

Supplemental Material

Table S1. Search Strategy.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

§A maximum of 9 points could be awarded.

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

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

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

- ² Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between total fruit and vegetable intake and incident CVD ($p < 0.001$).
- ³ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and incident CVD ($p = 0.004$).
- ⁴ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.90) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.97) crosses the MID.
- ⁵ No downgrade for inconsistency as analyses for inconsistency could not be performed due to < 2 observations available.
- ⁶ Downgrade for serious indirectness as evidence is based on 1 cohort of female health-professionals residing in the USA and may not be generalizable to different populations.
- ⁷ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.95 to 1.27) includes both clinically important benefit ($RR \leq 0.95$) and harm ($RR \geq 1.05$).
- ⁸ No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. < 10 observations available).
- ⁹ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.80) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.96) crosses the MID.
- ¹⁰ Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between citrus fruit intake and CVD incidence ($p = 0.033$).
- ¹¹ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.93 to 1.07) includes both clinically important benefit ($RR < 0.95$) and harm ($RR \geq 1.05$).
- ¹² Downgrade for serious indirectness as evidence is based on a predominately ($> 78\%$) female population and may not be generalizable to different populations.
- ¹³ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between pomes intake and incident CVD ($p = 0.043$).
- ¹⁴ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity ($I^2 = 85\%$, $p = 0.01$), which could not be explored through sensitivity due to only 2 observations available.
- ¹⁵ Downgrade for serious indirectness as evidence is based on a predominately (97%) female populations of which most are health professionals, and may not be generalizable to different populations.
- ¹⁶ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.57) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.10) crosses the MID.
- ¹⁷ Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity ($I^2 = 52\%$, $p = 0.04$). Although the removal of Buil-Cosiales et al. 2016 during sensitivity analysis did partially explain the heterogeneity ($I^2 = 27\%$, $p = 0.22$), the presence of residual heterogeneity could not be excluded.
- ¹⁸ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.90 to 1.08) includes both clinically important benefit ($RR \leq 0.95$) and harm ($RR \geq 1.05$).
- ¹⁹ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.76) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.
- ²⁰ Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between green leafy vegetables intake and CVD mortality ($p = 0.01$).
- ²¹ Downgrade for serious indirectness as evidence is based on a predominately (88%) female population and may not be generalizable to different populations.
- ²² Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.78 to 1.20) includes both clinically important benefit ($RR \leq 0.95$) and harm ($RR \geq 1.05$).

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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



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

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

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

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

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

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

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

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

³ No downgrade for inconsistency as the presence of inter-study heterogeneity (I²=53%, p=0.002) was explained by the removal of Dauchet et al. 2010 (I²=0%, p=0.5)

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

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

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

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

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

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

¹⁰ Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I²=74%, p=0.008), which could not be explained by sensitivity analyses.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

- ¹⁸ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.77 to 1.21) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).
- ¹⁹ Downgrade for serious indirectness as evidence is based on a predominately (82.1%) female populations and may not be generalizable to different populations.
- ²⁰ Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I²=88%, p<0.00001). Although heterogeneity could be partially explained by the removal of Blekkenhorst et al. 2017 (I²=47%, p=0.13) during sensitivity analyses, the presence of residual heterogeneity could not be excluded.
- ²¹ Downgrade for serious indirectness as evidence is based on a predominately (95.4%) female populations and may not be generalizable to different populations.
- ²² Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.
- ²³ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.37 to 1.58) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).
- ²⁴ No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.
- ²⁵ Downgrade for serious indirectness as evidence is based on 1 female cohort and may not be generalizable to different populations.
- ²⁶ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.80 to 1.06) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).
- ²⁷ Downgrade for serious risk of bias as 39.3% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9).
- ²⁸ Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I²=88%, p<0.00001) which could not be explained by sensitivity analyses.
- ²⁹ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.
- ³⁰ Downgrade for serious risk of bias as 36.8% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9)
- ³¹ Downgrade for serious risk of bias as 48.0% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 1/9)
- ³² Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.82) includes the minimally important difference (MID) of 5% while the upper bound of the 95% CI (RR, 1.04) crosses the MID.

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

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

- ¹ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit and vegetable intake and stroke incidence (p=0.002).
- ² Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit intake and stroke incidence (p<0.001).
- ³ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I²=50%, p=0.006) that could not be explained during sensitivity analysis.
- ⁴ Upgrade for a dose-response gradient, as the MKSPLINE analysis revealed a significant non-linear inverse relationship between vegetable intake and stroke incidence with a departure from linearity at 1.5 servings/day (p=0.012)
- ⁵ No downgrade for inconsistency as the presence of inter-study heterogeneity (I²=50%, p=0.08) was explained by the removal of Hirvonen et al. 2000 – cerebral infraction (I²=0%, p=0.41) during sensitivity analysis.
- ⁶ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.94 to 1.13) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05)
- ⁷ No downgrade for publication bias as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (i.e. <10 observations available).
- ⁸ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I²=51%, p=0.04). Although the removal of Larsson et al. 2013 (I²=37%, p=0.14) or Yamada et al. 2011 (I²=39%, p=0.12) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.
- ⁹ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between citrus fruit intake and stroke incidence (p=0.033) and an MKSPLINE analysis revealed a significant non-linear inverse relationship between citrus fruit intake and stroke incidence (p=0.039).
- ¹⁰ No downgrade for inconsistency as the presence of inter-study heterogeneity (I²=73%, p=0.02) was explained by the removal of Scheffers et al. 2019 (I²=0%, p=0.47)
- ¹¹ Downgrade for serious imprecision, as the upper bound of the 95% CIs (RR, 0.99) crosses the MID (RR<0.95).
- ¹² Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between pommes intake and stroke incidence (p=0.003). MKSPLINE analyses could not be conducted due to small sample size.
- ¹³ Downgrade for serious indirectness as evidence is based on cohorts residing in Northern Europe and may not be generalizable to different populations.
- ¹⁴ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.80) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.99) crosses the MID.
- ¹⁵ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I²=62%, p=0.02). Although the removal of Larsson et al. 2013 (during sensitivity analysis did partially explain the heterogeneity (I²=40%, p=0.16), the presence of residual heterogeneity could not be excluded.
- ¹⁶ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.91 to 1.05) includes both clinically important benefit (RR<0.95) and harm (RR≥1.05).
- ¹⁶ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.79) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.98) crosses the MID.
- ¹⁷ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between green leafy vegetable intake and stroke incidence (p=0.008). MKSPLINE analyses could not be conducted due to small sample size.
- ¹⁸ No downgrade for inconsistency as analyses for inconsistency could not be performed due to <2 observations available.
- ¹⁹ Downgrade for serious indirectness as evidence is based on only 1 cohort of females for USA and may not be generalizable to different populations.
- ²⁰ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between tomato intake and stroke incidence (p=0.002). MKSPLINE analyses could not be conducted due to small sample size.

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

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

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

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

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

³ Downgrade for serious inconsistency as there was evidence of substantial inter-study heterogeneity (I²=75%, p<0.00001) which could not be explained by sensitivity analyses.

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

⁵ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I²=62%, p=0.0010). Although the removal of Wang et al. 2013 (I²=43%, p=0.05) or Leanders et al. 2014 (I²=48%, p=0.02) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

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

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

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

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

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

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

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

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

¹⁴ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity (I²=82%, p=0.0001). Although the removal of Wang et al. 2016 (I²=40%, p=0.17) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.

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

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

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

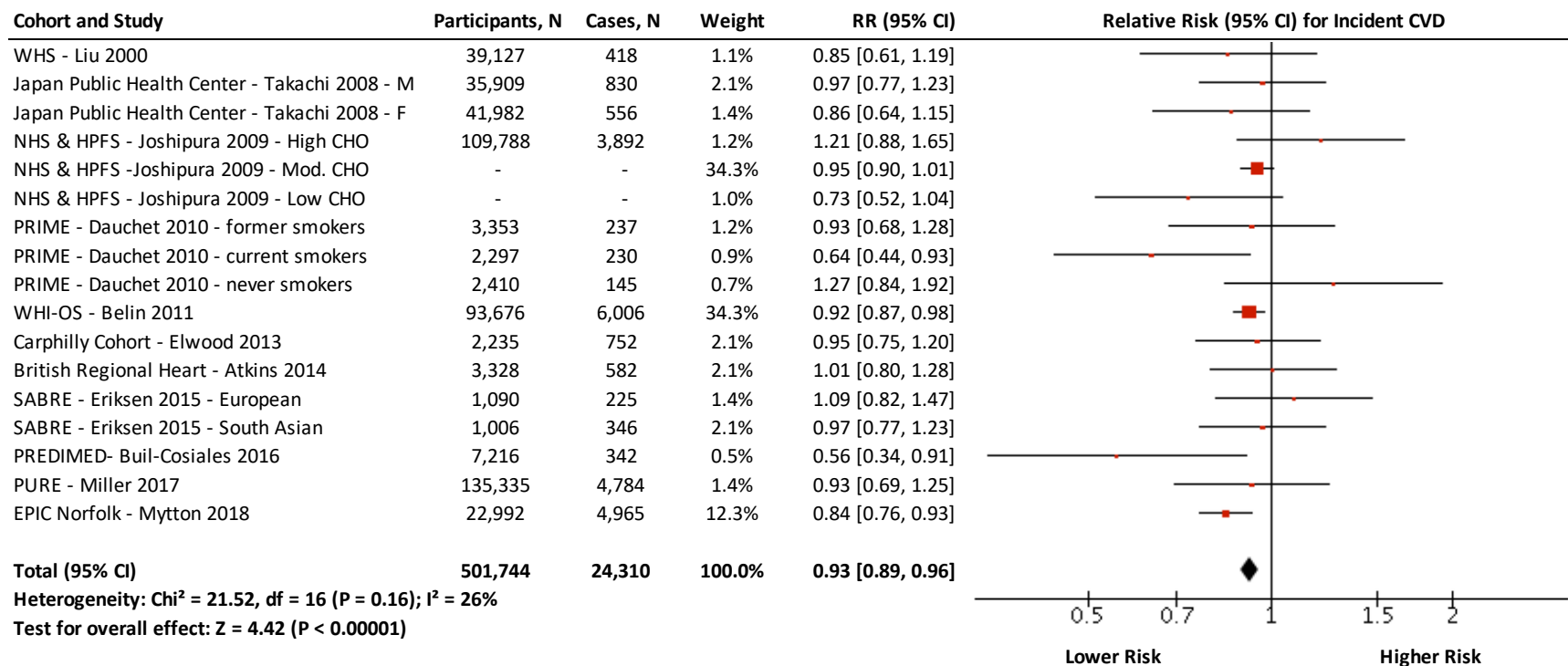
¹⁸ Downgrade for serious risk of bias as 62% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).

