semi-quantitative FFQ, 131 items Validated semi-quantitative FFQ, 131 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). In NHS and HPFS, questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review	Baked, boiled or mashed potatoes (pooled) French fries (pooled)	Almost never to 1-3 serv/mo 1 serv/wk 2-4 ≥5 Every 3 serv/wk Almost never 1-3 serv/mo 1 serv/wk 2-4 ≥5 Every 3 serv/wk	1.00 1.02 (0.95, 1.09) 1.03 (0.96, 1.10) 1.08 (1.00, 1.16) 1.04 (1.01, 1.08) 1.00 1.11 (1.06, 1.17) 1.17 (1.11, 1.24) 1.26 (1.18, 1.35) 1.32 (1.13, 1.55) 1.19 (1.13, 1.25)	Age, ethnicity, smoking status, alcohol intake, multivitamin use, physical activity, a family history of diabetes, menopausal status, postmenopausal hormone use, oral contraceptive use (NHS and NHS II), total energy intake, modified aHEI score, baked, boiled or mashed potatoes (for french fries), and french fries (for baked, boiled, or

		Median follow-up 20.02							mashed potatoes) and baseline BMI
Alperet DJ et al, 2017, Singapore	The Singapore Chinese Health Study (SCHS)	1993-2010, 10.89 years follow-up	45 411 participants, age 45-74 years, 5207 cases	Validated semi-quantitative FFQ, 165 items	Self-reported, validated by linkage with a nationwide hospital-based discharge database and supplementary questionnaire	Total whole-fruit (all)	0.1 serv/wk 1.5 3.0 5.5 9.6 16.6 25.3 Per 3 serv/wk	1.00 1.10 (0.92, 1.30) 1.15 (1.00, 1.32) 1.11 (0.98, 1.27) 1.06 (0.93, 1.21) 1.08 (0.93, 1.25) 1.08 (0.91, 1.27) 0.99 (0.98, 1.01)	Age, sex, dialect group, year of baseline interview, total daily energy intake, physical activity, education, smoking, alcohol intake, BMI, total vegetable intake, unsweetened soy intake, saturated fat intake, dairy intake, soft drink consumption, coffee intake, black and green tea intake, fruit- and vegetable-juice intake, mutually adjusted for individual fruits Juice: adjusted for all the above, included dietary fiber, but not adjusted for fruit- and vegetable-juice intake
						Total whole-fruit (men)	0.0 serv/wk 1.5 3.0 5.5 9.7 16.7 25.5 Per 3 serv/wk	1.00 1.01 (0.75, 1.35) 1.24 (0.99, 1.56) 1.25 (1.00, 1.54) 1.16 (0.94, 1.43) 1.24 (0.99, 1.56) 1.33 (1.04, 1.71) 1.01 (0.99, 1.03)	
						Total whole-fruit (women)	0.2 serv/wk 1.5 3.0 5.5 9.6 16.6 25.1 Per 3 serv/wk	1.00 1.14 (0.92, 1.41) 1.11 (0.93, 1.32) 1.04 (0.88, 1.23) 1.00 (0.85, 1.18) 0.97 (0.81, 1.17) 0.88 (0.71, 1.11) 0.97 (0.96, 0.99)	
						Temperate fruit (all) <i>apples, pears, apricots, peaches, grapes, persimmon</i>	0.0 serv/wk 0.5 1.3 2.9 5.0 8.1	1.00 0.95 (0.85, 1.06) 0.98 (0.88, 1.09) 0.96 (0.87, 1.05) 0.94 (0.83, 1.05) 0.86 (0.77, 0.97)	
						Temperate fruit (men)	0.0 serv/wk 0.5 1.3 2.9	1.00 0.99 (0.85, 1.16) 1.02 (0.87, 1.19)	

						5.0	1.03 (0.89, 1.19)	
						8.1	0.99 (0.83, 1.17)	
					Temperate fruit (women)	0.0 serv/wk	0.97 (0.82, 1.16)	
						0.5	1.00	
						1.4	0.91 (0.78, 1.05)	
						3.0	0.95 (0.82, 1.09)	
						5.1	0.90 (0.79, 1.03)	
					Apple (all)	8.1	0.89 (0.76, 1.04)	
						0.0 serv/wk	0.79 (0.67, 0.92)	
						0.5	1.00	
						1.0	0.97 (0.89, 1.05)	
						2.5	0.93 (0.85, 1.02)	
						5.0	0.93 (0.86, 1.01)	
					Apple (men)	7.0	0.90 (0.79, 1.03)	
						Per 3 serv/wk	0.82 (0.74, 0.92)	
						0.0 serv/wk	0.93 (0.90, 0.97)	
						0.5	1.00	
						1.0	0.93 (0.81, 1.06)	
						2.5	0.98 (0.85, 1.12)	
						5.0	0.94 (0.83, 1.07)	
					Apple (women)	7.0	0.94 (0.77, 1.14)	
						Per 3 serv/wk	0.95 (0.80, 1.13)	
						0.0 serv/wk	0.98 (0.92, 1.04)	
						0.5	1.00	
						1.0	0.99 (0.88, 1.11)	
						2.5	0.91 (0.81, 1.02)	
						5.0	0.92 (0.83, 1.03)	
						7.0	0.87 (0.74, 1.03)	
					Pear (all)	Per 3 serv/wk	0.75 (0.64, 0.87)	
						0.0 serv/wk	0.90 (0.86, 0.95)	
						0.5	1.00	
						1.0	0.99 (0.93, 1.06)	
						2.5	1.02 (0.94, 1.11)	
						Per 3 serv/wk	1.07 (0.97, 1.18)	
					Pear (men)	0.0 serv/wk	1.08 (0.99, 1.19)	
						0.5	1.00	
						1.0	1.03 (0.93, 1.14)	

						2.5	1.01 (0.88, 1.14)	
						Per 3 serv/wk	1.05 (0.90, 1.23)	
						0.0 serv/wk	1.07 (0.93, 1.23)	
						0.5	1.00	
						1.0	0.97 (0.89, 1.06)	
						2.5	1.04 (0.93, 1.15)	
						Per 3 serv/wk	1.09 (0.96, 1.23)	
						0.0 serv/wk	1.10 (0.97, 1.23)	
						0.3	1.00	
						1.3	0.95 (0.89, 1.01)	
						2.0	0.98 (0.88, 1.08)	
						Per 3 serv/wk	0.86 (0.75, 0.99)	
						0.0 serv/wk	0.87 (0.76, 0.99)	
						0.5	1.00	
						1.3	0.97 (0.88, 1.06)	
						2.0	1.02 (0.87, 1.19)	
						Per 3 serv/wk	0.81 (0.65, 1.01)	
						0.0 serv/wk	0.87 (0.71, 1.07)	
						0.3	1.00	
						1.3	0.94 (0.86, 1.02)	
						2.0	0.95 (0.83, 1.08)	
						Per 3 serv/wk	0.89 (0.75, 1.06)	
						0.0 serv/wk	0.87 (0.73, 1.03)	
						0.5	1.00	
						1.1	0.97 (0.89, 1.06)	
						2.5	0.99 (0.90, 1.09)	
						5.1	1.01 (0.93, 1.09)	
						7.1	0.98 (0.88, 1.10)	
						Per 3 serv/wk	1.01 (0.90, 1.12)	
						0.0 serv/wk	1.00 (0.97, 1.04)	
						0.5	1.00	
						1.1	0.96 (0.84, 1.11)	
						2.5	1.03 (0.89, 1.20)	
						5.1	1.07 (0.94, 1.22)	
						7.1	1.02 (0.85, 1.21)	
						0.0 serv/wk	1.07 (0.91, 1.26)	
						0.5	1.00	

							1.1	0.99 (0.88, 1.11)	
							2.5	0.97 (0.86, 1.09)	
							5.1	0.97 (0.87, 1.08)	
							7.1	0.96 (0.83, 1.12)	
						Oranges (all)	0.0 serv/wk	0.96 (0.84, 1.11)	
							0.6	1.00	
							1.0	0.96 (0.88, 1.05)	
							2.5	1.00 (0.92, 1.10)	
							5.0	1.01 (0.94, 1.09)	
							7.0	1.07 (0.95, 1.21)	
							Per 3 serv/wk	1.01 (0.91, 1.13)	
						Oranges (men)	0.0 serv/wk	1.02 (0.98, 1.06)	
							0.6	1.00	
							1.0	0.93 (0.81, 1.07)	
							2.5	1.02 (0.89, 1.18)	
							5.0	1.09 (0.96, 1.22)	
							7.0	1.10 (0.92, 1.32)	
							Per 3 serv/wk	1.05 (0.89, 1.23)	
						Oranges (women)	0.0 serv/wk	1.03 (0.98, 1.09)	
							0.6	1.00	
							1.0	0.99 (0.89, 1.12)	
							2.5	1.00 (0.89, 1.12)	
							5.0	0.98 (0.88, 1.08)	
							7.0	1.06 (0.90, 1.24)	
							Per 3 serv/wk	1.00 (0.87, 1.15)	
						Tangerine (all)	0.0 serv/wk	1.01 (0.96, 1.06)	
							0.1	1.00	
							1.5	1.05 (0.99, 1.11)	
							4.1	0.91 (0.77, 1.06)	
							Per 3 serv/wk	0.90 (0.79, 1.04)	
						Tangerine (men)	0.0 serv/wk	0.90 (0.81, 1.00)	
							0.1	1.00	
							1.5	1.03 (0.94, 1.13)	
							4.1	0.94 (0.73, 1.20)	
							Per 3 serv/wk	0.87 (0.70, 1.08)	
						Tangerine (women)	0.0 serv/wk	0.90 (0.77, 1.05)	
							0.1	1.00	

						1.5	1.06 (0.98, 1.15)	
						4.1	0.89 (0.72, 1.10)	
						Per 3 serv/wk	0.90 (0.75, 1.08)	
					Tropical fruit (all)	0.0 serv/wk	0.88 (0.77, 1.01)	
						0.6	1.00	
						1.4	1.02 (0.91, 1.16)	
						2.8	1.05 (0.93, 1.18)	
						5.0	1.05 (0.94, 1.17)	
						10.0	1.01 (0.89, 1.14)	
					Tropical fruit (men)	0.0 serv/wk	1.08 (0.95, 1.22)	
						0.6	1.00	
						1.4	1.20 (0.97, 1.50)	
						2.8	1.12 (0.91, 1.38)	
						5.0	1.19 (0.98, 1.45)	
						10.1	1.16 (0.95, 1.41)	
					Tropical fruit (women)	0.0 serv/wk	1.24 (1.01, 1.53)	
						0.6	1.00	
						1.4	0.95 (0.81, 1.10)	
						2.8	1.02 (0.88, 1.17)	
						5.0	0.98 (0.86, 1.13)	
						9.5	0.94 (0.80, 1.10)	
					Banana (all)	0.0 serv/wk	0.99 (0.83, 1.17)	
						0.5	1.00	
						1.0	0.99 (0.92, 1.07)	
						2.5	0.96 (0.89, 1.05)	
						5.0	0.96 (0.87, 1.05)	
						7.0	1.04 (0.91, 1.19)	
						Per 3 serv/wk	1.09 (0.93, 1.29)	
					Banana (men)	0.0 serv/wk	1.03 (0.98, 1.08)	
						0.6	1.00	
						1.0	1.13 (0.99, 1.28)	
						2.5	1.12 (0.98, 1.28)	
						5.0	1.06 (0.92, 1.23)	
						7.0	1.19 (0.99, 1.43)	
						Per 3 serv/wk	1.49 (1.20, 1.84)	
					Banana (women)	0.0 serv/wk	1.11 (1.04, 1.19)	
						0.5	1.00	

						1.0	0.93 (0.85, 1.03)	
						2.5	0.89 (0.80, 0.99)	
						5.0	0.91 (0.81, 1.03)	
						7.0	0.96 (0.78, 1.18)	
						Per 3 serv/wk	0.77 (0.59, 1.01)	
					Papaya	0.0 serv/wk	0.94 (0.87, 1.01)	
						0.5	1.00	
						1.0	1.00 (0.93, 1.07)	
						2.5	0.92 (0.85, 1.00)	
						5.0	0.94 (0.85, 1.03)	
						Per 3 serv/wk	0.89 (0.78, 1.02)	
					Papaya (men)	0.0 serv/wk	0.94 (0.88, 1.00)	
						0.5	1.00	
						1.0	1.01 (0.90, 1.13)	
						2.5	0.91 (0.80, 1.04)	
						5.0	0.94 (0.81, 1.08)	
						Per 3 serv/wk	0.83 (0.68, 1.00)	
					Papaya (women)	0.0 serv/wk	0.91 (0.83, 1.00)	
						0.5	1.00	
						1.0	1.00 (0.91, 1.09)	
						2.5	0.93 (0.83, 1.03)	
						5.0	0.94 (0.82, 1.07)	
						Per 3 serv/wk	0.97 (0.80, 1.17)	
					Watermelon (all)	0.0 serv/wk	0.97 (0.88, 1.06)	
						0.5	1.00	
						1.0	1.05 (0.98, 1.12)	
						2.5	1.06 (0.97, 1.15)	
						5.0	1.10 (0.98, 1.24)	
						Per 3 serv/wk	1.10 (0.92, 1.32)	
					Watermelon (men)	0.0 serv/wk	1.08 (0.98, 1.18)	
						0.5	1.00	
						1.0	1.05 (0.95, 1.17)	
						2.5	1.10 (0.97, 1.26)	
						5.0	1.14 (0.97, 1.34)	
						Per 3 serv/wk	1.17 (0.92, 1.49)	
					Watermelon (women)	0.0 serv/wk	1.11 (0.98, 1.26)	
						0.5	1.00	

						1.0	1.05 (0.96, 1.14)	
						2.5	1.02 (0.91, 1.15)	
						5.0	1.08 (0.91, 1.28)	
						Per 3 serv/wk	1.01 (0.76, 1.36)	
					Honeydew melon (all)	0.0 serv/wk	1.05 (0.91, 1.20)	
						0.3	1.00	
						1.0	1.03 (0.97, 1.10)	
						2.5	0.94 (0.85, 1.04)	
						Per 3 serv/wk	1.05 (0.92, 1.19)	
					Honeydew melon (men)	0.0 serv/wk	1.02 (0.90, 1.16)	
						0.3	1.00	
						1.0	0.98 (0.89, 1.09)	
						2.5	0.88 (0.76, 1.03)	
						Per 3 serv/wk	0.98 (0.82, 1.18)	
					Honeydew melon (women)	0.0 serv/wk	0.92 (0.76, 1.10)	
						0.3	1.00	
						1.0	1.06 (0.98, 1.15)	
						2.5	0.99 (0.87, 1.13)	
						Per 3 serv/wk	1.10 (0.92, 1.32)	
					Total juice (all)	0.0 serv/wk	1.12 (0.94, 1.34)	
						0.5	1.00	
						1.0	1.03 (0.95, 1.12)	
						2.5	1.13 (1.04, 1.24)	
						7.0	1.05 (0.93, 1.18)	
						Per 3 serv/wk	1.16 (1.00, 1.34)	
					Total juice (men)	0.0 serv/wk	1.08 (1.02, 1.16)	
						0.5	1.00	
						1.0	1.09 (0.96, 1.23)	
						2.5	1.16 (1.03, 1.32)	
						5.5	1.09 (0.93, 1.29)	
						Per 3 serv/wk	1.15 (0.93, 1.41)	
					Total juice (women)	0.0 serv/wk	1.09 (1.00, 1.20)	
						0.5	1.00	
						1.0	0.99 (0.89, 1.11)	
						2.5	1.11 (0.99, 1.25)	
						7.0	1.01 (0.86, 1.20)	
						Per 3 serv/wk	1.16 (0.94, 1.42)	

								1.07 (0.98, 1.18)	
Auerbach BJ et al, 2017, USA	The Women's Health Initiative (WHI)	1993-1998 to 2005, 7.8 years follow-up	114 219 women, age 50-79 years, 11 488 cases	Validated semi-quantitative FFQ, 122 items	Self-reported, validated by medication inventory and fasting plasma glucose levels	100% fruit juice Whole fruit Citrus fruits	≤4 serv/wk 5-6 1 serv/d 2-3 ≥4 ≤4 serv/wk 5-6 1 serv/d 2-3 ≥4 ≤4 serv/wk 5-6 1 serv/d 2-3	1.00 1.01 (0.97, 1.07) 0.97 (0.93, 1.02) 0.97 (0.87, 1.08) 0.82 (0.53, 1.27) 1.00 1.03 (0.97, 1.08) 1.00 (0.94, 1.06) 1.04 (0.96, 1.11) 0.93 (0.73, 1.18) 1.00 0.93 (0.87, 0.99) 0.96 (0.85, 1.08) 0.98 (0.65, 1.47)	Age, education level, race/ethnicity, smoking status, physical activity, body mass index, hormone replacement therapy status, study arm, and total energy intake
Bahadoran Z et al, 2017, Iran	Tehran Lipid and Glucose Study (TLGS)	2006-2008 to 2012-2014, 6 years follow-up	3052 participants, age ≥19 years, 150 cases	Validated FFQ, 168 items	Fasting plasma glucose or medication use	Allium vegetables	1.0 g/wk 10 g/wk 39 g/wk Per each 10 g/wk	1.00 1.05 (0.69, 1.61) 0.86 (0.57, 1.31) 0.95 (0.91, 1.05)	Age, diabetes risk score, physical activity, and dietary pattern scores
Du H et al, 2017, China	The China Kadoorie Biobank Study (CKB)	2004-2008 to 2013-2014 7 years follow-up	482 591 participants, age 30-79 years, 9504 cases	Administered laptop-based questionnaire on diet	Linkage with local disease and death registries, health insurance databases	Fresh fruit consumption	Never/rarely Monthly 1-3 d/wk 4-6 d/wk Daily	1.00 0.99 (0.90, 1.09) 0.93 (0.84, 1.02) 0.93 (0.83, 1.04) 0.88 (0.83, 0.93)	Age, sex, region, education, income, alcohol, smoking, physical activity, survey season, BMI, family history of diabetes, dairy products, meat, preserved vegetables
Huang M et al, 2017, USA	The Women's Health Initiative (WHI)	1993-1998 to 2010, 8.4 years follow-up	64 850 women, age 50-79 years, 4675 cases	Validated semi-quantitative FFQ, 122 items	Self-report, validated by medical record review and laboratory data	Fruit drinks	<1 serv/wk 1 serv/wk - <1 serv/d ≥ 1 serv/d	1.00 0.99 (0.85, 1.15) 1.33 (0.89, 1.98)	Age, race, marital status, family income, education, family history of diabetes, BMI, change in BMI, WHR, systolic

									blood pressure, insurance status, antihypertensive use, antihyperlipidemic use, hormone replacement therapy use, calibrated energy, sugar-sweetened beverages, glycemic load, glycemic index, Alternate Healthy Eating Index, cardiovascular history, hysterectomy history, smoking status, physical activity, sitting time, alcohol consumption
Lv J et al, 2017, China	China Kadoorie Biobank (CKB)	2004-2008 to 2013, 7.2 years follow-up	461 211 participants, age 30-79 years, 8784 cases	Validated qualitative FFQ	Linkage with local disease and death registries	Vegetables and fruits	Less than daily (either or both) Daily (both)	1.00 0.91 (0.85, 0.97)	Age, sex, education, marital status, family history of diabetes, smoking, alcohol consumption, physical activity and intakes of vegetables, fruits, red meat and wheat, BMI, WHR
Mamluk L, 2017, USA	The	1995-1996 to 2004-2006,	401 909 participants,	Validated self-reported FFQ, 124-items	Self-administered questionnaires	Fruit intake	0.82 portions/d 1.99 3.24	1.00 0.96 (0.91, 1.02) 0.95 (0.91, 0.99)	Age, sex, BMI, physical activity, energy intake,

	NIH-AARP Diet and Health Study (NIH-AARP)	10.6 years follow-up	age >50 years, 22 782 cases		or in interviews	Vegetable intake	7.73 Total intake 1 portion/d 1.04 portions/d 2.02 3.20 6.41 Total intake 1 portion/d	0.95 (0.91, 0.99) 1.00 (0.99, 1.01) 1.00 0.92 (0.87, 0.97) 0.88 (0.84, 0.94) 0.92 (0.87, 0.97) 1.00 (0.99, 1.01)	alcohol consumption, education, smoking	
						Leafy green vegetables	0.65 portions/wk 1.98 3.10 8.06 Total intake 1 portion/d	1.00 0.90 (0.86, 0.94) 0.89 (0.85, 0.94) 0.87 (0.84, 0.90) 0.98 (0.98, 0.99)		
						Cabbage	0.32 portions/wk 1.63 3.90 9.79 Total intake 1 portion/d	1.00 1.06 (1.01, 1.12) 1.09 (1.00, 1.18) 1.07 (0.94, 1.21) 1.02 (1.01, 1.03)		
Mamluk L, 2017, Greece	EPIC-elderly Greece	1994-ongoing 10 years follow-up	7567 participants, age >50 years, 1077 cases	Validated FFQ, 200 items	Self-administered questionnaires or in interviews	Fruit intake	1.06 portions/d 2.08 3.28 5.29 Total intake 1 portion/d	1.00 1.12 (0.77, 1.64) 1.09 (0.77, 1.54) 1.09 (0.77, 1.55) 1.00 (0.96, 1.04)		Age, sex, BMI, physical activity, energy intake, alcohol consumption, education, smoking
						Vegetable intake	1.15 portions/d 2.12 3.39 5.61 Total intake 1 portion/d	1.00 1.96 (0.81, 4.77) 2.29 (0.99, 5.36) 2.15 (0.93, 5.03) 0.99 (0.95, 1.04)		
						Leafy green vegetables	0.87 portions/wk 2.13 3.13 6.18	1.00 1.23 (0.89, 1.71) 1.55 (1.14, 2.11) 1.52 (1.13, 2.04)		

						Cabbage	Total intake 1 portion/d 0.84 portions/wk 2.06 3.06 4.88 Total intake 1 portion/d	1.02 (0.99, 1.04) 1.00 0.93 (0.77, 1.11) 1.21 (1.07, 1.44) 1.09 (0.85, 1.41) 1.02 (0.98, 1.07)	
Chen GC et al, 2018, Singapore	Singapore Chinese Health Study (SCHS)	1993-2010, 10.894 years follow-up	45 411 participants, age 45-74 years, 5207 cases	Validated semi-quantitative FFQ, 165 items	Self-reported, validated by linkage with a nationwide hospital-based discharge database and supplementary questionnaire	Total vegetables	57.431 g/d 83.286 105.459 132.489 184.357	1.00 1.16 (1.06, 1.26) 0.98 (0.89, 1.07) 1.02 (0.93, 1.11) 1.08 (0.98, 1.18)	Age, sex, dialect group, year of baseline interview, energy intake, physical activity, education, smoking, alcohol, soft drink, coffee, energy-adjusted intakes of red meat, poultry, fish, nuts and seeds, soya products and wholegrains, BMI, history of hypertension
						Light green vegetables	14.181 g/d 22.094 28.989 37.608 55.001	1.00 0.99 (0.90, 1.08) 0.98 (0.90, 1.08) 1.02 (0.93, 1.11) 0.95 (0.87, 1.04)	
						Dark green leafy vegetables	13.946 g/d 23.505 32.201 43.484 65.735	1.00 0.96 (0.88, 1.04) 1.03 (0.94, 1.12) 0.96 (0.88, 1.05) 1.05 (0.96, 1.15)	
						Cruciferous vegetables	18.882 g/d 30.243 40.428 53.278 79.211	1.00 0.97 (0.89, 1.06) 1.02 (0.94, 1.12) 0.90 (0.82, 0.98) 0.97 (0.88, 1.06)	
						Yellow vegetables	0.938 g/d 3.525 5.954 9.480 18.568	1.00 0.94 (0.87, 1.03) 0.95 (0.87, 1.03) 1.05 (0.96, 1.14) 0.97 (0.88, 1.06)	
						Potatoes	0.023 g/d 1.802 3.604 5.876	1.00 1.02 (0.94, 1.11) 0.97 (0.89, 1.06) 1.02 (0.94, 1.11)	

						Tomatoes	11.517 0.579 g/d 2.898 5.249 8.226 17.315	0.95 (0.87, 1.04) 1.00 1.02 (0.93, 1.11) 1.08 (0.99, 1.18) 1.09 (1.00, 1.19) 1.06 (0.97, 1.16)	
						Preserved vegetables	1.488 g/d 3.839 5.719 8.461 16.375	1.00 0.91 (0.84, 1.00) 0.95 (0.87, 1.04) 0.99 (0.90, 1.08) 0.97 (0.89, 1.06)	
Farhadnejad H et al, 2018, Iran	Tehran Lipid and Glucose Study (TLGS)	2006-2008 to 2012-2015, 6 years follow-up	1981 participants, age 18-75 years, 132 cases	Validated FFQ, 168 items	Fasting plasma glucose levels (ADA criteria)	Total potato	7.30 g/d 16.05 29.22 55.50	1.00 0.60 (0.34, 1.01) 0.75 (0.45, 1.26) 0.46 (0.25, 0.84)	Age, sex, BMI, physical activity, smoking, family history of diabetes, hypertension, serum triglycerides, high-density lipoprotein cholesterol, daily intakes of energy, saturated fat and food groups intake, including fruit, whole grains, vegetables, nuts and legumes
						Boiled potato	2.42 g/d 10.38 20.76 36.3	1.00 0.65 (0.39, 1.08) 0.74 (0.43, 1.28) 0.47 (0.26, 0.85)	
						Fried potato	1.30 g/d 4.66 10.33 25.71	1.00 0.82 (0.50, 1.35) 0.60 (0.35, 1.03) 0.50 (0.25, 1.07)	
Ma L et al, 2018, USA	The Nurses' Health Study (NHS)	1984-2012, 23.636 years follow-up	71 256 women, age 30-55 years, 7586 cases	Validated FFQ, 116 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes criteria	Total cruciferous vegetables	<1 serv/wk 1-3 4-6 ≥1 serv/d Every 2 serv/wk	1.00 1.14 (1.04, 1.25) 1.23 (1.11, 1.36) 1.22 (1.07, 1.38) 1.03 (1.01, 1.05)	
						Broccoli	<0.5 serv/wk 0.5-1 2-3 ≥4 Every 2 serv/wk	1.00 1.03 (0.95, 1.11) 1.07 (1.00, 1.15) 0.92 (0.77, 1.09) 1.01 (0.96, 1.05)	
						Cabbage	Never/almost never	1.00	

					(after 1998). Questionnaire -confirmed diagnosis of T2D was reconfirmed by medical record review.	Cauliflower	<0.5 serv/wk 0.5-1 ≥1 Every 2 serv/wk Never/almost never	1.12 (1.00, 1.24) 1.22 (1.09, 1.36) 1.25 (1.12, 1.39) 1.10 (1.04, 1.17) 1.00	hypertension, hypercholesterolemi a, BMI, total energy intake, the modified alternate healthy eating index score
						Brussel sprouts	<0.5 serv/wk 0.5-1 ≥1 Every 2 serv/wk Never/almost never	0.99 (0.91, 1.08) 1.04 (0.96, 1.14) 1.07 (0.98, 1.17) 1.05 (0.99, 1.10) 1.00	
						Kale, mustard or chard greens	<0.5 serv/wk 0.5-1 ≥1 Every 2 serv/wk Never/almost never	1.08 (1.03, 1.14) 1.14 (1.06, 1.24) 1.27 (1.16, 1.40) 1.28 (1.16, 1.40) 1.00	
							<0.5 serv/wk 0.5-1 ≥1 Every 2 serv/wk	1.03 (0.96, 1.10) 0.98 (0.84, 1.15) 1.04 (0.87, 1.24) 1.04 (0.89, 1.21)	
Ma L et al, 2018, USA	The Nurses' Health Study II (NHS II)	1991-2013, 20.180 years follow-up	88 293 women, age 24-44 years, 5438 cases	Validated FFQ, 131 items	Self-reported/ supplemental questionnaire/ the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998)	Total cruciferous vegetables	<1 serv/wk 1-3 4-6 ≥ 1 serv/d Every 2 serv/wk	1.00 1.00 (0.93, 1.07) 1.10 (1.00, 1.20) 1.10 (0.98, 1.24) 1.02 (1.00, 1.04)	Age, race/ethnicity, family history of diabetes, smoking status, alcohol intake, physical activity,
						Broccoli	<0.5 serv/wk 0.5-1 2-3 ≥4 Every 2 serv/wk	1.00 0.91 (0.82, 1.01) 0.98 (0.92, 1.05) 1.06 (0.91, 1.23) 1.00 (0.96, 1.04)	menopausal status and postmenopausal hormone use, oral contraceptive use,
						Cabbage	Never/almost never <0.5 serv/wk 0.5-1 ≥1 Every 2 serv/wk	1.00 0.97 (0.91, 1.04) 0.95 (0.85, 1.07) 1.13 (1.04, 1.23) 1.05 (0.99, 1.11)	multivitamin use, hypertension, hypercholesterolemi a, BMI, total energy intake, and the
						Cauliflower	Never/almost never <0.5 serv/wk 0.5-1	1.00 0.95 (0.88, 1.02) 0.91 (0.81, 1.02)	modified alternate healthy eating index score

						Brussel sprouts	≥ 1 Every 2 serv/wk Never/almost never <0.5 serv/wk 0.5-1 ≥ 1 Every 2 serv/wk Never/almost never <0.5 serv/wk 0.5-1 ≥ 1 Every 2 serv/wk	1.05 (0.98, 1.14) 1.05 (1.00, 1.11) 1.00 1.04 (0.97, 1.11) 1.09 (0.93, 1.27) 1.09 (0.98, 1.22) 1.11 (1.01, 1.23) 1.00 1.05 (0.95, 1.16) 1.20 (0.93, 1.54) 1.16 (0.97, 1.38) 1.07 (1.00, 1.16)	
Ma L et al, 2018, USA	The Health Professionals Follow-up Study (HPFS)	1986-2012, 20.254 years follow-up	41 358 men, age 40-75 years, 3543 cases	Validated FFQ, 131 items	Self-reported/supplemental questionnaire/the National Diabetes Data group criteria (before 1997) or American Diabetes criteria (after 1998). Questionnaire-confirmed diagnosis of T2D was reconfirmed by medical record review	Total cruciferous vegetables	<1 serv/wk 1-3 4-6 ≥ 1 serv/d Every 2 serv/wk <0.5 serv/wk 0.5-1 2-3 ≥ 4 Every 2 serv/wk Never/almost never <0.5 serv/wk 0.5-1 ≥ 1 Every 2 serv/wk Never/almost never <0.5 serv/wk 0.5-1 ≥ 1 Every 2 serv/wk Never/almost never <0.5 serv/wk 0.5-1 ≥ 1 Every 2 serv/wk	1.00 0.98 (0.88, 1.09) 1.04 (0.92, 1.18) 1.17 (1.00, 1.36) 1.03 (1.01, 1.06) 1.00 1.07 (0.97, 1.19) 1.02 (0.93, 1.11) 1.38 (1.10, 1.72) 1.03 (0.98, 1.09) 1.00 0.99 (0.86, 1.13) 1.11 (0.94, 1.32) 1.09 (0.97, 1.23) 1.00 (0.99, 1.02) 1.00 0.92 (0.84, 1.02) 1.00 (0.89, 1.12) 1.01 (0.90, 1.12) 1.04 (0.96, 1.13) 1.00 1.01 (0.94, 1.09) 1.11 (0.98, 1.25) 1.16 (1.03, 1.31) 1.11 (1.00, 1.24)	Age, race/ethnicity, family history of diabetes, smoking status, alcohol intake, physical activity, multivitamin use, hypertension, hypercholesterolemia, BMI, total energy intake, and the modified alternate healthy eating index score

						Kale, mustard or chard greens	Never/almost never <0.5 serv/wk	1.00 1.04 (0.95, 1.14)	
							0.5-1	1.07 (0.88, 1.30)	
							≥1	1.09 (0.90, 1.31)	
							Every 2 serv/wk	1.08 (0.94, 1.24)	

Supplementary Table 4. Serving sizes

Exposure	Serving size (g/d) ^a	Serving size (g/d) ^b
Main exposures		
Fruit and vegetables		
Fruits	-	80
Vegetables	-	80
Subtypes of fruit		
Apples	138	-
Apples and pears	138	-
Bananas	114	-
Berries	-	75
Blueberries	-	70
Cantaloupe	134	-
Citrus fruits	-	110
Fruit drinks	-	250
Fruit juice	-	250
100% fruit juice	-	250
Grapefruit	120	-
Grapes and raisins	-	49
Oranges	131	-
Peaches, plums and apricots	87	-
Prunes	-	85
Strawberries	75	-
Watermelon	-	286
Subtypes of vegetables		
Allium vegetables	-	160
Boiled potato	-	202
Broccoli	78	-
Brussel sprouts	78	-
Cabbage	68	-
Cauliflower	62	-
Cruciferous vegetables	-	72
Green leafy vegetables	-	73
Kale, mustard and chard greens	-	73
Potatoes	202	-
Tomatoes	122	-
Yellow vegetables	-	93

^a Serving sizes retrieved from Lee et al. (2009)

^b Estimated values based on Lee et al. (2009)

Lee, J. E., Mannisto, S., Spiegelman, D., Hunter, D. J., Bernstein, L., van den Brandt, P. A., . . . Smith-Warner, S. A. (2009). Intakes of fruit, vegetables, and carotenoids and renal cell cancer risk: a pooled analysis of 13 prospective studies. *Cancer Epidemiol Biomarkers Prev*, 18(6), 1730-1739. doi:10.1158/1055-9965.epi-09-0045

Bahadoran, 2017	*	*	*	*		*	*	*	7
Du, 2017	*	*		*	*	*	*	*	7
Huang, 2017		*	*	*	*	*	*	*	7
Lv, 2017	*	*	*	*	*	*	*	*	9
Mamluk, 2017	*	*	*	*	*	*		*	7
Mamluk, 2017	*	*	*	*	*	*		*	7
Chen, 2018	*	*	*	*	*	*	*	*	8
Farhadnejad, 2018	*	*	*	*	*	*	*	*	8
Ma, 2018		*	*	*	*	*	*	*	7
Ma, 2018		*	*	*	*	*	*	*	7
Ma, 2018		*	*	*	*	*	*	*	7

a

^a A study can be awarded a maximum of one star for each numbered item within the Selection and Outcome categories.

^b A maximum of two stars can be given for Comparability. One point was allocated if the study adjusted for age, with an additional point given if adjusted for any other additional factor.

Supplementary Table 6. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable intake and type 2 diabetes

Fruit and vegetables (n=8)		Fruits (n=16)		Vegetables (n=13)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
0	1.00	0	1.00	24	1.00
100	0.97 (0.94-1.00)	100	0.91 (0.87-0.96)	100	0.94 (0.89-0.99)
200	0.95 (0.89-1.00)	200	0.88 (0.82-0.94)	200	0.89 (0.80-0.99)
300	0.92 (0.84-1.01)	300	0.88 (0.82-0.94)	300	0.87 (0.77-0.99)
400	0.91 (0.82-1.01)	400	0.90 (0.85-0.95)	400	0.88 (0.76-1.00)
500	0.90 (0.81-1.01)	500	0.92 (0.86-0.97)	500	0.89 (0.77-1.02)
600	0.90 (0.81-1.00)	600	0.94 (0.87-1.00)	600	0.90 (0.78-1.04)
700	0.91 (0.82-1.00)				
800	0.91 (0.82-1.01)				
p _{nonlinearity}	0.13	p _{nonlinearity}	0.001	p _{nonlinearity}	0.01

Supplementary Table 7. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Apples (n=2)		Apples and pears (n=4)		Bananas (n=5)		Berries (n=5)		Blueberries (n=3)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
0	1.00	0	1.00	0	1.00	0	1.00	1	1.00
50	0.91 (0.83-1.01)	50	0.90 (0.83-0.97)	20	0.95 (0.91-1.00)	10	0.93 (0.83-1.03)	10	0.86 (0.81-0.91)
100	0.86 (0.75-0.99)	100	0.87 (0.80-0.95)	40	0.92 (0.85-1.00)	20	0.89 (0.76-1.04)	20	0.79 (0.72-0.86)
150	0.83 (0.74-0.95)	150	0.87 (0.78-0.96)	60	0.91 (0.83-1.01)	30	0.89 (0.78-1.03)	30	0.76 (0.69-0.83)
200	0.82 (0.73-0.92)			80	0.91 (0.81-1.03)	40	0.92 (0.84-1.01)	40	0.76 (0.68-0.84)
250	0.81 (0.71-0.93)			100	0.92 (0.80-1.06)	50	0.97 (0.89-1.07)		
				120	0.92 (0.78-1.10)	60	1.05 (0.86-1.27)		
				140	0.93 (0.77-1.13)	70	1.13 (0.82-1.55)		
p_{nonlinearity}	0.37	p_{nonlinearity}	0.07	p_{nonlinearity}	0.04	p_{nonlinearity}	0.23	p_{nonlinearity}	0.003

Supplementary Table 8. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Cantaloupe (n=3)		Citrus fruits (n=6)		Oranges (n=4)		Grapefruit (n=3)		Grapes and raisins (n=4)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
2	1.00	0	1.00	0	1.00	2	1.00	0	1.00
10	1.05 (1.01-1.09)	50	1.01 (0.97-1.06)	20	0.99 (0.95-1.03)	20	0.97 (0.92-1.02)	10	0.88 (0.83-0.94)
20	1.09 (1.02-1.16)	100	1.02 (0.96-1.09)	40	0.98 (0.93-1.04)	40	0.95 (0.87-1.03)	20	0.83 (0.77-0.90)
30	1.12 (1.03-1.21)	150	1.03 (0.96-1.11)	60	0.99 (0.93-1.05)	60	0.94 (0.86-1.02)	30	0.83 (0.76-0.90)
40	1.14 (1.04-1.24)	200	1.04 (0.94-1.15)	80	0.99 (0.94-1.05)	80	0.93 (0.85-1.02)	40	0.84 (0.74-0.97)
50	1.14 (1.05-1.25)	250	1.05 (0.92-1.21)	100	1.00 (0.95-1.06)	100	0.93 (0.84-1.03)		
60	1.14 (1.05-1.25)	300	1.06 (0.89-1.28)	120	1.01 (0.96-1.08)				
70	1.14 (1.05-1.23)	330	1.07 (0.87-1.31)	130	1.02 (0.96-1.09)				
80	1.13 (1.05-1.22)								
pnonlinearity	0.04	pnonlinearity	0.94	pnonlinearity	0.41	pnonlinearity	0.49	pnonlinearity	0.01

Supplementary Table 9. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Peaches, plums, apricots (n=3)		Prunes (n=3)		Strawberries (n=3)		Watermelon (n=2)		Allium vegetables (n=3)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
1	1.00	0	1.00	1	1.00	g/d	RR (95% CI)	0	1.00
10	1.01 (0.97-1.05)	10	0.90 (0.82-0.98)	10	1.00 (0.94-1.06)	0	1.00	5	0.81 (0.67-0.97)
20	1.01 (0.94-1.08)	20	0.85 (0.74-0.97)	20	1.01 (0.91-1.12)	50	0.94 (0.74-1.21)	10	0.72 (0.55-0.96)
30	1.00 (0.92-1.09)	30	0.84 (0.74-0.96)	30	1.03 (0.90-1.19)	100	0.92 (0.62-1.35)	15	0.71 (0.52-0.96)
40	0.98 (0.89-1.08)	40	0.86 (0.77-0.97)	40	1.06 (0.90-1.26)	150	0.92 (0.63-1.35)	20	0.72 (0.53-0.97)
50	0.96 (0.86-1.07)	50	0.91 (0.79-1.05)	50	1.10 (0.90-1.36)	200	0.96 (0.72-1.27)	23	0.72 (0.53-0.99)
60	0.93 (0.83-1.06)			60	1.15 (0.89-1.49)	220	0.97 (0.77-1.24)		
70	0.91 (0.78-1.05)								
74	0.89 (0.76-1.05)								
pnonlinearity	0.29	pnonlinearity	0.06	pnonlinearity	0.39	pnonlinearity	0.61	pnonlinearity	0.045

Supplementary Table 10. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Broccoli (n=4)		Brussel sprouts (n=3)		Cabbage (n=6)		Cauliflower (n=3)		Cruciferous vegetables (n=8)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
0	1.00	1	1.00	0.8	1.00	1	1.00	0	1.00
20	1.01 (0.93-1.09)	2	1.02 (1.00-1.05)	20	1.15 (1.03-1.28)	2	0.98 (0.95-1.01)	20	0.97 (0.90-1.05)
40	1.03 (0.98-1.09)	4	1.05 (1.01-1.09)	40	1.20 (1.01-1.42)	4	0.96 (0.90-1.02)	40	0.96 (0.84-1.09)
60	1.07 (0.93-1.23)	6	1.07 (1.01-1.14)	60	1.23 (0.97-1.56)	6	0.96 (0.89-1.03)	60	0.96 (0.82-1.12)
80	1.11 (0.85-1.44)	8	1.10 (1.03-1.17)	80	1.26 (0.92-1.73)	8	0.98 (0.91-1.05)	80	0.97 (0.82-1.13)
100	1.14 (0.78-1.65)	10	1.12 (1.04-1.21)	100	1.29 (0.87-1.92)	10	1.01 (0.95-1.08)	100	0.99 (0.85-1.15)
		12	1.15 (1.06-1.25)	110	1.30 (0.84-2.02)	11	1.03 (0.97-1.09)	120	1.01 (0.87-1.17)
		14	1.18 (1.07-1.29)					140	1.04 (0.89-1.20)
pnonlinearity	0.81	pnonlinearity	0.98	pnonlinearity	0.04	pnonlinearity	0.03	pnonlinearity	0.32

Supplementary Table 10. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Green leafy vegetables (n=8)		Kale, mustard and chard greens (n=3)		Tomatoes (n=3)		Yellow vegetables (n=4)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
1.6	1.00	1	1.00	0	1.00	0	1.00
20	0.92 (0.82-1.04)	2	1.02 (0.99-1.05)	20	0.91 (0.68-1.23)	20	0.84 (0.66-1.07)
40	0.87 (0.70-1.07)	4	1.04 (0.99-1.10)	40	0.88 (0.57-1.37)	40	0.77 (0.55-1.08)
60	0.85 (0.66-1.09)	6	1.06 (0.98-1.13)	60	0.89 (0.56-1.40)	60	0.75 (0.53-1.05)
80	0.84 (0.65-1.10)	8	1.07 (0.99-1.16)	80	0.92 (0.61-1.38)	80	0.75 (0.56-1.01)
100	0.85 (0.66-1.10)	10	1.08 (0.99-1.18)	100	0.96 (0.68-1.34)	100	0.77 (0.59-1.00)
120	0.85 (0.66-1.11)	12	1.09 (0.99-1.20)				
140	0.86 (0.66-1.12)	13	1.10 (0.99-1.21)				
p _{nonlinearity}	0.21	p _{nonlinearity}	0.63	p _{nonlinearity}	0.50	p _{nonlinearity}	0.27

Supplementary Table 11. Relative risks (95% confidence intervals) from nonlinear analysis of fruit and vegetable subtypes and type 2 diabetes

Potatoes (n=8)		Boiled potato (n=2)		Fruit juice (n=7)		Fruit drinks (n=5)	
g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)	g/d	RR (95% CI)
0	1.00	2.4	1.00	0	1.00	0	1.00
50	0.98 (0.89-1.08)	20	0.85 (0.58-1.25)	200	1.03 (0.97-1.09)	100	1.07 (0.98-1.19)
100	0.99 (0.84-1.16)	40	0.74 (0.35-1.58)	400	1.05 (0.97-1.14)	200	1.17 (1.00-1.37)
150	1.02 (0.85-1.23)	60	0.66 (0.21-2.08)	600	1.06 (0.98-1.16)	300	1.28 (1.00-1.65)
200	1.08 (0.89-1.31)	80	0.60 (0.12-2.91)	800	1.08 (1.00-1.16)	400	1.41 (0.97-2.05)
250	1.15 (0.95-1.40)	100	0.55 (0.07-4.39)	1000	1.09 (1.02-1.17)	500	1.54 (0.93-2.57)
300	1.22 (1.00-1.50)	120	0.52 (0.04-7.06)	1200	1.10 (1.04-1.17)		
325	1.26 (1.02-1.55)	140	0.49 (0.02-12.02)	1400	1.12 (1.06-1.18)		
		160	0.47 (0.01-21.39)	1600	1.13 (1.07-1.19)		
				1800	1.14 (1.09-1.20)		
pnonlinearity	0.15	pnonlinearity	0.71	pnonlinearity	0.65	pnonlinearity	0.83

Supplementary Table 12. Subgroup analyses of fruit and vegetable intake and type 2 diabetes, dose-response

		Fruit and vegetables, 200 g/day					
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P</i> _h ^a	<i>P</i> _h ^b	
All studies		7	0.98 (0.95-1.01)	55.4	0.03		
Duration of follow-up							
	<10 years follow-up	3	1.01 (0.97-1.05)	0	0.75	0.14	
	≥10 years follow-up	4	0.95 (0.91-1.00)	46.3	0.13		
Gender							
	Men	1	0.89 (0.76-1.03)			0.46	
	Women	3	1.00 (0.97-1.04)	0	0.62		
	Men and women	3	0.95 (0.90-1.01)	45.8	0.16		
Geographic location							
	Europe	2	0.95 (0.91-1.00)	3.2	0.31	0.32	
	America	4	0.99 (0.94-1.04)	54.2	0.09		
	Asia	1	1.00 (0.92-1.09)				
	Australia						
Number of cases							
	Cases <1.000	2	0.96 (0.85-1.08)	49.0	0.16	0.95	
	Cases 1.000-<2.000	3	0.98 (0.90-1.06)	69.5	0.04		
	Cases ≥2.000	2	0.98 (0.95-1.01)	0	0.36		
Study quality							
	0-3	0				NC	
	4-6	0					
	7-9	7	0.98 (0.95-1.01)	55.4	0.03		
Adjustment for confounders							
	Age	Yes	7	0.98 (0.95-1.01)	55.4	0.03	NC
		No	0				
	Education	Yes	4	0.95 (0.89-1.02)	59.3	0.06	0.28
		No	3	1.00 (0.96-1.03)	0	0.95	
	Ethnicity	Yes	0				NC
		No	7	0.98 (0.95-1.01)	55.4	0.03	
	Family history	Yes	4	0.99 (0.96-1.02)	0	0.50	0.46
		No	3	0.96 (0.89-1.04)	67.6	0.05	
	Body mass index	Yes	7	0.98 (0.95-1.01)	55.4	0.03	NC
		No	0				
	Waist circumference/WHR	Yes	2	0.97 (0.83-1.13)	70	0.07	0.69
		No	5	0.98 (0.94-1.01)	32.5	0.21	
	Hypertension	Yes	2	1.00 (0.96-1.05)	0	0.98	0.45
		No	5	0.97 (0.92-1.01)	52.8	0.08	
	Alcohol	Yes	7	0.98 (0.95-1.01)	55.4	0.03	NC
		No	0				
	Smoking	Yes	7	0.98 (0.95-1.01)	55.4	0.03	NC
		No	0				
	Physical activity	Yes	7	0.98 (0.95-1.01)	55.4	0.03	NC
		No	0				
	Meat consumption	Yes	1	0.99 (0.95-1.04)			0.74
		No	6	0.97 (0.93-1.02)	46.5	0.10	
	Soft drink	Yes	1	0.99 (0.95-1.04)			0.74
		No	6	0.97 (0.93-1.02)	46.5	0.10	
	Whole grain	Yes	1	0.99 (0.95-1.04)			0.74
		No	6	0.97 (0.93-1.02)	46.5	0.10	
	Coffee	Yes	2	0.99 (0.95-1.04)	0	0.81	0.56
		No	5	0.97 (0.92-1.02)	55.1	0.06	
	Energy intake	Yes	6	0.99 (0.96-1.01)	3.2	0.40	0.09
		No	1	0.88 (0.80-0.98)			

n = number of studies^a *P* for heterogeneity within each subgroup^b *P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Table 13. Subgroup analyses of fruit intake and type 2 diabetes, dose-response

		Fruits, 200 g/day					
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P</i> _h ^a	<i>P</i> _h ^b	
All studies		16	0.96 (0.92-1.01)	71.6	<0.0001		
Duration of follow-up							
	<10 years follow-up	7	0.99 (0.88-1.11)	86.6	<0.0001	0.54	
	≥10 years follow-up	9	0.98 (0.97-0.99)	0	0.54		
Gender							
	Men	2	0.92 (0.82-1.05)	0	0.39	0.53	
	Women	6	1.01 (0.95-1.07)	43.8	0.11		
	Men and women	8	0.94 (0.87-1.01)	82.3	<0.0001		
Geographic location							
	Europe	4	0.95 (0.89-1.03)	0	0.62	0.82	
	America	7	0.98 (0.95-1.02)	21.7	0.26		
	Asia	4	0.95 (0.82-1.11)	92.1	<0.0001		
	Australia	1	0.84 (0.32-2.23)				
Number of cases							
	Cases <1.000	4	0.99 (0.90-1.09)	0	0.65	0.17	
	Cases 1.000-<2.000	4	1.05 (1.00-1.12)	0	0.65		
	Cases ≥2.000	8	0.93 (0.87-0.99)	83.5	<0.0001		
Study quality							
	0-3	0				NC	
	4-6	0					
	7-9	16	0.96 (0.92-1.01)	71.6	<0.0001		
Adjustment for confounders							
	Age	Yes	16	0.96 (0.92-1.01)	71.6	<0.0001	NC
		No	0				
	Education	Yes	10	0.98 (0.92-1.04)	80.8	<0.0001	0.52
		No	6	0.95 (0.90-1.00)	0	0.48	
	Ethnicity	Yes	5	0.97 (0.92-1.03)	79.0	<0.0001	0.60
		No	11	0.96 (0.90-1.02)	15.9	0.31	
	Family history	Yes	9	0.91 (0.84-0.99)	64.2	<0.0001	0.03
		No	7	1.00 (0.97-1.03)	30.1	0.20	
	Body mass index	Yes	16	0.96 (0.92-1.01)	71.6	<0.0001	NC
		No	0				
	Waist circumference/WHR	Yes	4	1.07 (1.00-1.14)	0	0.81	0.07
		No	12	0.94 (0.90-0.99)	75.3	<0.0001	
	Hypertension	Yes	3	1.04 (0.98-1.10)	0	0.53	0.16
		No	13	0.95 (0.90-1.00)	74.3	<0.0001	
	Alcohol	Yes	11	0.98 (0.92-1.04)	78.5	<0.0001	0.46
		No	5	0.95 (0.88-1.01)	29.2	0.23	
	Smoking	Yes	15	0.96 (0.92-1.01)	73.4	<0.0001	0.79
		No	1	0.84 (0.32-2.23)			
	Physical activity	Yes	15	0.97 (0.92-1.01)	71.9	<0.0001	0.37
		No	1	0.81 (0.57-1.14)			
	Meat consumption	Yes	2	0.92 (0.68-1.24)	97.1	<0.0001	0.36
		No	14	0.98 (0.97-1.00)	0	0.65	
	Soft drink	Yes	1	0.98 (0.90-1.06)			0.89
		No	15	0.96 (0.92-1.01)	73.5	<0.0001	
	Whole grain	Yes	0				NC
		No	16	0.96 (0.92-1.01)	71.6	<0.0001	
	Coffee	Yes	2	0.99 (0.93-1.06)	0	0.57	0.63
		No	14	0.96 (0.91-1.01)	75.2	<0.0001	
	Energy intake	Yes	15	0.99 (0.97-1.01)	8.0	0.36	0
		No	1	0.79 (0.73-0.84)			

n = number of studies

^a *P* for heterogeneity within each subgroup

^b *P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Table 14. Subgroup analyses of vegetable intake and type 2 diabetes, dose-response

		Vegetables, 200 g/day				
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P_h</i> ^a	<i>P_h</i> ^b
All studies		12	0.98 (0.94-1.02)	48.3	0.03	
Duration of follow-up						
	<10 years follow-up	5	0.94 (0.84-1.05)	69.7	0.01	0.43
	≥10 years follow-up	7	1.00 (0.98-1.02)	0	0.49	
Gender						
	Men	1	0.82 (0.62-1.09)			0.76
	Women	4	0.98 (0.89-1.08)	79.3	0.002	
	Men and women	7	1.00 (0.97-1.02)	0	0.55	
Geographic location						
	Europe	4	0.95 (0.88-1.02)	0	0.53	0.55
	America	4	1.01 (0.99-1.03)	0	0.76	
	Asia	3	0.91 (0.80-1.04)	65.9	0.05	
	Australia	1	0.59 (0.20-1.73)			
Number of cases						
	Cases <1.000	4	0.86 (0.75-0.99)	0	0.77	0.09
	Cases 1.000-<2.000	4	0.96 (0.87-1.06)	74.7	0.008	
	Cases ≥2.000	3	1.00 (0.98-1.03)	0	0.63	
Study quality						
	0-3	0				NC
	4-6	0				
	7-9	12	0.98 (0.94-1.02)	48.3	0.03	
Adjustment for confounders						
Age	Yes	12	0.98 (0.94-1.02)	48.3	0.03	NC
	No	0				
Education	Yes	8	0.96 (0.91-1.02)	58.3	0.02	0.59
	No	4	1.00 (0.94-1.06)	24.0	0.27	
Ethnicity	Yes	1	0.59 (0.20-1.73)			0.39
	No	11	0.98 (0.94-1.02)	50.9	0.03	
Family history	Yes	6	0.98 (0.92-1.05)	27.9	0.23	0.95
	No	6	0.97 (0.91-1.03)	64.7	0.02	
Body mass index	Yes	12	0.98 (0.94-1.02)	48.3	0.03	NC
	No	0				
Waist circumference/WHR	Yes	4	0.90 (0.75-1.07)	70.3	0.02	0.12
	No	8	1.00 (0.98-1.02)	0	0.64	
Hypertension	Yes	4	0.94 (0.84-1.05)	72.3	0.01	0.31
	No	8	1.00 (0.98-1.02)	0	0.44	
Alcohol	Yes	11	0.98 (0.94-1.02)	49.6	0.03	0.31
	No	1	0.77 (0.50-1.17)			
Smoking	Yes	11	0.98 (0.94-1.02)	50.9	0.03	0.39
	No	1	0.59 (0.20-1.73)			
Physical activity	Yes	11	0.98 (0.94-1.02)	49.6	0.03	0.31
	No	1	0.77 (0.50-1.17)			
Meat consumption	Yes	3	0.96 (0.83-1.11)	85	0.001	0.78
	No	9	1.00 (0.98-1.02)	0	0.49	
Soft drink	Yes	2	1.03 (0.97-1.09)	0	0.87	0.24
	No	10	0.96 (0.91-1.01)	53.9	0.02	
Whole grain	Yes	2	1.03 (0.97-1.09)	0	0.87	0.24
	No	10	0.96 (0.91-1.01)	53.9	0.02	
Coffee	Yes	3	1.01 (0.95-1.08)	13.5	0.32	0.48
	No	9	0.96 (0.91-1.02)	56.2	0.02	
Energy intake	Yes	12	0.98 (0.94-1.02)	48.3	0.03	NC
	No	0				

n = number of studies

^a *P* for heterogeneity within each subgroup

^b *P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Table 15. Subgroup analyses of potato intake and type 2 diabetes, dose-response

		Potatoes, 100 g/day					
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P</i> _h ^a	<i>P</i> _h ^b	
All studies		8	1.08 (1.02-1.15)	55.4	0.03		
Duration of follow-up							
	<10 years follow-up	3	0.95 (0.78-1.16)	60.1	0.08	0.08	
	≥10 years follow-up	5	1.11 (1.08-1.15)	0	0.43		
Gender							
	Men	1	1.12 (1.04-1.20)			0.64	
	Women	3	1.09 (1.02-1.15)	57.5	0.01		
	Men and women	4	0.94 (0.71-1.24)	71.3	0.02		
Geographic location							
	Europe	1	1.17 (1.02-1.35)			0.20	
	America	4	1.10 (1.05-1.14)	39.5	0.18		
	Asia	2	0.46 (0.19-1.13)	43.1	0.19		
	Australia	1	0.97 (0.81-1.15)				
Number of cases							
	Cases <1.000	3	0.99 (0.74-1.33)	76.6	0.01	0.45	
	Cases 1.000-<2.000	1	1.00 (0.91-1.10)				
	Cases ≥2.000	4	1.11 (1.07-1.15)	6.7	0.36		
Study quality							
	0-3	0				NC	
	4-6	0					
	7-9	8	1.08 (1.02-1.15)	55.4	0.03		
Adjustment for confounders							
	Age	Yes	8	1.08 (1.02-1.15)	55.4	0.03	NC
		No	0				
	Education	Yes	2	0.92 (0.72-1.18)	12.0	0.29	0.28
		No	6	1.10 (1.04-1.16)	56.7	0.04	
	Ethnicity	Yes	4	1.10 (1.06-1.15)	10.7	0.34	0.42
		No	4	0.99 (0.78-1.24)	71.7	0.01	
	Family history	Yes	7	1.09 (1.03-1.15)	55.9	0.03	0.36
		No	1	0.65 (0.32-1.32)			
	Body mass index	Yes	8	1.08 (1.02-1.15)	55.4	0.03	NC
		No	0				
	Waist circumference/WHR	Yes	1	0.97 (0.81-1.15)			0.38
		No	7	1.09 (1.03-1.16)	56.1	0.03	
	Hypertension	Yes	2	0.93 (0.69-1.27)	28.2	0.24	0.17
		No	6	1.10 (1.05-1.17)	49.2	0.08	
	Alcohol	Yes	6	1.08 (1.03-1.14)	44.5	0.11	0.65
		No	2	0.61 (0.14-2.73)	83.7	0.01	
	Smoking	Yes	7	1.09 (1.03-1.16)	56.1	0.03	0.38
		No	1	0.97 (0.81-1.15)			
	Physical activity	Yes	7	1.07 (1.00-1.14)	59.1	0.02	0.51
		No	1	1.17 (1.02-1.35)			
	Meat consumption	Yes	1	0.65 (0.32-1.32)			0.36
		No	7	1.09 (1.03-1.15)	55.9	0.03	
	Soft drink	Yes	1	0.65 (0.32-1.32)			0.36
		No	7	1.09 (1.03-1.15)	55.9	0.03	
	Whole grain	Yes	2	0.46 (0.19-1.13)	43.1	0.19	0.09
		No	6	1.09 (1.05-1.14)	36.9	0.16	
	Coffee	Yes	1	0.65 (0.32-1.32)			0.36
		No	7	1.09 (1.03-1.15)	55.9	0.03	
	Energy intake	Yes	8	1.08 (1.02-1.15)	55.4	0.03	NC
		No	0				

n = number of studies

^a *P* for heterogeneity within each subgroup

^b *P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Table 16. Subgroup analyses of cruciferous vegetable intake and type 2 diabetes, dose-response

		Cruciferous vegetables, 100 g/day				
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P</i> _h ^a	<i>P</i> _h ^b
All studies		8	0.96 (0.84-1.09)	80.9	<0.0001	
Duration of follow-up						
	<10 years follow-up	3	0.75 (0.51-1.11)	82.8	0.003	0.20
	≥10 years follow-up	5	1.07 (0.97-1.18)	67.5	0.02	
Gender						
	Men	2	0.85 (0.42-1.73)	79.6	0.03	0.99
	Women	4	0.94 (0.76-1.16)	87.4	0	
	Men and women	2	0.91 (0.81-1.03)	0	0.79	
Geographic location						
	Europe	1	0.55 (0.29-1.04)			0.53
	America	4	1.13 (1.07-1.19)	0	0.53	
	Asia	3	0.75 (0.54-1.05)	83.4	0.002	
	Australia					
Number of cases						
	Cases <1.000	2	0.77 (0.50-1.19)	47.1	0.17	0.14
	Cases 1.000-<2.000	2	0.68 (0.33-1.39)	90.8	0.001	
	Cases ≥2.000	4	1.09 (1.00-1.19)	61.1	0.05	
Study quality						
	0-3	0				NC
	4-6	0				
	7-9	8	0.96 (0.84-1.09)	80.9	<0.0001	
Adjustment for confounders						
	Age					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				
	Education					
	Yes	3	0.64 (0.38-1.08)	85.2	0.001	0.05
	No	5	1.10 (1.03-1.18)	29.5	0.23	
	Ethnicity					
	Yes	3	1.13 (1.07-1.20)	0	0.75	0.03
	No	5	0.78 (0.62-0.99)	73.1	0.005	
	Family history					
	Yes	6	1.08 (0.99-1.17)	51.3	0.07	0.15
	No	2	0.67 (0.34-1.30)	91.6	0.001	
	Body mass index					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				
	Waist circumference/WHR					
	Yes	2	0.48 (0.35-0.66)	0	0.68	0.005
	No	6	1.06 (0.98-1.15)	57.1	0.04	
	Hypertension					
	Yes	7	0.98 (0.86-1.11)	81.6	<0.0001	0.38
	No	1	0.55 (0.29-1.04)			
	Alcohol					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				
	Smoking					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				
	Physical activity					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				
	Meat consumption					
	Yes	2	0.67 (0.34-1.30)	91.6	0.001	0.15
	No	6	1.08 (0.99-1.17)	51.3	0.07	
	Soft drink					
	Yes	1	0.92 (0.80-1.06)			0.96
	No	7	0.96 (0.83-1.11)	81.2	<0.0001	
	Whole grain					
	Yes	1	0.92 (0.80-1.06)			0.96
	No	7	0.96 (0.83-1.11)	81.2	<0.0001	
	Coffee					
	Yes	2	0.91 (0.81-1.03)	0	0.79	0.99
	No	6	0.97 (0.84-1.14)	82.8	<0.0001	
	Energy intake					
	Yes	8	0.96 (0.84-1.09)	80.9	<0.0001	NC
	No	0				

n = number of studies

^a*P* for heterogeneity within each subgroup

^b*P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Table 17. Subgroup analyses of green leafy vegetable intake and type 2 diabetes, dose-response

		Green leafy vegetables, 100 g/day					
		<i>n</i>	RR (95% CI)	I ² (%)	<i>P</i> _h ^a	<i>P</i> _h ^b	
All studies		8	0.96 (0.91-1.01)	75.0	<0.0001		
Duration of follow-up							
	<10 years follow-up	3	0.86 (0.76-0.96)	19.0	0.29	0.18	
	≥10 years follow-up	5	0.98 (0.93-1.03)	78.7	0.001		
Gender							
	Men	0					
	Women	3	0.87 (0.81-0.94)	0	0.41	0.06	
	Men and women	5	1.00 (0.94-1.05)	76.2	0.002		
Geographic location							
	Europe	2	0.87 (0.57-1.32)	73.6	0.05	0.66	
	America	3	0.95 (0.88-1.01)	48.5	0.14		
	Asia	3	0.90 (0.70-1.15)	78.2	0.01		
	Australia	0					
Number of cases							
	Cases <1.000	1	0.70 (0.44-1.12)			0.57	
	Cases 1.000-<2.000	3	0.93 (0.80-1.08)	83.2	0.003		
	Cases ≥2.000	4	0.95 (0.86-1.05)	66.3	0.03		
Study quality							
	0-3	0				NC	
	4-6	0					
	7-9	8	0.96 (0.91-1.01)	75.0	<0.0001		
Adjustment for confounders							
	Age	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Education	Yes	5	0.98 (0.92-1.03)	81.9	<0.0001	0.44
		No	3	0.89 (0.82-0.97)	0	0.47	
	Ethnicity	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Family history	Yes	3	0.89 (0.82-0.97)	0	0.47	0.44
		No	5	0.98 (0.92-1.03)	81.9	<0.0001	
	Body mass index	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Waist circumference/WHR	Yes	1	0.82 (0.72-0.93)			0.18
		No	7	0.98 (0.93-1.02)	71.2	0.002	
	Hypertension	Yes	4	0.92 (0.78-1.08)	67.8	0.03	0.81
		No	4	0.97 (0.92-1.02)	81.9	0.001	
	Alcohol	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Smoking	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Physical activity	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				
	Meat consumption	Yes	3	0.92 (0.79-1.07)	76.0	0.02	0.68
		No	5	0.98 (0.94-1.03)	73.1	0.005	
	Soft drink	Yes	2	0.98 (0.78-1.22)	81.1	0.02	0.63
		No	6	0.96 (0.91-1.01)	77.4	0.001	
	Whole grain	Yes	2	0.98 (0.78-1.22)	81.1	0.02	0.63
		No	6	0.96 (0.91-1.01)	77.4	0.001	
	Coffee	Yes	3	0.86 (0.76-1.14)	70.4	0.03	0.93
		No	5	0.96 (0.91-1.02)	80.2	<0.0001	
	Energy intake	Yes	8	0.96 (0.91-1.01)	75.0	<0.0001	NC
		No	0				

n = number of studies^a *P* for heterogeneity within each subgroup^b *P* for heterogeneity between subgroups with meta-regression analysis

NC not calculatable

Supplementary Figures

Supplementary Figures of main exposures, high vs. low, linear and nonlinear dose response analyses

Supplementary Figure 1. Fruit and vegetables and type 2 diabetes, high vs. low

Supplementary Figure 2. Fruits and type 2 diabetes, high vs. low

Supplementary Figure 3. Vegetables and type 2 diabetes, high vs. low

Supplementary figures of subtypes of fruit, high vs. low, linear and nonlinear dose response analyses

Supplementary Figure 4. Apples and type 2 diabetes, high vs. low

Supplementary Figure 5. Apples and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 6. Apples and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 7. Apples and pears and type 2 diabetes, high vs. low

Supplementary Figure 8. Apples and pears and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 9. Apples and pears and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 10. Bananas and type 2 diabetes, high vs. low

Supplementary Figure 11. Bananas and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 12. Bananas and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 13. Berries and type 2 diabetes, high vs. low

Supplementary Figure 14. Berries and type 2 diabetes, dose-response analysis per 50 g/d

Supplementary Figure 15. Berries and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 16. Blueberries and type 2 diabetes, high vs. low

Supplementary Figure 17. Blueberries and type 2 diabetes, dose-response analysis per 50 g/d

Supplementary Figure 18. Blueberries and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 19. Cantaloupe and type 2 diabetes, high vs. low

Supplementary Figure 20. Cantaloupe and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 21. Cantaloupe and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 22. Citrus fruits and type 2 diabetes, high vs. low

Supplementary Figure 23. Citrus fruits and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 24. Citrus fruits and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 25. Fruit drinks and type 2 diabetes, high vs. low

Supplementary Figure 26. Fruit drinks and type 2 diabetes, dose-response analysis per 250 g/d

Supplementary Figure 27. Fruit juice and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 28. Fruit juice and type 2 diabetes, high vs. low

Supplementary Figure 29. Fruit juice and type 2 diabetes, dose-response analysis per 250 g/d

Supplementary Figure 30. Fruit juice and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 31. Grapefruit and type 2 diabetes, high vs. low

Supplementary Figure 32. Grapefruit and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 33. Grapefruit and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 34. Grapes and raisins and type 2 diabetes, high vs. low

Supplementary Figure 35. Grapes and raisins and type 2 diabetes, dose-response analysis per 50 g/d

Supplementary Figure 36. Grapes and raisins and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 37. Oranges and type 2 diabetes, high vs. low

Supplementary Figure 38. Oranges and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 39. Oranges and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 40. Peaches, plums and apricots and type 2 diabetes, high vs. low

Supplementary Figure 41. Peaches, plums and apricots and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 42. Peaches, plums and apricots and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 43. Prunes and type 2 diabetes, high vs. low

Supplementary Figure 44. Prunes and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 45. Prunes and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 46. Strawberries and type 2 diabetes, high vs. low

Supplementary Figure 47. Strawberries and type 2 diabetes, dose-response analysis per 50 g/d

Supplementary Figure 48. Strawberries and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 49. Watermelon and type 2 diabetes, high vs. low

Supplementary Figure 50. Watermelon and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 51. Watermelon and type 2 diabetes, nonlinear dose-response analysis

Supplementary figures of subtypes of vegetables

Supplementary Figure 52. Allium vegetables and type 2 diabetes, high vs. low

Supplementary Figure 53. Allium vegetables and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 54. Allium vegetables and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 55. Broccoli and type 2 diabetes, high vs. low

Supplementary Figure 56. Broccoli and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 57. Broccoli and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 58. Brussel sprouts and type 2 diabetes, high vs. low

Supplementary Figure 59. Brussel sprouts and type 2 diabetes, dose-response analysis per 10 g/d

Supplementary Figure 60. Brussel sprouts and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 61. Cabbage and type 2 diabetes, high vs. low

Supplementary Figure 62. Cabbage and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 63. Cabbage and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 64. Cauliflower and type 2 diabetes, high vs. low

Supplementary Figure 65. Cauliflower and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 66. Cauliflower and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 67. Cruciferous vegetables and type 2 diabetes, high vs. low

Supplementary Figure 68. Cruciferous vegetables and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 69. Cruciferous vegetables and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 70. Green leafy vegetables and type 2 diabetes, high vs. low

Supplementary Figure 71. Green leafy vegetables and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 72. Green leafy vegetables and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 73. Kale, mustard and chard greens and type 2 diabetes, high vs. low

Supplementary Figure 74. Kale, mustard and chard greens and type 2 diabetes, dose-response analysis per 10 g/d

Supplementary Figure 75. Kale, mustard and chard greens and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 76. Potatoes, boiled and type 2 diabetes, high vs. low

Supplementary Figure 77. Potatoes, boiled and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 78. Potatoes, boiled and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 79. Potatoes, total and type 2 diabetes, high vs. low

Supplementary Figure 80. Potatoes, total and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 81. Potatoes, total and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 82. Tomatoes and type 2 diabetes, high vs. low

Supplementary Figure 83. Tomatoes and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 84. Tomatoes and type 2 diabetes, nonlinear dose-response analysis

Supplementary Figure 85. Yellow vegetables and type 2 diabetes, high vs. low

Supplementary Figure 86. Yellow vegetables and type 2 diabetes, dose-response analysis per 100 g/d

Supplementary Figure 87. Yellow vegetables and type 2 diabetes, nonlinear dose-response analysis

Funnel plots

Supplementary Figure 88. Funnel plot of fruit and vegetables and type 2 diabetes

Supplementary Figure 89. Funnel plot of fruits and type 2 diabetes

Supplementary Figure 90. Funnel plot of vegetables and type 2 diabetes

Supplementary Figure 91. Funnel plot of potatoes and type 2 diabetes

Supplementary Figure 92. Funnel plot of green leafy vegetables and type 2 diabetes

Supplementary Figure 93. Funnel plot of cruciferous vegetables and type 2 diabetes

Influence analyses

Supplementary Figure 94. Influence analysis of fruit and vegetables and type 2 diabetes

Supplementary Figure 95. Influence analysis of fruits and type 2 diabetes

Supplementary Figure 96. Influence analysis of vegetables and type 2 diabetes

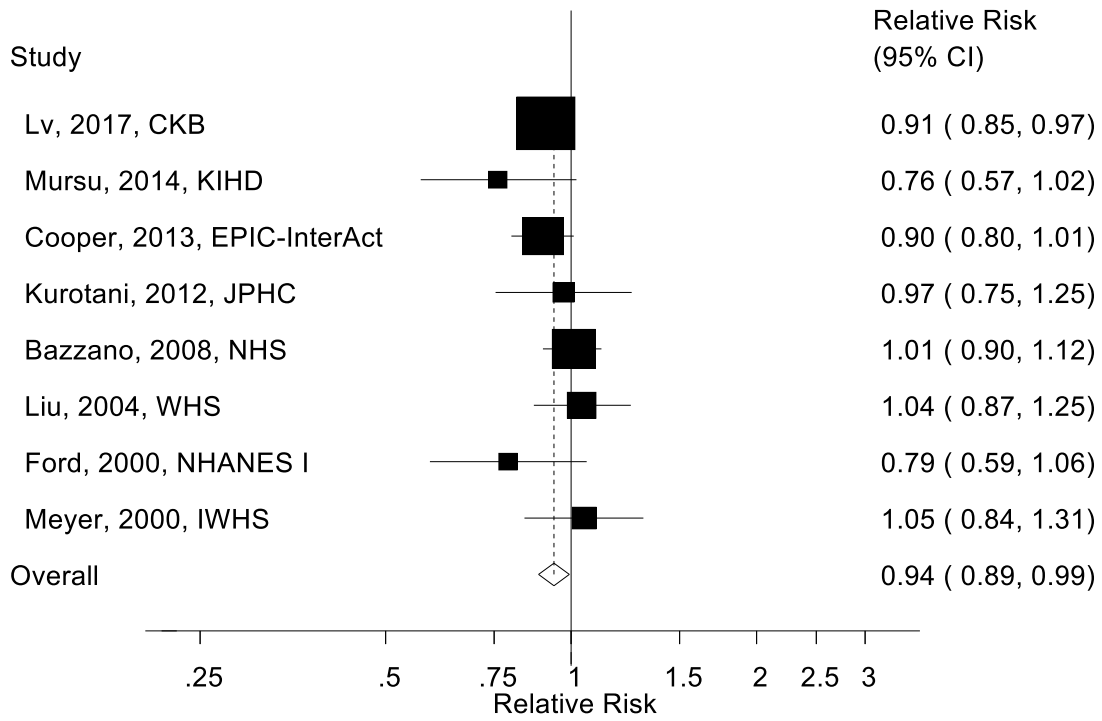
Supplementary Figure 97. Influence analysis of potatoes and type 2 diabetes

Supplementary Figure 98. Influence analysis of cruciferous vegetables and type 2 diabetes

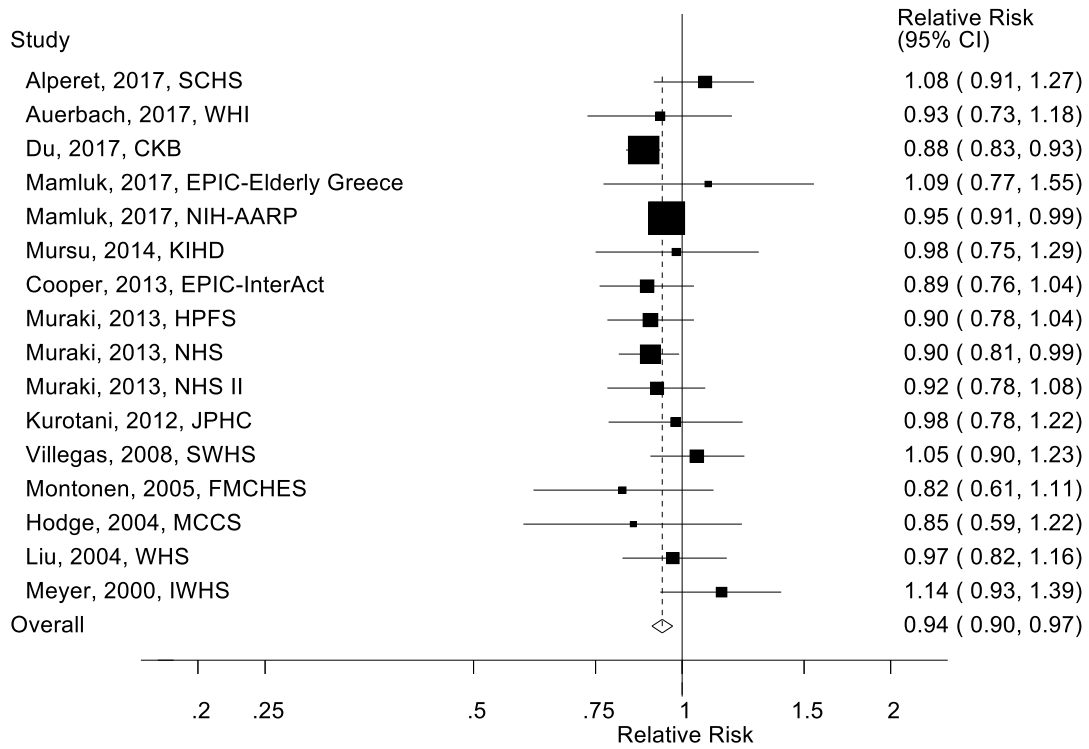
Supplementary Figure 99. Influence analysis of green leafy vegetables and type 2 diabetes

Supplementary Figure 95. Influence analysis of fruits drinks and type 2 diabetes (excluding Montonen because of extreme result)

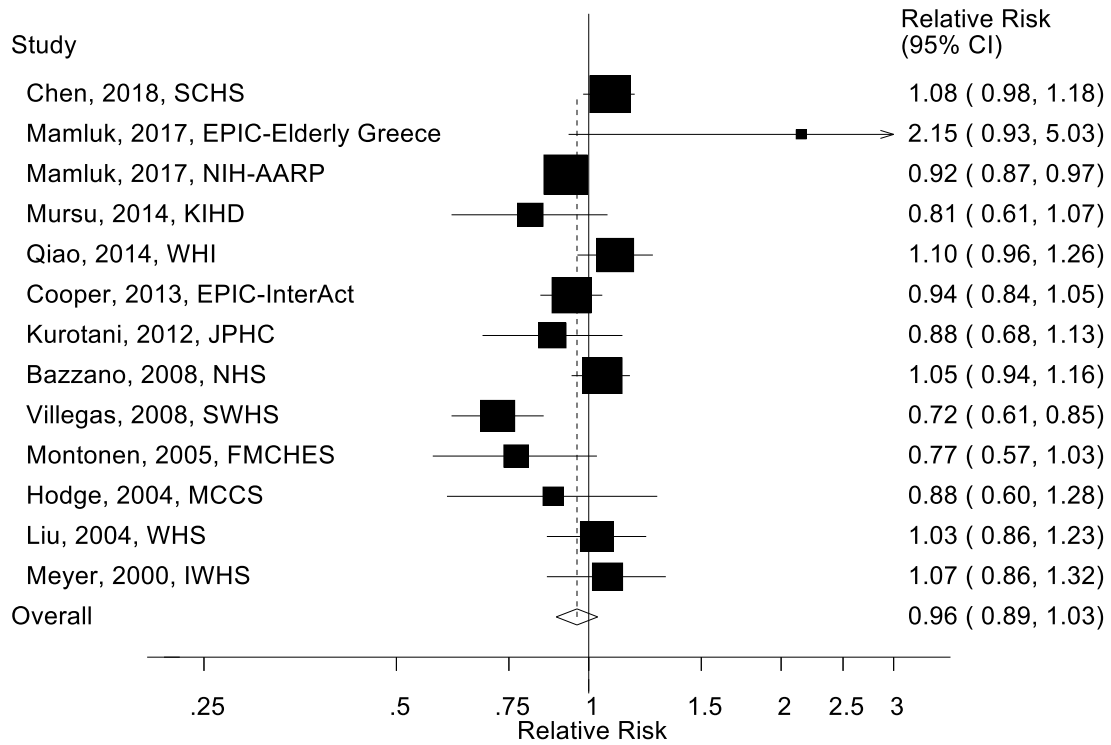
Supplementary Figure 1. Fruit and vegetables and type 2 diabetes, high vs. low