- ¹⁹ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between fruit juice intake and CHD mortality ($p=0.002$). MKSPLINE analyses could not be conducted due to small sample size.
- ²⁰ Downgrade for serious indirectness as evidence is based on a predominately (76%) female population and may not be generalizable to different populations.
- ²¹ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.53 to 1.02) includes both clinically important benefit ($RR<0.95$) and harm ($RR\geq 1.05$).
- ²² Downgrade for serious indirectness as evidence is based on a predominately (87%) female population and may not be generalizable to different populations.
- ²³ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.77) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.09) crosses the MID.
- ²⁴ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity ($I^2=96\%$, $p<0.00001$).
- ²⁵ Downgrade for serious imprecision, as the lower and upper bound of the 95% CIs (RR, 0.79 to 1.24) includes both clinically important benefit ($RR<0.95$) and harm ($RR\geq 1.05$).
- ²⁶ Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between carrots intake and stroke mortality ($p<0.001$).
- ²⁷ Downgrade for serious risk of bias as 79.4% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).
- ²⁸ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.85) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.01) crosses the MID.
- ²⁹ Downgrade for serious risk of bias as 50.0% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).
- ³⁰ Downgrade for serious inconsistency given evidence of substantial inter-study heterogeneity ($I^2=50\%$, $p=0.09$). Although the removal of Appleby et al. 2002 ($I^2=36\%$, $p=0.20$) or Wang et al. 2016 ($I^2=25\%$, $p=0.05$) during sensitivity analysis did partially explain the heterogeneity, the presence of residual heterogeneity could not be excluded.
- ³¹ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.83) includes the MID of 5% while the upper bound of the 95% CI (RR, 0.97) crosses the MID.
- ³² Upgrade for a dose-response gradient, as the GLST analysis revealed a significant linear inverse relationship between green leafy vegetable intake and CHD mortality ($p=0.032$). MKSPLINE analyses could not be conducted due to small sample size.
- ³³ Downgrade for serious risk of bias as 60.4% of effect estimate is based on Iso et al. 2007, which presented with a high risk of bias (Newcastle-Ottawa Score: 5/9).
- ³⁴ Downgrade for serious imprecision, as the lower bound of the 95% CI (RR, 0.94) includes the MID of 5% while the upper bound of the 95% CI (RR, 1.12) crosses the MID.
- ³⁵ Dose-response gradient could not be assessed due to insufficient dose ranges available to determine the presence of a linear/non-linear dose response.

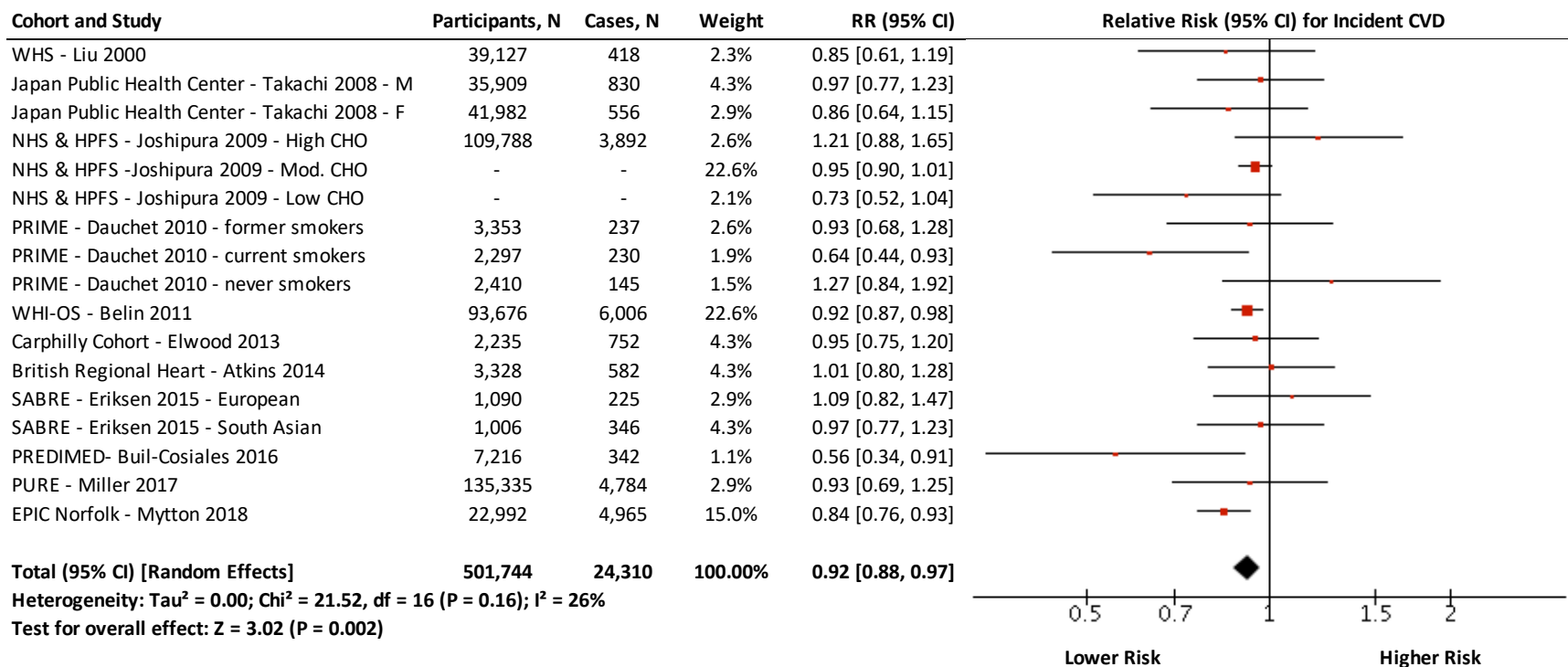
Figure S1. Relation between total fruit and vegetable intake and cardiovascular disease incidence (highest vs. lowest level of intake).

TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects



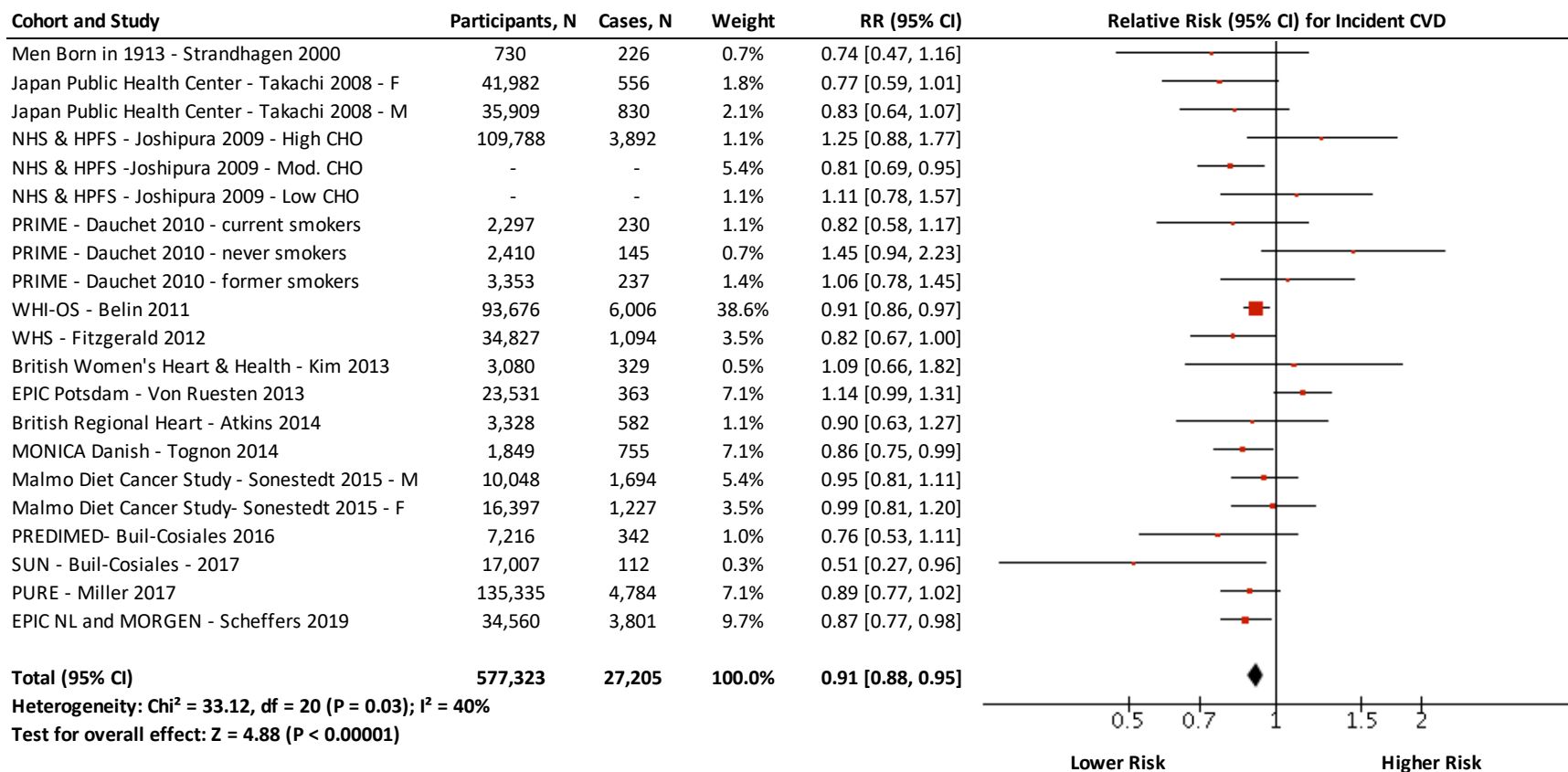
B. Random Effects



All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

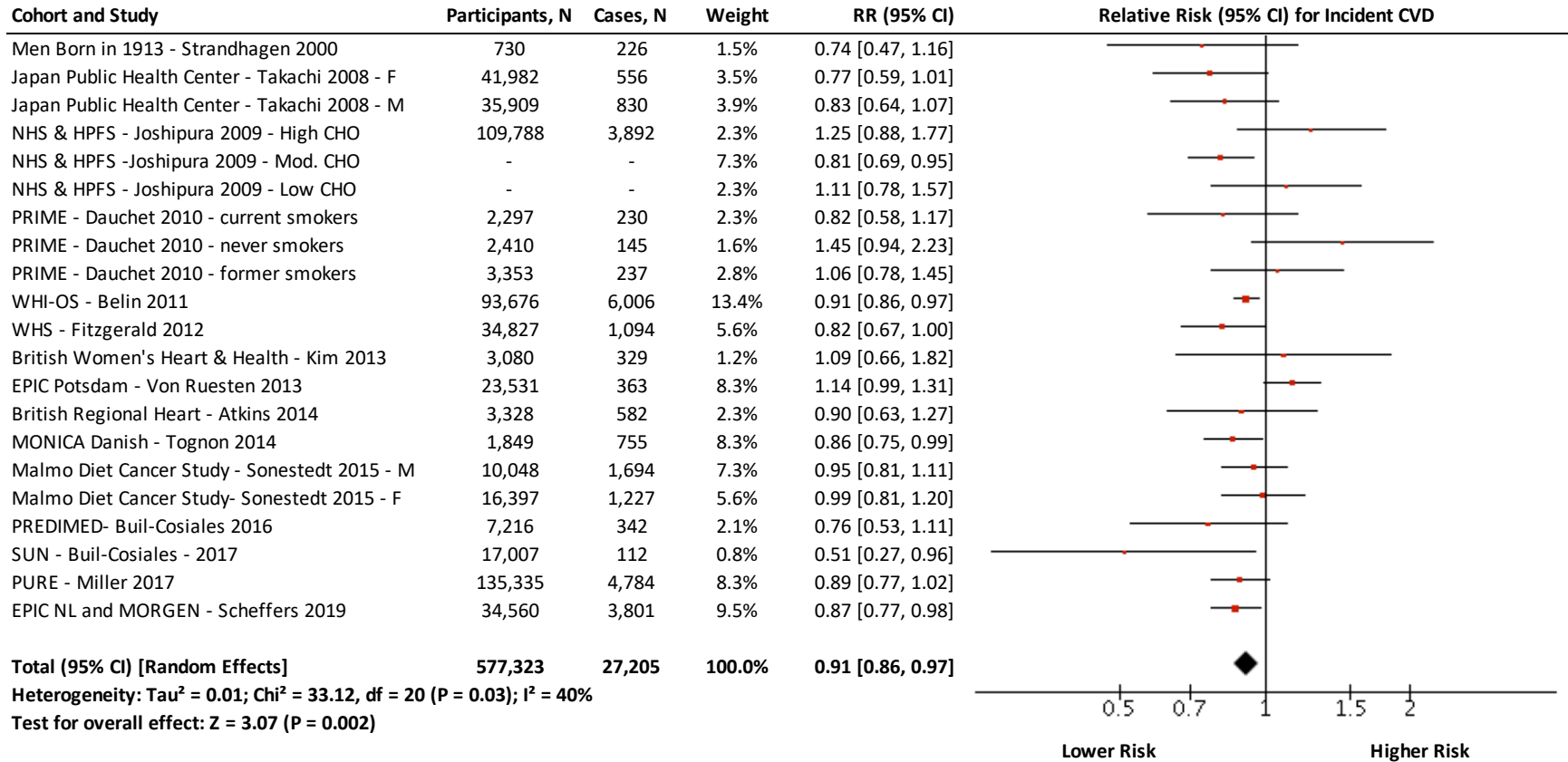


Figure S2. Relation between fruit intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: Chi² = 27.44, df = 18 (P = 0.07); I² = 34%
Test for overall effect: Z = 3.51 (P = 0.0004)

B. Random Effects

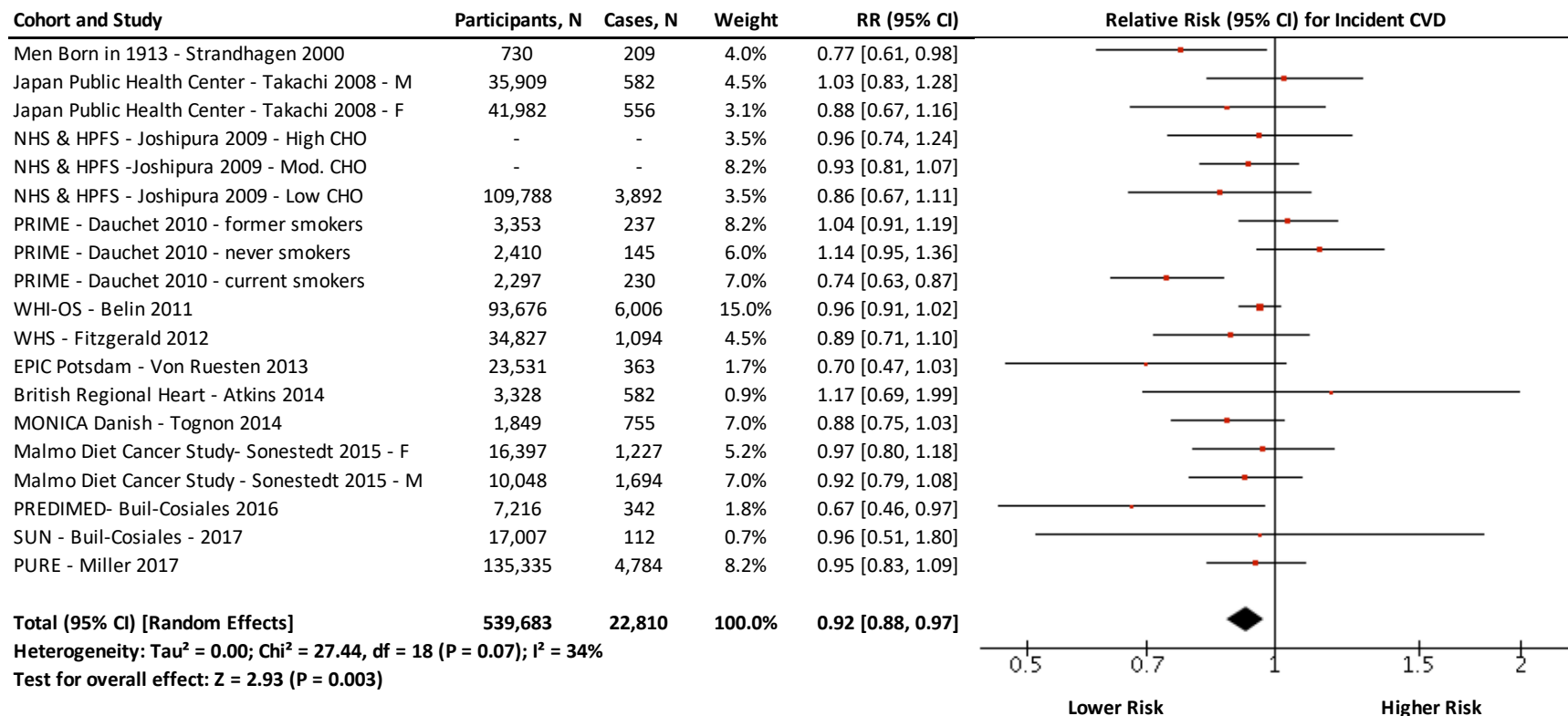


Figure S3. Relation between vegetable intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

BERRIES AND CARDIOVASCULAR DISEASE INCIDENCE

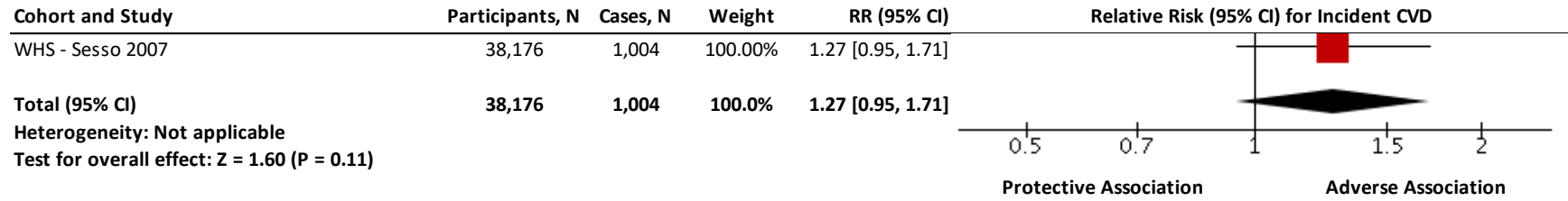


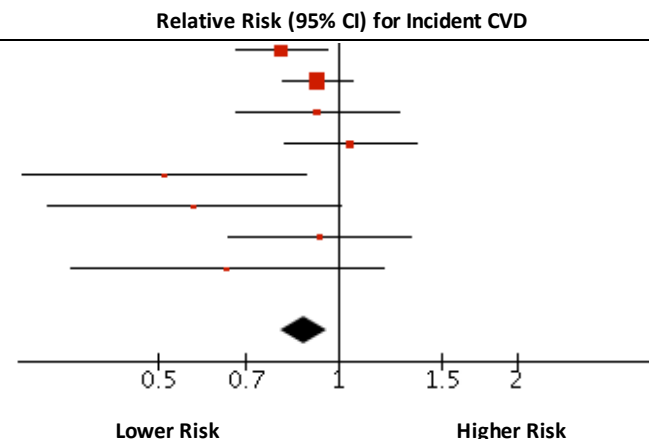
Figure S4. Relation between intake of berries and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

CITRUS FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 10.49$, $\text{df} = 7$ ($P = 0.16$); $I^2 = 33\%$
 Test for overall effect: $Z = 2.95$ ($P = 0.003$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.01$; $\text{Chi}^2 = 10.49$, $\text{df} = 7$ ($P = 0.16$); $I^2 = 33\%$
 Test for overall effect: $Z = 2.40$ ($P = 0.02$)

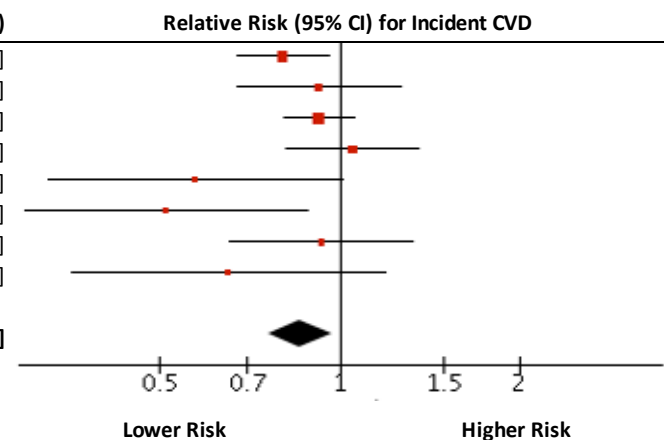


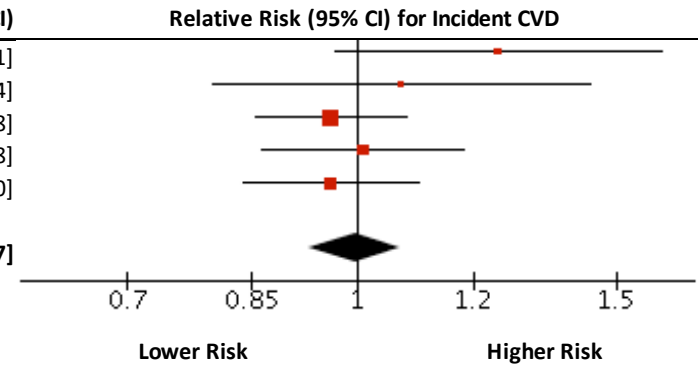
Figure S5. Relation between citrus fruit intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

FRUIT JUICE AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 3.86$, $\text{df} = 4$ ($P = 0.42$); $I^2 = 0\%$
 Test for overall effect: $Z = 0.06$ ($P = 0.95$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 3.86$, $\text{df} = 4$ ($P = 0.42$); $I^2 = 0\%$
 Test for overall effect: $Z = 0.06$ ($P = 0.95$)