Supplementary Figure 2. Fruits and type 2 diabetes, high vs. low

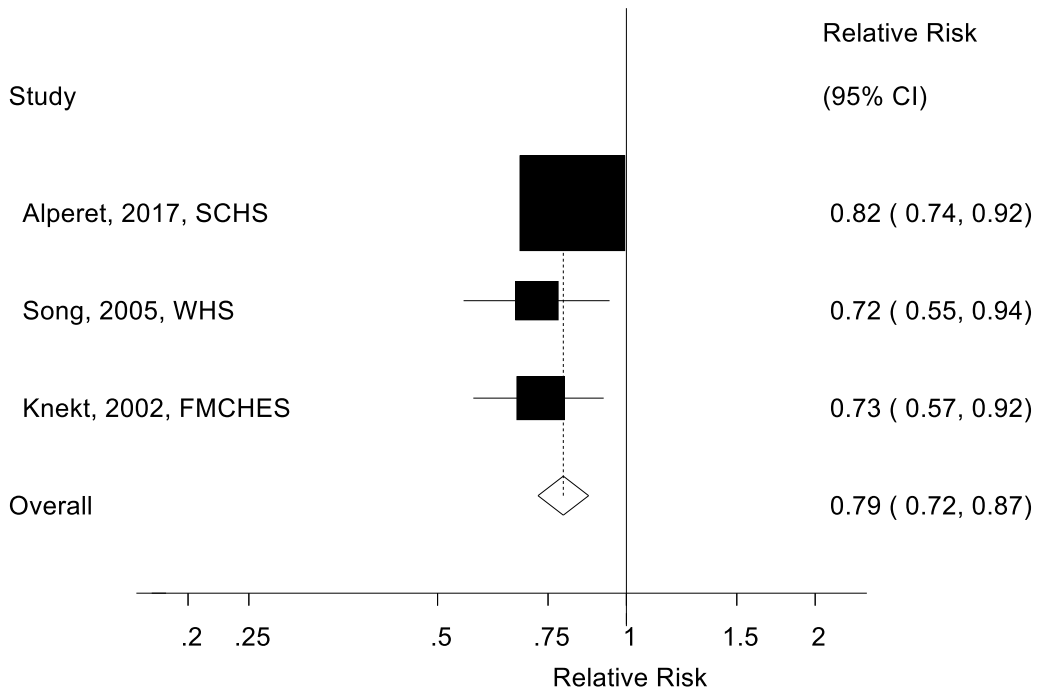


Supplementary Figure 3. Vegetables and type 2 diabetes, high vs. low

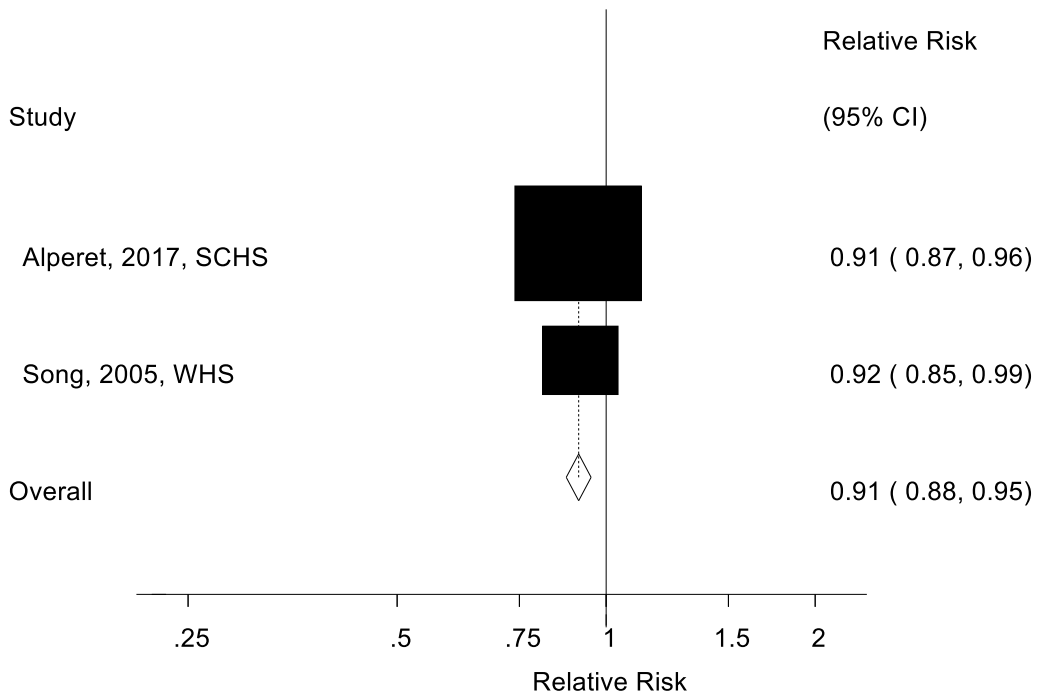


Supplementary figures of subtypes of fruit, high vs. low, linear and nonlinear dose response analyses

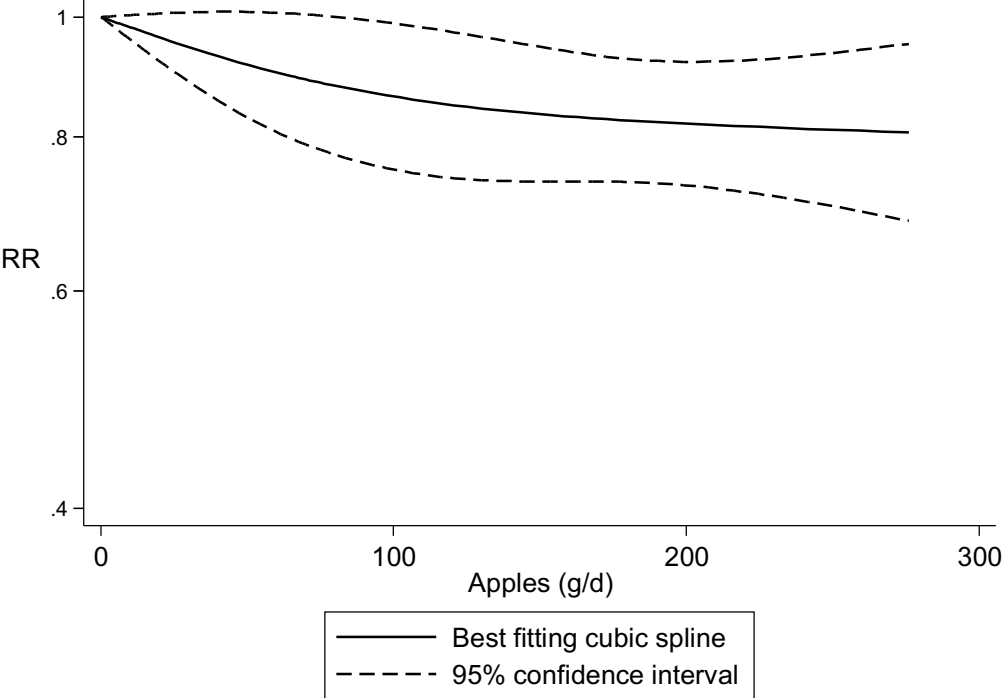
Supplementary Figure 4. Apples and type 2 diabetes, high vs. low



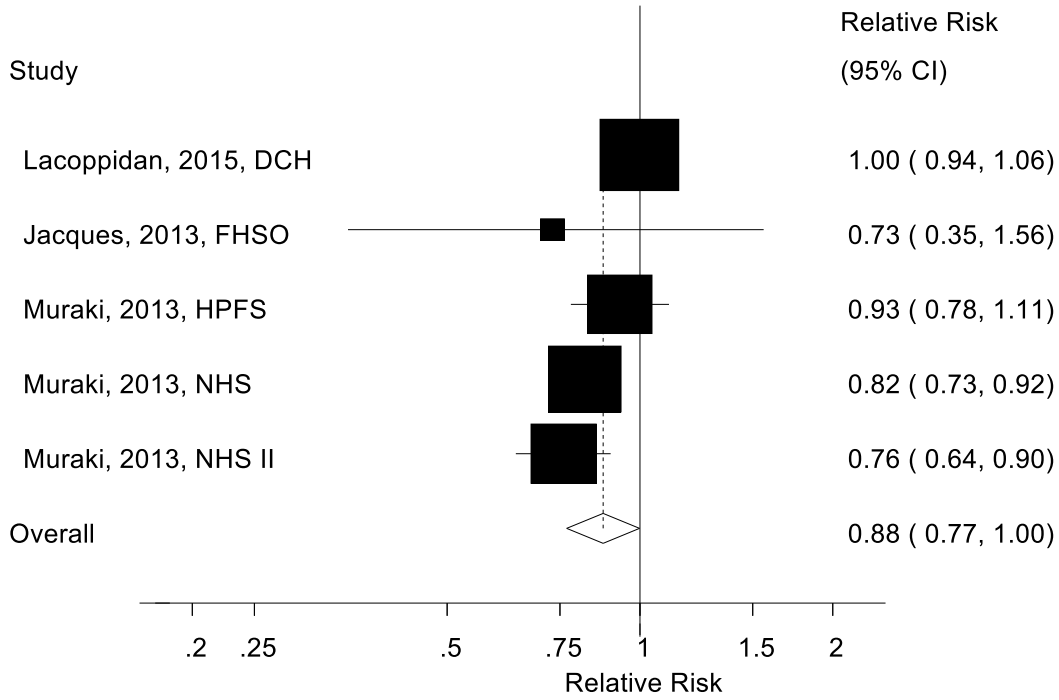
Supplementary Figure 5. Apples and type 2 diabetes, dose-response analysis per 100 g/d



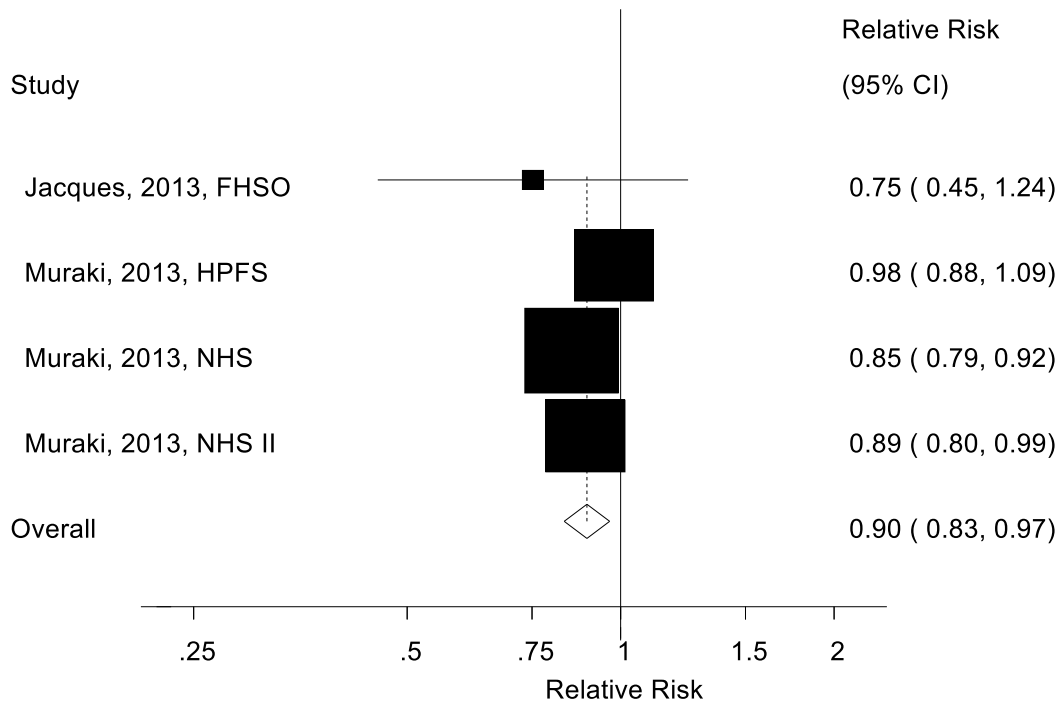
Supplementary Figure 6. Apples and type 2 diabetes, nonlinear dose-response analysis



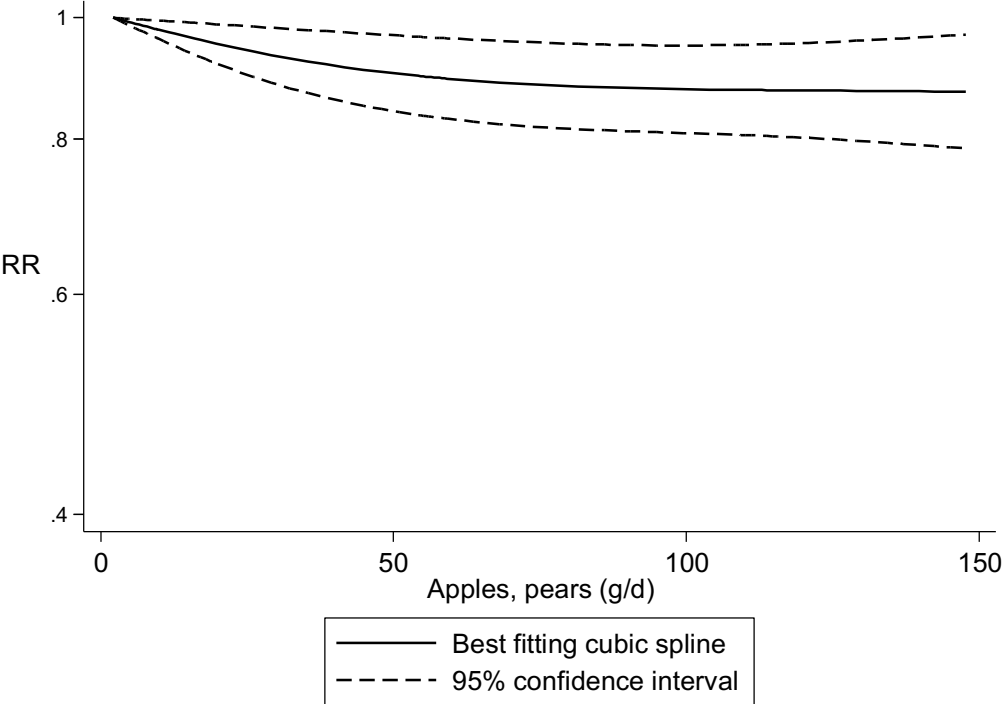
Supplementary Figure 7. Apples and pears and type 2 diabetes, high vs. low



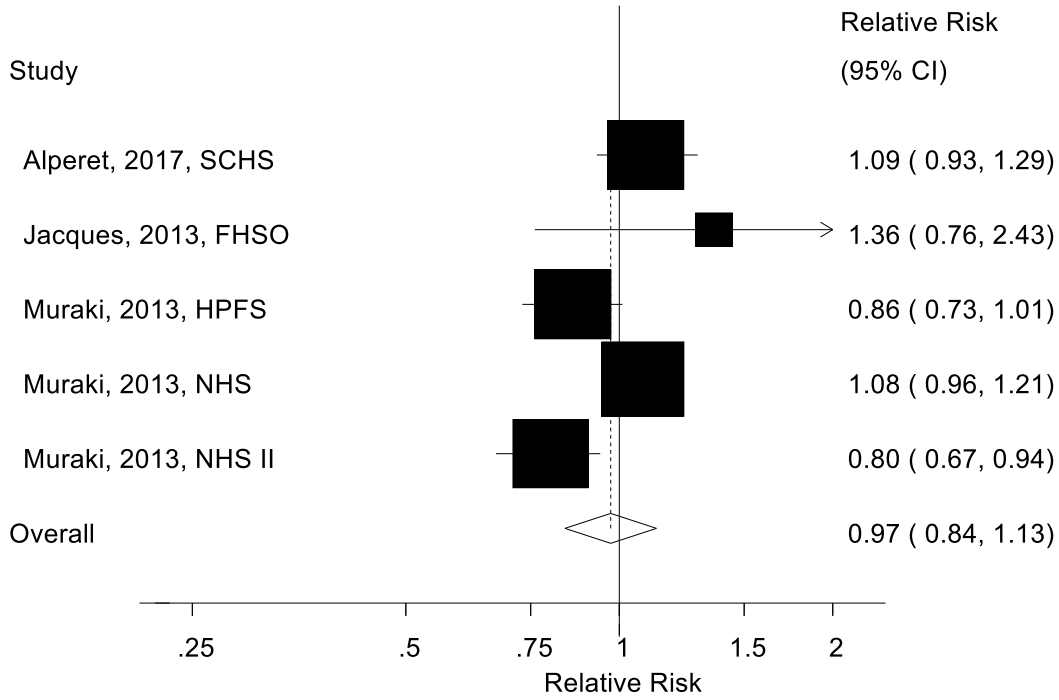
Supplementary Figure 8. Apples and pears and type 2 diabetes, dose-response analysis per 100 g/d



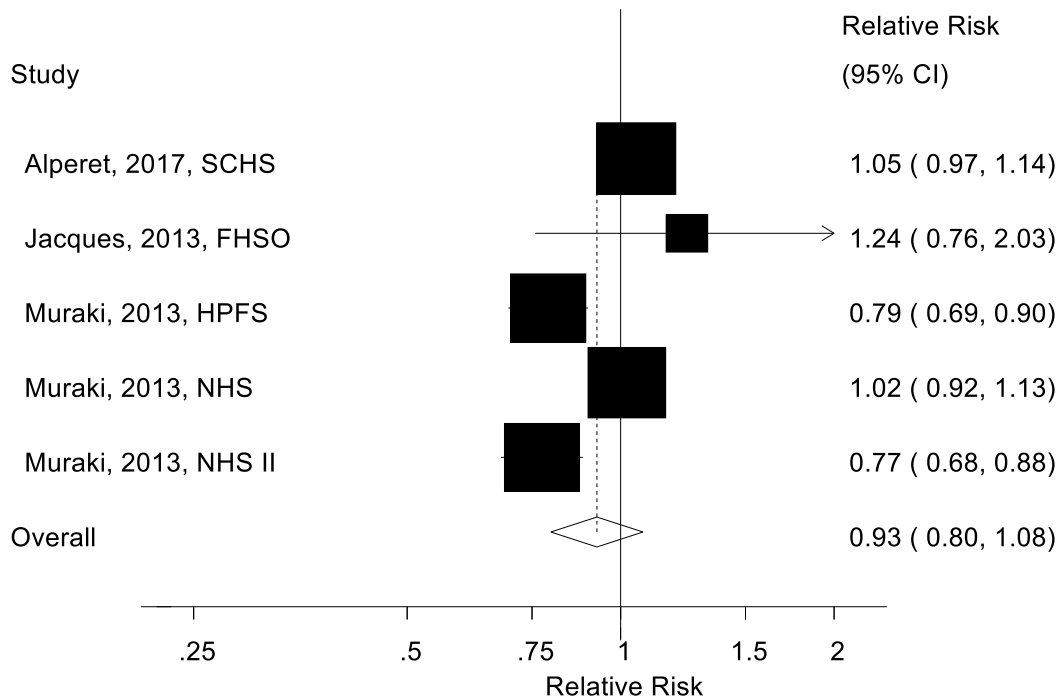
Supplementary Figure 9. Apples and pears and type 2 diabetes, nonlinear dose-response analysis



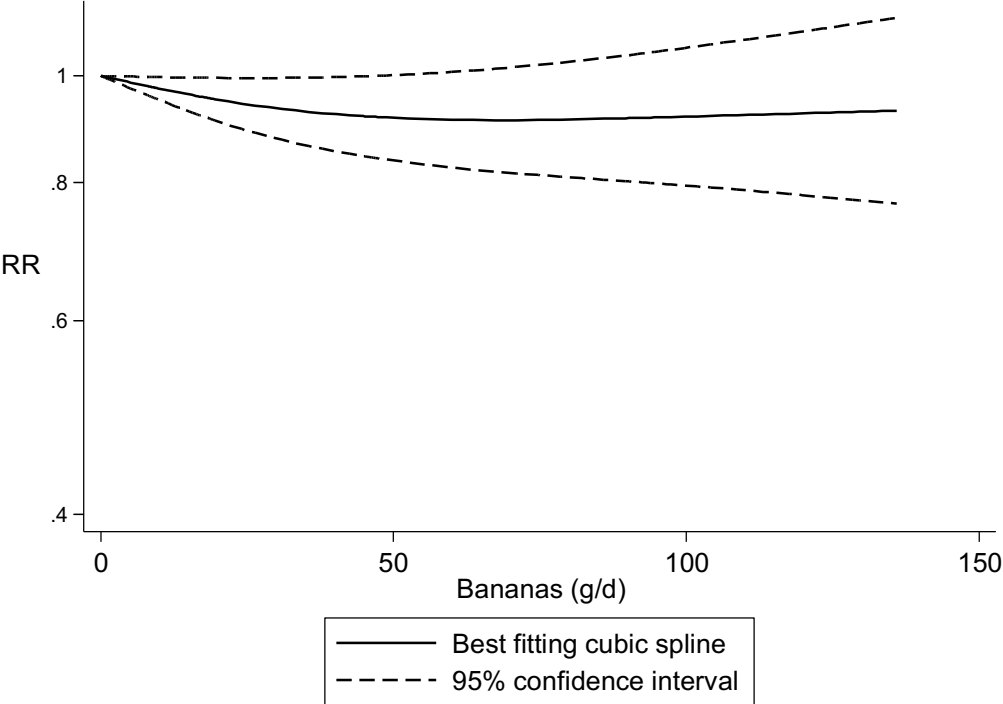
Supplementary Figure 10. Bananas and type 2 diabetes, high vs. low



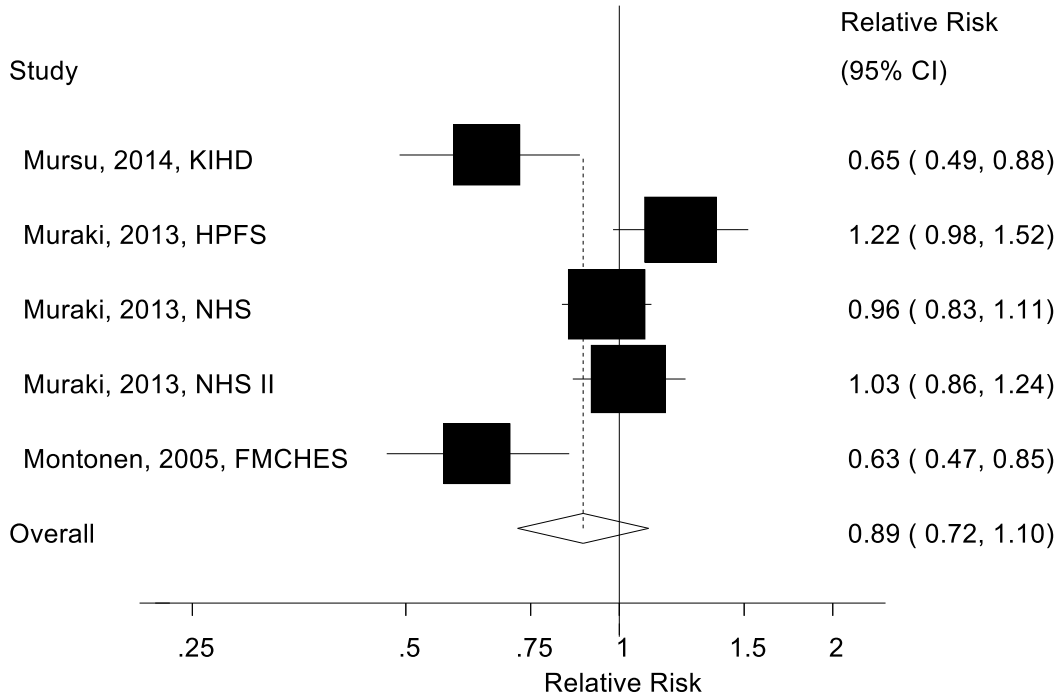
Supplementary Figure 11. Bananas and type 2 diabetes, dose-response analysis per 100 g/d



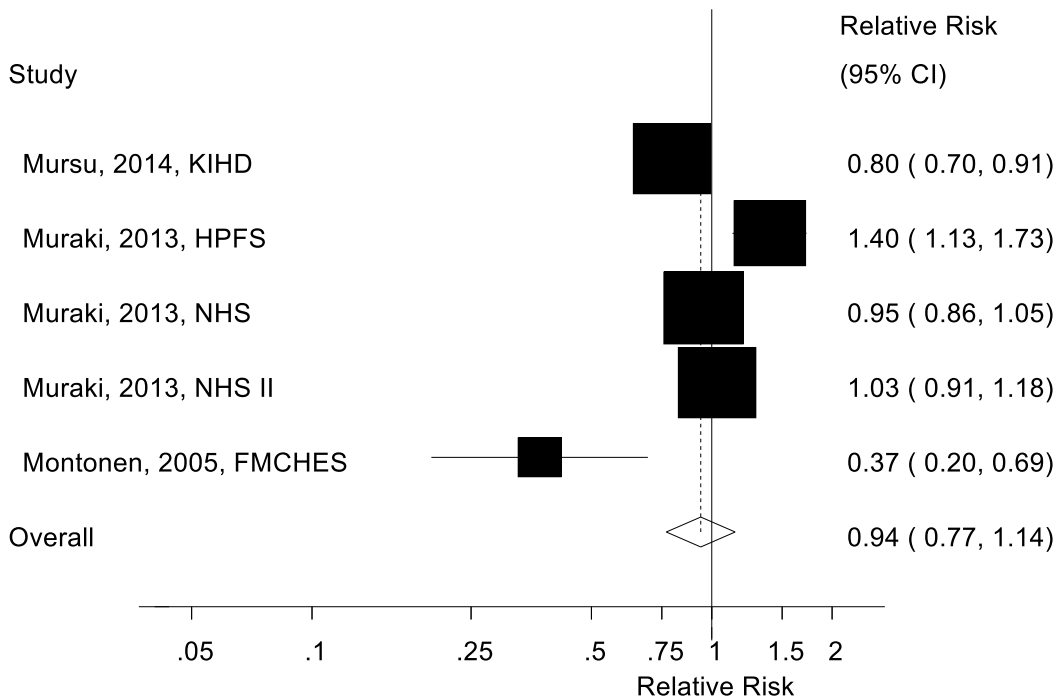
Supplementary Figure 12. Bananas and type 2 diabetes, nonlinear dose-response analysis



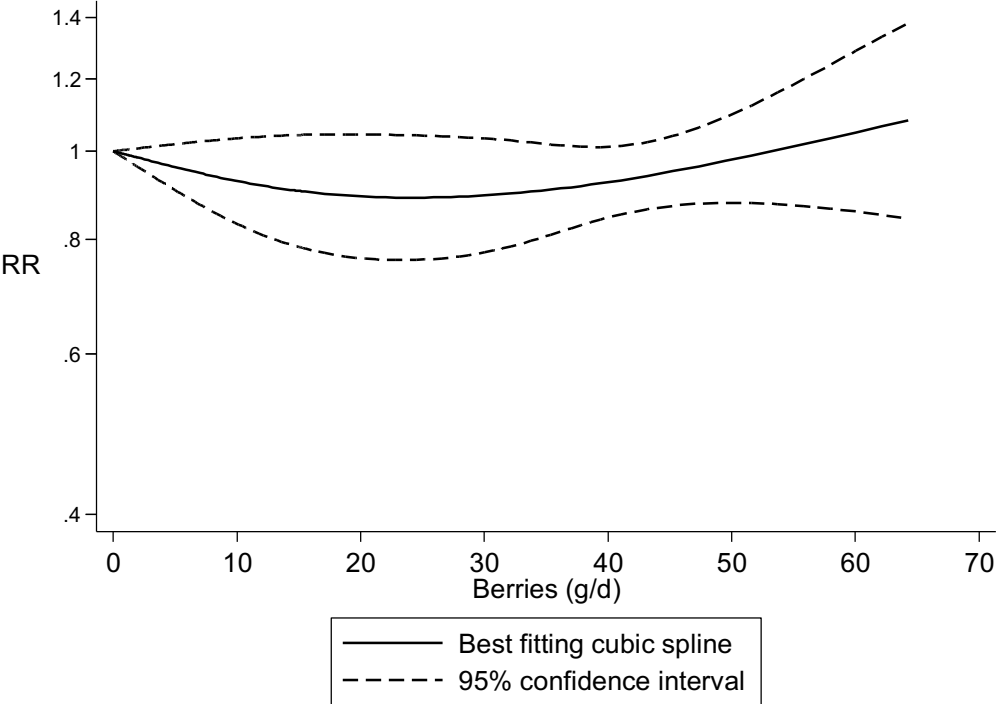
Supplementary Figure 13. Berries and type 2 diabetes, high vs. low



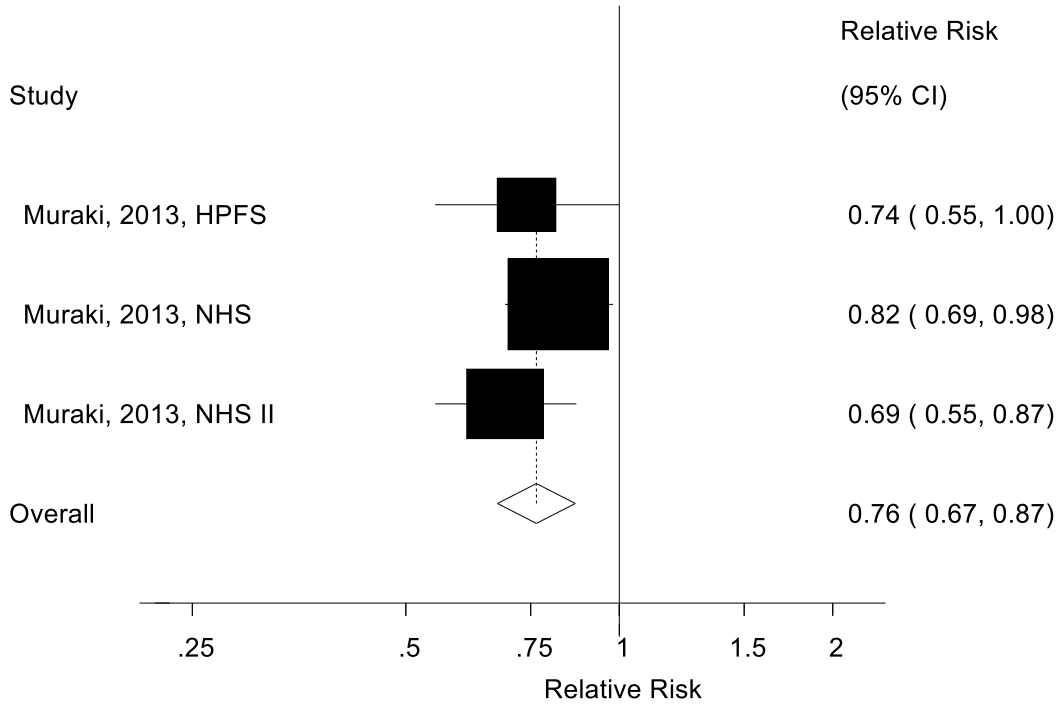
Supplementary Figure 14. Berries and type 2 diabetes, dose-response analysis per 50 g/d



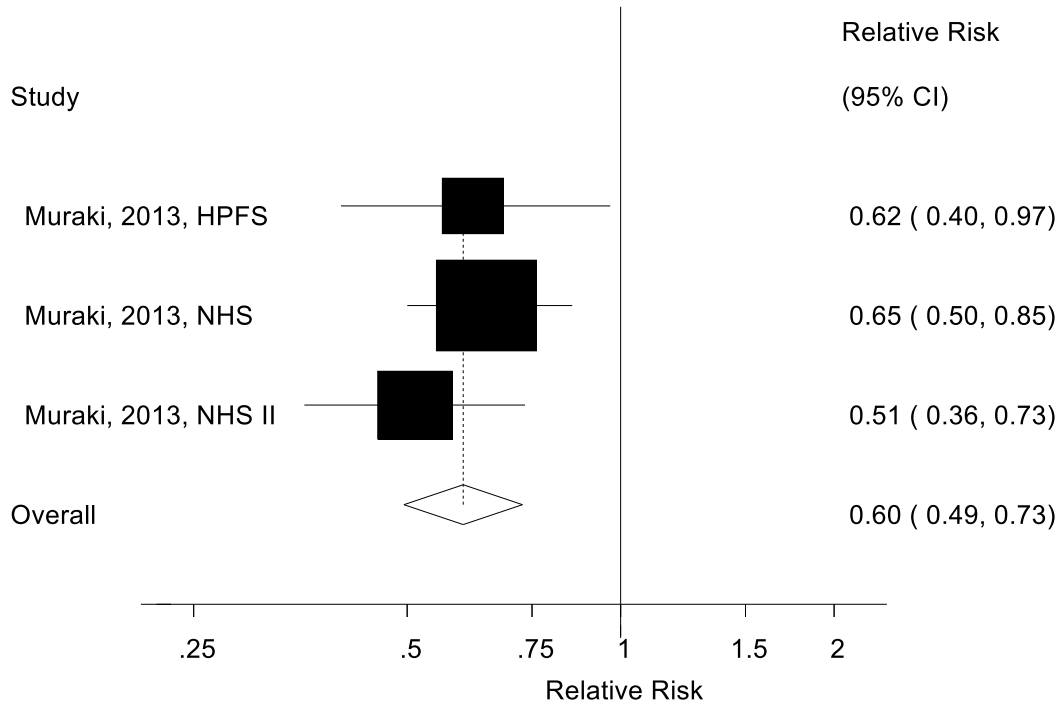
Supplementary Figure 15. Berries and type 2 diabetes, nonlinear dose-response analysis



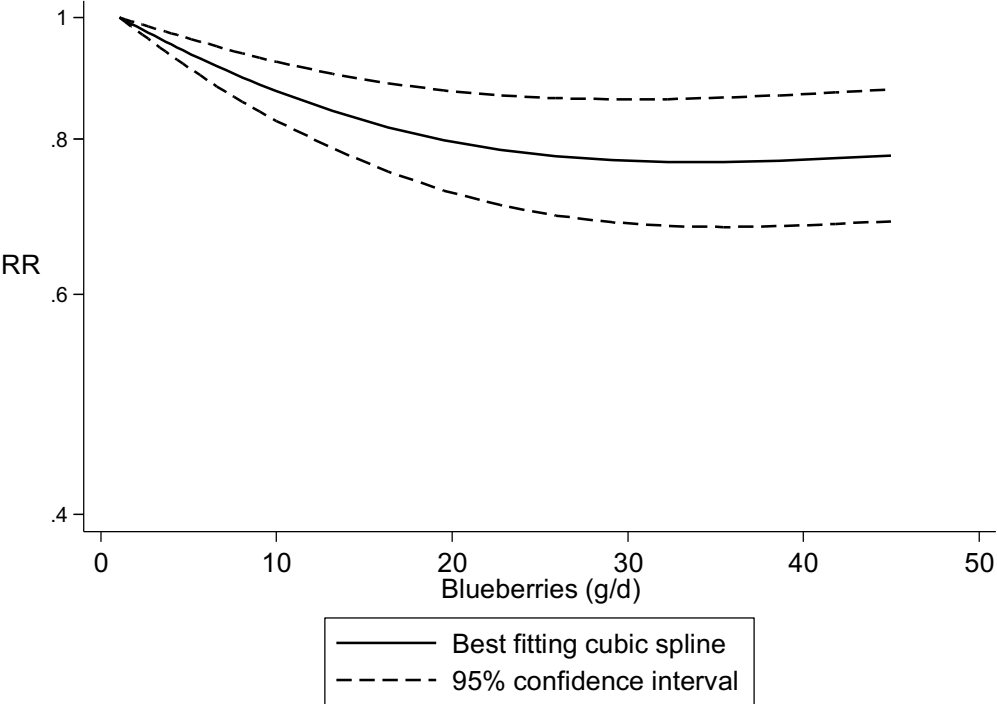
Supplementary Figure 16. Blueberries and type 2 diabetes, high vs. low



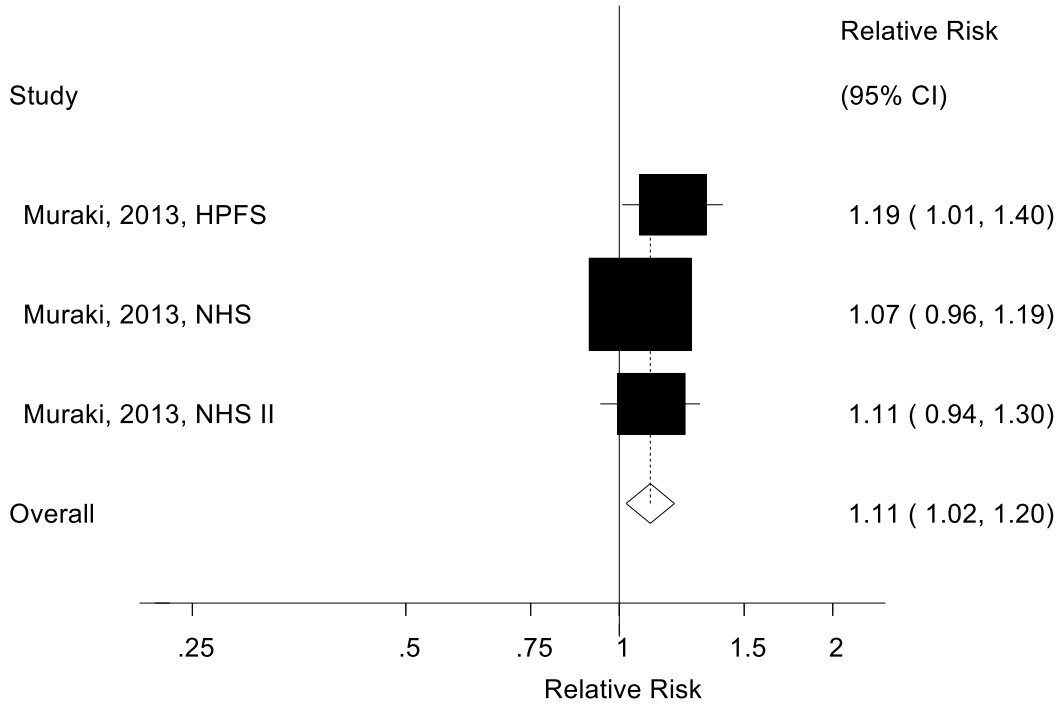
Supplementary Figure 17. Blueberries and type 2 diabetes, dose-response analysis per 50 g/d



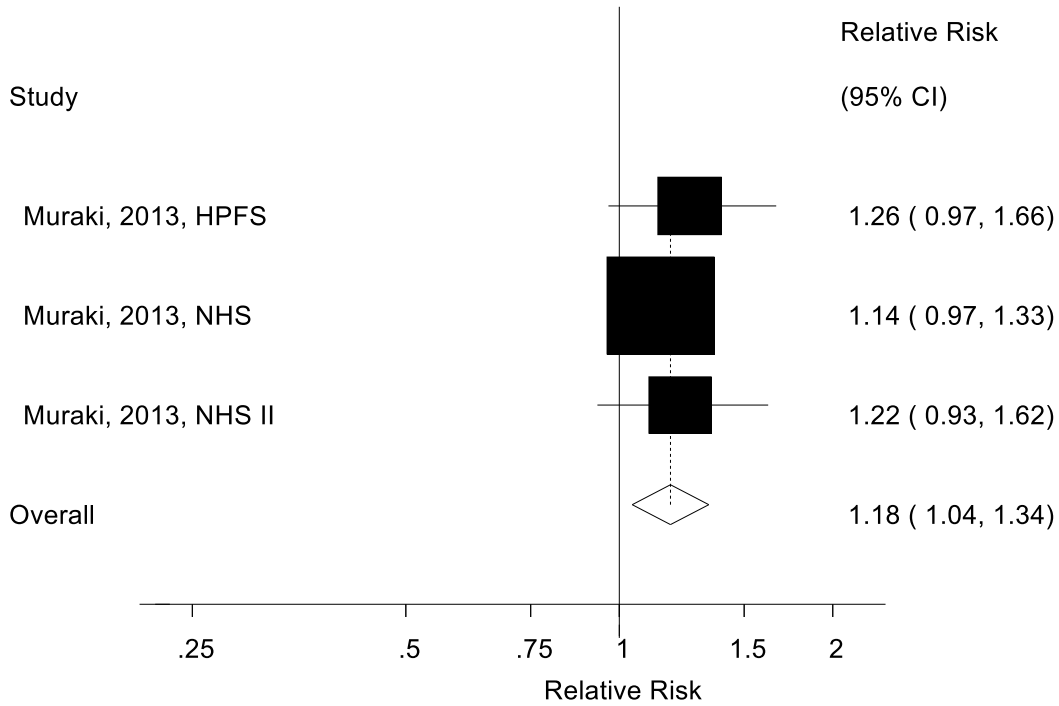
Supplementary Figure 18. Blueberries and type 2 diabetes, nonlinear dose-response analysis



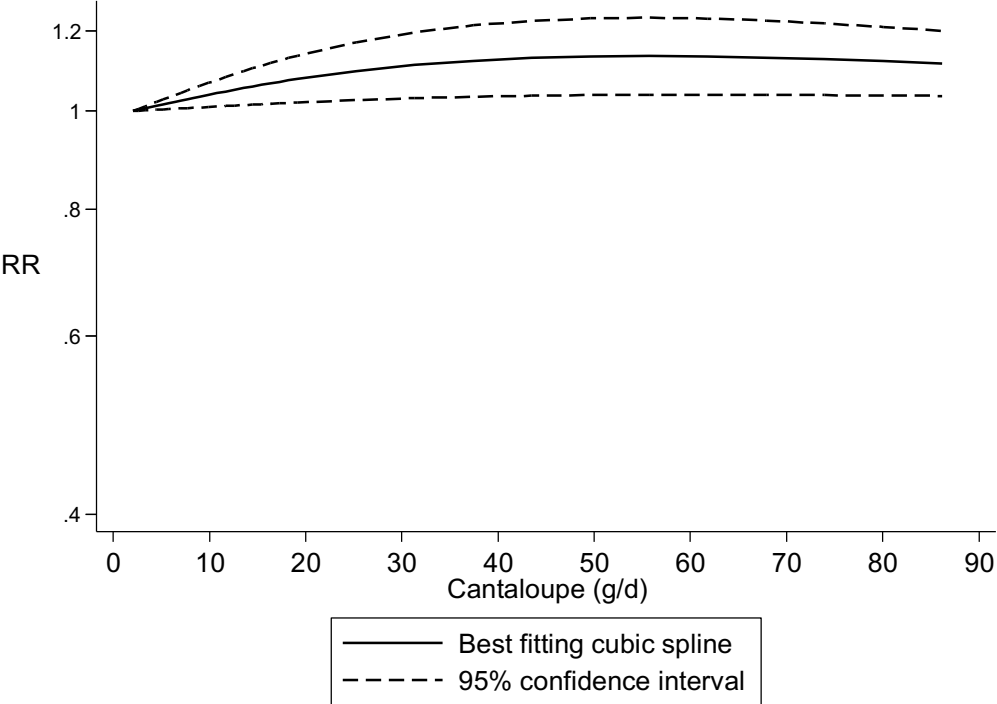
Supplementary Figure 19. Cantaloupe and type 2 diabetes, high vs. low



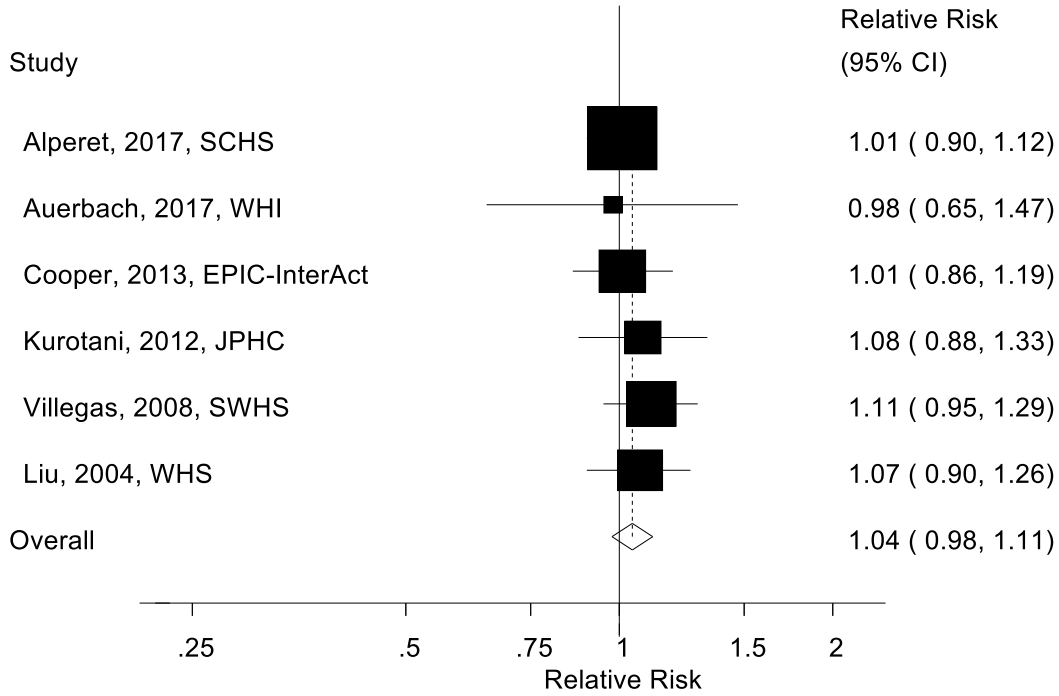
Supplementary Figure 20. Cantaloupe and type 2 diabetes, dose-response analysis per 100 g/d



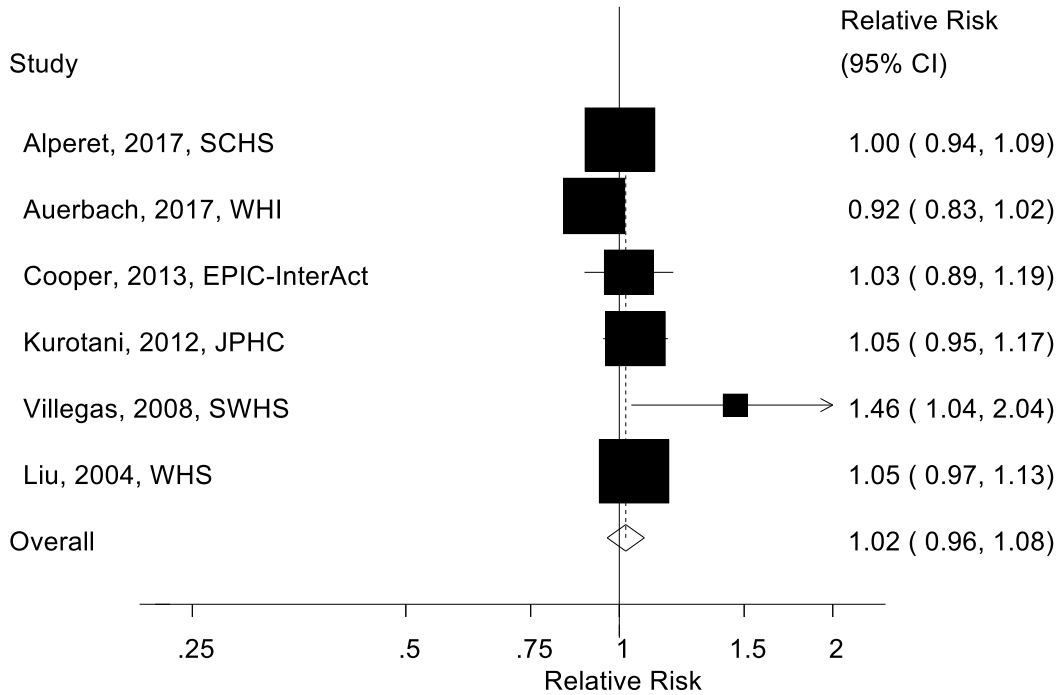
Supplementary Figure 21. Cantaloupe and type 2 diabetes, nonlinear dose-response analysis



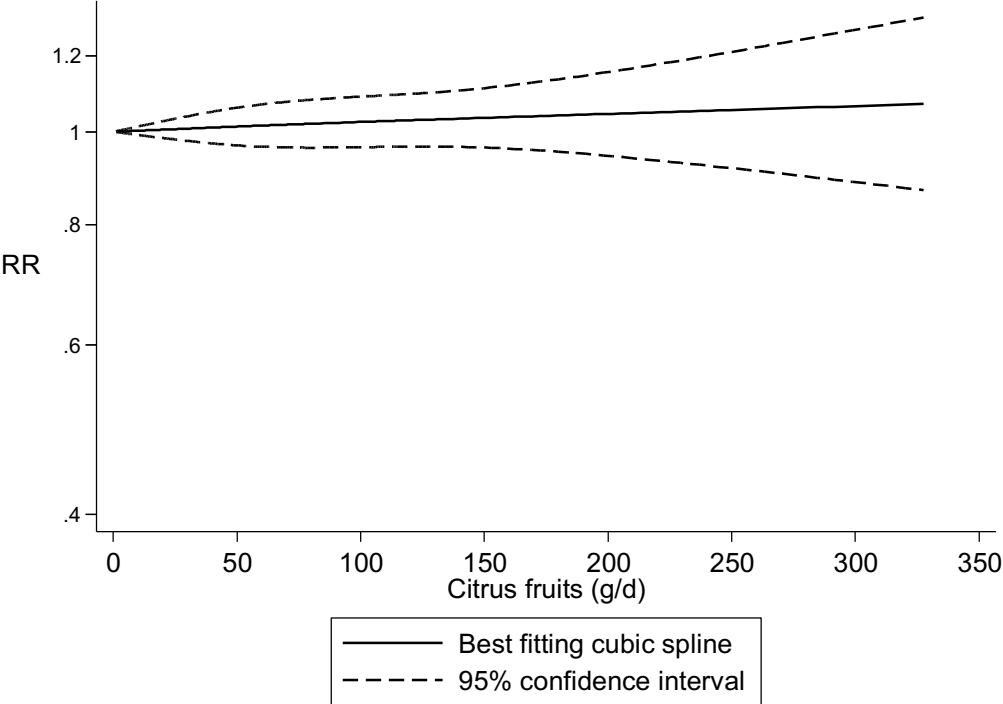
Supplementary Figure 22. Citrus fruits and type 2 diabetes, high vs. low



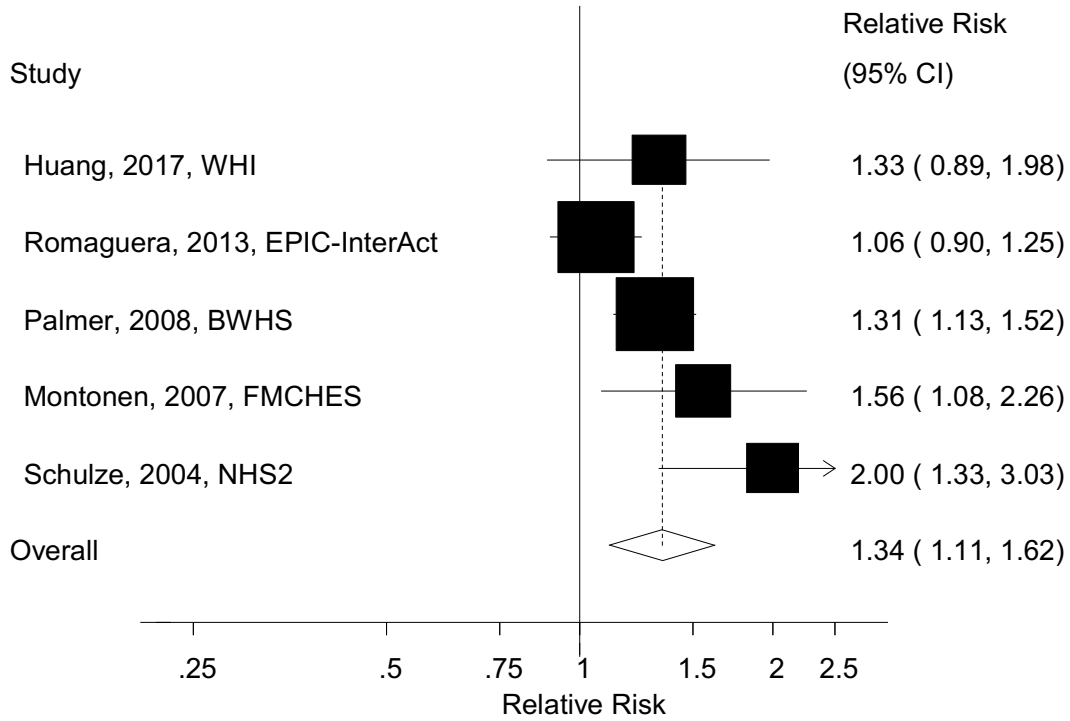
Supplementary Figure 23. Citrus fruits and type 2 diabetes, dose-response analysis per 100 g/d



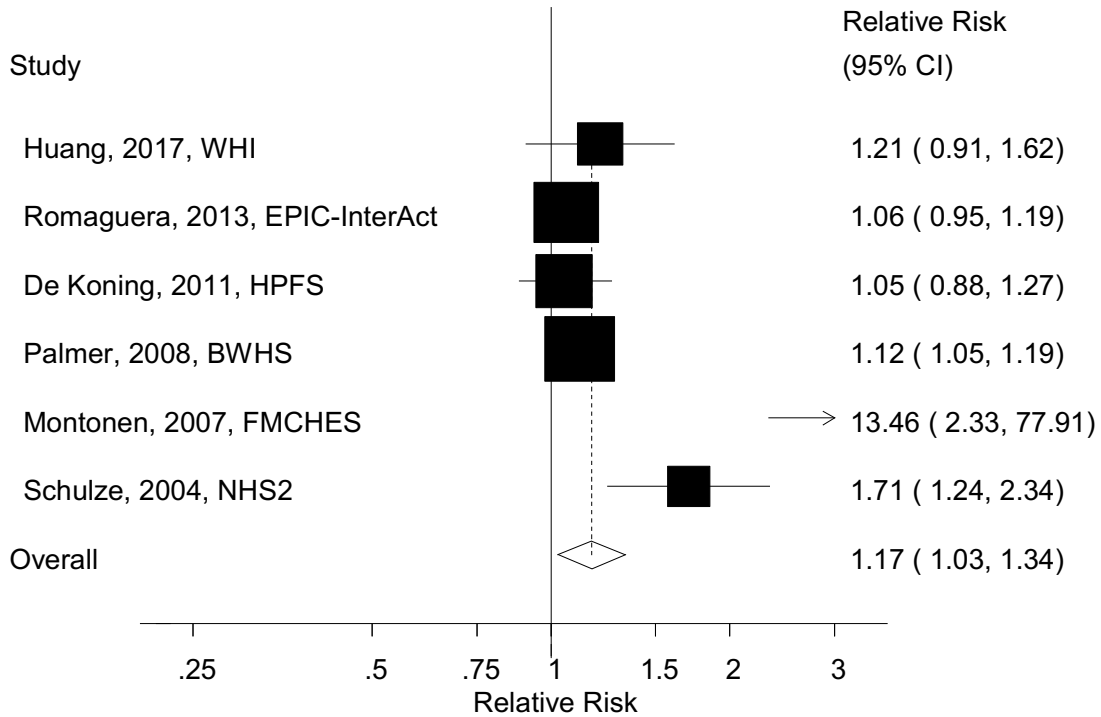
Supplementary Figure 24. Citrus fruits and type 2 diabetes, nonlinear dose-response analysis



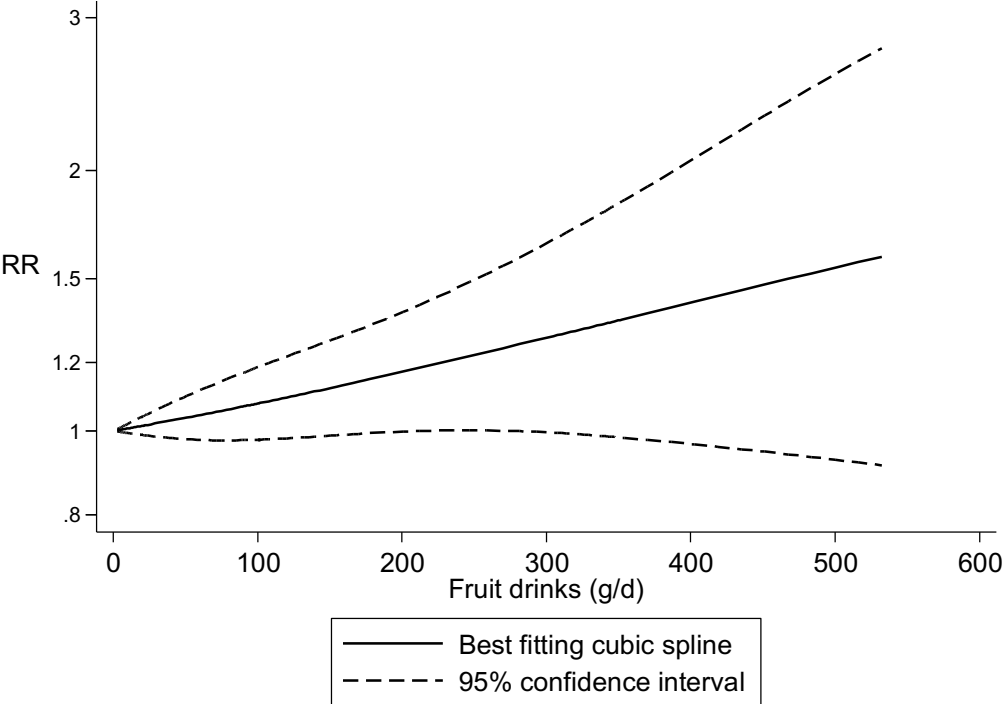
Supplementary Figure 25. Fruit drinks and type 2 diabetes, high vs. low



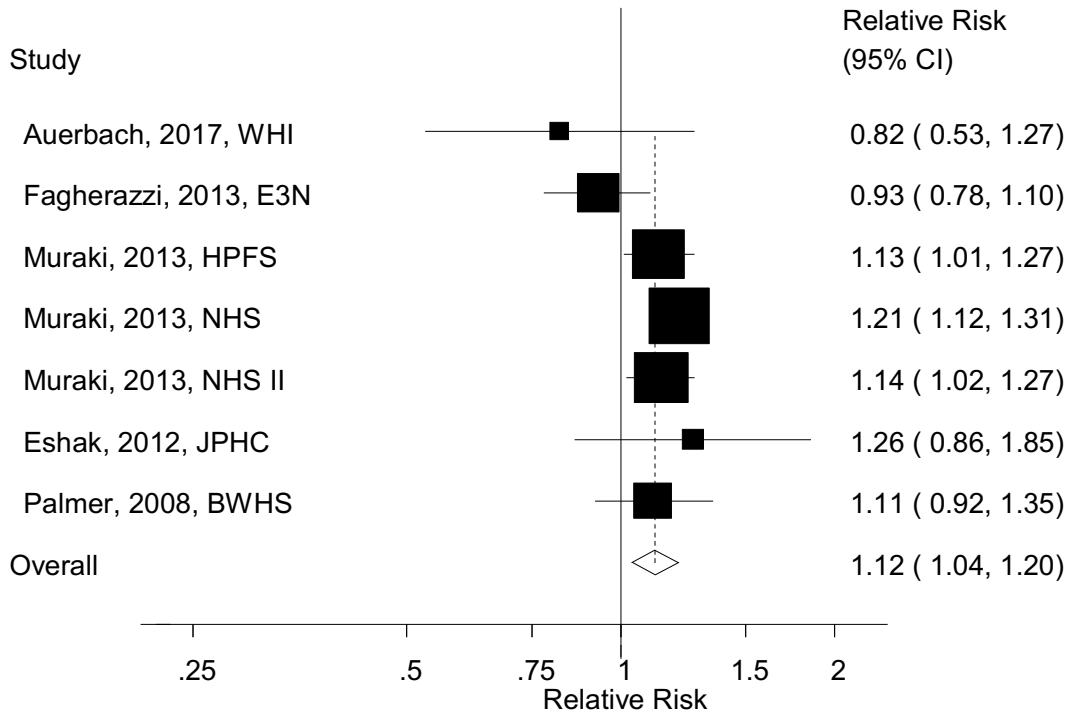
Supplementary Figure 26. Fruit drinks and type 2 diabetes, dose-response analysis per 250 g/d



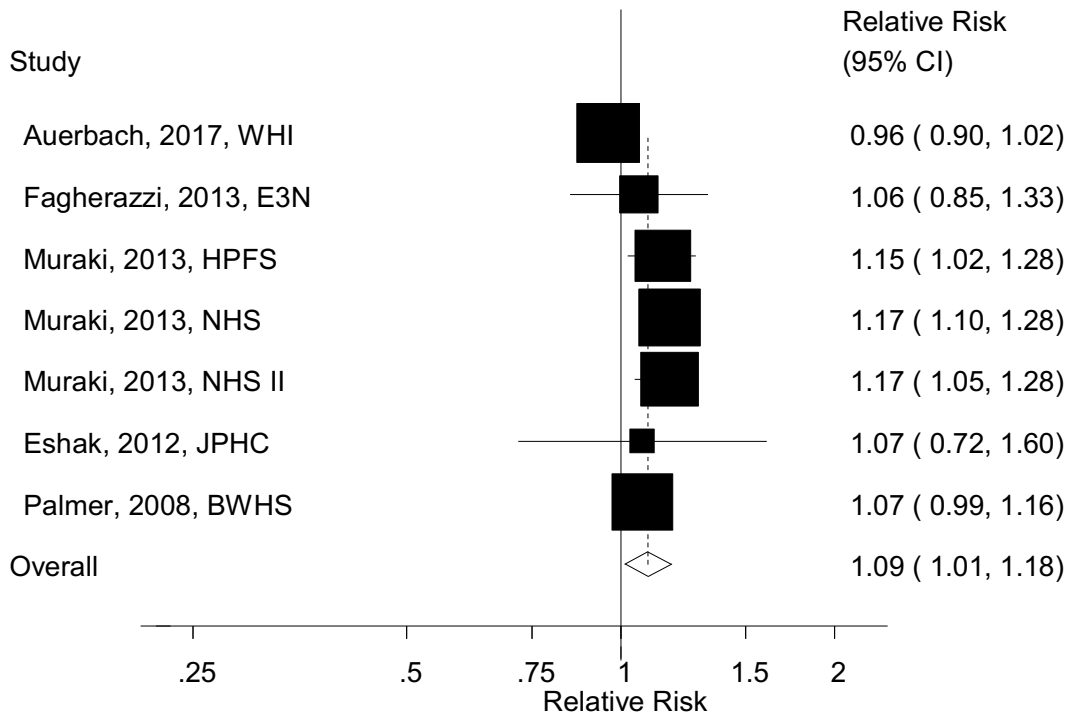
Supplementary Figure 27. Fruit juice and type 2 diabetes, nonlinear dose-response analysis



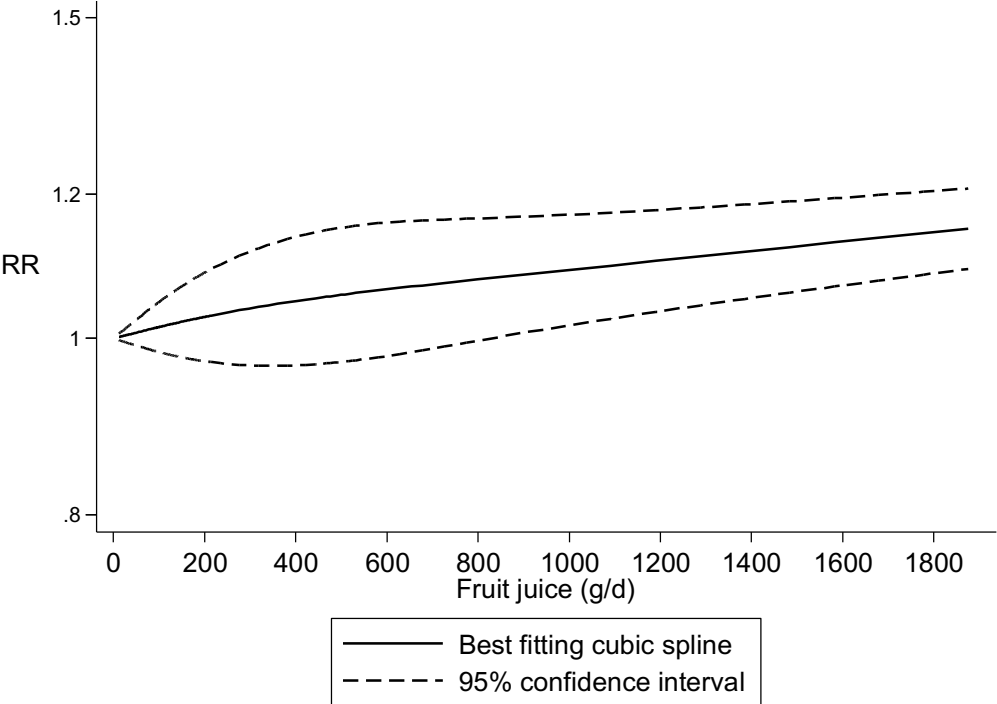
Supplementary Figure 28. Fruit juice and type 2 diabetes, high vs. low



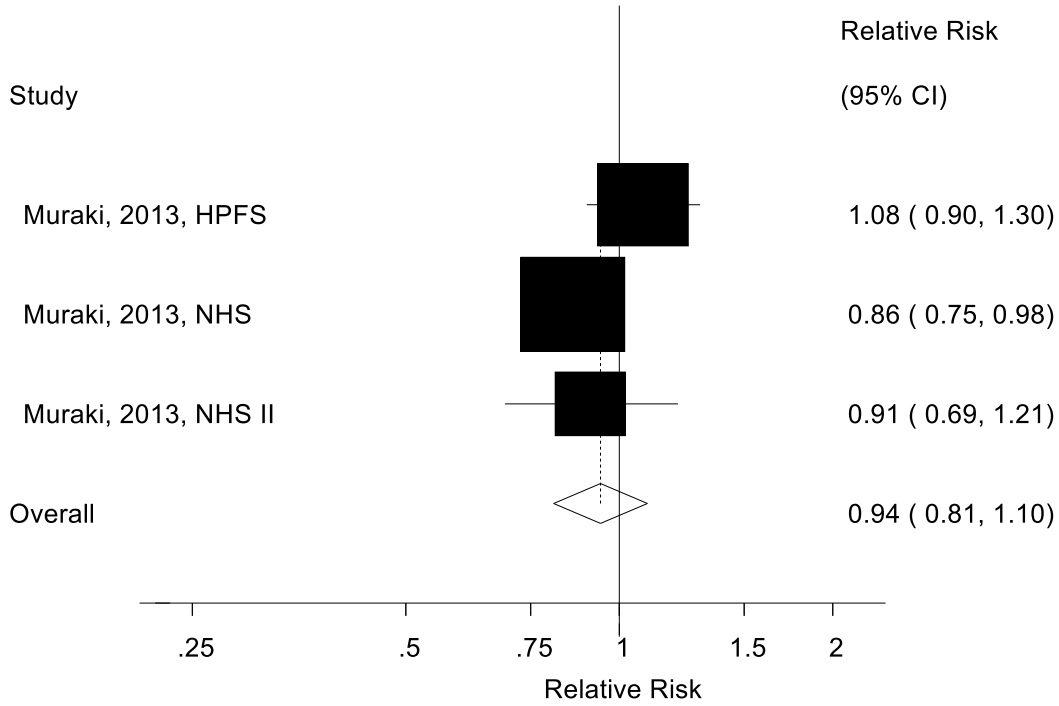
Supplementary Figure 29. Fruit juice and type 2 diabetes, dose-response analysis per 250 g/d



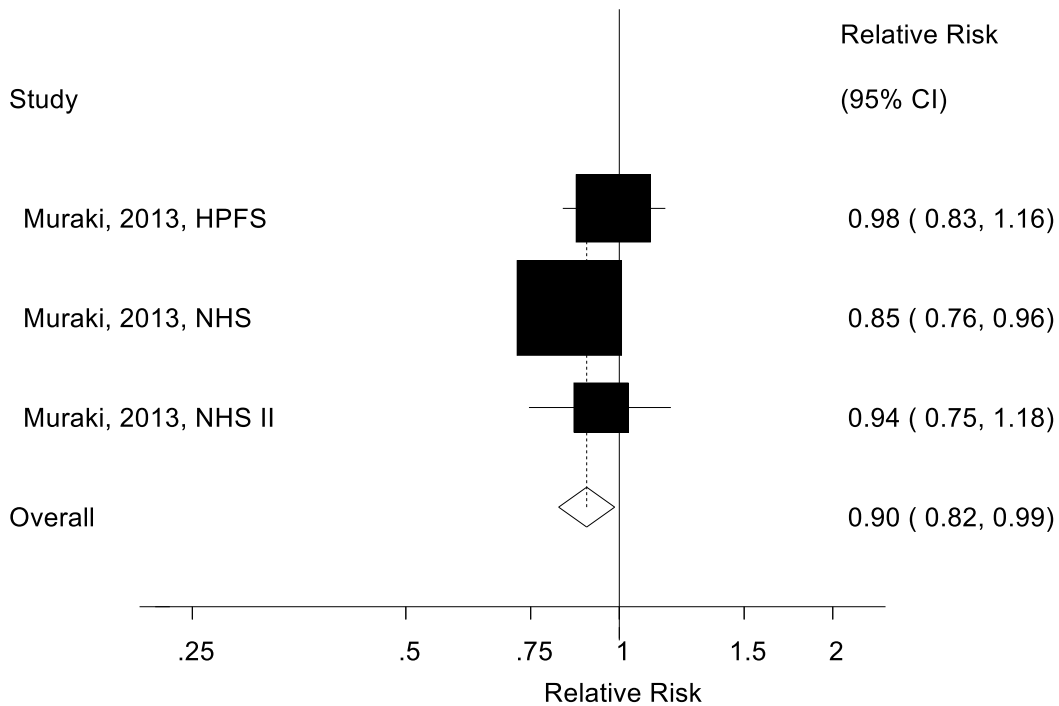
Supplementary Figure 30. Fruit juice and type 2 diabetes, nonlinear dose-response analysis



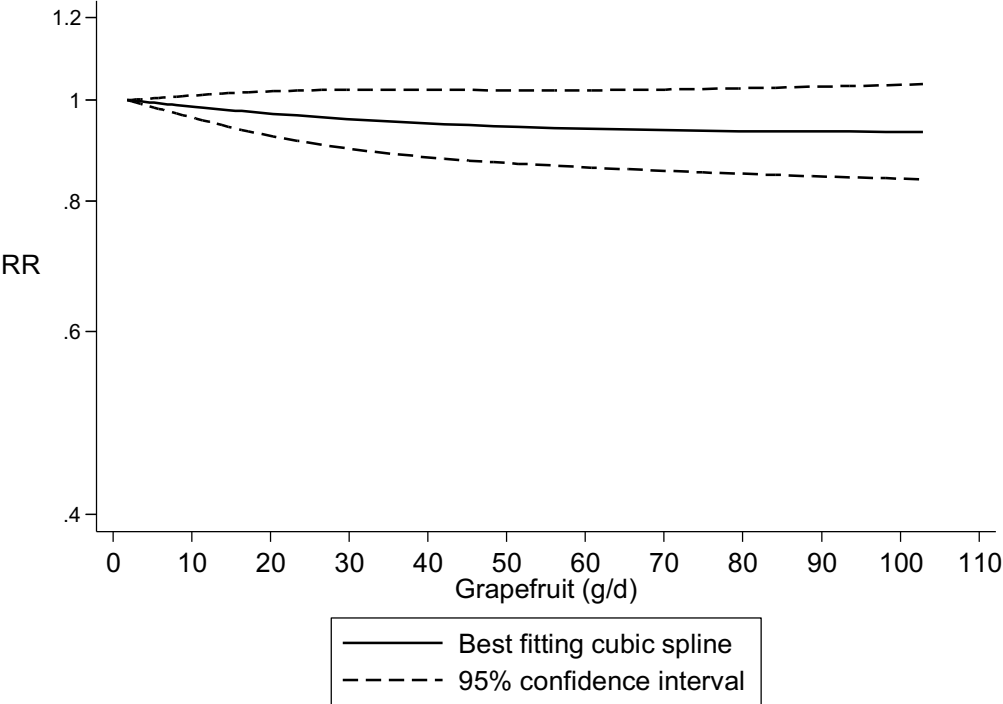
Supplementary Figure 31. Grapefruit and type 2 diabetes, high vs. low



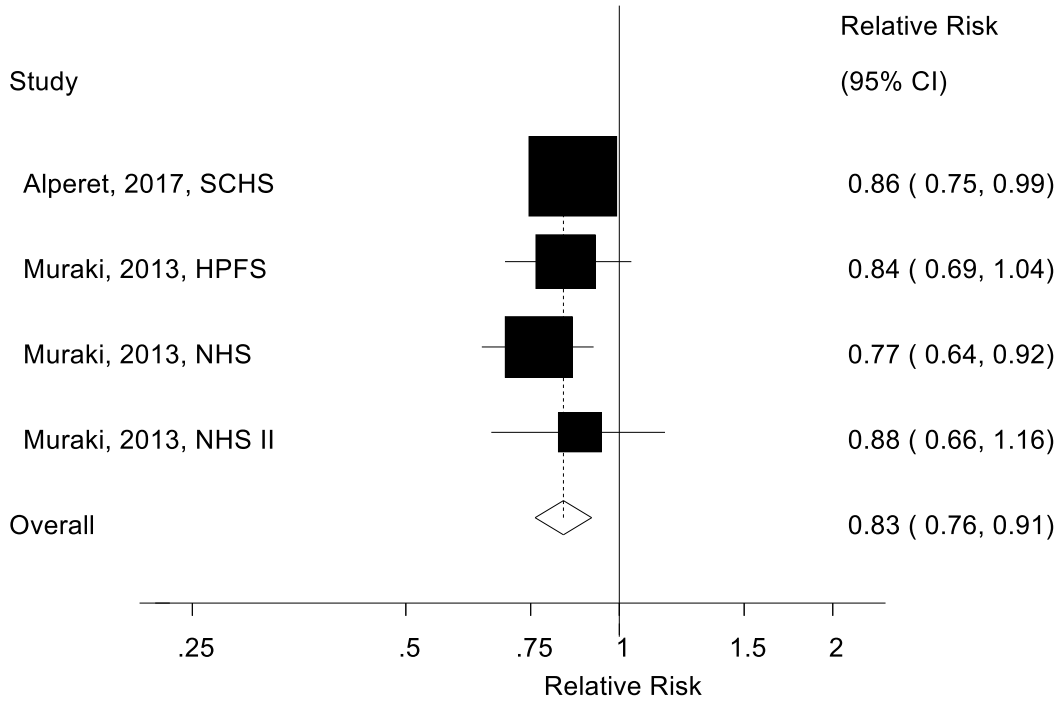
Supplementary Figure 32. Grapefruit and type 2 diabetes, dose-response analysis per 100 g/d



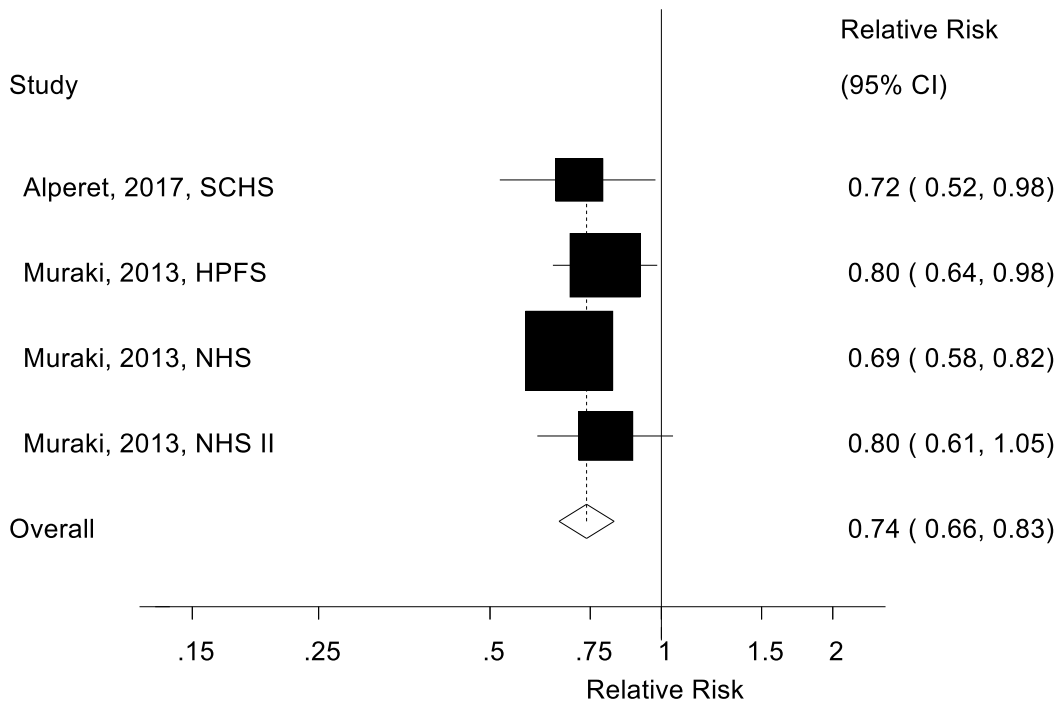
Supplementary Figure 33. Grapefruit and type 2 diabetes, nonlinear dose-response analysis



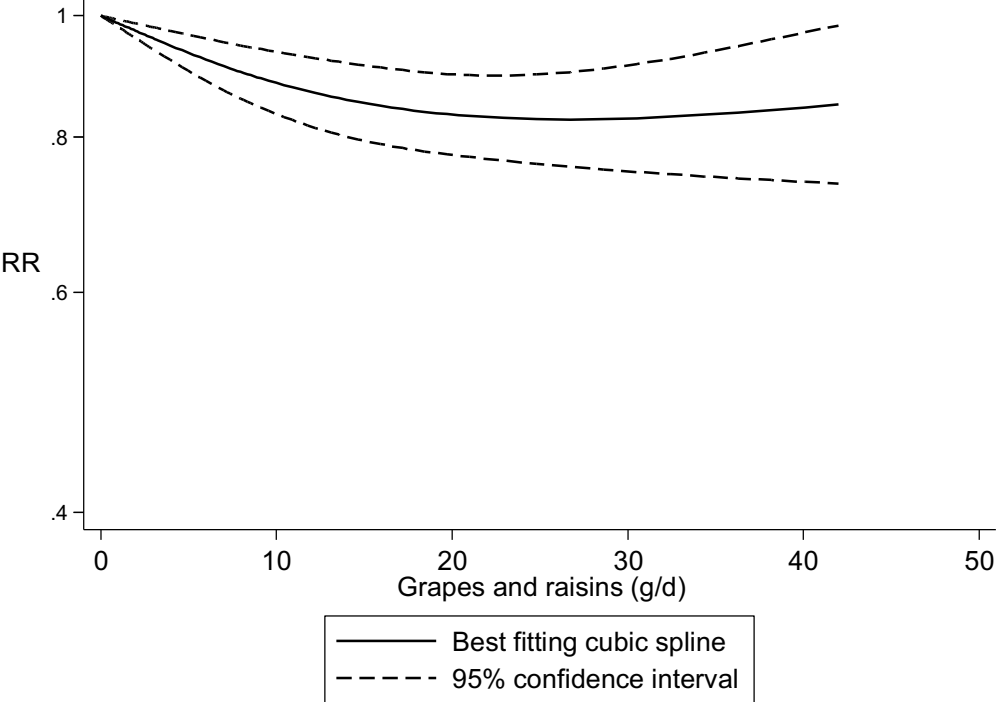
Supplementary Figure 34. Grapes and raisins and type 2 diabetes, high vs. low



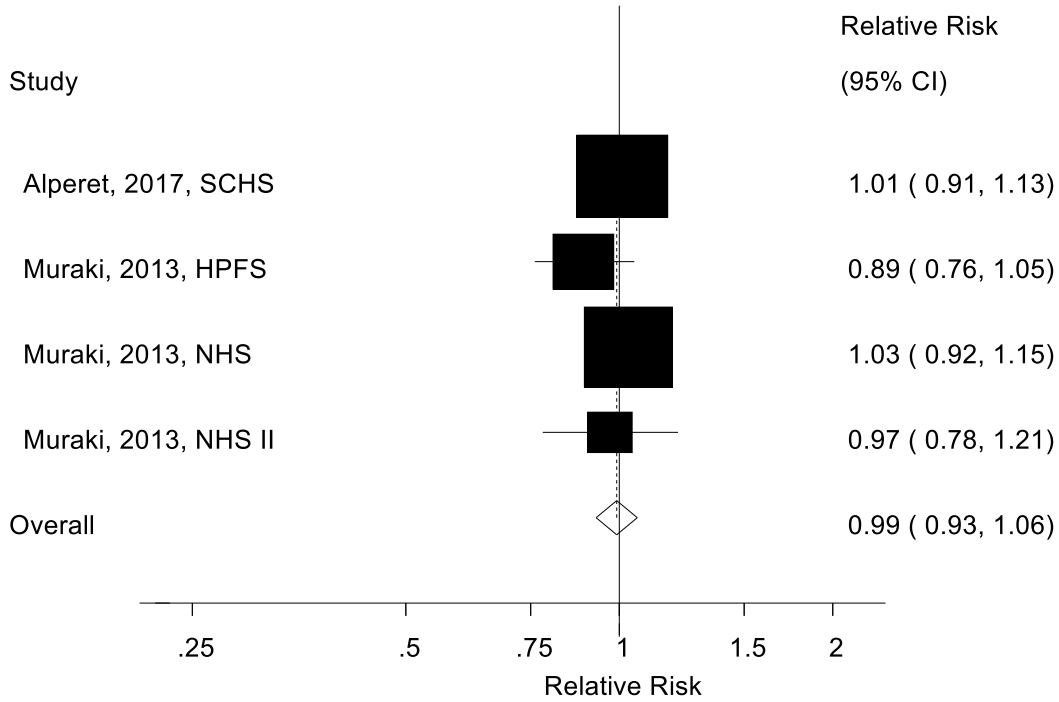
Supplementary Figure 35. Grapes and raisins and type 2 diabetes, dose-response analysis per 50 g/d