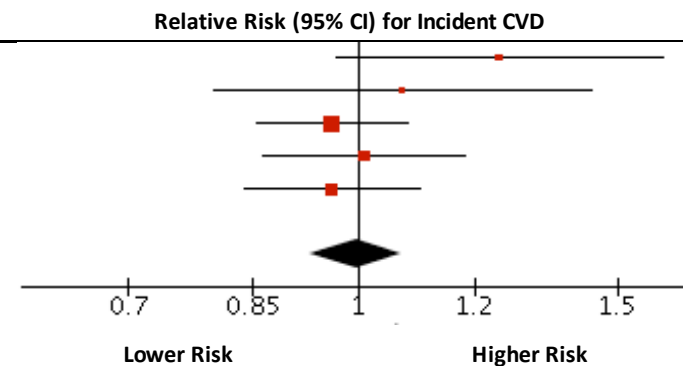


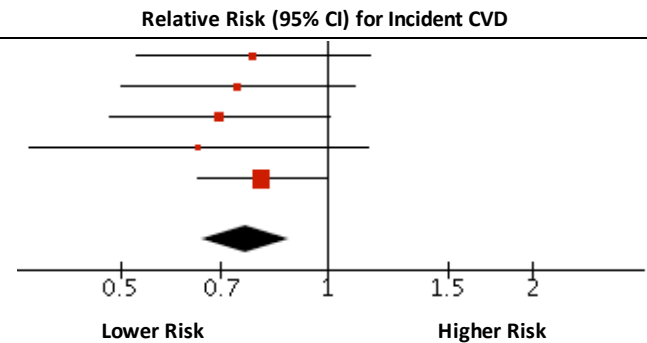
Figure S6. Relation between fruit juice intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

POMMES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 0.77$, $\text{df} = 4$ ($P = 0.94$); $I^2 = 0\%$
 Test for overall effect: $Z = 3.58$ ($P = 0.0003$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 0.77$, $\text{df} = 4$ ($P = 0.94$); $I^2 = 0\%$
 Test for overall effect: $Z = 3.58$ ($P = 0.0003$)

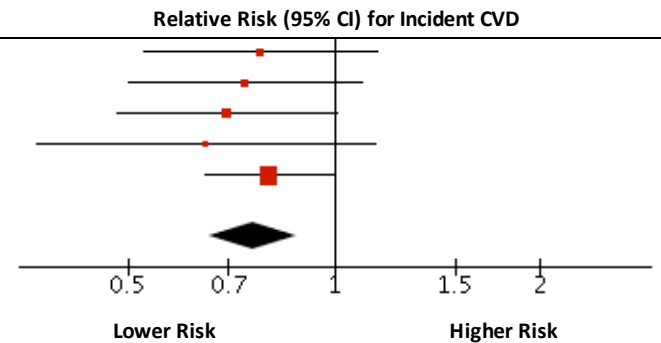
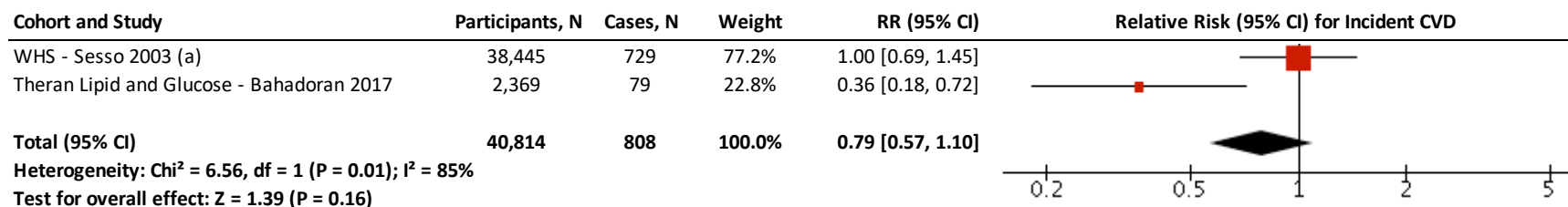


Figure S7. Relation between pommes intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

ALLIUM VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

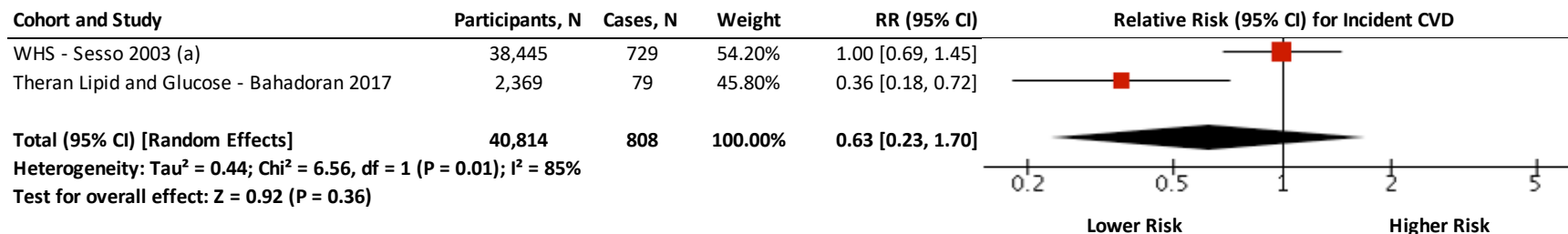


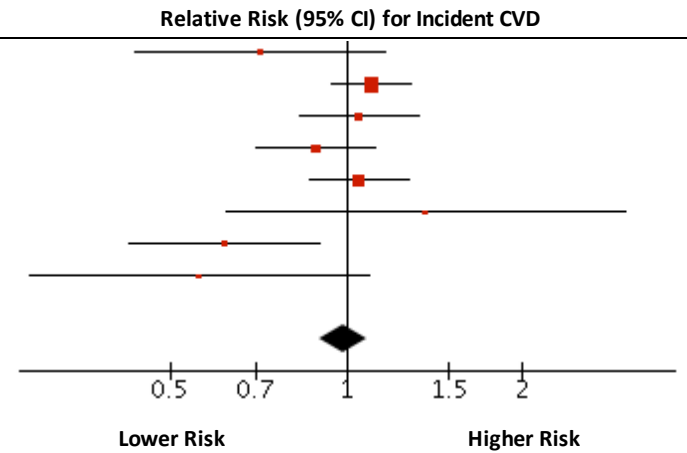
Figure S8. Relation between intake of allium vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and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

CRUCIFEROUS VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 14.65$, $\text{df} = 7$ ($P = 0.04$); $I^2 = 52\%$
 Test for overall effect: $Z = 0.28$ ($P = 0.78$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.02$; $\text{Chi}^2 = 14.65$, $\text{df} = 7$ ($P = 0.04$); $I^2 = 52\%$
 Test for overall effect: $Z = 0.91$ ($P = 0.36$)

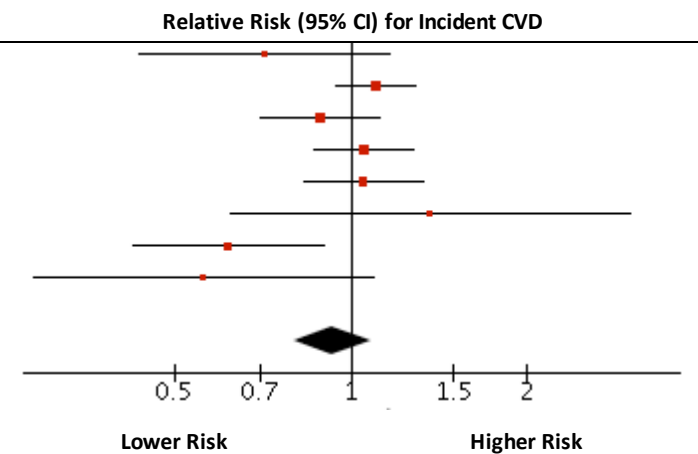


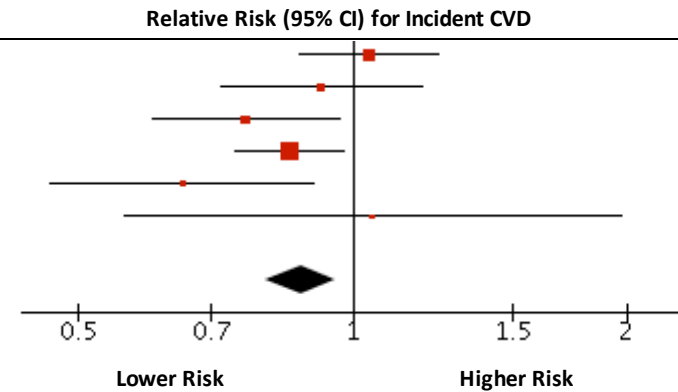
Figure S9. Relation between intake of cruciferous vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

GREEN LEAFY VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 8.69$, $\text{df} = 5$ ($P = 0.12$); $I^2 = 42\%$
 Test for overall effect: $Z = 2.93$ ($P = 0.003$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.01$; $\text{Chi}^2 = 8.69$, $\text{df} = 5$ ($P = 0.12$); $I^2 = 42\%$
 Test for overall effect: $Z = 2.16$ ($P = 0.03$)

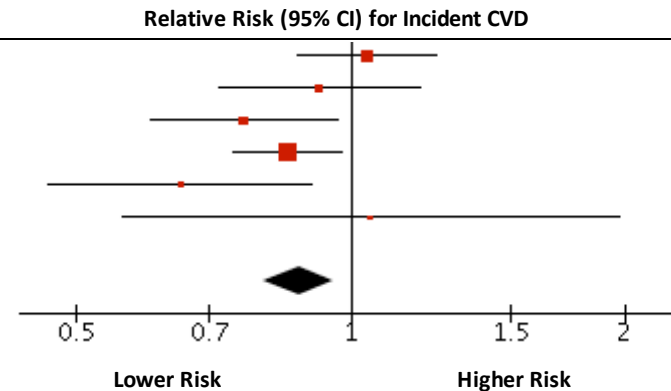


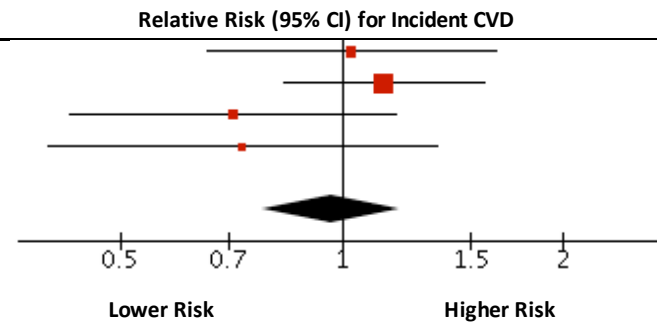
Figure S10. Relation between intake of green leafy vegetables and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

TOMATOES AND CARDIOVASCULAR DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 3.31$, $\text{df} = 3$ ($P = 0.35$); $I^2 = 9\%$
 Test for overall effect: $Z = 0.29$ ($P = 0.77$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.01$; $\text{Chi}^2 = 3.31$, $\text{df} = 3$ ($P = 0.35$); $I^2 = 9\%$
 Test for overall effect: $Z = 0.35$ ($P = 0.73$)