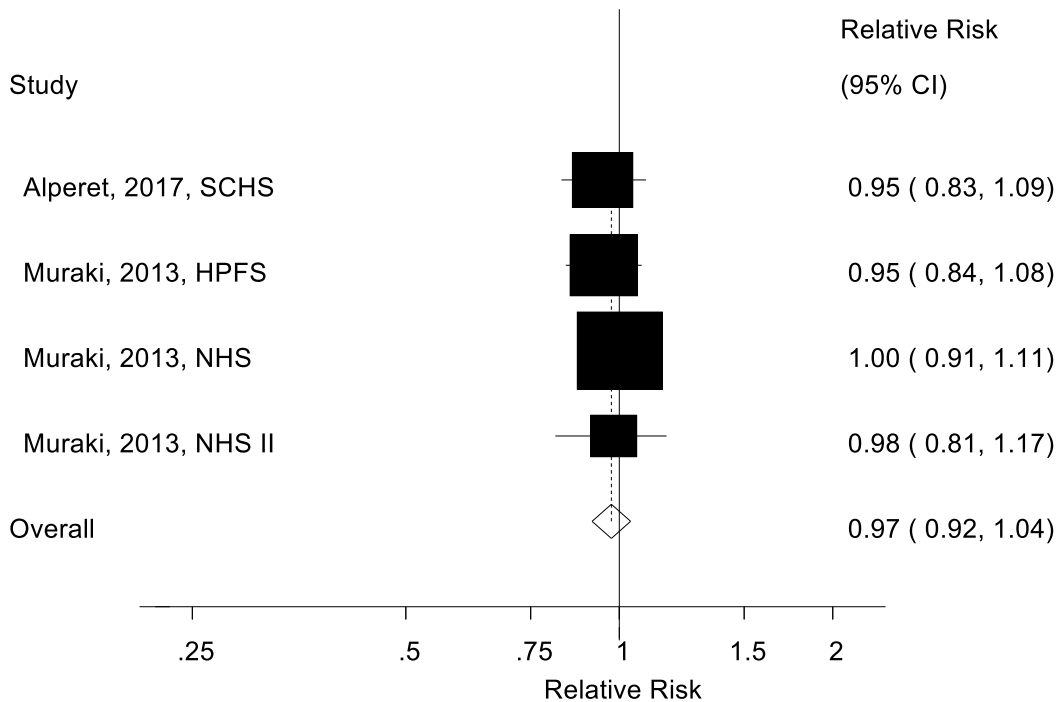
Supplementary Figure 36. Grapes and raisins and type 2 diabetes, nonlinear dose-response analysis



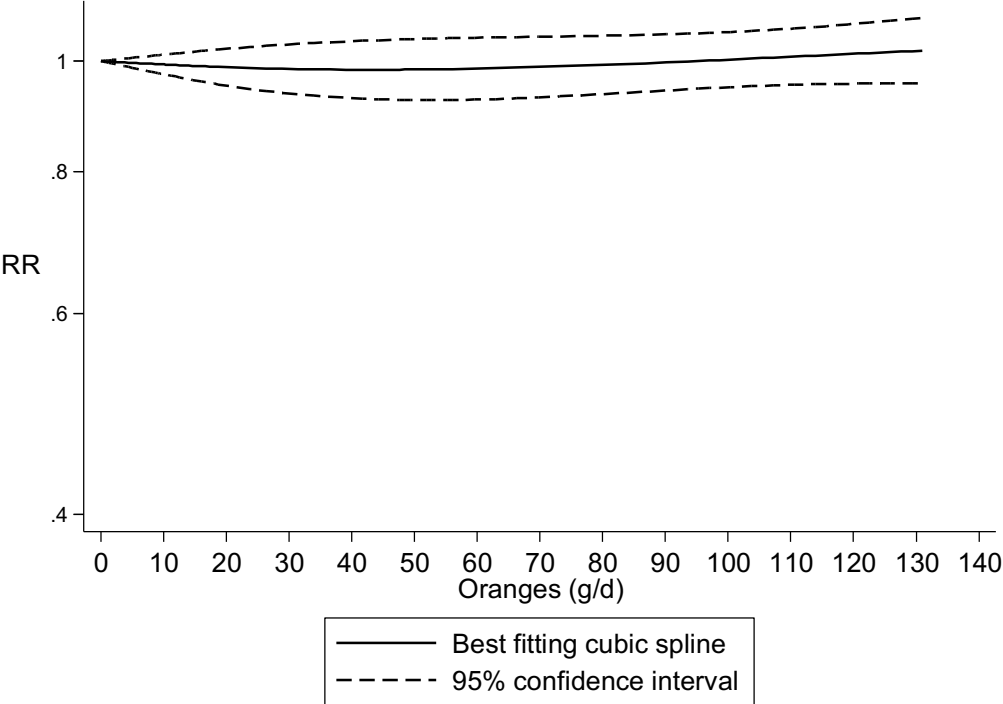
Supplementary Figure 37. Oranges and type 2 diabetes, high vs. low



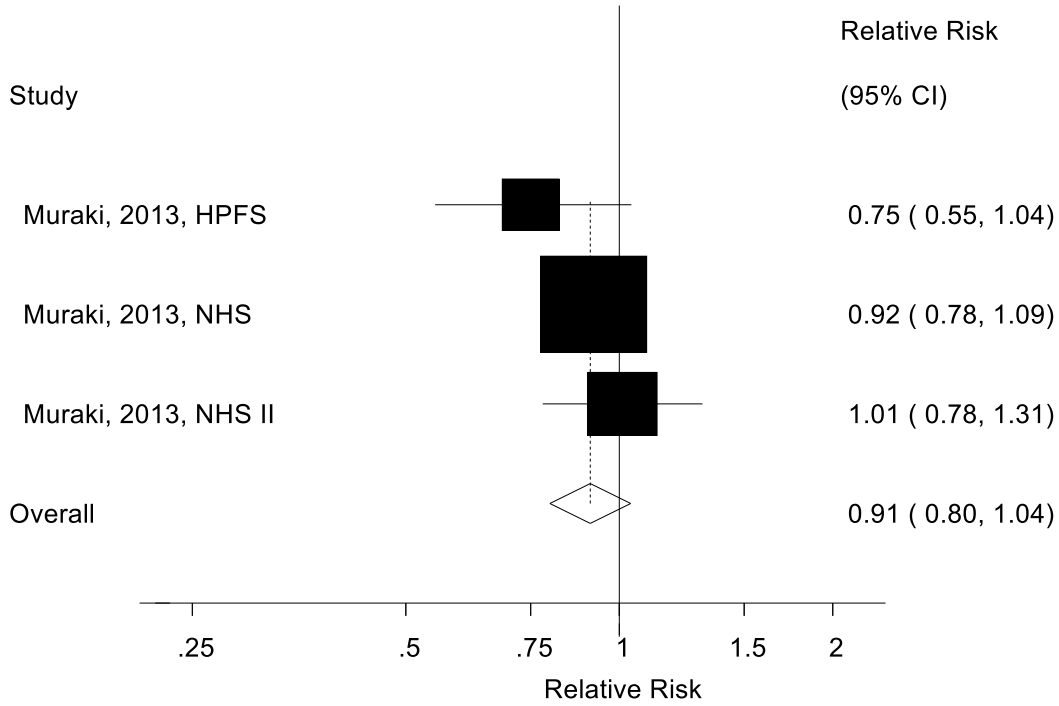
Supplementary Figure 38. Oranges and type 2 diabetes, dose-response analysis per 100 g/d



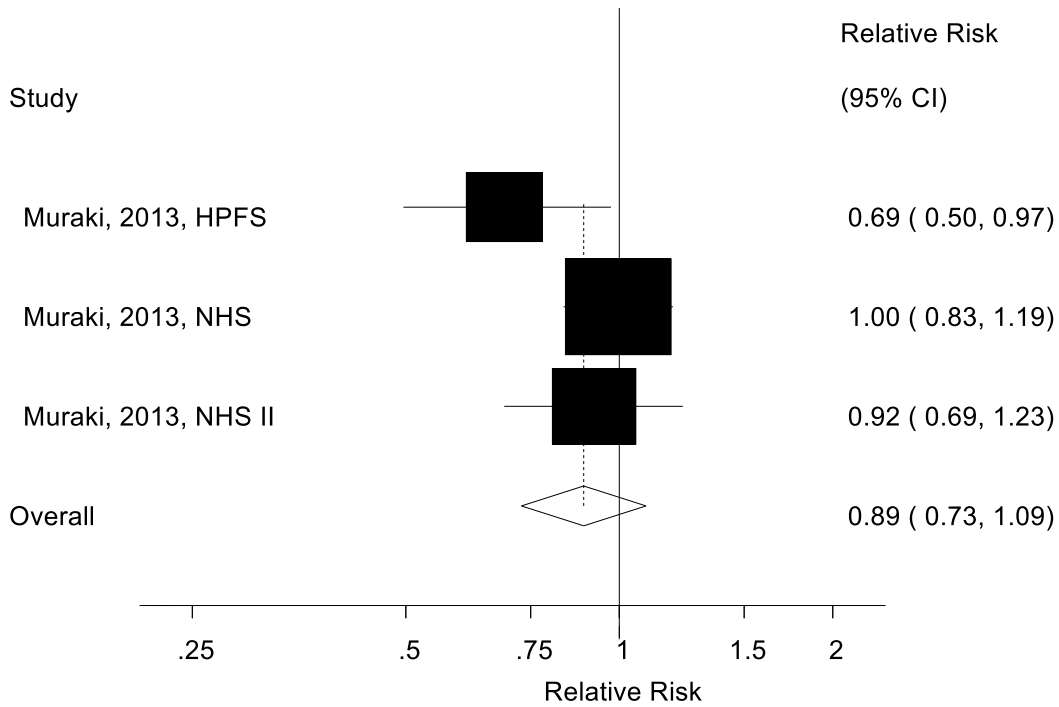
Supplementary Figure 39. Oranges and type 2 diabetes, nonlinear dose-response analysis



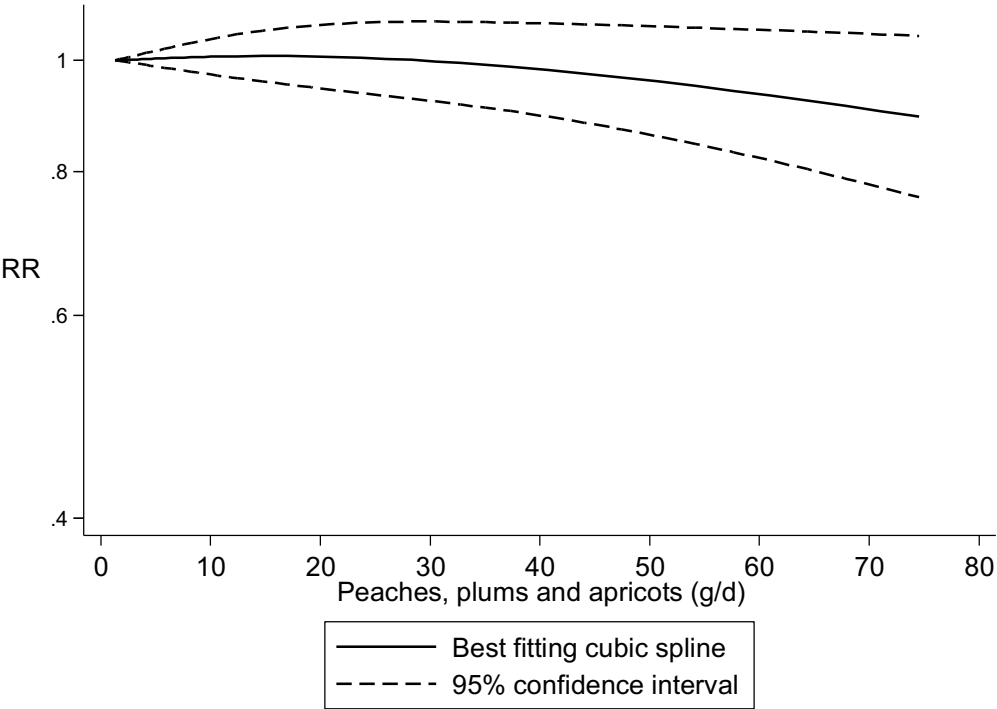
Supplementary Figure 40. Peaches, plums and apricots and type 2 diabetes, high vs. low



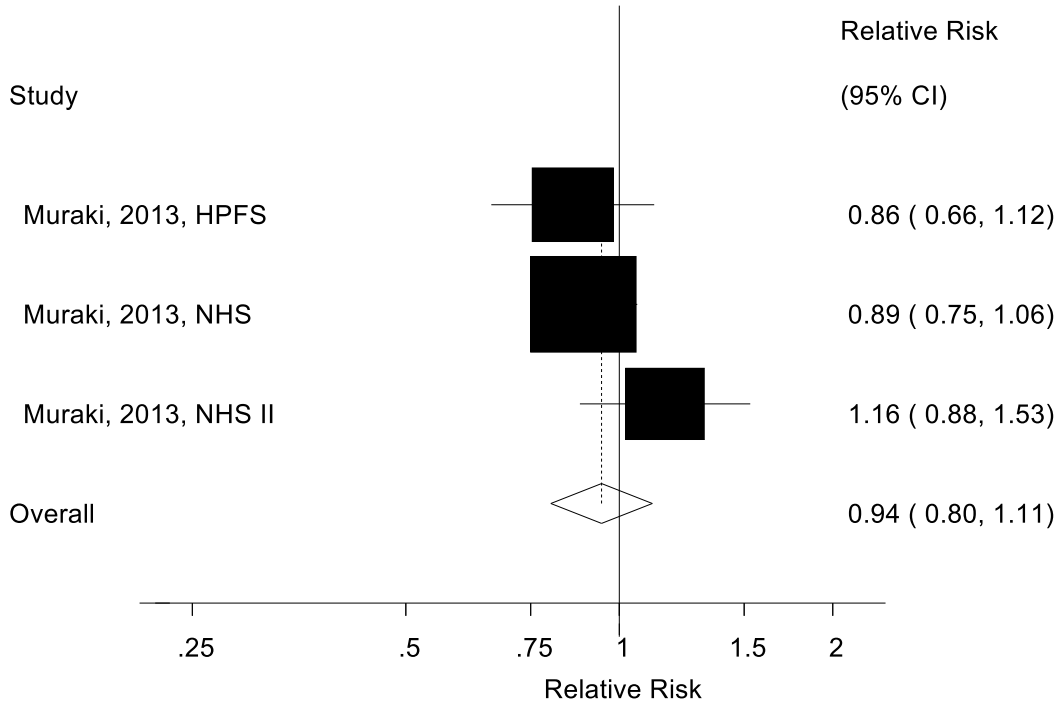
Supplementary Figure 41. Peaches, plums and apricots and type 2 diabetes, dose-response analysis per 100 g/d



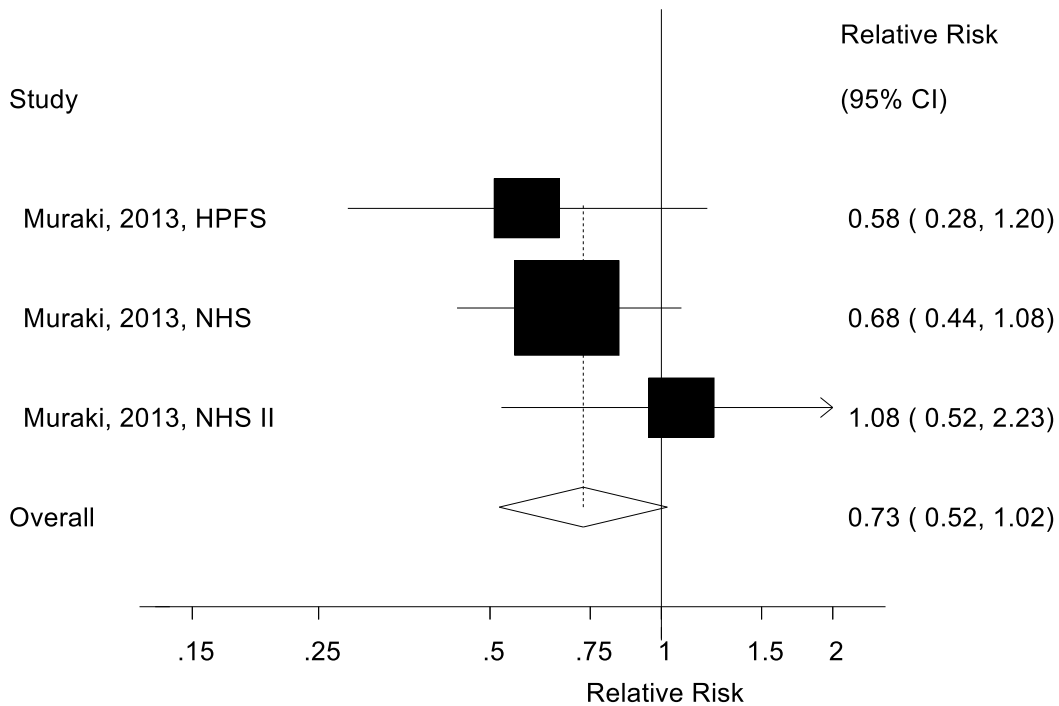
Supplementary Figure 42. Peaches, plums and apricots and type 2 diabetes, nonlinear dose-response analysis



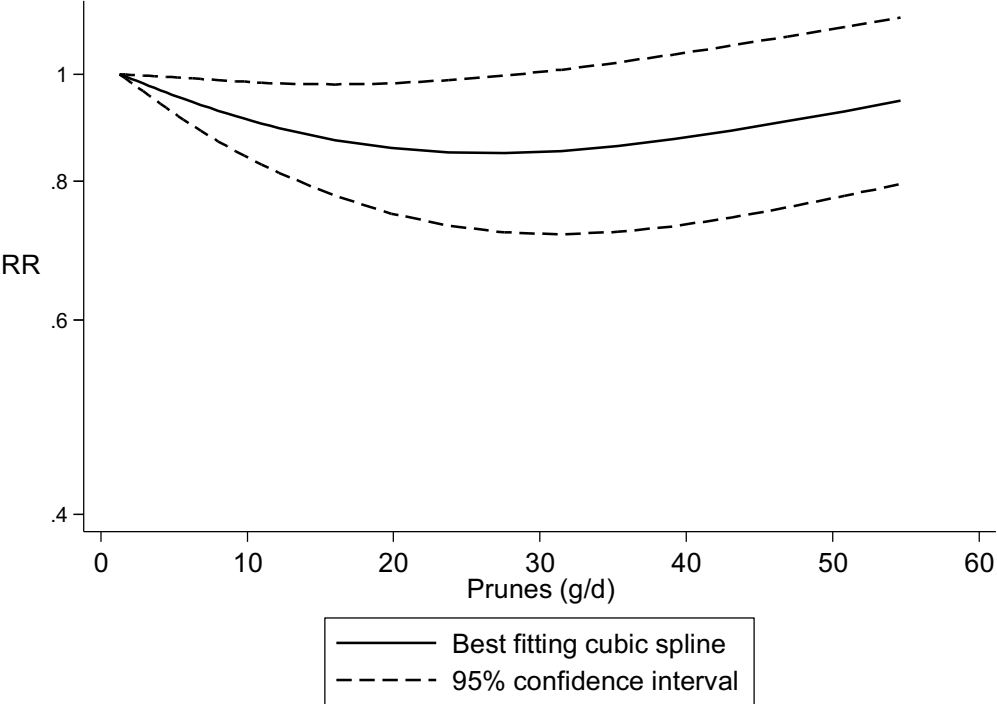
Supplementary Figure 43. Prunes and type 2 diabetes, high vs. low



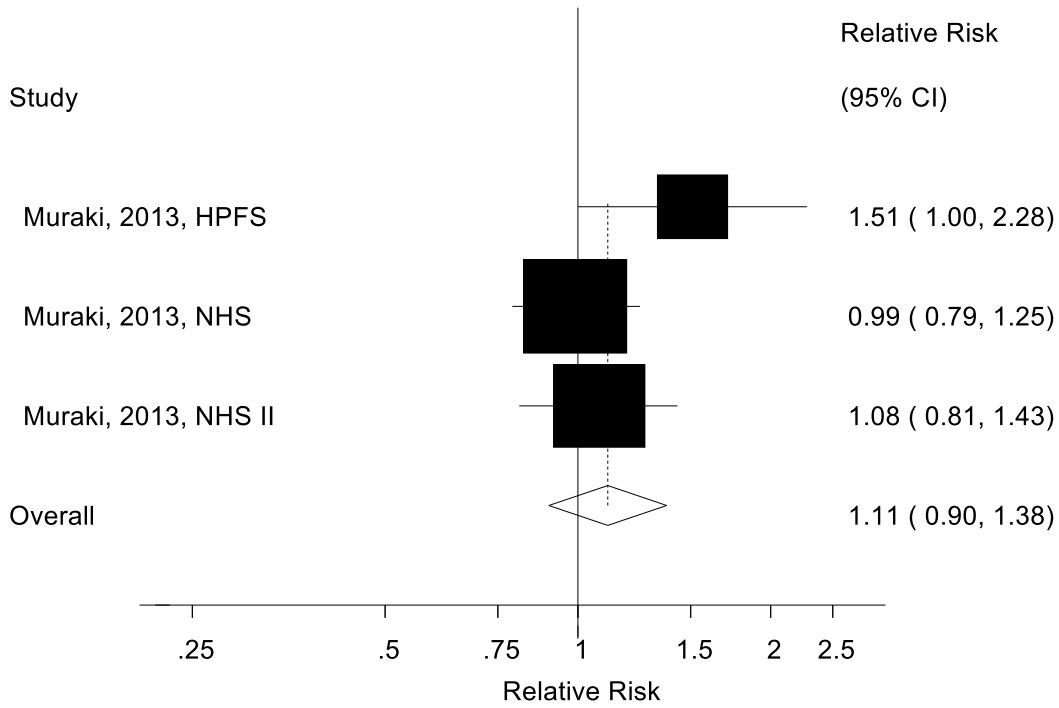
Supplementary Figure 44. Prunes and type 2 diabetes, dose-response analysis per 100 g/d



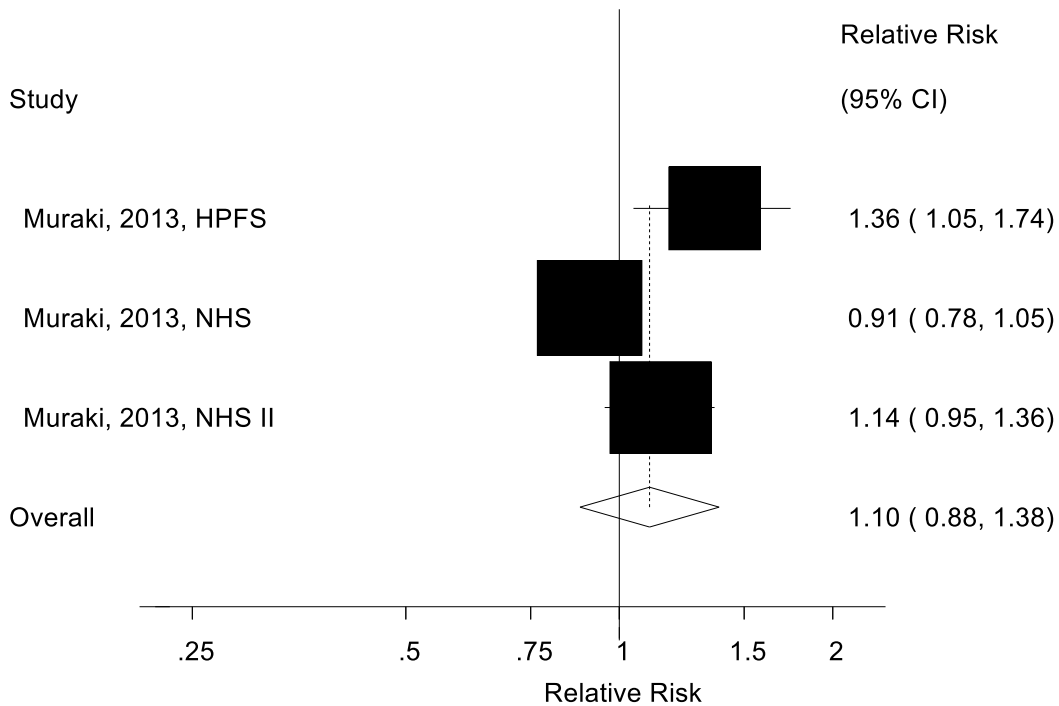
Supplementary Figure 45. Prunes and type 2 diabetes, nonlinear dose-response analysis



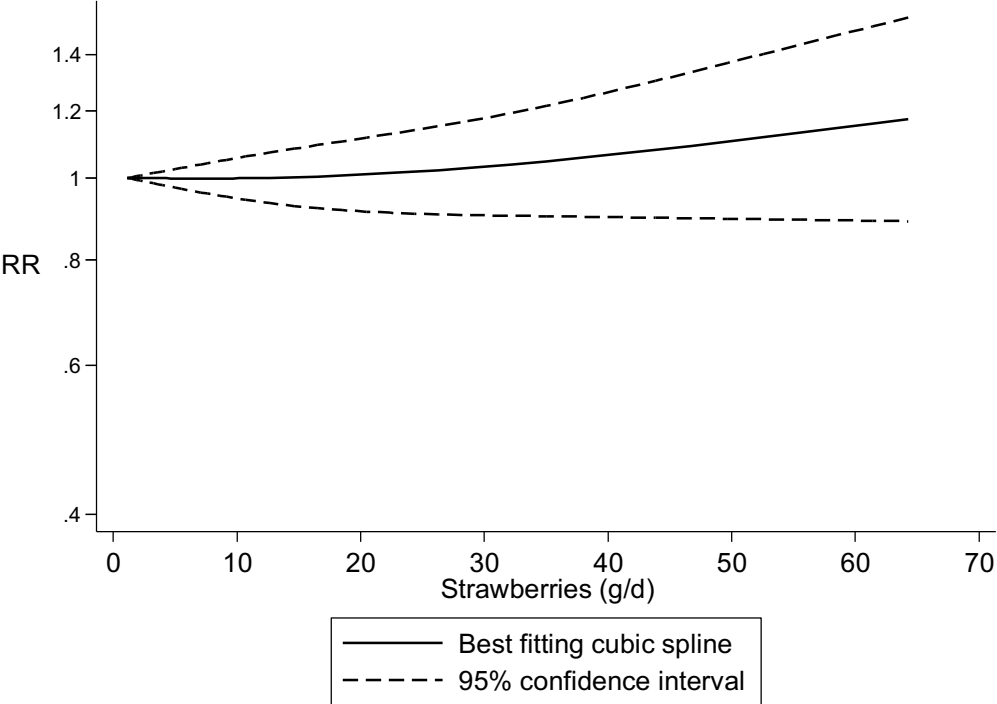
Supplementary Figure 46. Strawberries and type 2 diabetes, high vs. low



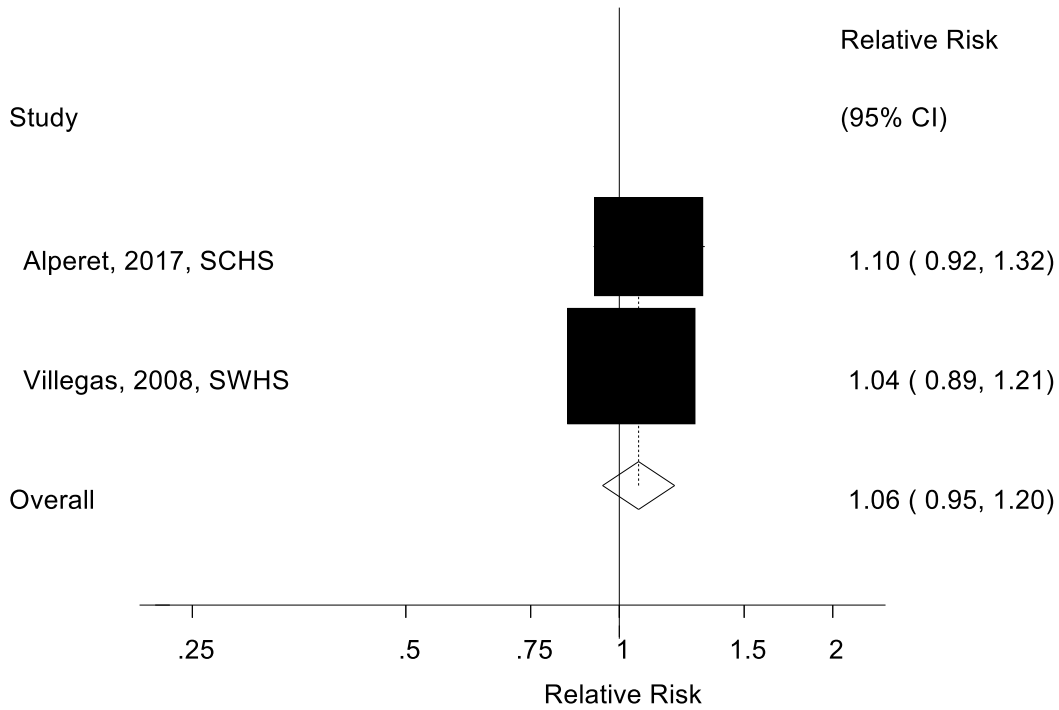
Supplementary Figure 47. Strawberries and type 2 diabetes, dose-response analysis per 50 g/d



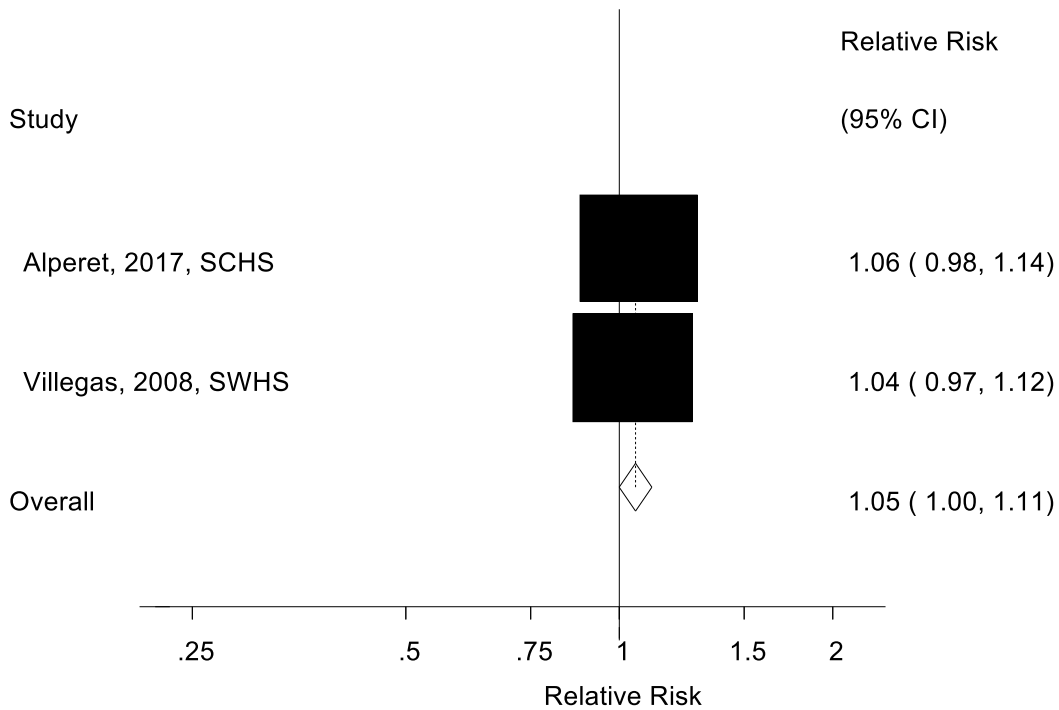
Supplementary Figure 48. Strawberries and type 2 diabetes, nonlinear dose-response analysis



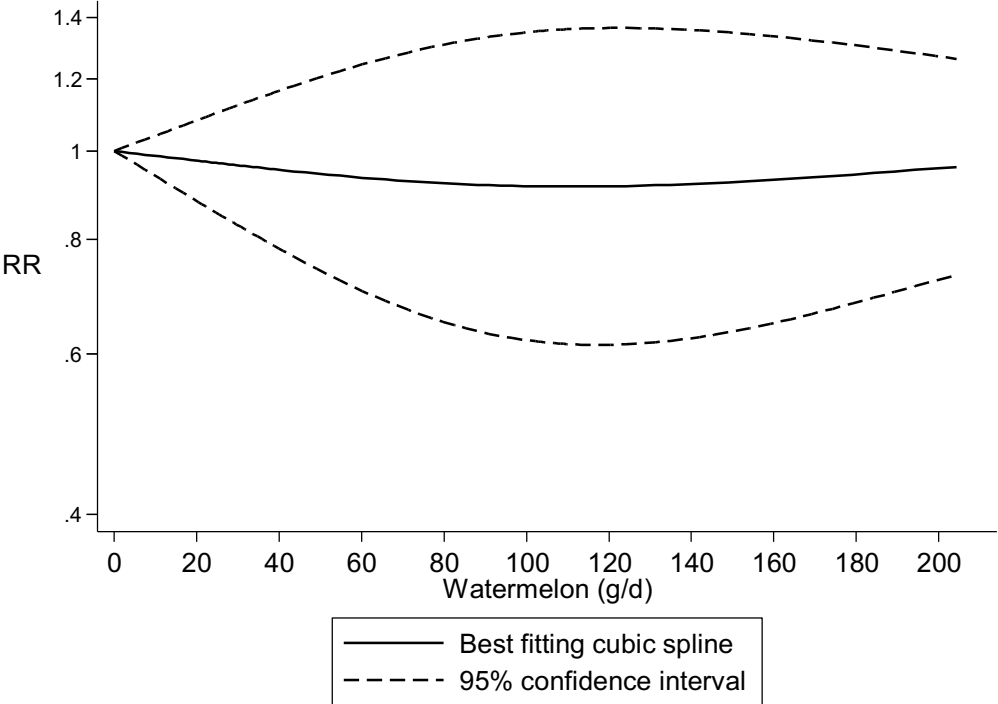
Supplementary Figure 49. Watermelon and type 2 diabetes, high vs. low



Supplementary Figure 50. Watermelon and type 2 diabetes, dose-response analysis per 100 g/d

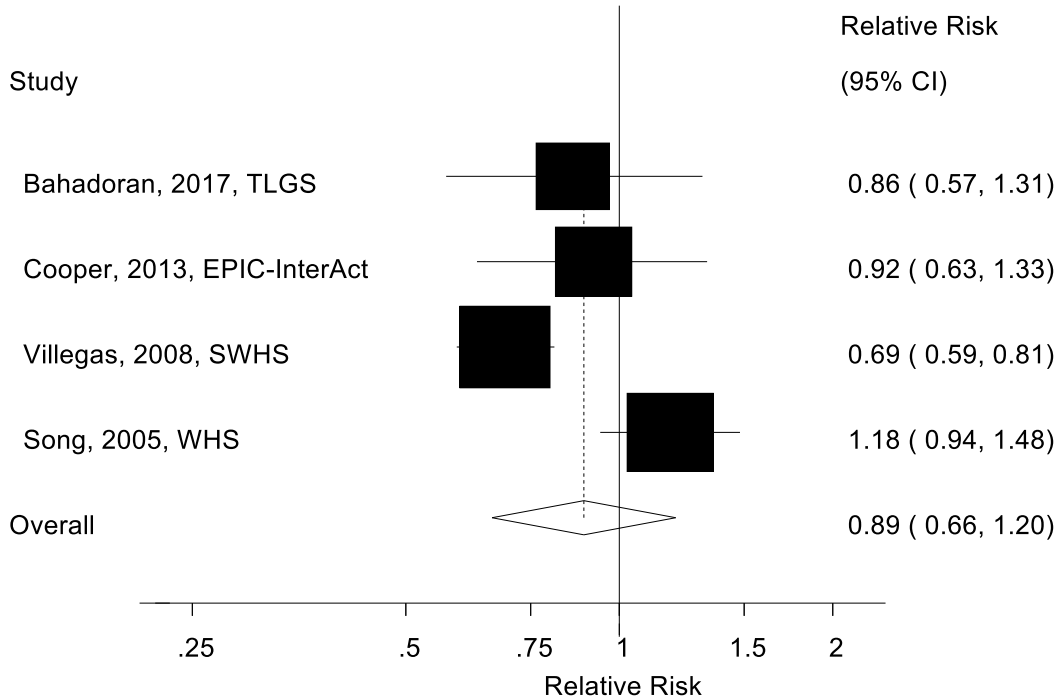


Supplementary Figure 51. Watermelon and type 2 diabetes, nonlinear dose-response analysis

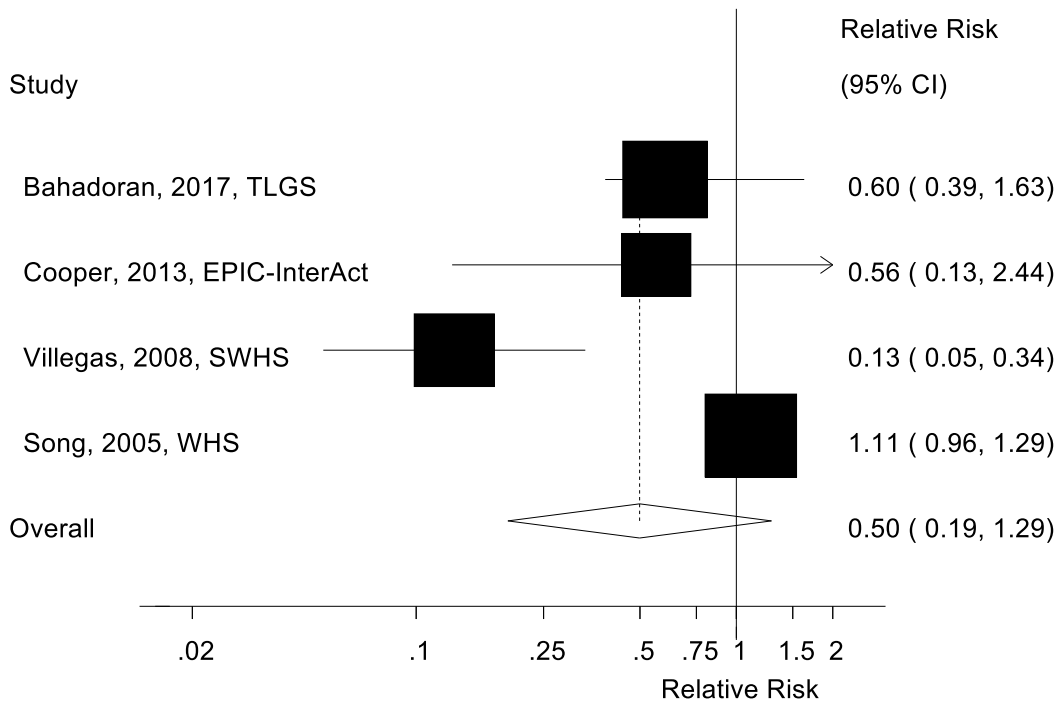


Supplementary figures of subtypes of vegetables

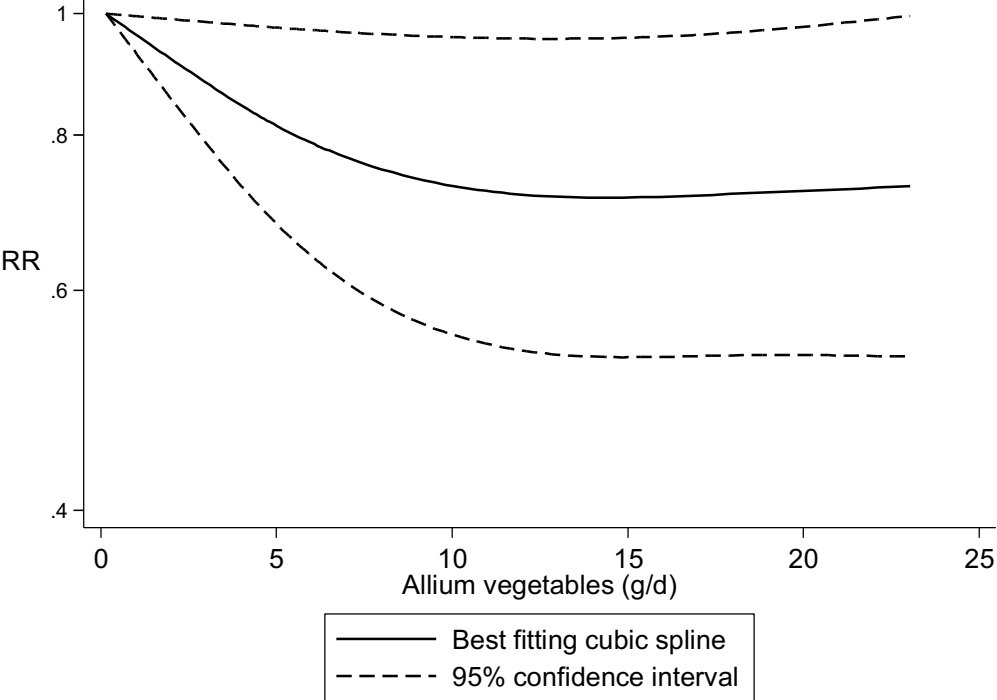
Supplementary Figure 52. Allium vegetables and type 2 diabetes, high vs. low



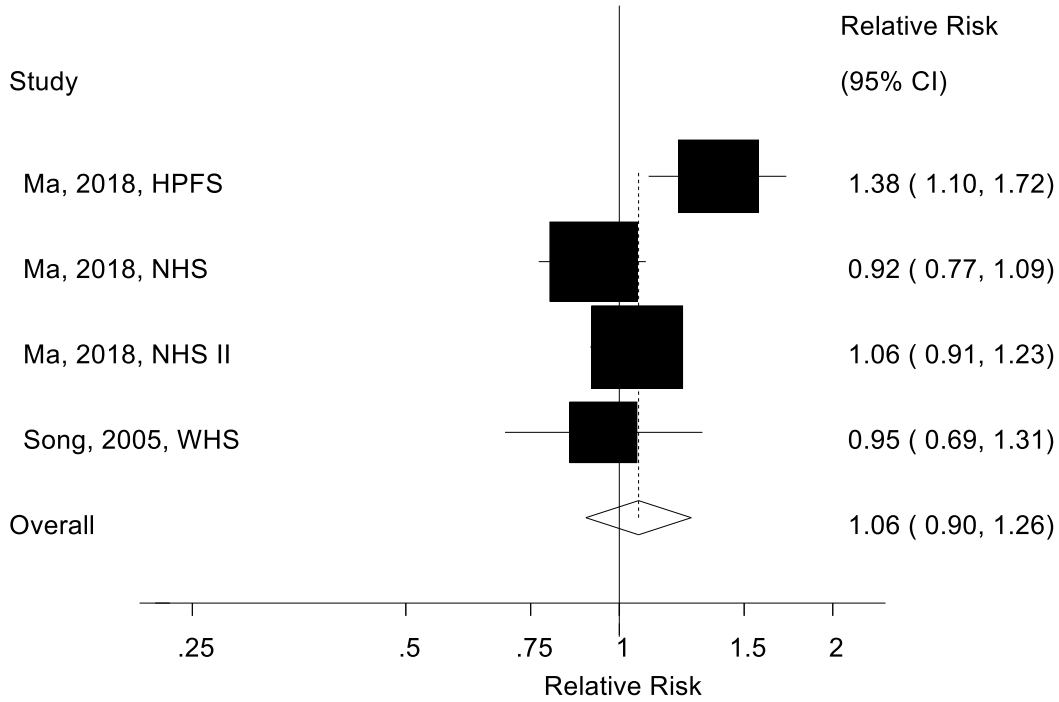
Supplementary Figure 53. Allium vegetables and type 2 diabetes, dose-response analysis per 100 g/d



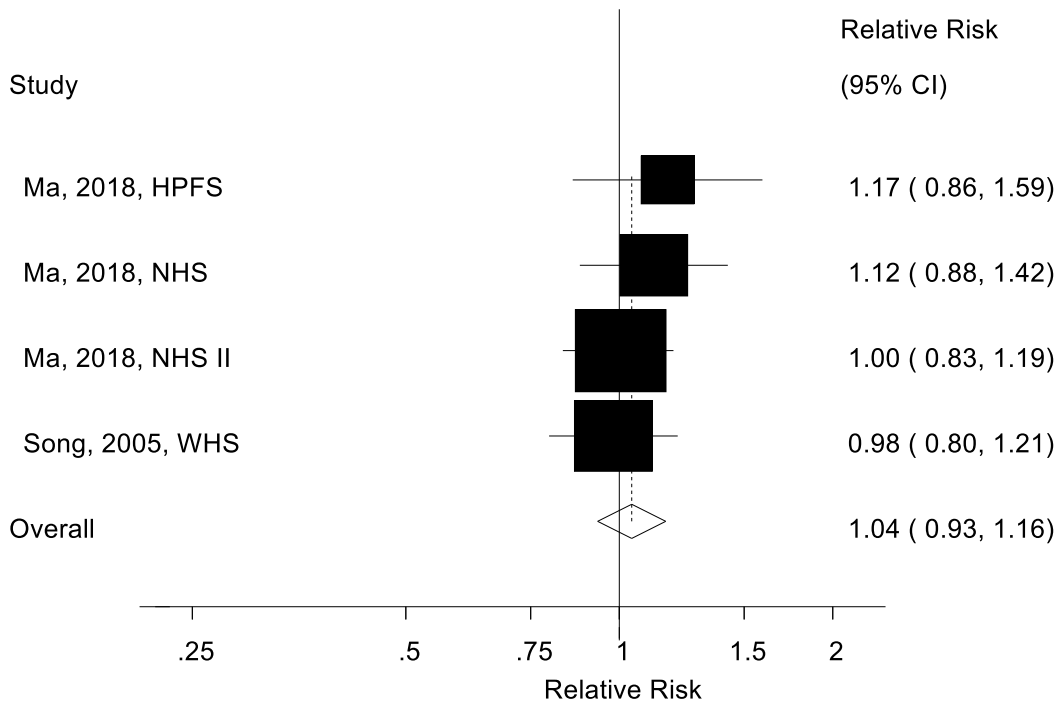
Supplementary Figure 54. Allium vegetables and type 2 diabetes, nonlinear dose-response analysis



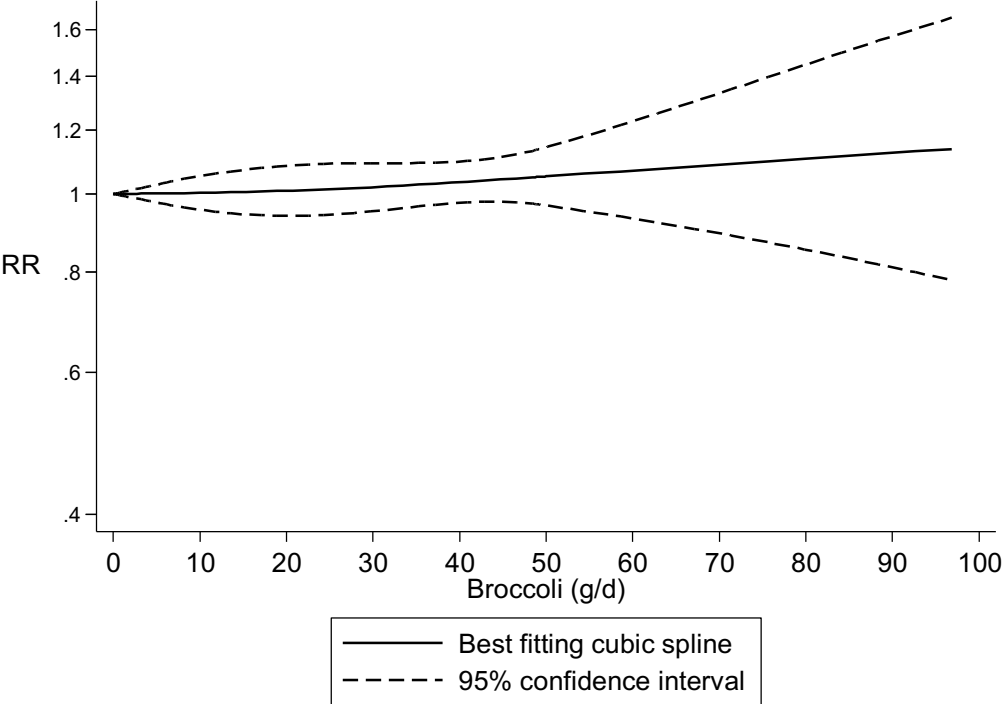
Supplementary Figure 58. Broccoli and type 2 diabetes, high vs. low



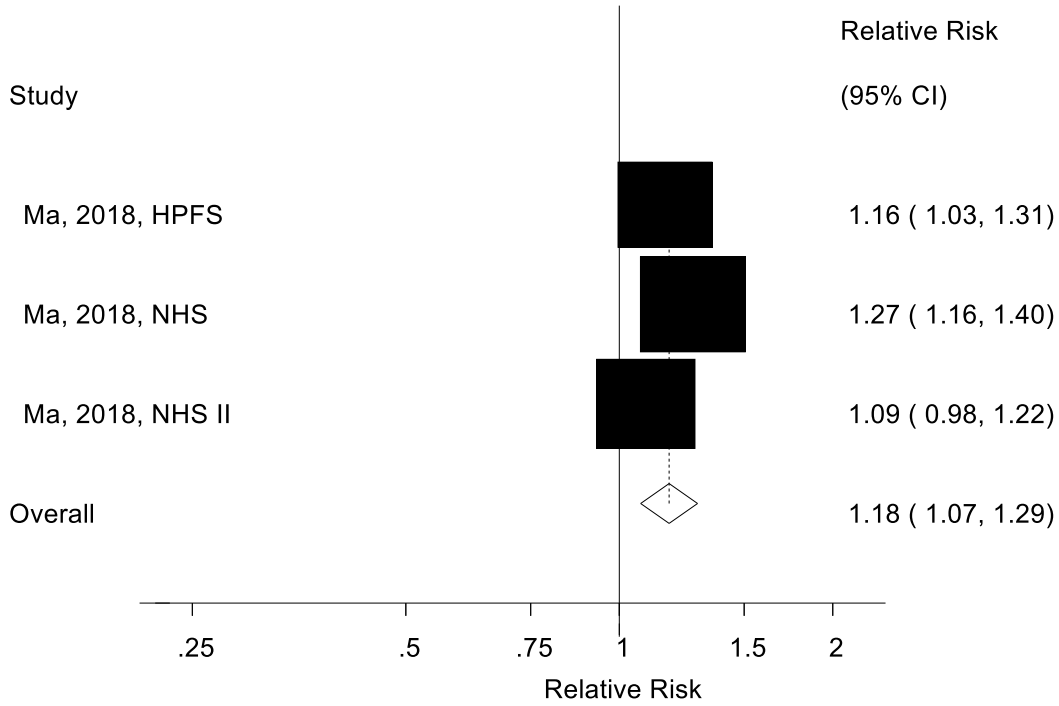
Supplementary Figure 59. Broccoli and type 2 diabetes, dose-response analysis per 100 g/d



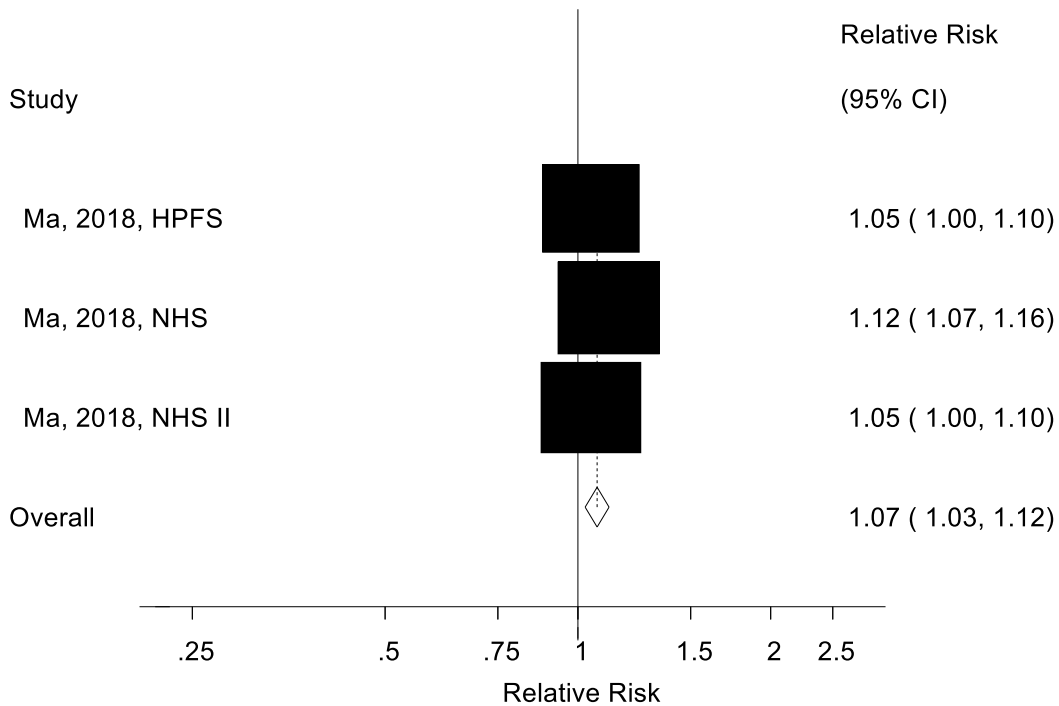
Supplementary Figure 60. Broccoli and type 2 diabetes, nonlinear dose-response analysis



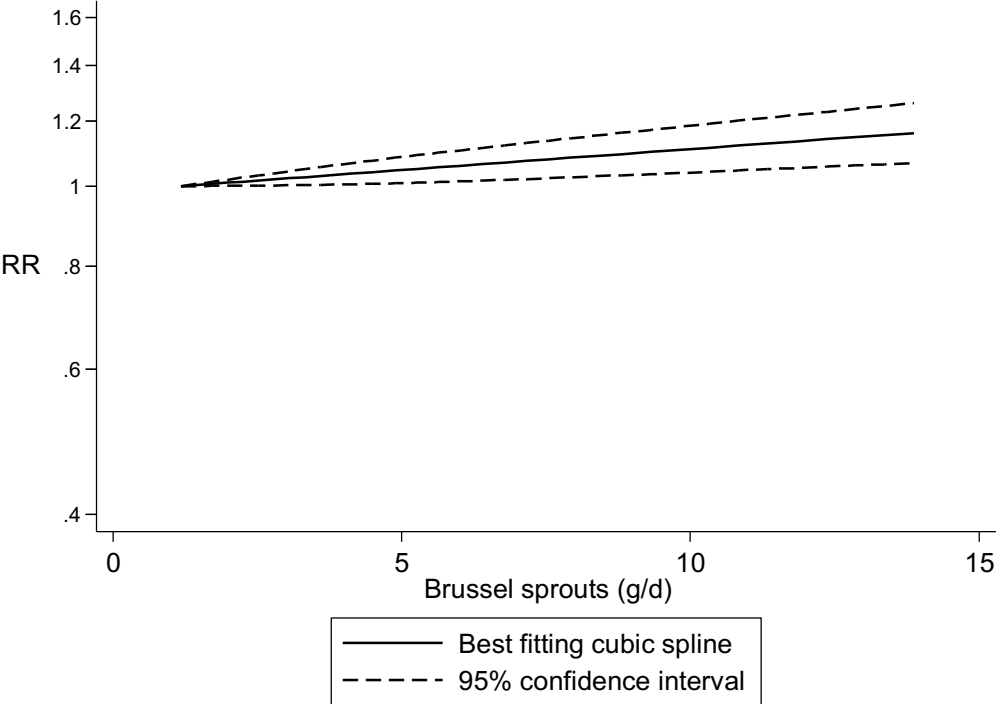
Supplementary Figure 61. Brussel sprouts and type 2 diabetes, high vs. low



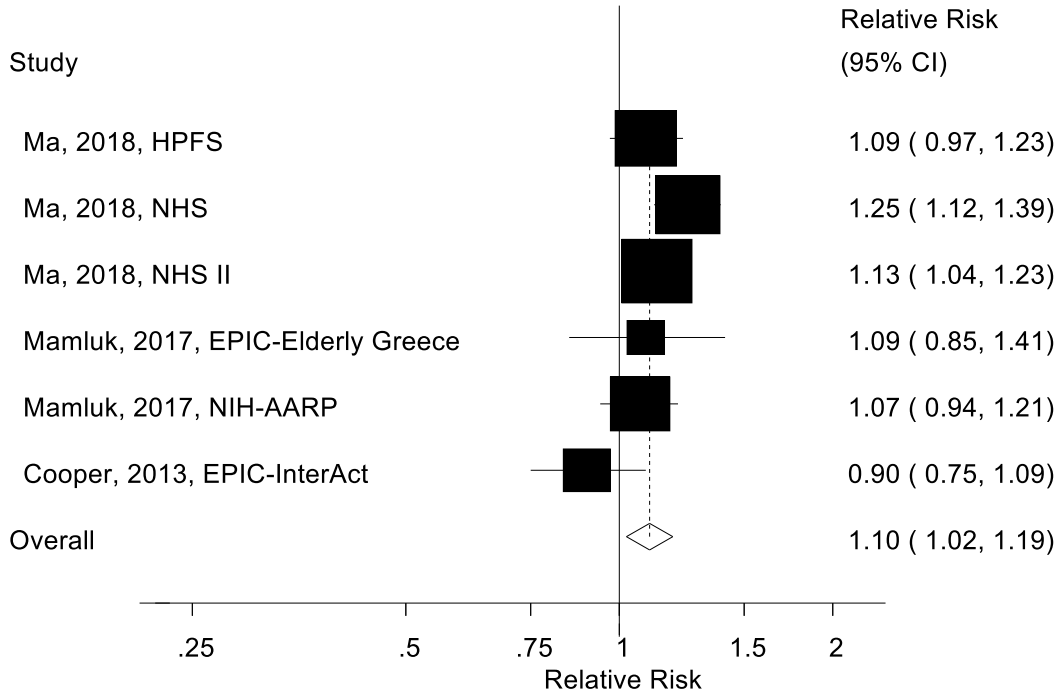
Supplementary Figure 62. Brussel sprouts and type 2 diabetes, dose-response analysis per 10 g/d



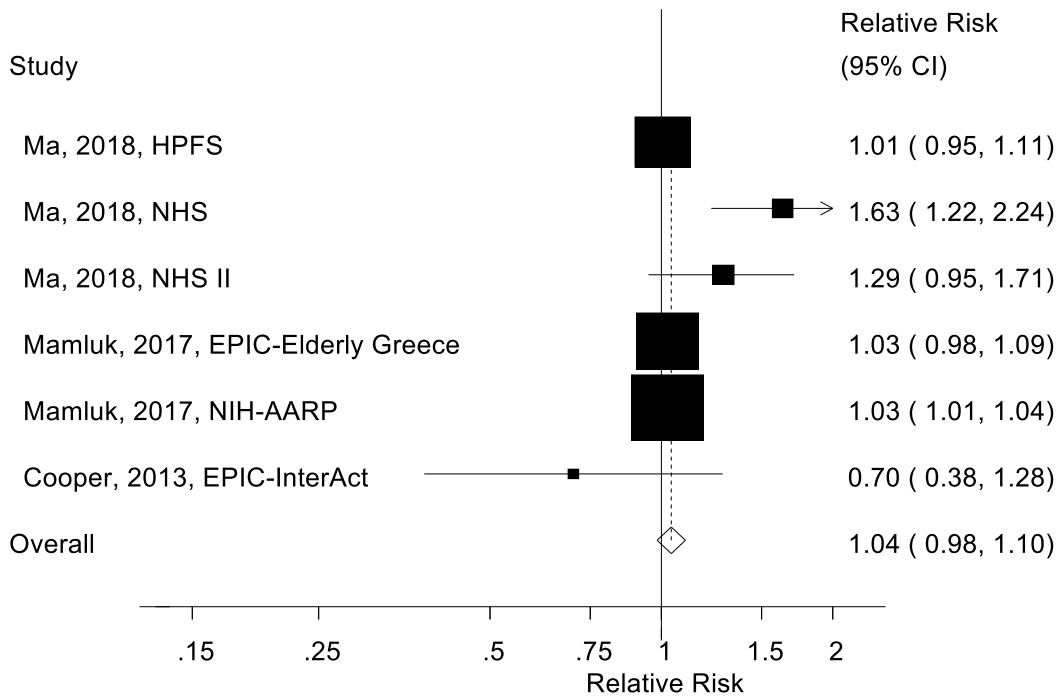
Supplementary Figure 63. Brussel sprouts and type 2 diabetes, nonlinear dose-response analysis



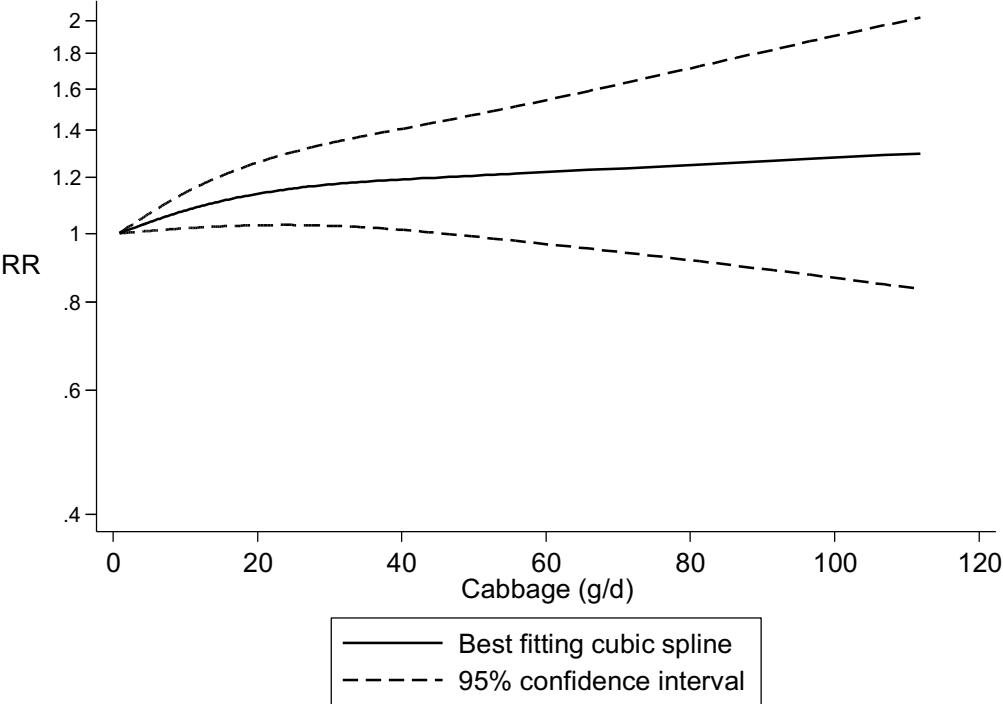
Supplementary Figure 64. Cabbage and type 2 diabetes, high vs. low



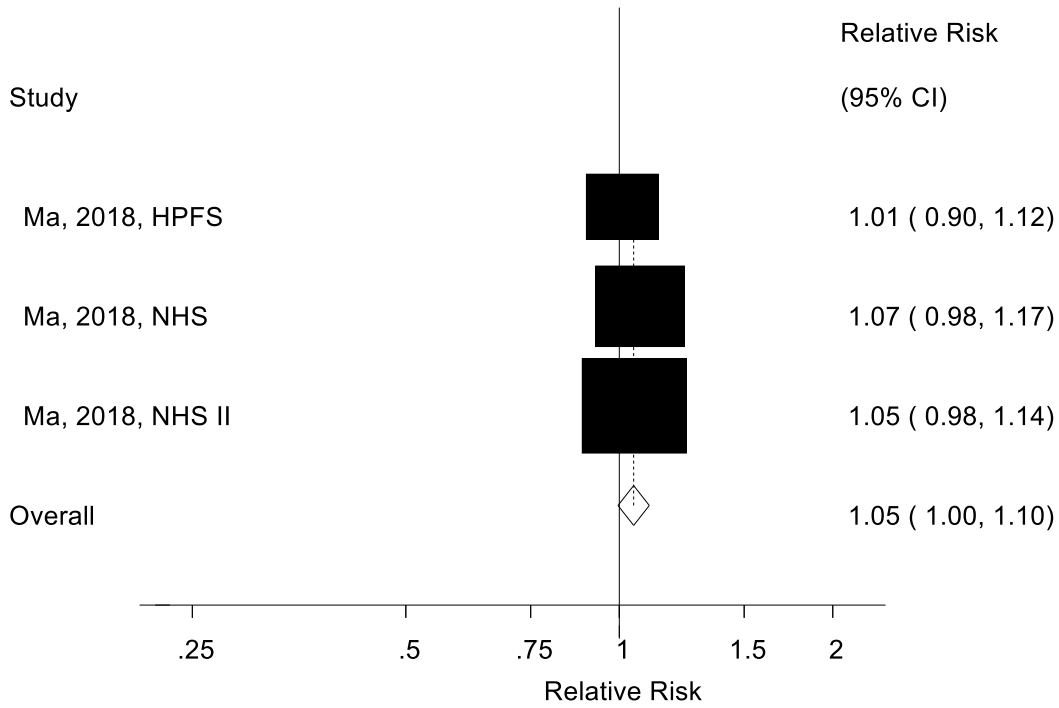
Supplementary Figure 65. Cabbage and type 2 diabetes, dose-response analysis per 100 g/d



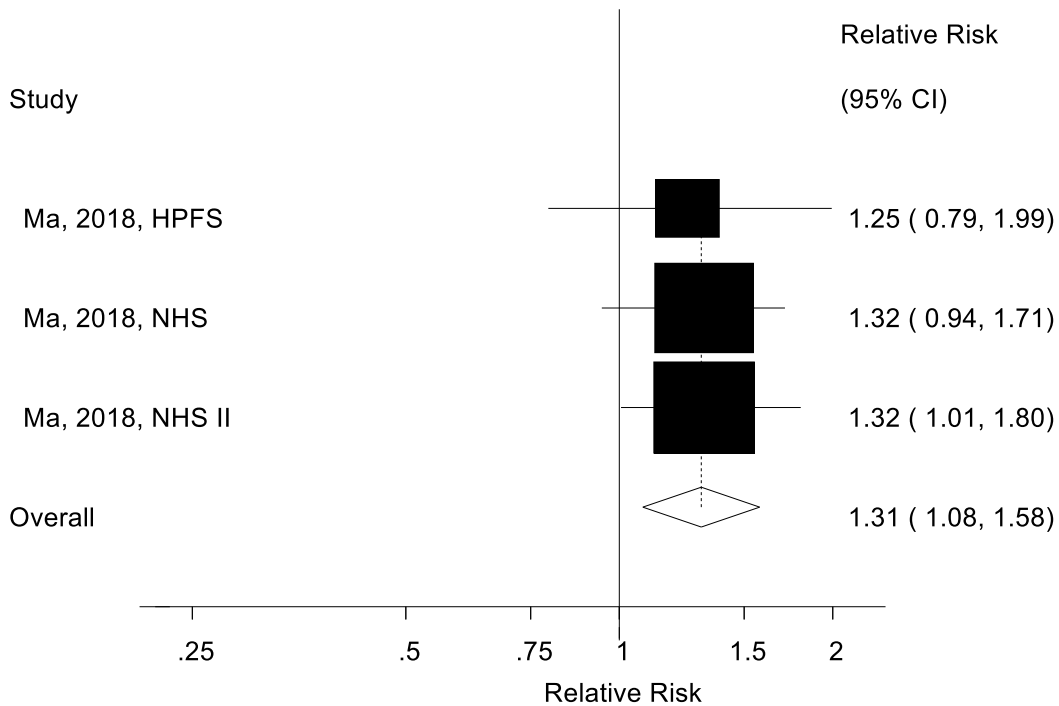
Supplementary Figure 66. Cabbage and type 2 diabetes, nonlinear dose-response analysis



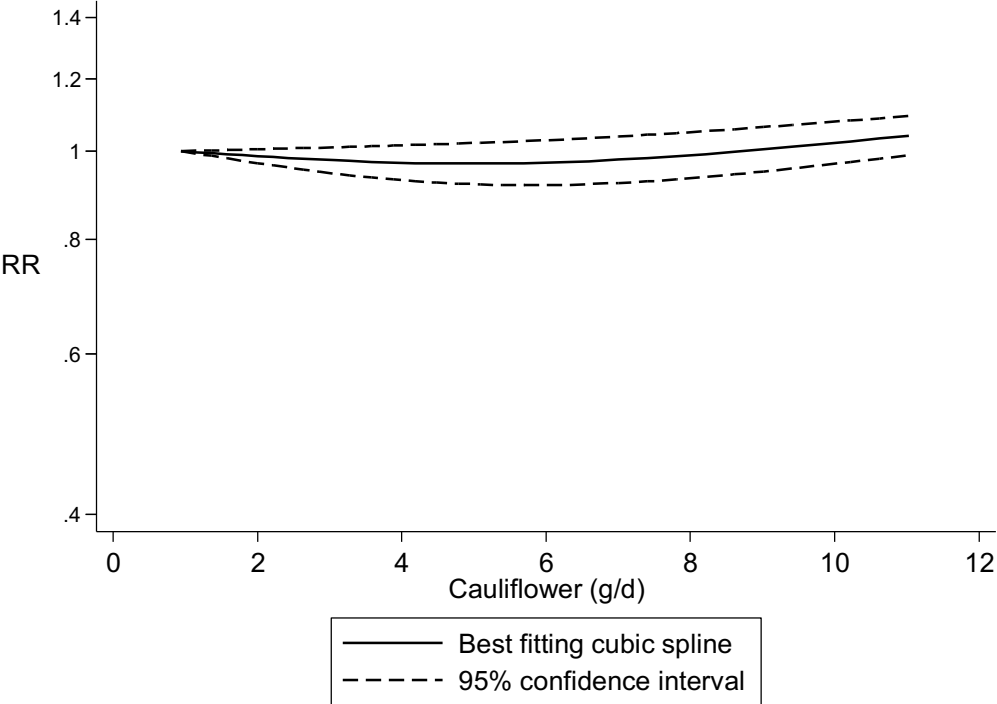
Supplementary Figure 67. Cauliflower and type 2 diabetes, high vs. low



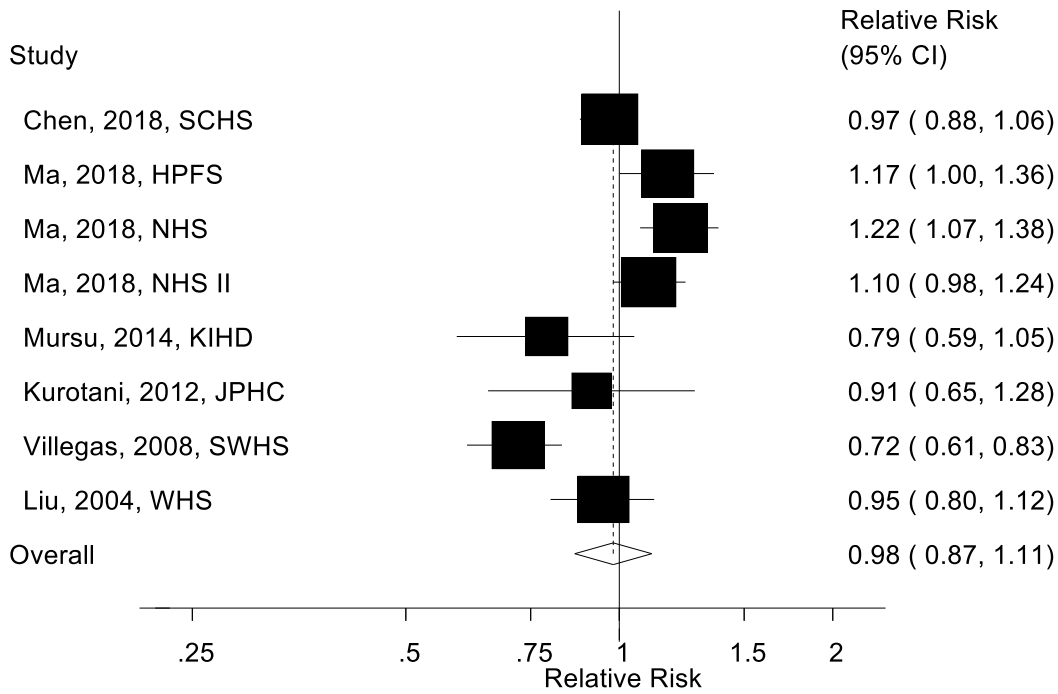
Supplementary Figure 68. Cauliflower and type 2 diabetes, dose-response analysis per 100 g/d



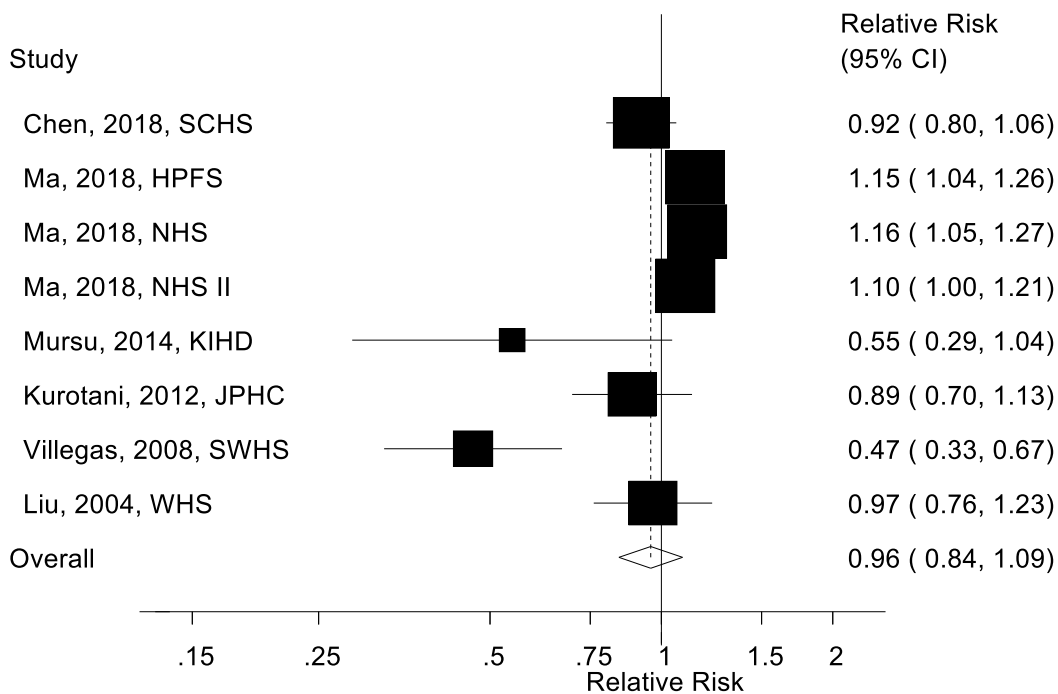
Supplementary Figure 69. Cauliflower and type 2 diabetes, nonlinear dose-response analysis



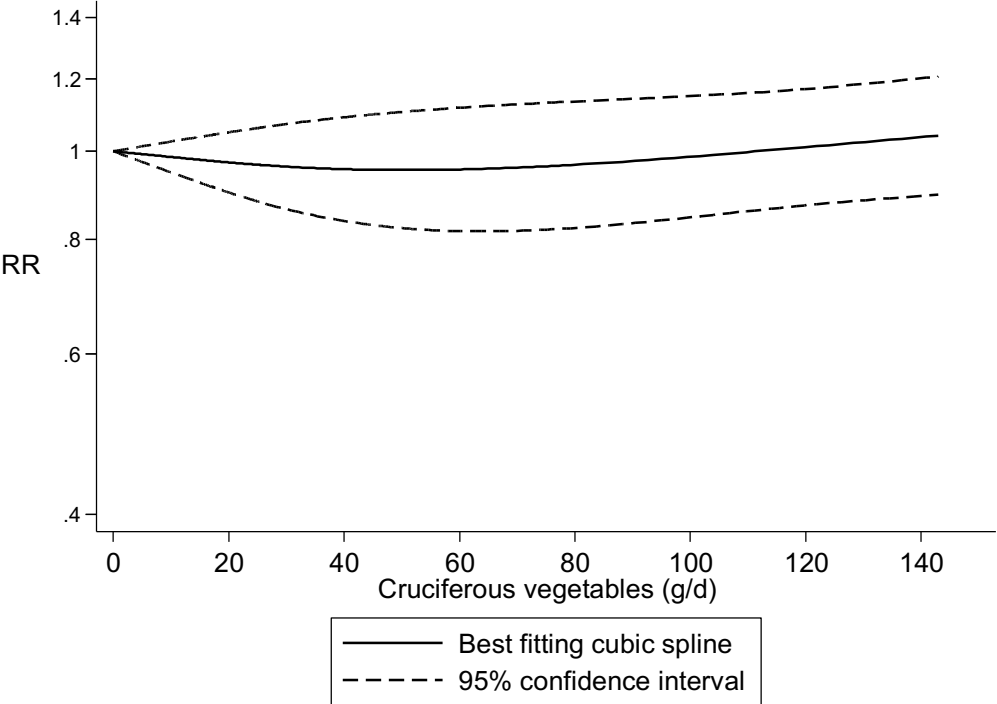
Supplementary Figure 70. Cruciferous vegetables and type 2 diabetes, high vs. low



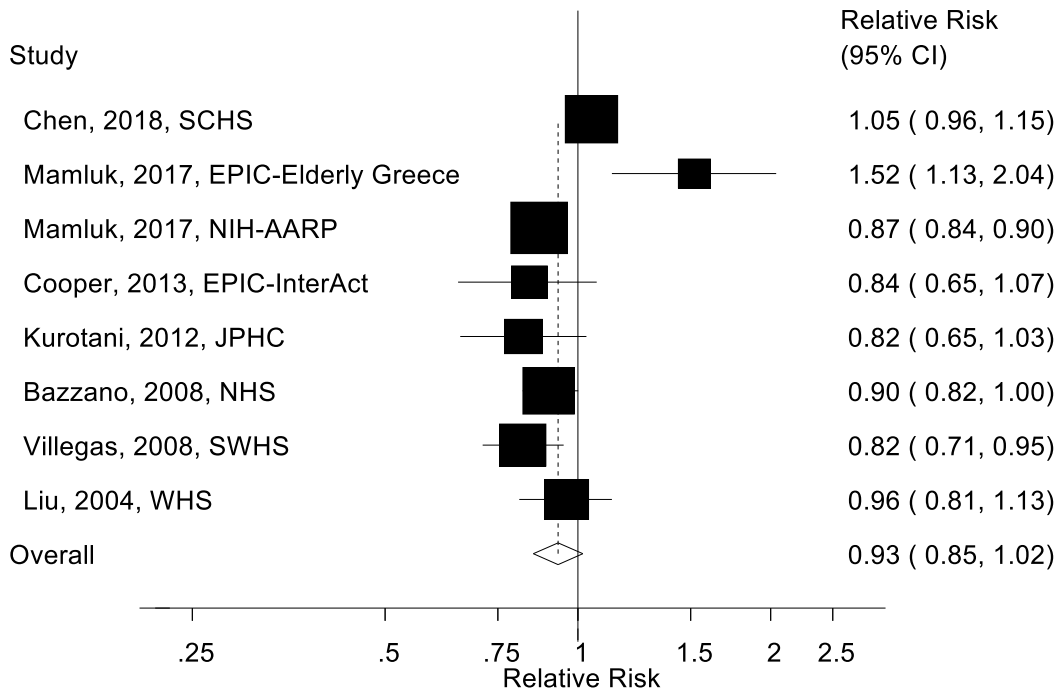
Supplementary Figure 71. Cruciferous vegetables and type 2 diabetes, dose-response analysis per 100 g/d