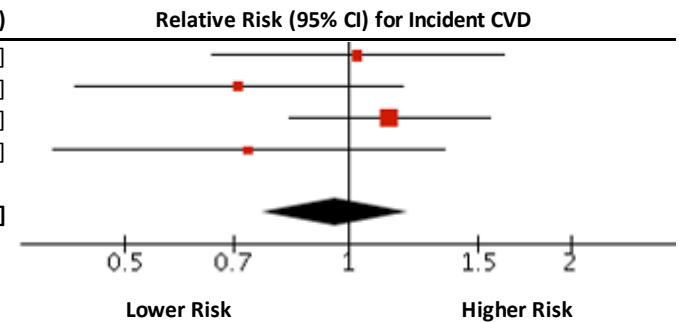
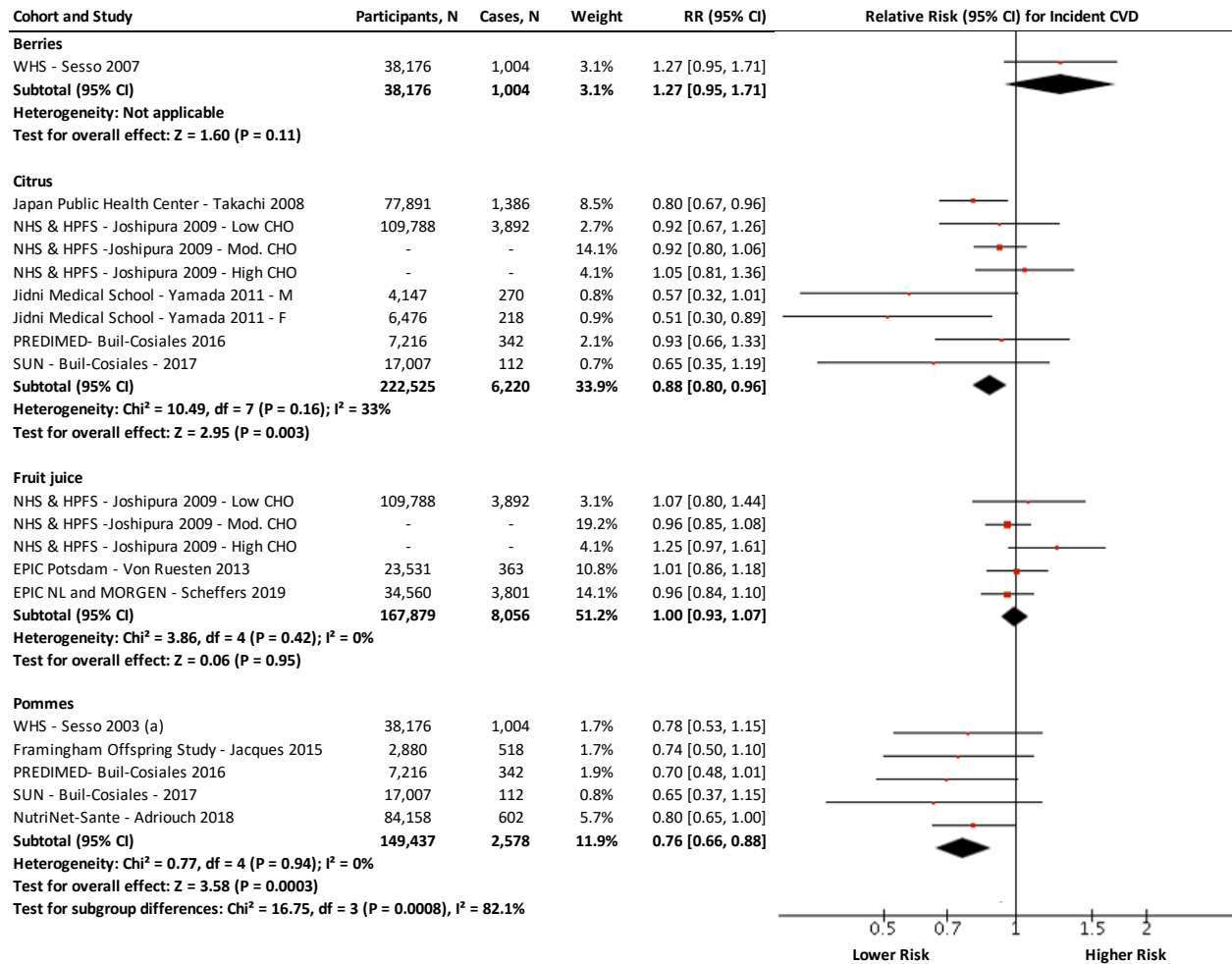


Figure S11. Relation between tomato intake and cardiovascular disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

A. Fixed Effects



B. Random Effects

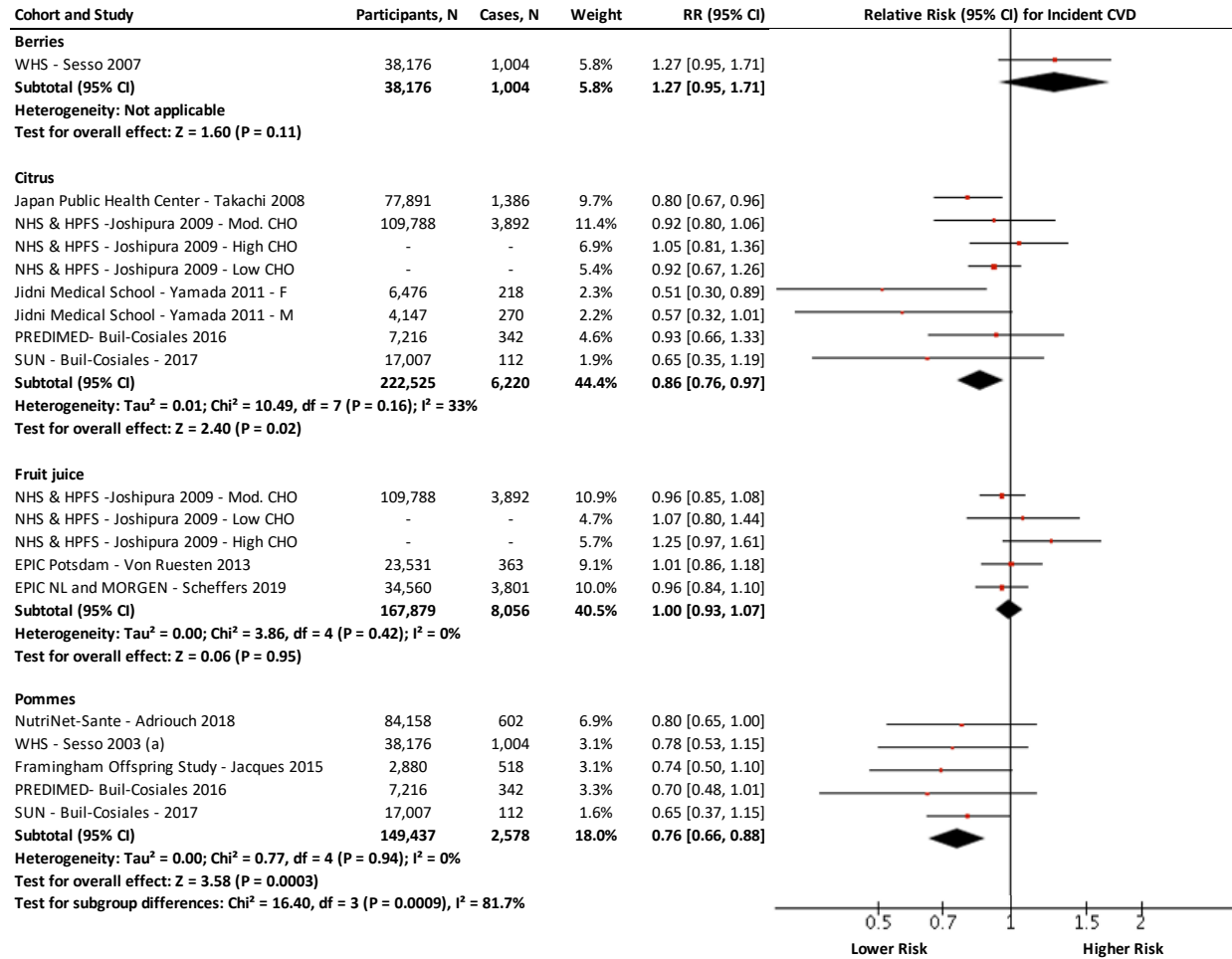
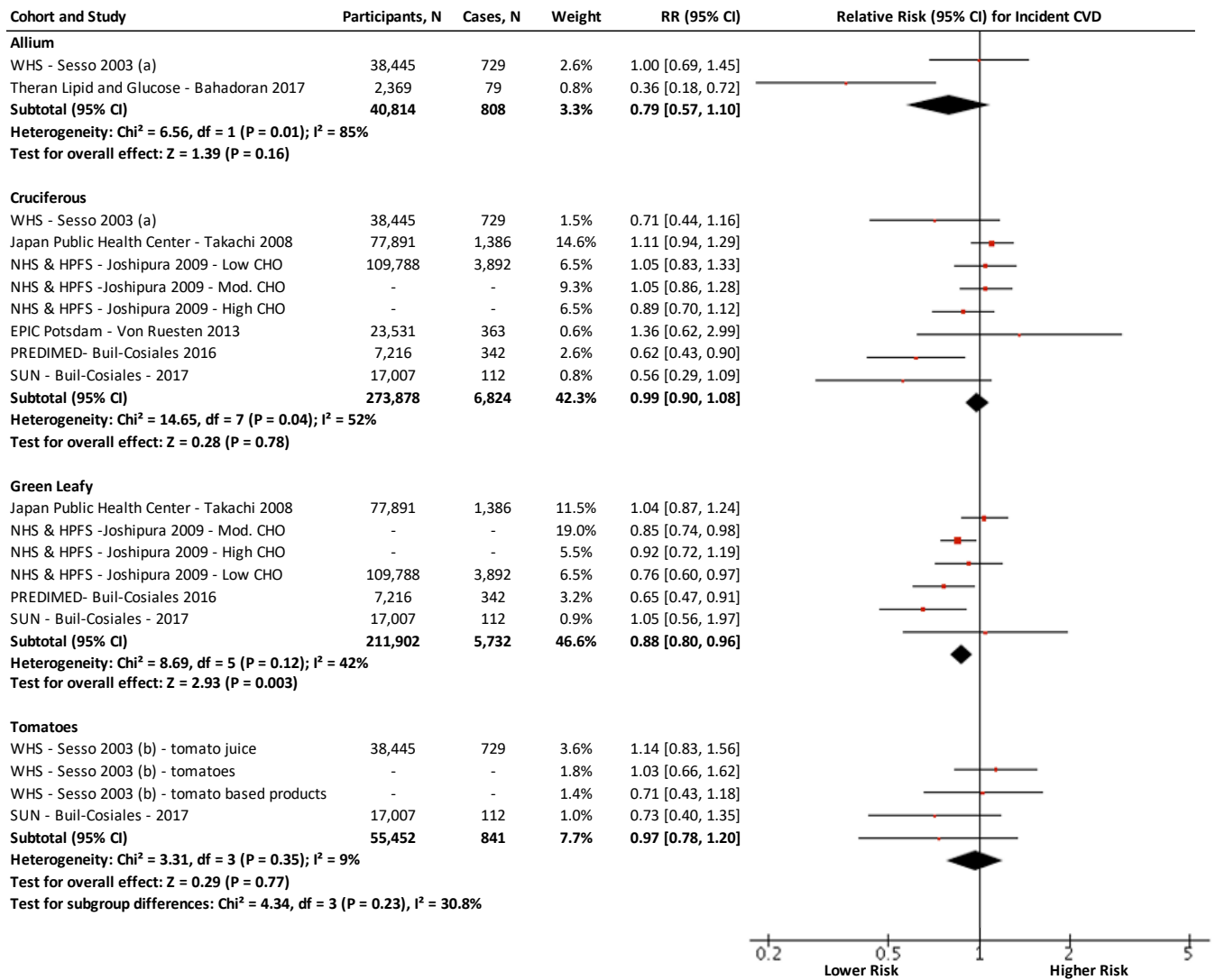