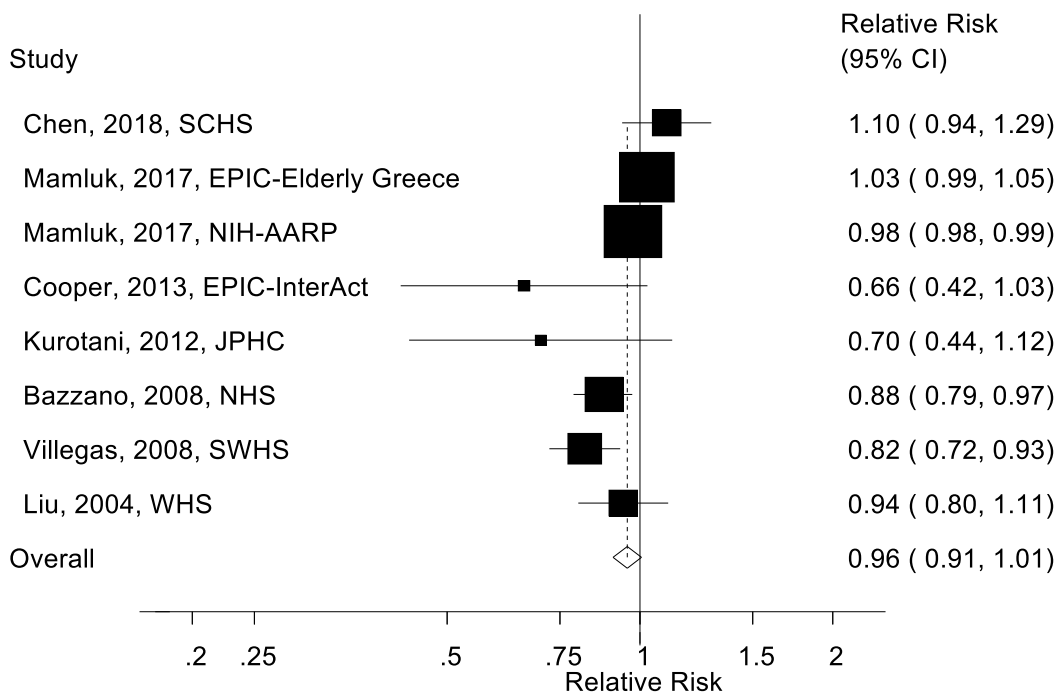
Supplementary Figure 72. Cruciferous vegetables and type 2 diabetes, nonlinear dose-response analysis



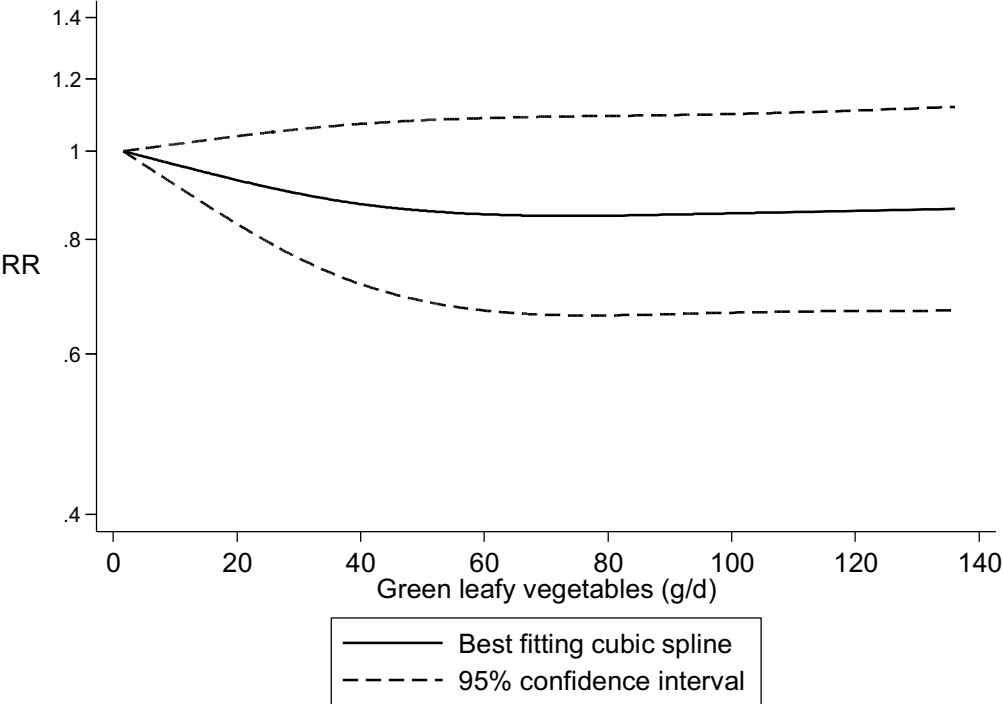
Supplementary Figure 73. Green leafy vegetables and type 2 diabetes, high vs. low



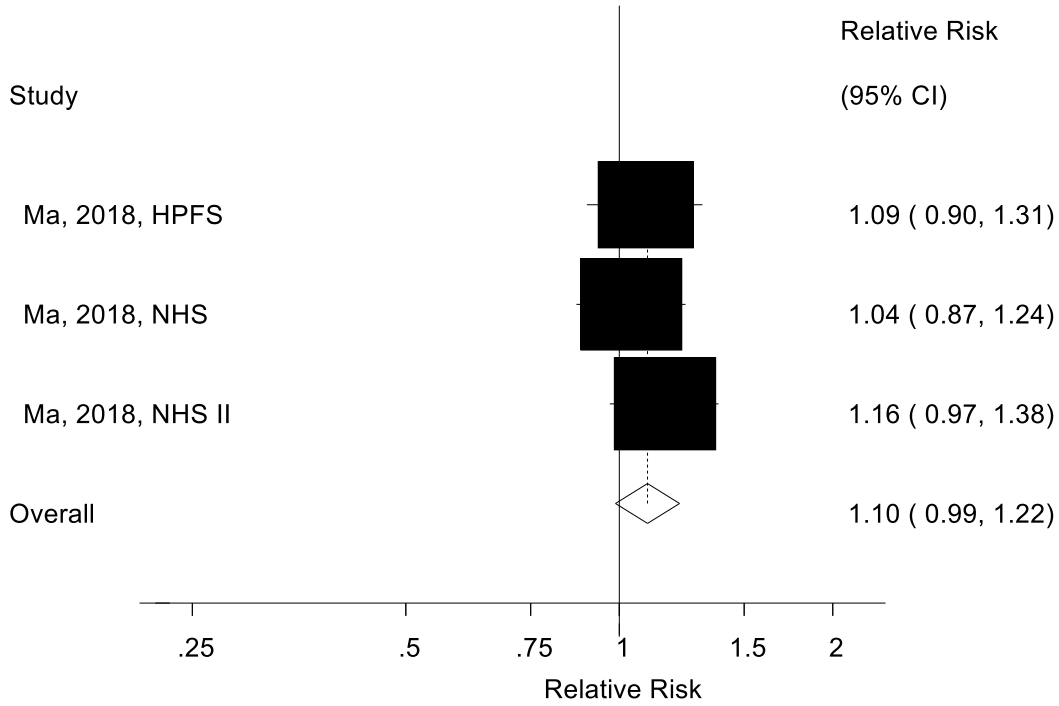
Supplementary Figure 74. Green leafy vegetables and type 2 diabetes, dose-response analysis per 100 g/d



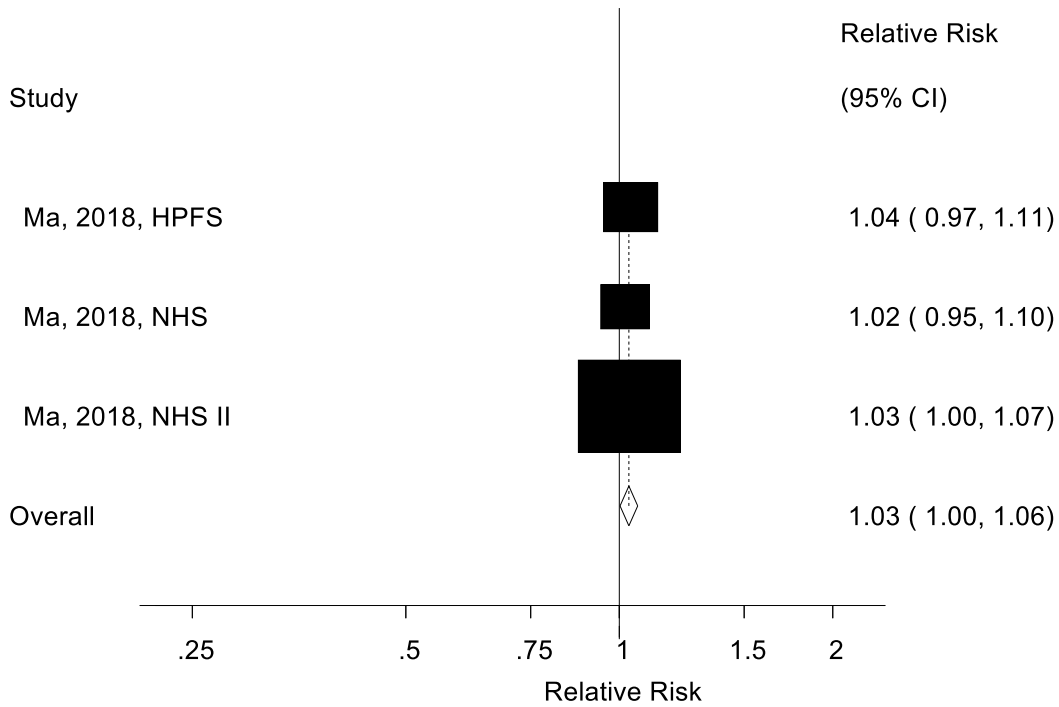
Supplementary Figure 75. Green leafy vegetables and type 2 diabetes, nonlinear dose-response analysis



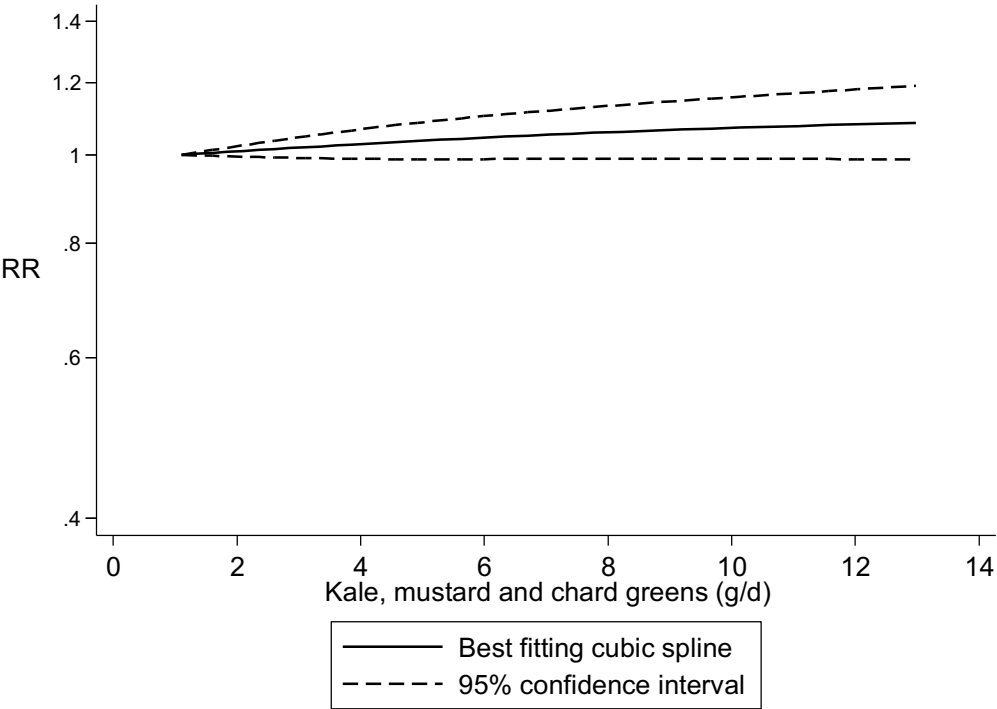
Supplementary Figure 76. Kale, mustard and chard greens and type 2 diabetes, high vs. low



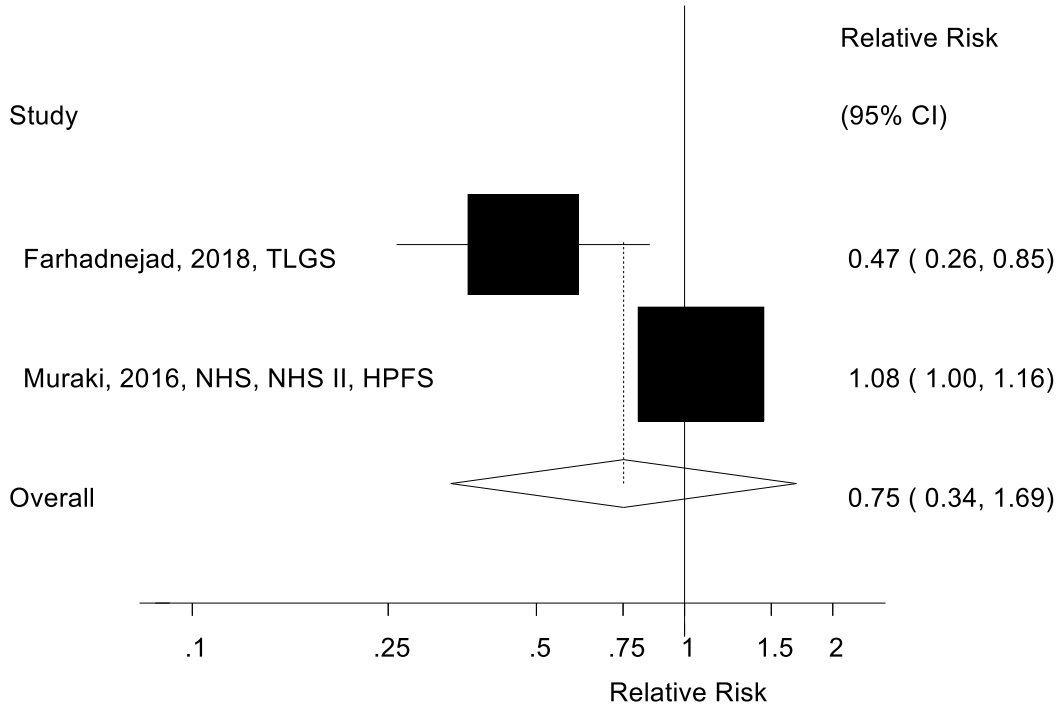
Supplementary Figure 77. Kale, mustard and chard greens and type 2 diabetes, dose-response analysis per 10 g/d



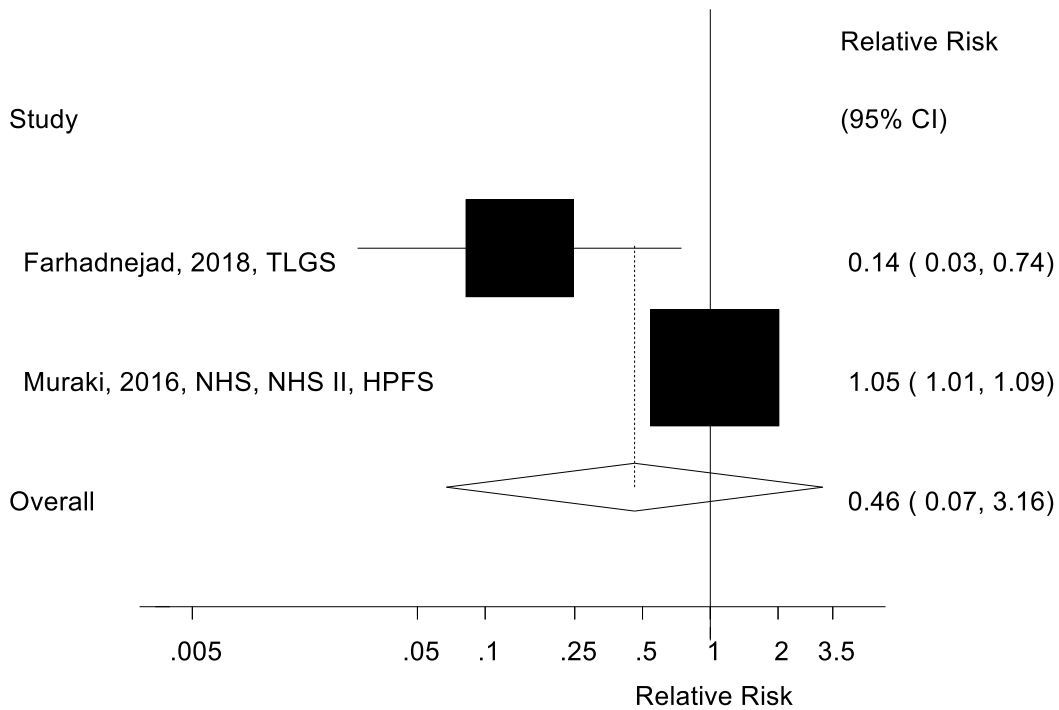
Supplementary Figure 78. Kale, mustard and chard greens and type 2 diabetes, nonlinear dose-response analysis



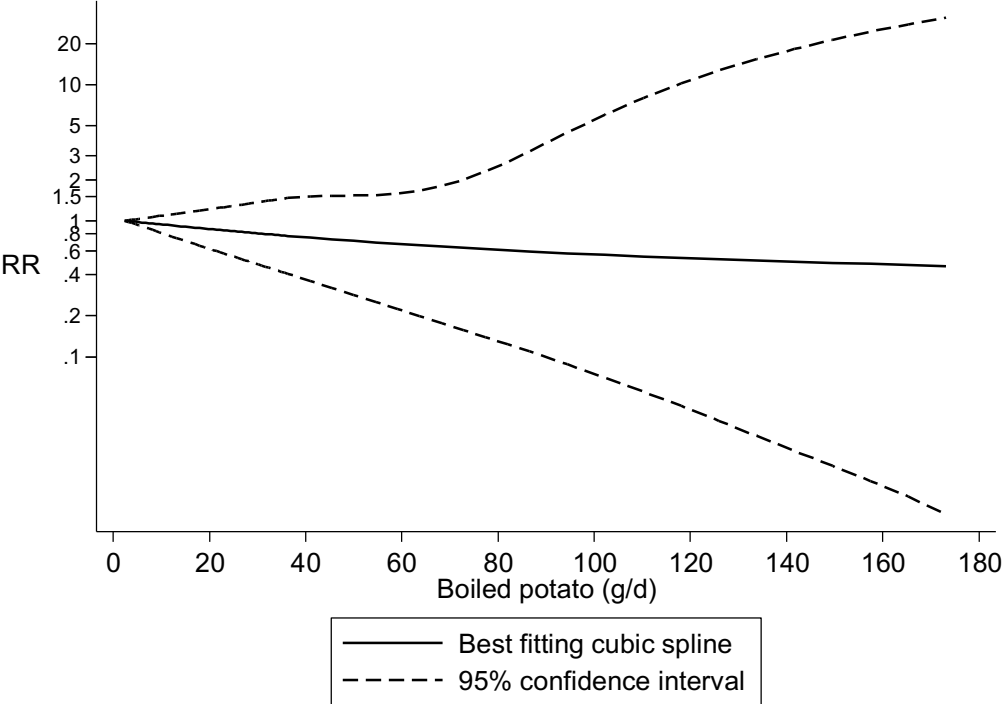
Supplementary Figure 55. Boiled potatoes and type 2 diabetes, high vs. low



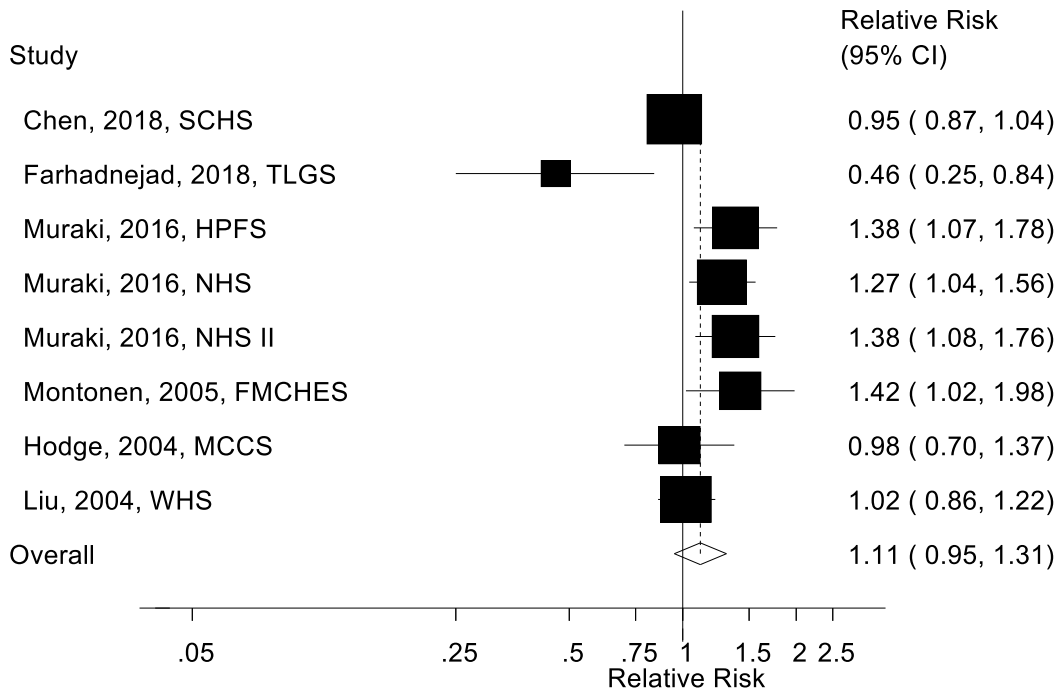
Supplementary Figure 56. Boiled potatoes and type 2 diabetes, dose-response analysis per 100 g/d



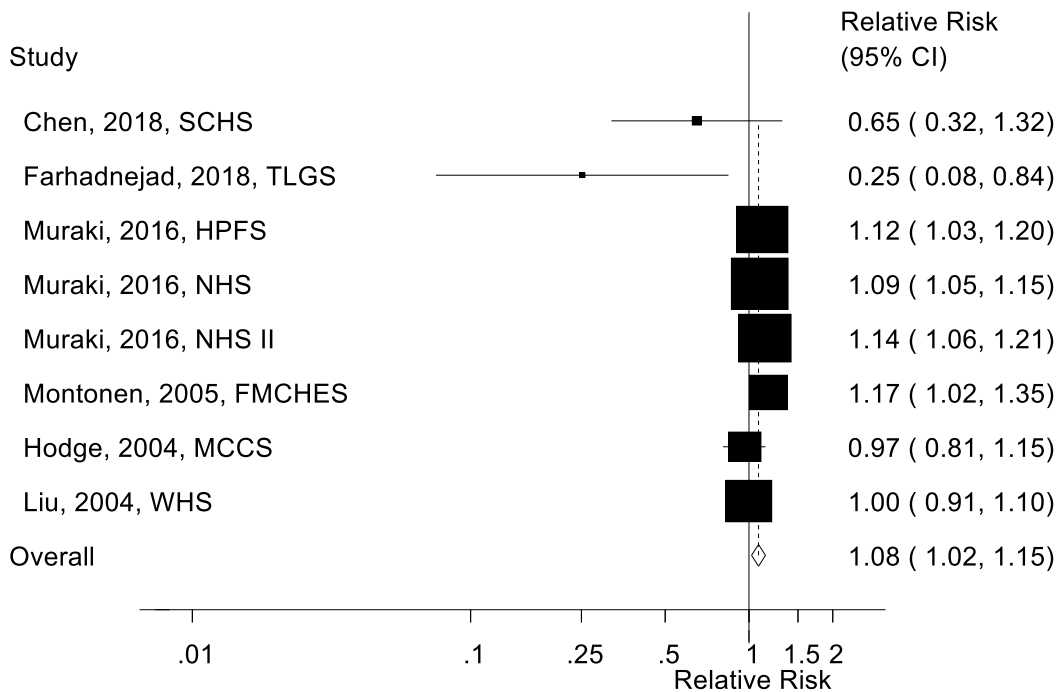
Supplementary Figure 57. Boiled potatoes and type 2 diabetes, nonlinear dose-response analysis



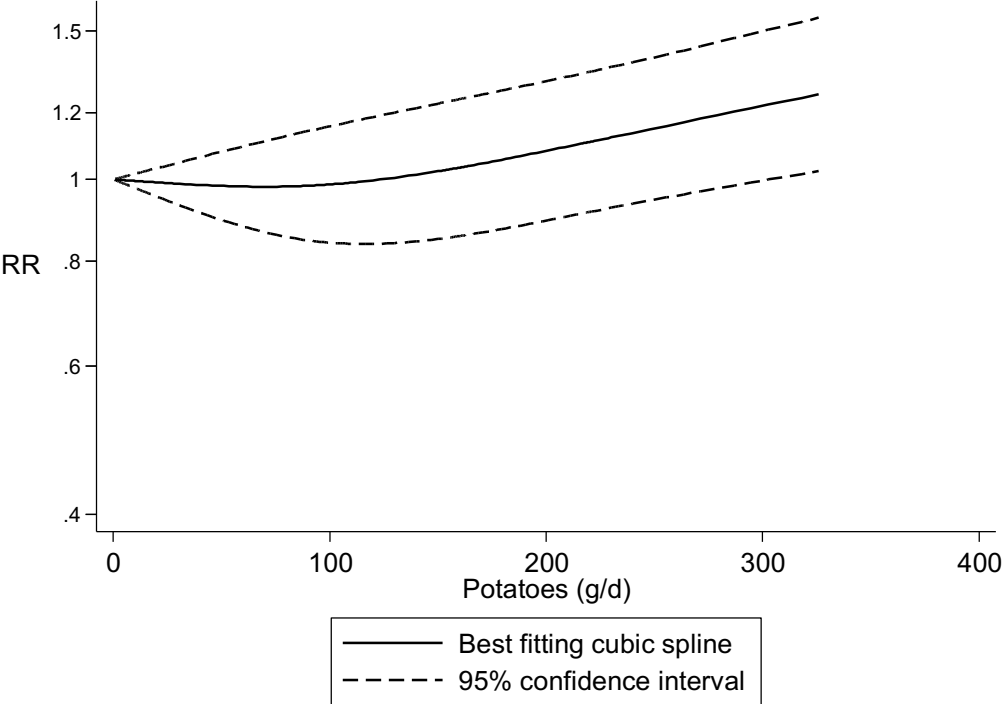
Supplementary Figure 79. Potatoes, total and type 2 diabetes, high vs. low



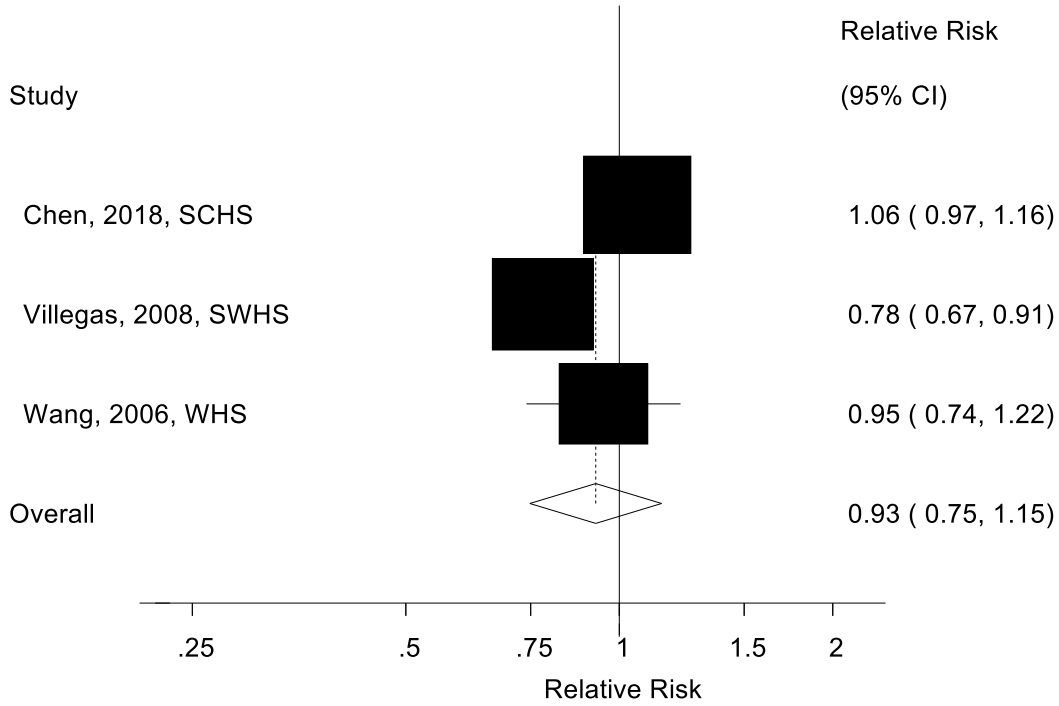
Supplementary Figure 80. Potatoes, total and type 2 diabetes, dose-response analysis per 100 g/d



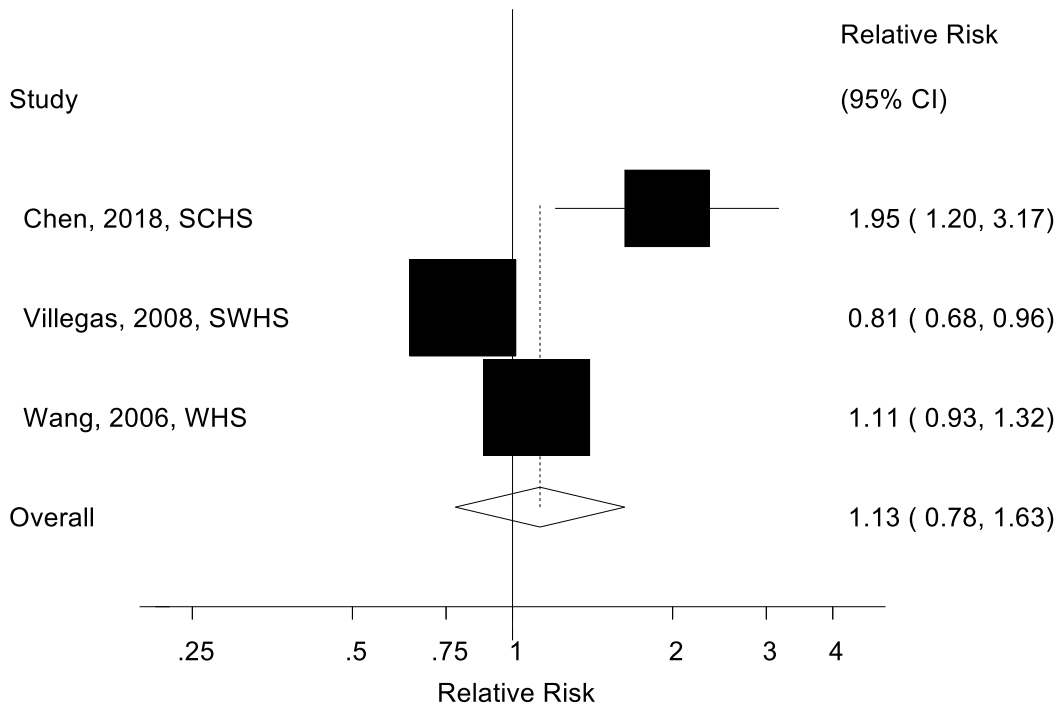
Supplementary Figure 81. Potatoes, total and type 2 diabetes, nonlinear dose-response analysis



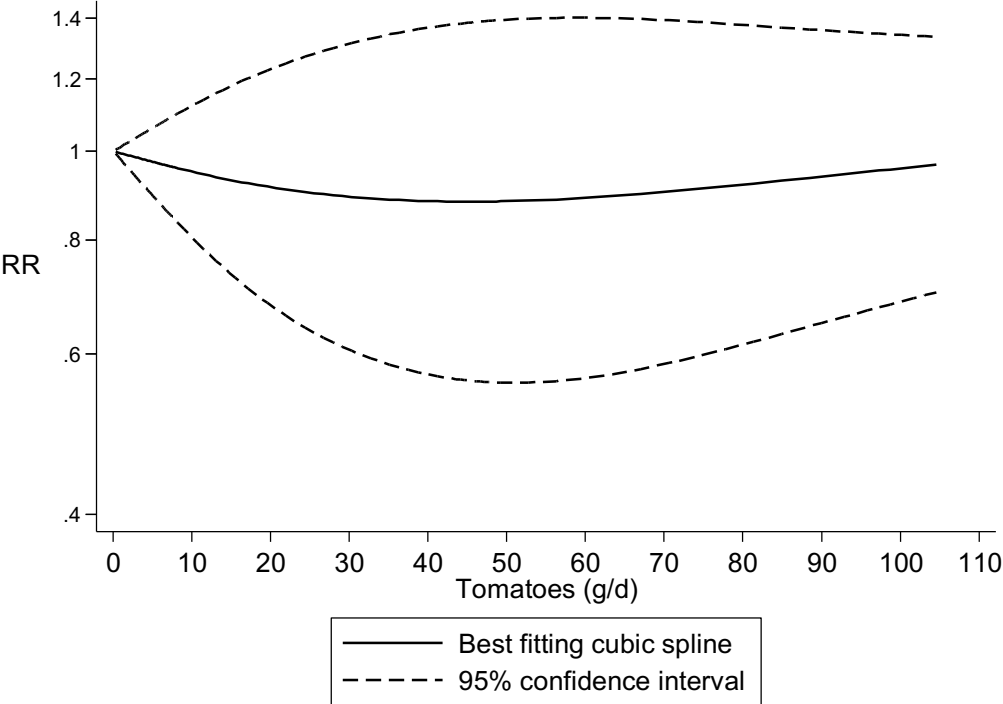
Supplementary Figure 82. Tomatoes and type 2 diabetes, high vs. low



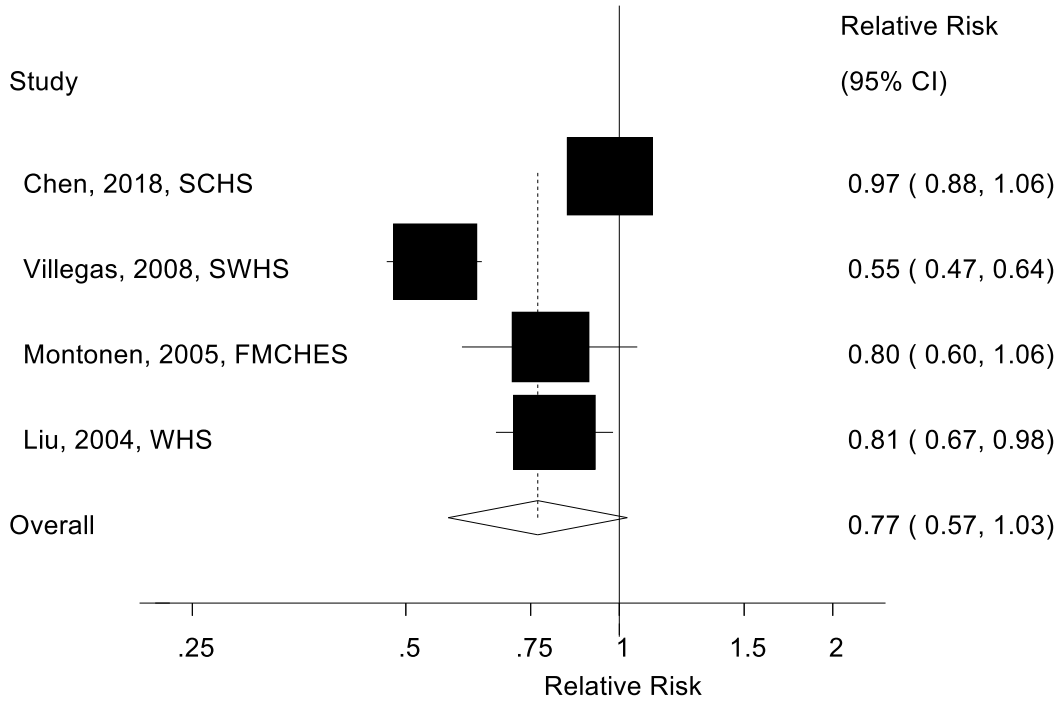
Supplementary Figure 83. Tomatoes and type 2 diabetes, dose-response analysis per 100 g/d



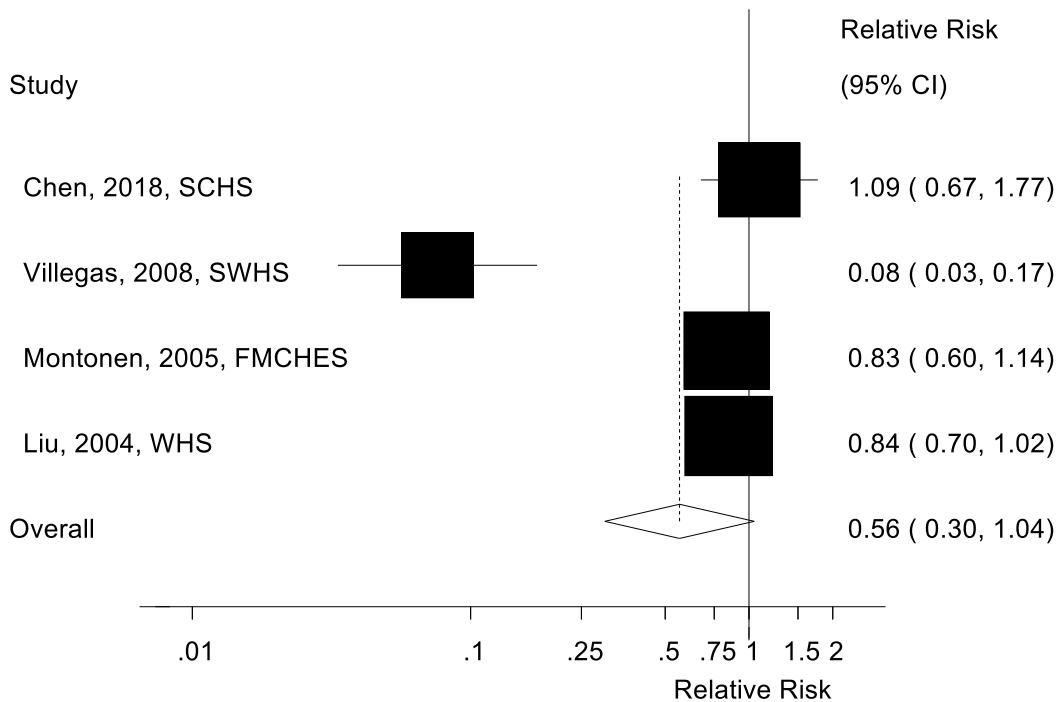
Supplementary Figure 84. Tomatoes and type 2 diabetes, nonlinear dose-response analysis



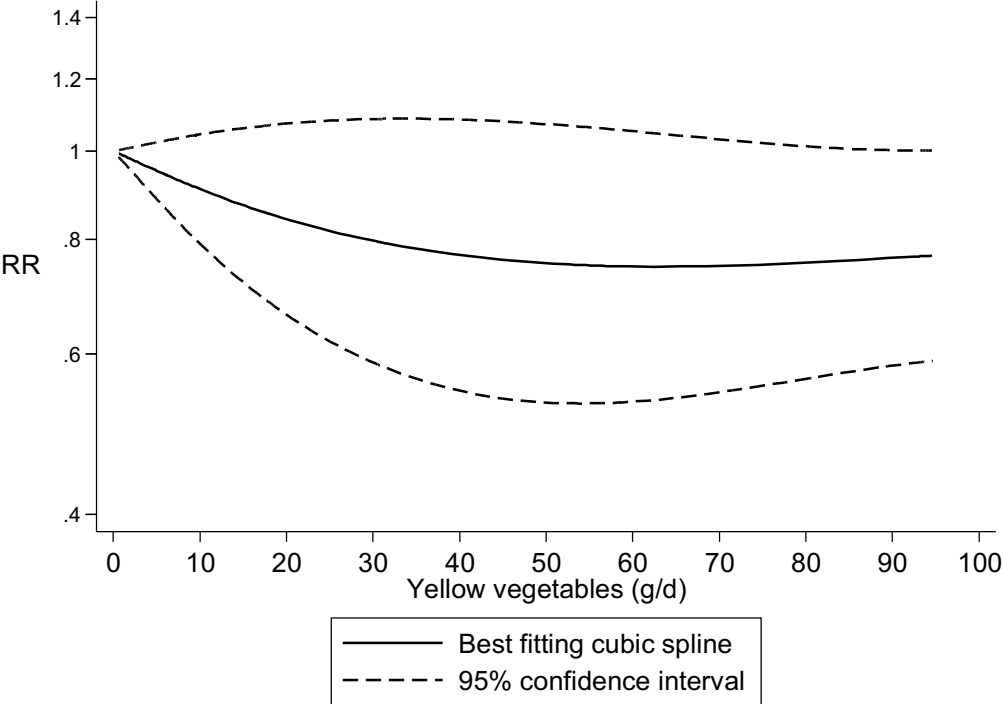
Supplementary Figure 85. Yellow vegetables and type 2 diabetes, high vs. low



Supplementary Figure 86. Yellow vegetables and type 2 diabetes, dose-response analysis per 100 g/d

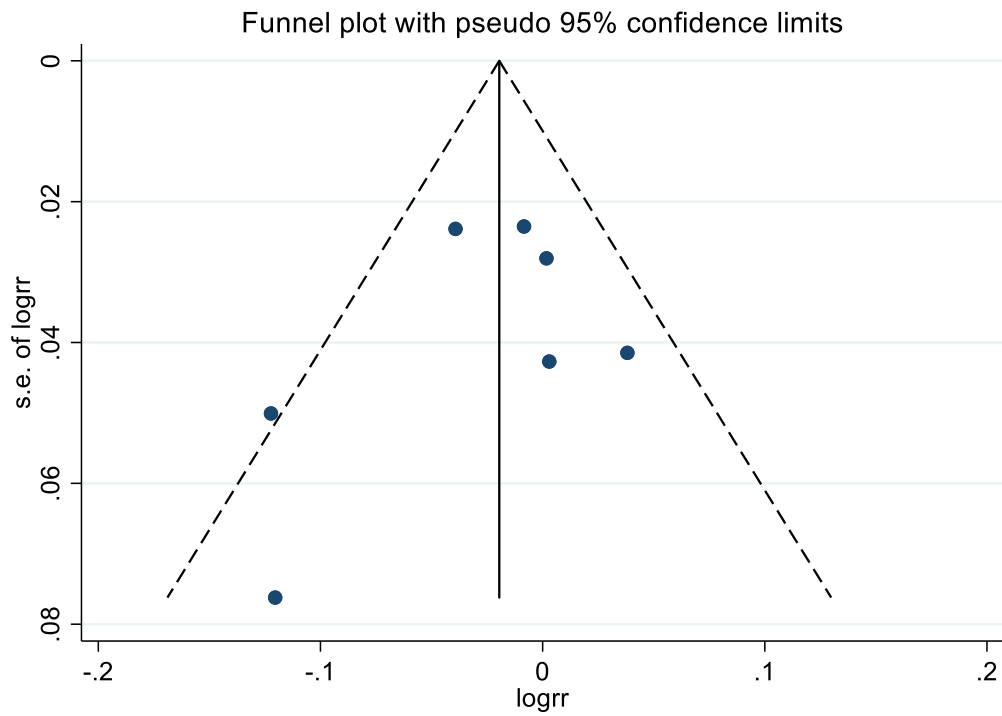


Supplementary Figure 87. Yellow vegetables and type 2 diabetes, nonlinear dose-response analysis

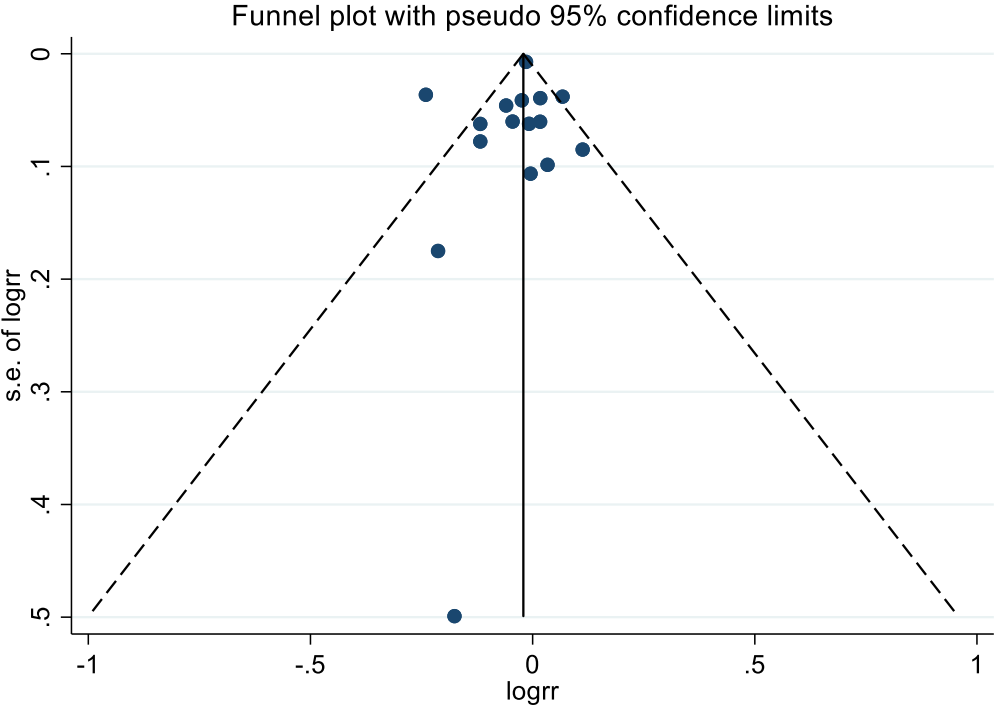


Funnel plots

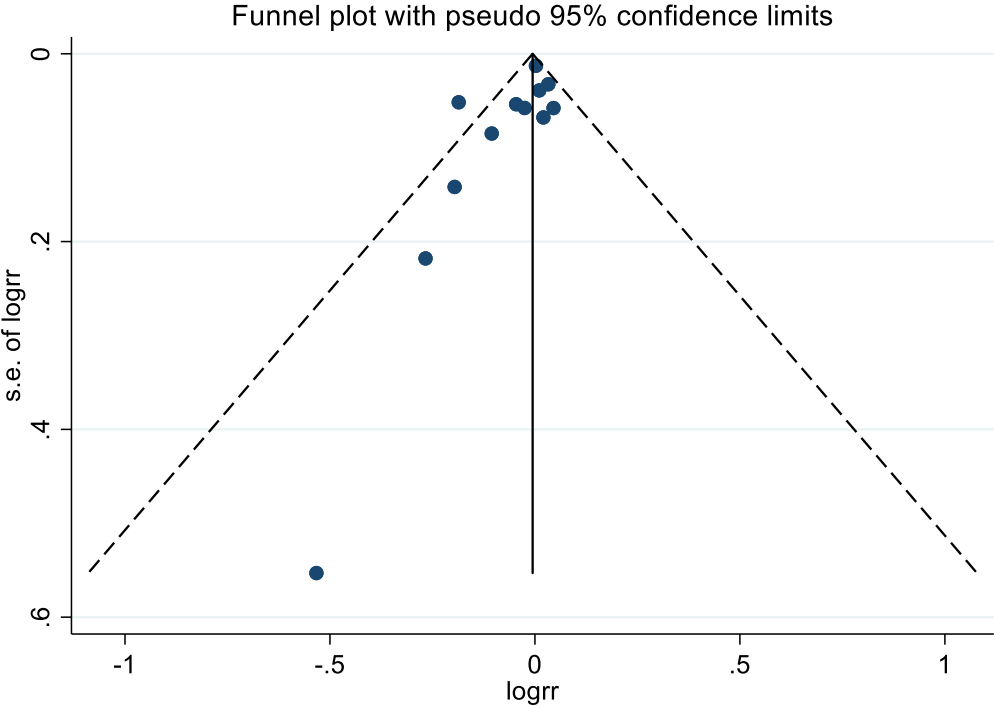
Supplementary Figure 88. Funnel plot of fruit and vegetables and type 2 diabetes



Supplementary Figure 89. Funnel plot of fruits and type 2 diabetes

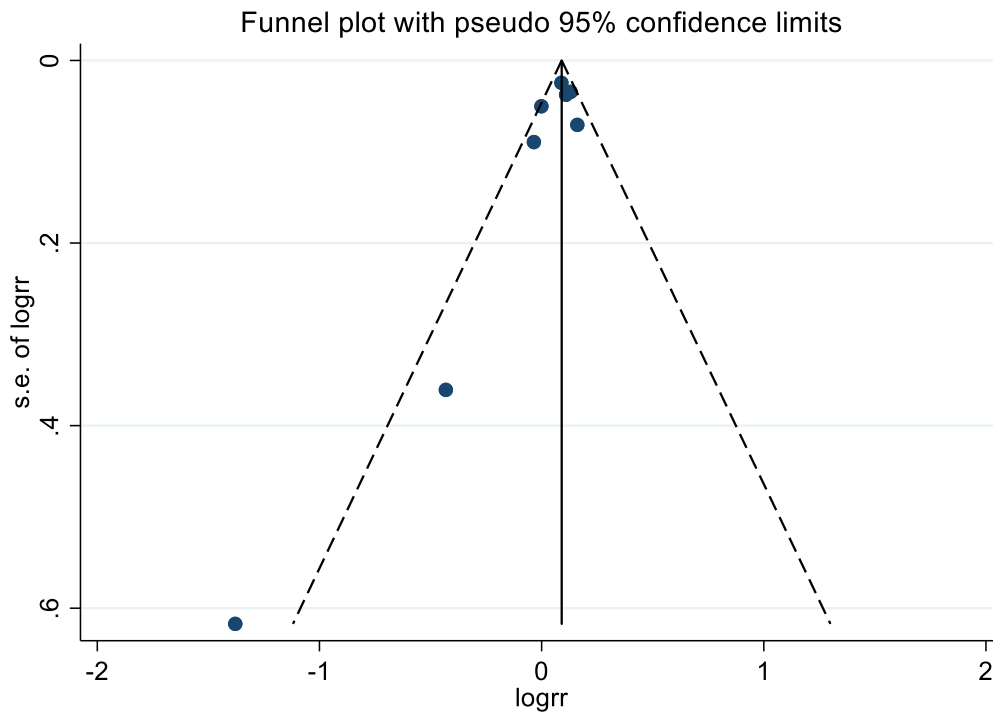


Supplementary Figure 90. Funnel plot of vegetables and type 2 diabetes



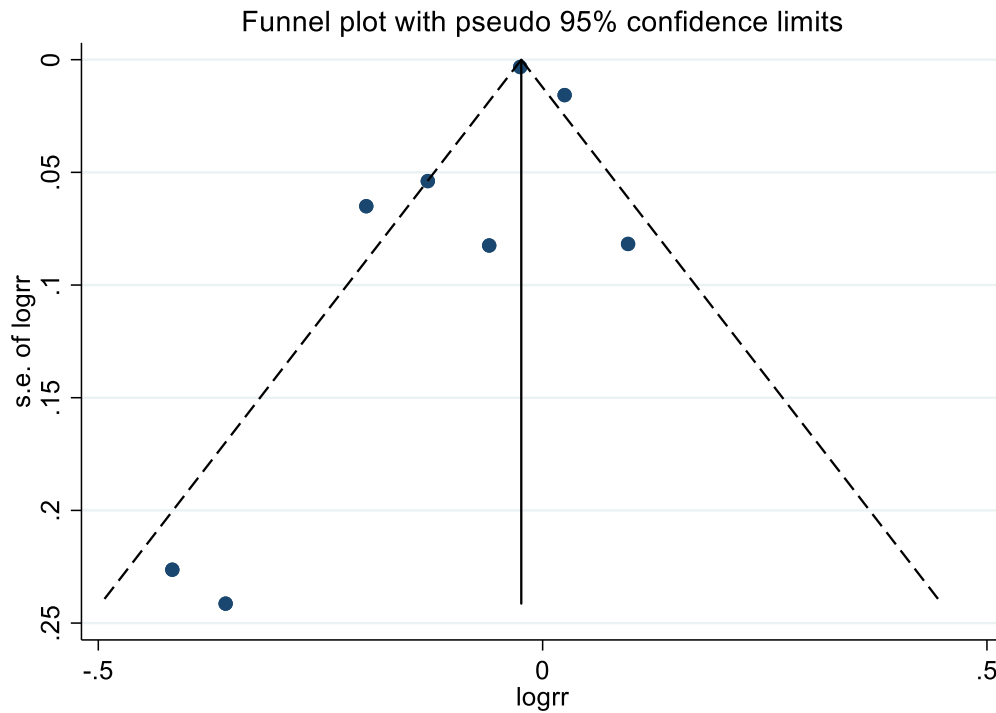
Egger's test attenuated from 0.08 to 0.12 when excluding the study by Hodge et al., which appeared to be an outlier. However, the summary RR was not materially altered, 0.98 (0.94-1.02, $I^2 = 50.9\%$, $P=0.03$).

Supplementary Figure 91. Funnel plot of potatoes and type 2 diabetes



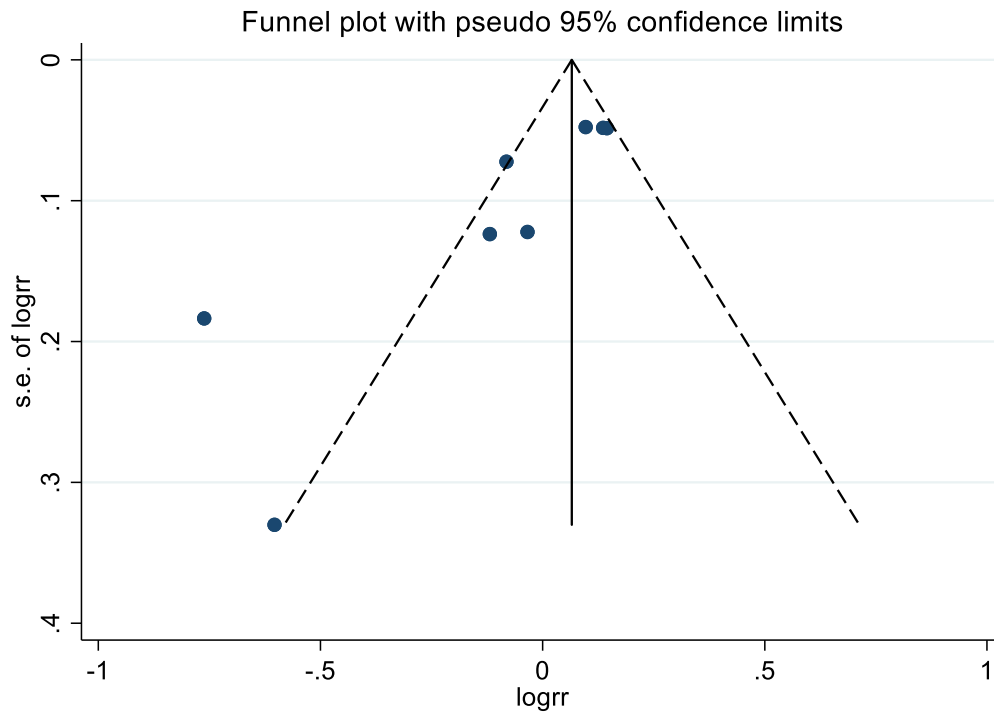
Although there was indication of publication bias with Egger's test ($P = 0.06$) and by inspection of the funnel plot, the asymmetry in the funnel plot indicated missing positive studies. Excluding one outlying study by Farhadnejad et al. attenuated Egger's test to 0.23, but did not substantially alter the results, summary RR = 1.09 (95% CI: 1.04-1.14, $I^2 = 40.2\%$, $P = 0.12$).

Supplementary Figure 92. Funnel plot of green leafy vegetables and type 2 diabetes



Although Egger's test was not significant ($P = 0.46$), there was some indication of asymmetry in the funnel plot. This appeared to be driven by the studies of Cooper et al and Kurotani et al. However, the results were not materially altered by exclusion of these two studies, summary RR = 0.96 (95% CI: 0.92-1.01, $I^2 = 78.4\%$).

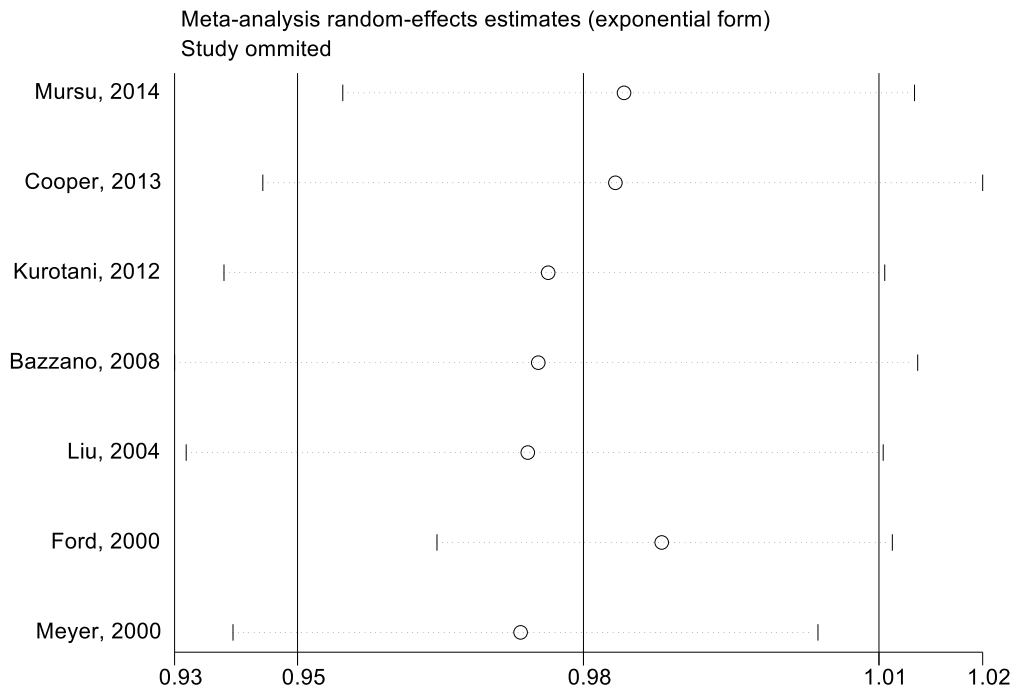
Supplementary Figure 93. Funnel plot of cruciferous vegetables and type 2 diabetes



There was evidence of publication bias with Egger's test ($P = 0.006$), which remained significant ($P = 0.05$) after exclusion of two apparently outlying studies (Mursu et al and Villegas et al), and the association remained non-significant, summary RR=1.06 (95% CI: 0.98-1.15, $I^2 = 57\%$), although the direction of the association changed.

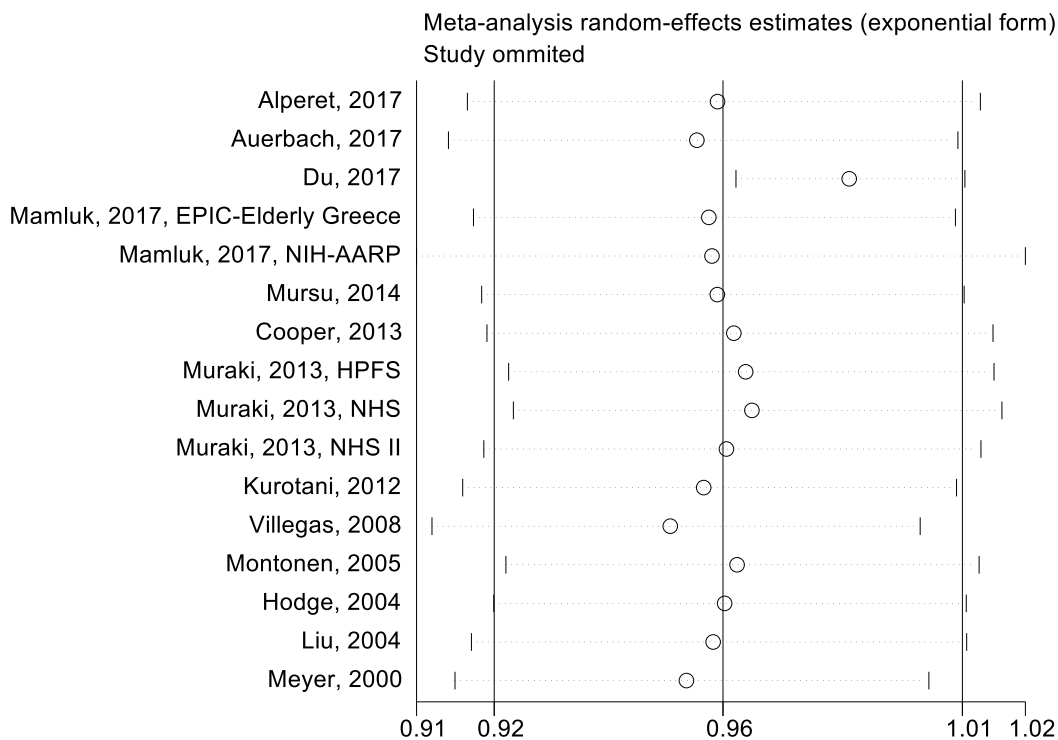
Influence analyses

Supplementary Figure 94. Influence analysis of fruit and vegetables and type 2 diabetes



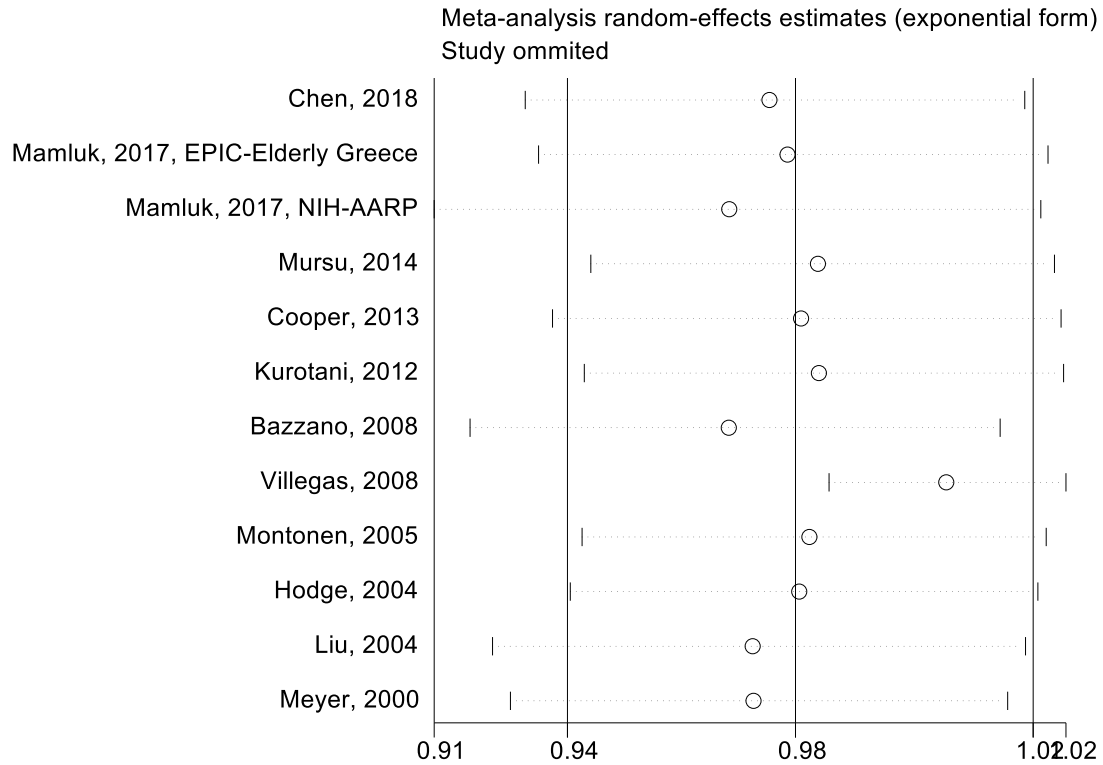
Study omitted	e ^{coef.}	[95% Conf. Interval]
Mursu, 2014	0.98320258	0.95193875 1.0154932
Cooper, 2013	0.98224336	0.94305086 1.0230646
Kurotani, 2012	0.97476596	0.93873465 1.0121802
Bazzano, 2008	0.97366518	0.93324012 1.0158414
Liu, 2004	0.97250021	0.93453473 1.0120081
Ford, 2000	0.98739189	0.96240878 1.0130235
Meyer, 2000	0.97170234	0.93973494 1.0047572
Combined	0.97869077	0.94691062 1.0115375

Supplementary Figure 95. Influence analysis of fruits and type 2 diabetes



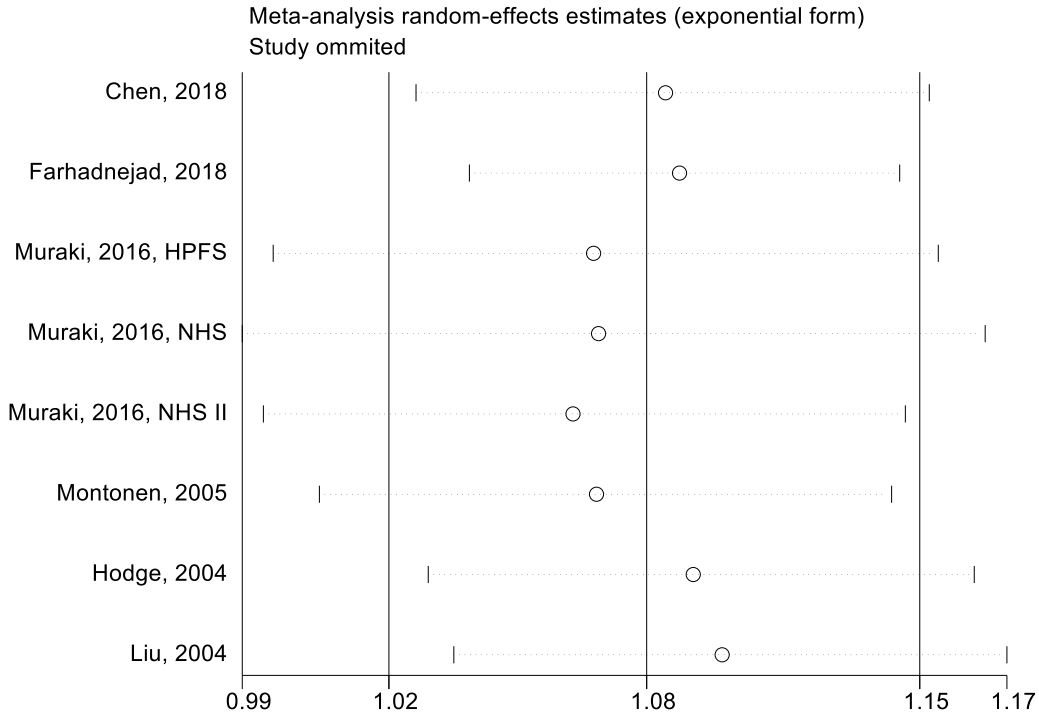
Study omitted	e ^{coef}	[95% Conf. Interval]
Alperet, 2017	0.9632051	0.91660255 1.012177
Auerbach, 2017	0.95935804	0.9130711 1.0079914
Du, 2017	0.98773992	0.96664274 1.0092975
Mamluk, 2017, EPIC-Elderly Greece	0.96158373	0.91773182 1.0075309
Mamluk, 2017, NIH-AARP	0.96217495	0.90712595 1.0205647
Mursu, 2014, KIHD	0.96316457	0.91926324 1.0091624
Cooper, 2013	0.96624291	0.92024559 1.0145394
Muraki, 2013	0.96845031	0.9242872 1.0147235
Muraki, 2013	0.96961492	0.92517221 1.0161926
Muraki, 2013	0.96486109	0.91966164 1.012282
Kurotani, 2012	0.96063113	0.91572422 1.0077401
Villegas, 2008	0.95439708	0.90999991 1.0009604
Montonen, 2005	0.966856	0.92377824 1.0119426
Hodge, 2004	0.96452886	0.92151368 1.0095519
Liu, 2004	0.96238476	0.91735351 1.0096265
Meyer, 2000	0.95741743	0.91428953 1.0025797
Combined	0.96423089	0.92160247 1.0088311

Supplementary Figure 96. Influence analysis of vegetables and type 2 diabetes



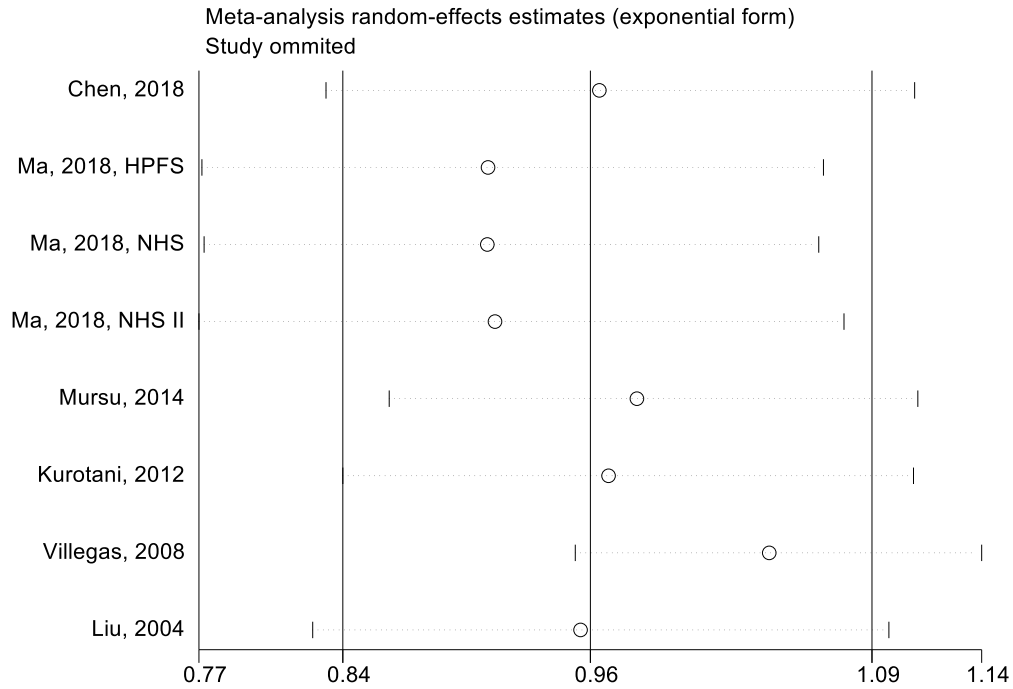
Study omitted	e ^{coef.}	[95% Conf. Interval]
Chen, 2018	0.9710921	0.92872405 1.0153929
Mamluk, 2017, EPIC-Elderly Greece	0.97423261	0.93104196 1.0194268
Mamluk, 2017, NIH-AARP	0.96411711	0.91292655 1.0181781
Mursu, 2014	0.97950399	0.94010854 1.0205504
Cooper, 2013	0.97658521	0.93346846 1.0216936
Kurotani, 2012	0.9796738	0.93898165 1.0221294
Bazzano, 2008	0.96404517	0.91915435 1.0111284
Villegas, 2008	1.0017768	0.98143595 1.0225393
Montonen, 2005	0.97802418	0.93858677 1.0191187
Hodge, 2004	0.97626317	0.93654597 1.0176647
Liu, 2004	0.9681868	0.92305547 1.0155247
Meyer, 2000	0.96833163	0.92615956 1.012424
Combined	0.97561402	0.93605213 1.016848

Supplementary Figure 97. Influence analysis of potatoes and type 2 diabetes



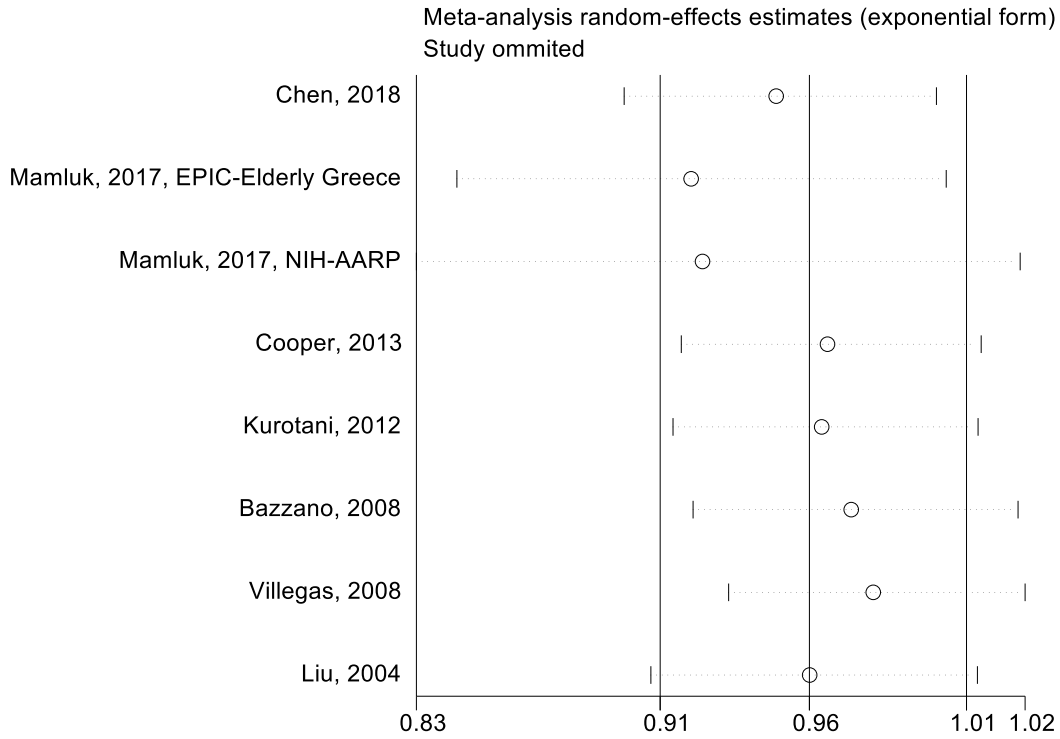
Study omitted	e ^{coef.}	[95% Conf. Interval]
Chen, 2018	1.0865777	1.0277396 1.1487839
Farhadnejad, 2018	1.089886	1.0403271 1.1418056
Muraki, 2016, HPFS	1.0696121	0.99405956 1.150907
Muraki, 2016, NHS	1.0707895	0.98676884 1.1619643
Muraki, 2016, NHS II	1.064756	0.99175006 1.1431361
Montonen, 2005	1.0703043	1.0049591 1.1398983
Hodge, 2004	1.093115	1.0306273 1.1593915
Liu, 2004	1.0999411	1.0366597 1.1670854
Combined	1.0821336	1.0213531 1.1465311

Supplementary Figure 98. Influence analysis of cruciferous vegetables and type 2 diabetes



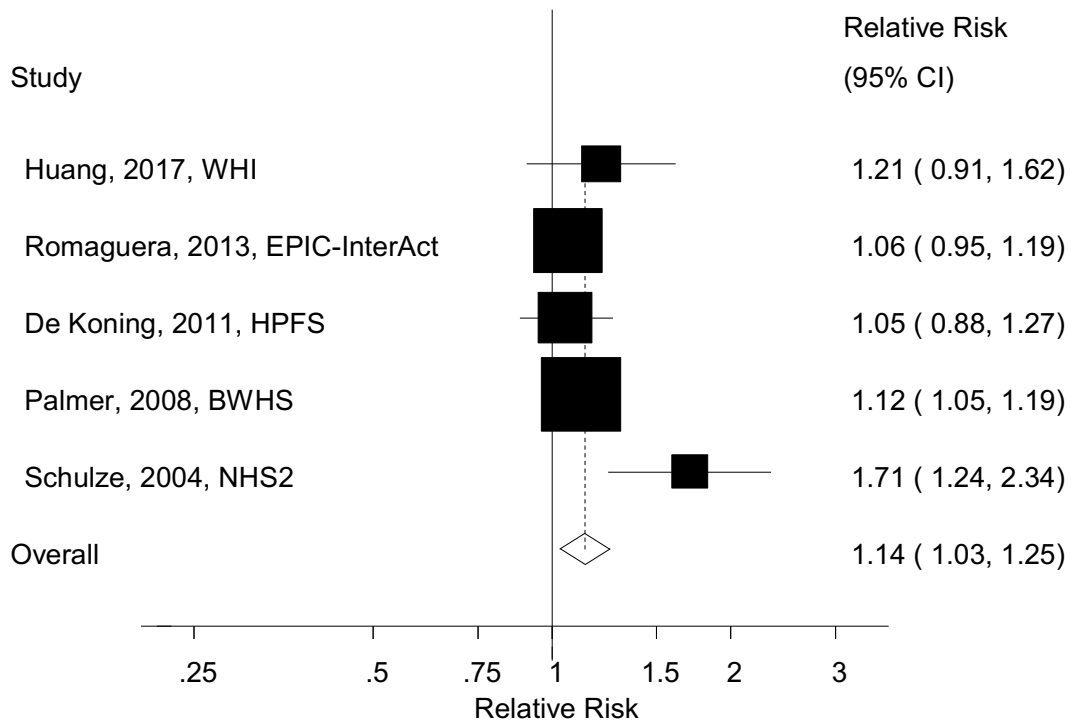
Study omitted	e ^{coef.}	[95% Conf. Interval]	
Chen, 2018	0.96210164	0.8343128	1.1094635
Ma, 2018, HPFS	0.91004604	0.77629888	1.0668364
Ma, 2018, NHS	0.90970498	0.77731478	1.0646435
Ma, 2018, NHS II	0.91330427	0.77489197	1.0764399
Mursu, 2014	0.97963929	0.86384547	1.1109545
Kurotani, 2012	0.96639293	0.84216845	1.1089413
Villegas, 2008	1.0414906	0.95082915	1.1407964
Liu, 2004	0.95329195	0.82808661	1.0974281
Combined	0.95789886	0.84217237	1.0895278

Supplementary Figure 99. Influence analysis of green leafy vegetables and type 2 diabetes



Study omitted	e ^{coef.}	[95% Conf. Interval]	
Chen, 2018	0.94474554	0.89619291	0.99592859
Mamluk, 2017, EPIC-Elderly Greece	0.9175899	0.84276497	0.99905813
Mamluk, 2017, NIH-AARP	0.92121667	0.82982373	1.0226753
Cooper, 2013	0.96114069	0.91443449	1.0102326
Kurotani, 2012	0.95928353	0.91180414	1.0092353
Bazzano, 2008	0.96871805	0.91820931	1.0220052
Villegas, 2008	0.97576261	0.9295674	1.0242535
Liu, 2004	0.95545352	0.90472227	1.0090294
Combined	0.95537042	0.90770012	1.0055443

Supplementary Figure 100. Influence analysis of fruits drinks and type 2 diabetes (excluding Montonen because of extreme result)



Study	ES	[95% Conf. Interval]	
Huang, 2017, WHI	1.210	0.907	1.615
Romaguera, 2013, EPI	1.063	0.951	1.188
De Koning, 2011, HPF	1.053	0.884	1.265
Palmer, 2008, BWHS	1.119	1.053	1.189
Schulze, 2004, NHS2	1.705	1.244	2.339
D+L pooled ES	1.136	1.030	1.252