Figure S12. Relation between sources of fruit and CVD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

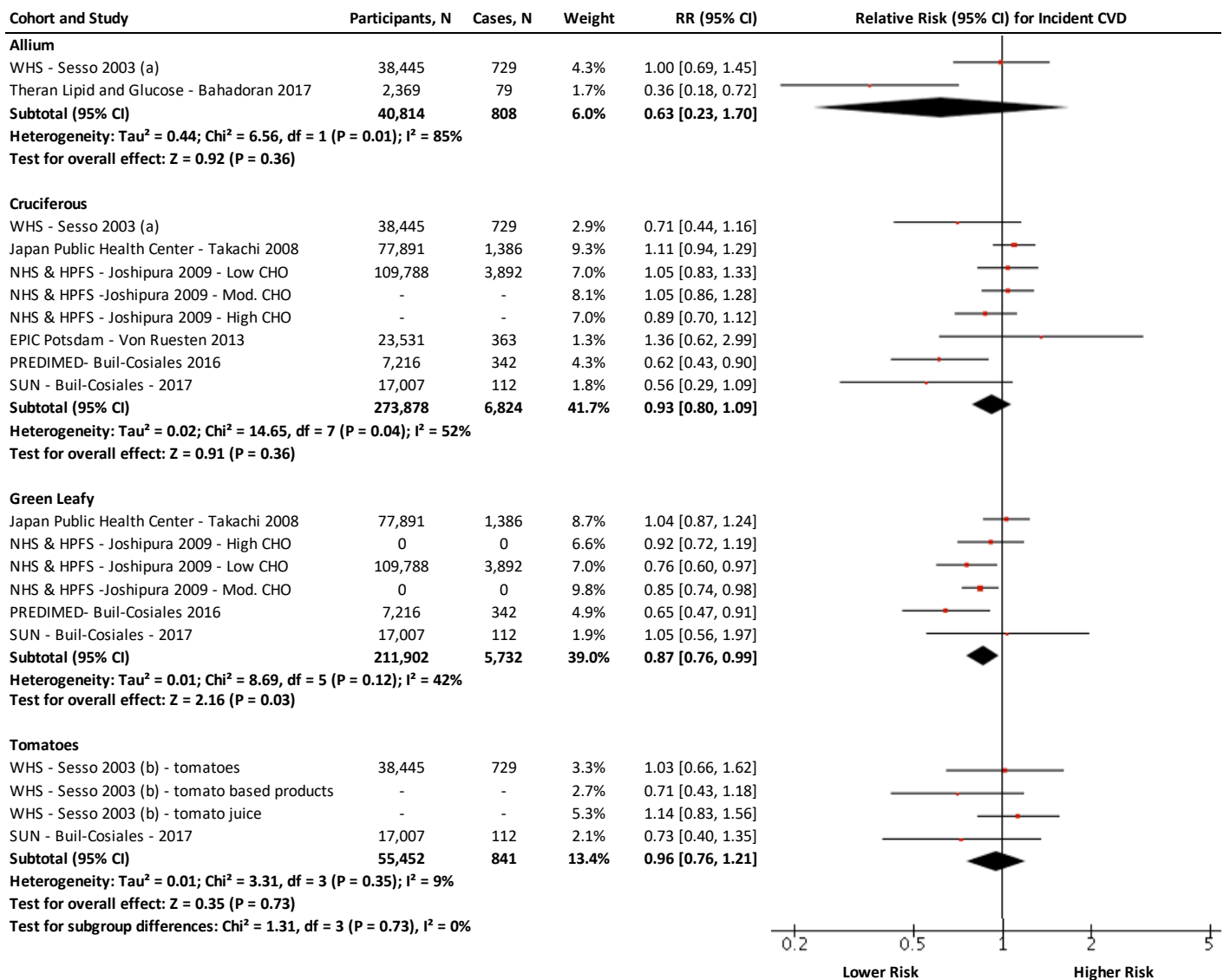
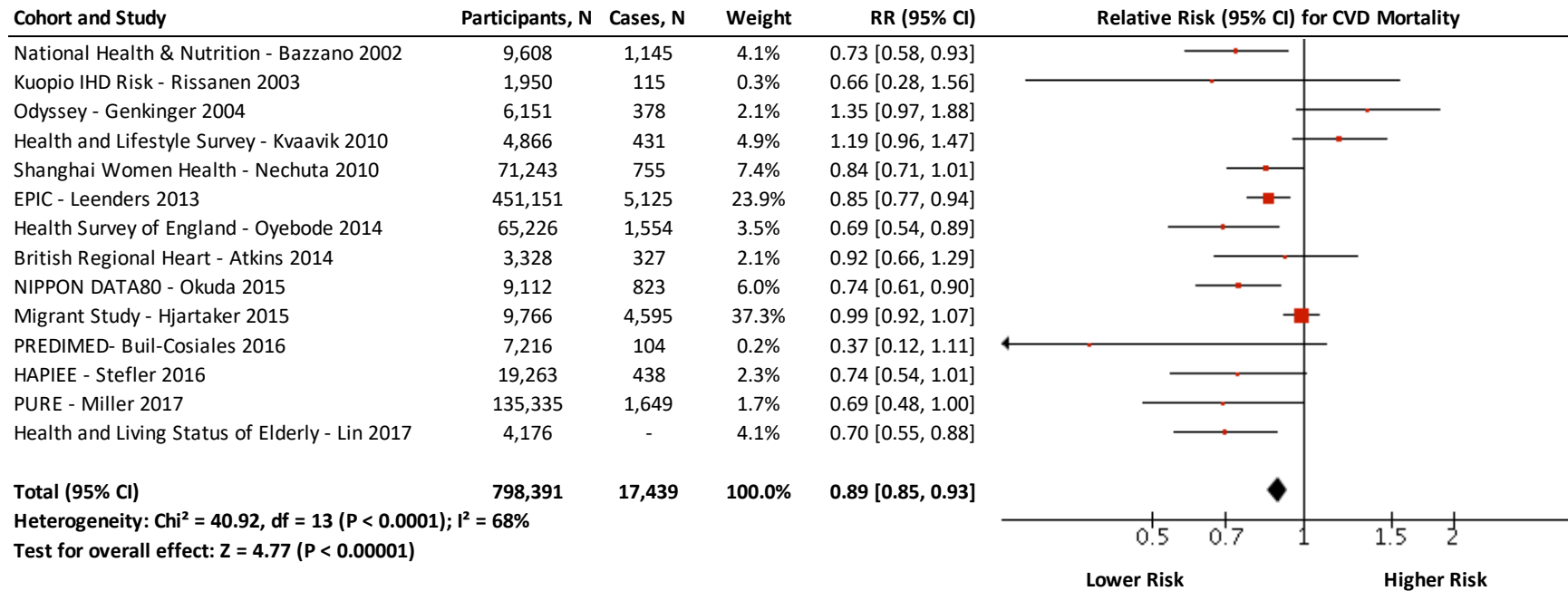


Figure S13. Relation between sources of vegetables and CVD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

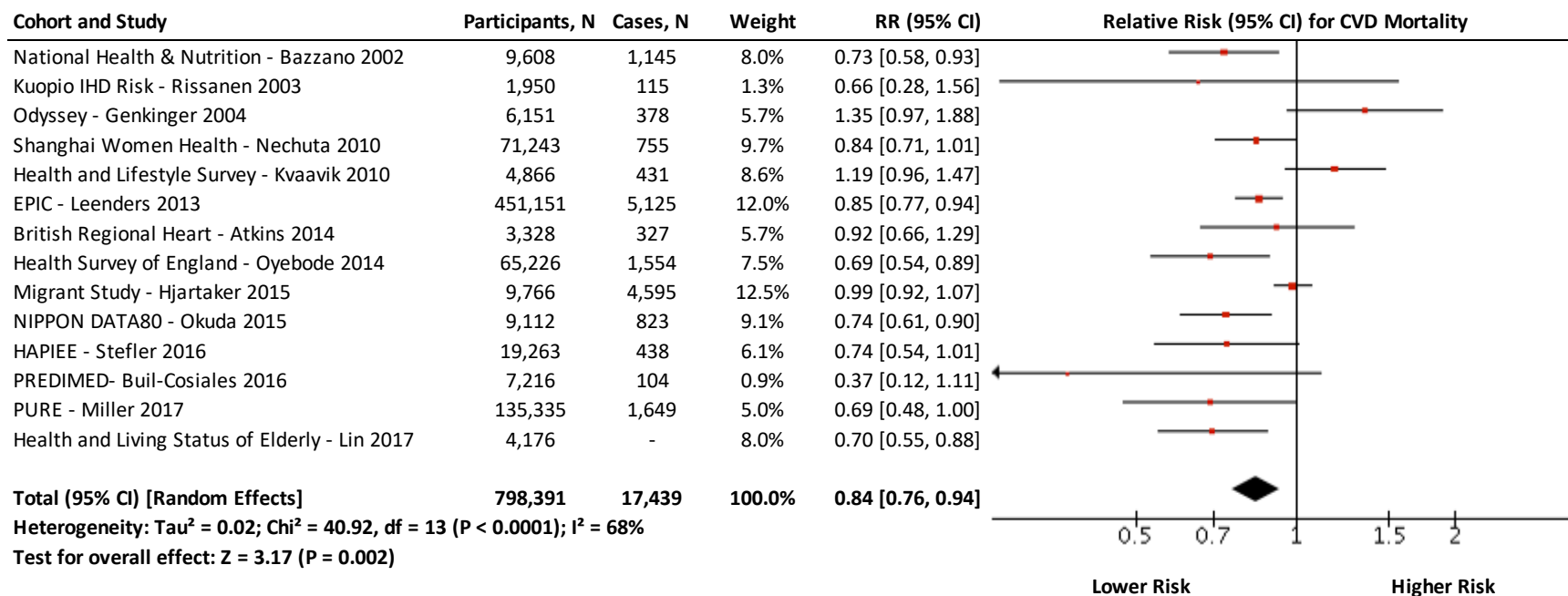
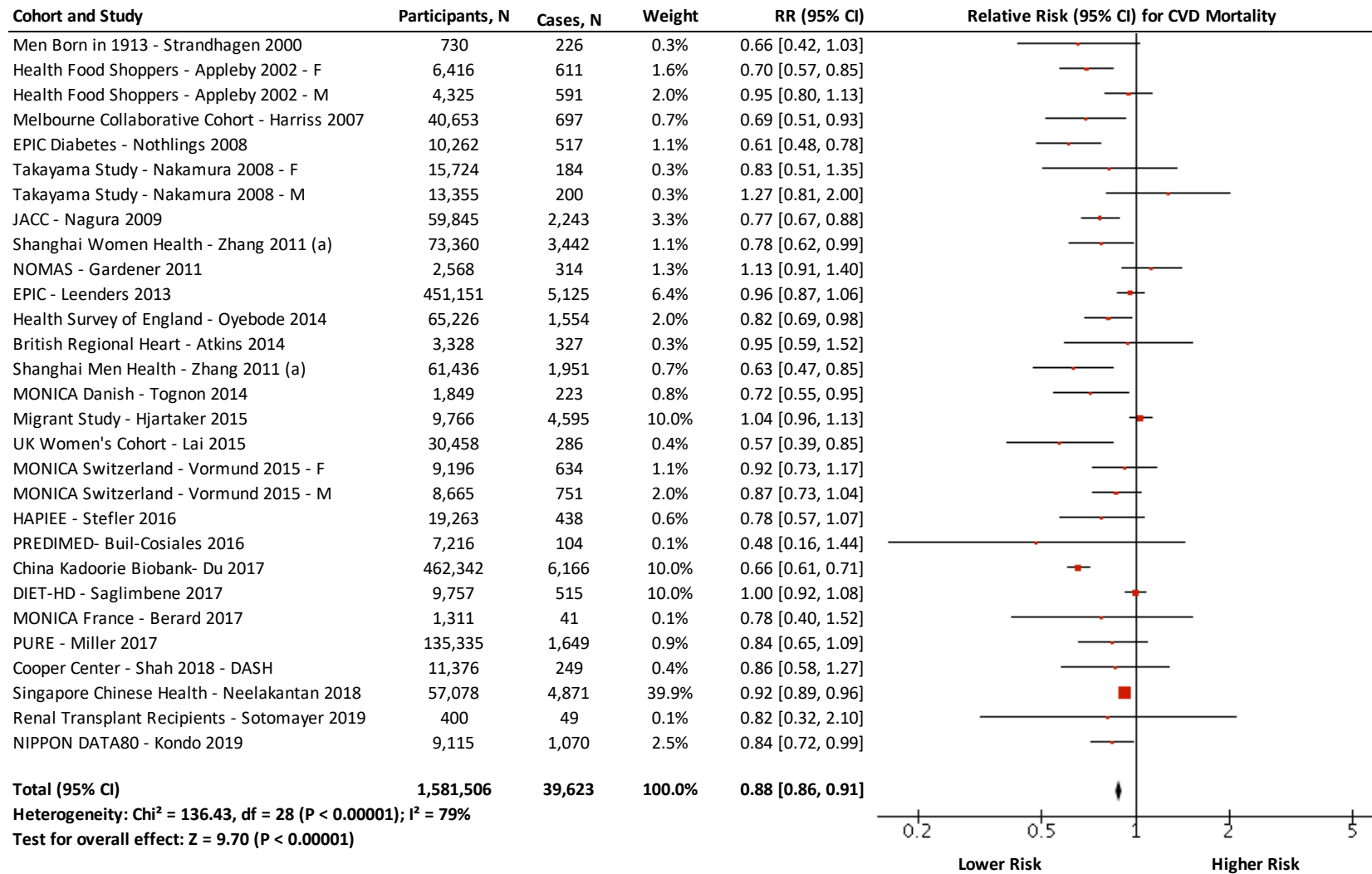


Figure S14. Relation between total fruit and vegetable intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effect

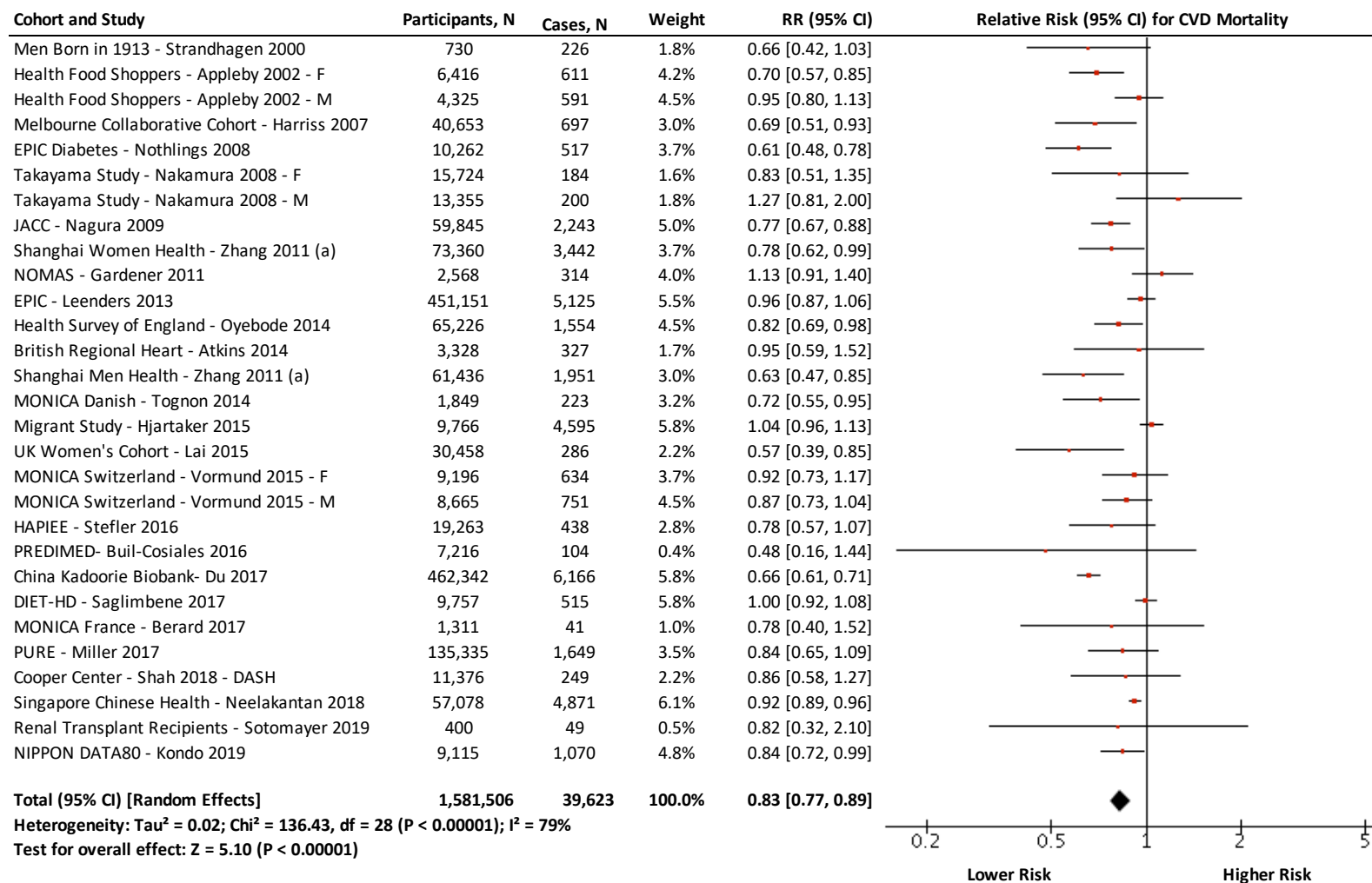
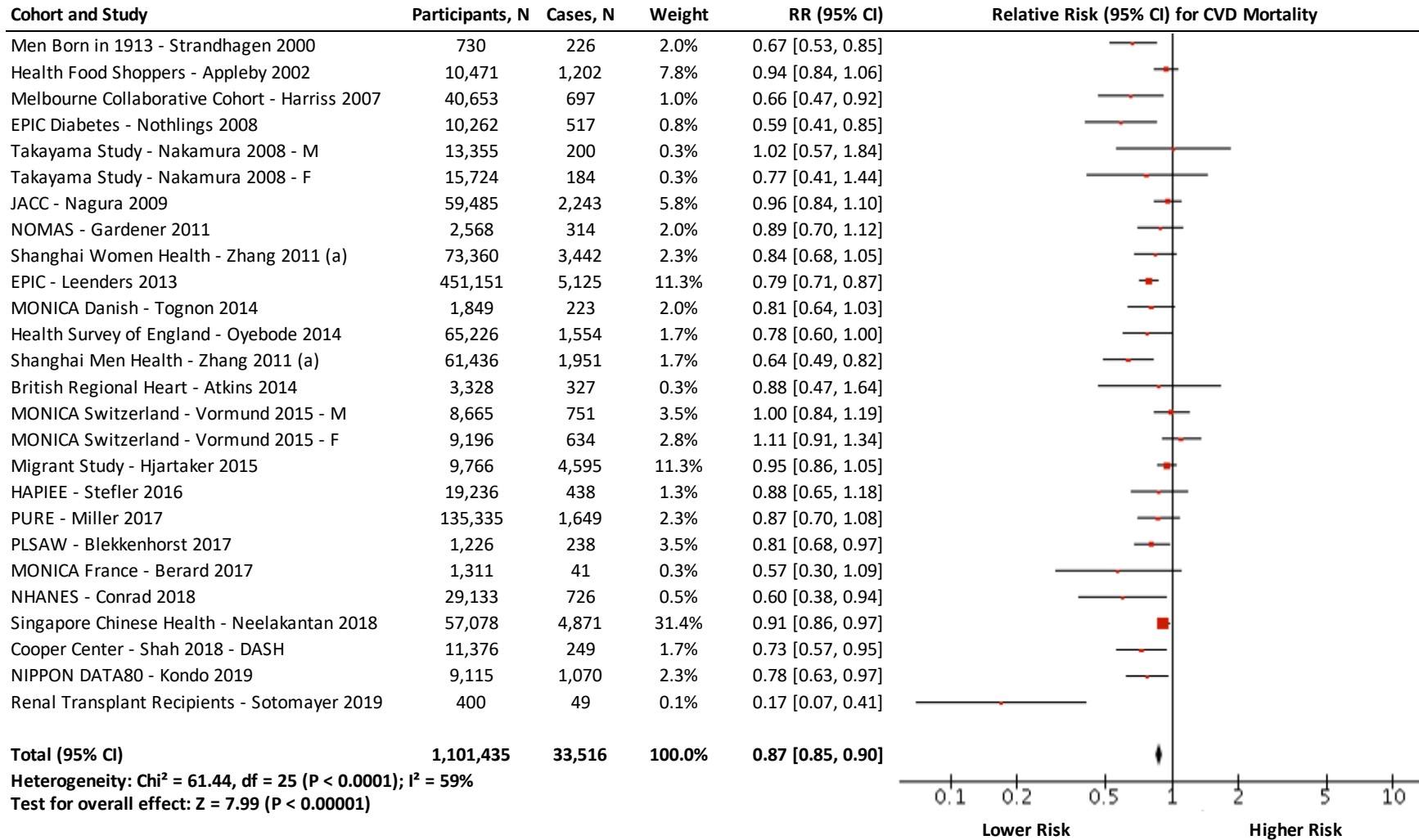


Figure S15. Relation between fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

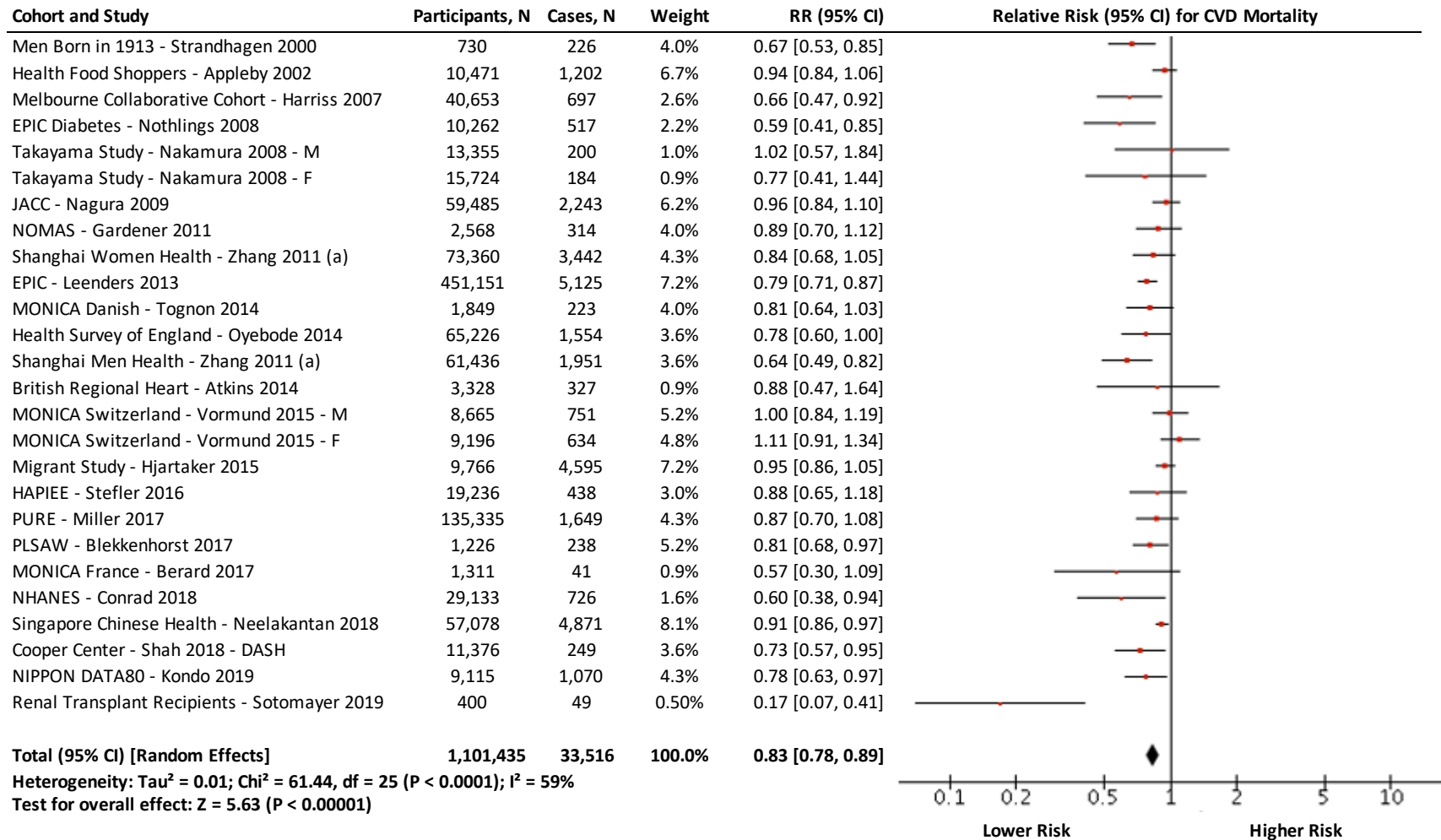
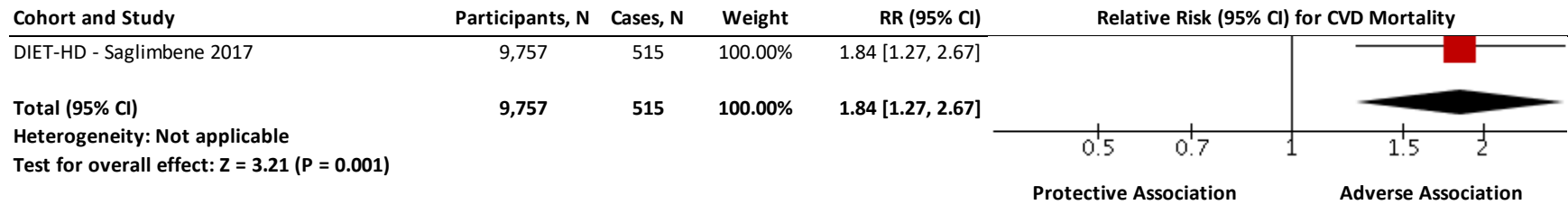


Figure S16. Relation between vegetable intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

APRICOTS AND CARDIOVASCULAR DISEASE MORTALITY



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

BANANAS AND CARDIOVASCULAR DISEASE MORTALITY

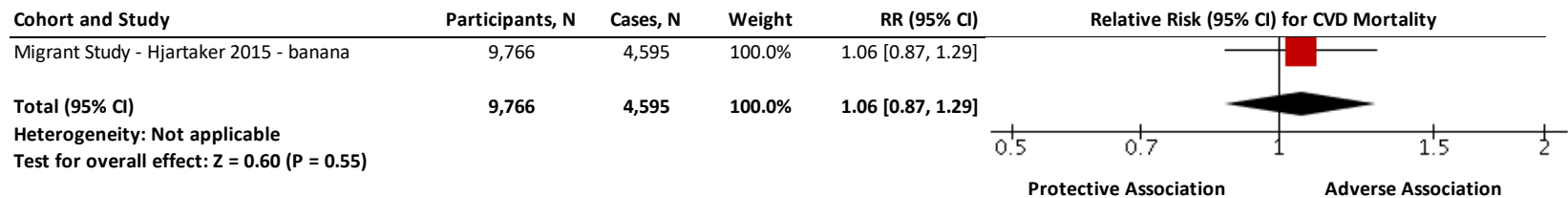


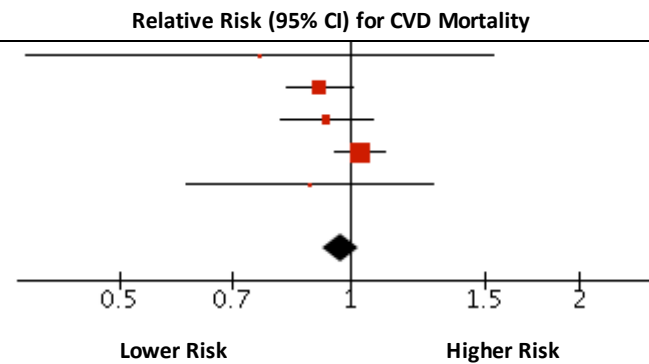
Figure S18. Relation between intake of bananas and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed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

BERRIES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 4.68$, $\text{df} = 4$ ($P = 0.32$); $I^2 = 15\%$
 Test for overall effect: $Z = 1.04$ ($P = 0.30$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 4.68$, $\text{df} = 4$ ($P = 0.32$); $I^2 = 15\%$
 Test for overall effect: $Z = 1.06$ ($P = 0.29$)

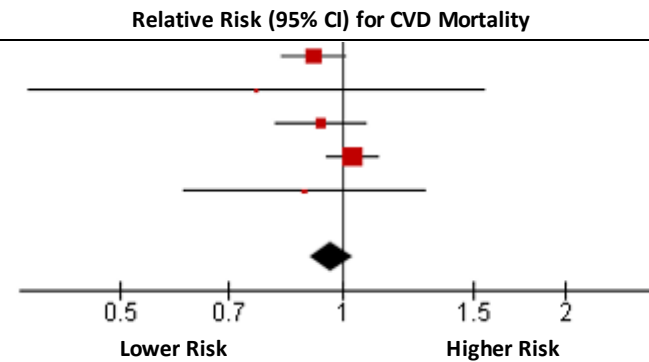
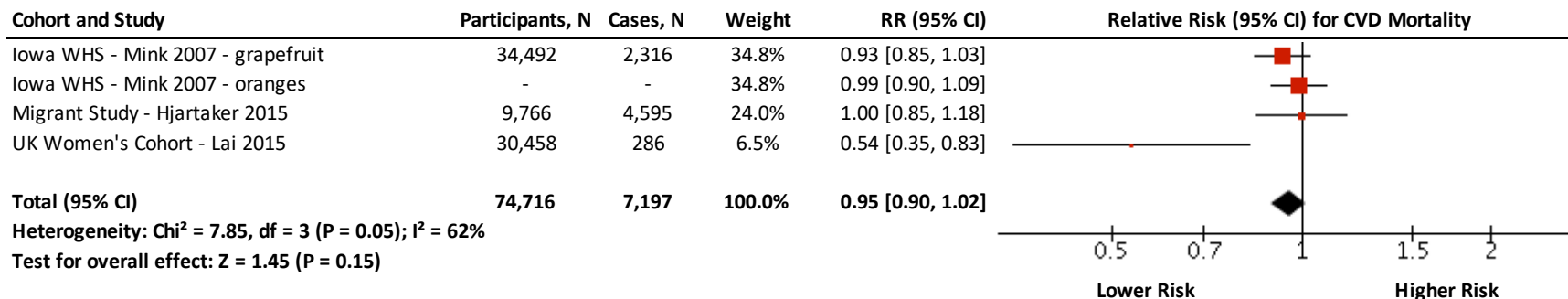


Figure S19. Relation between intake of berries and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CITRUS FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

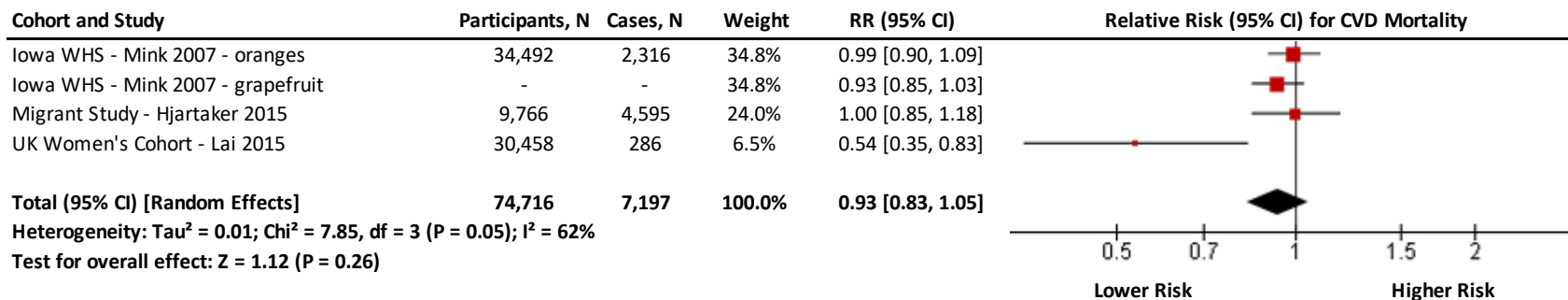
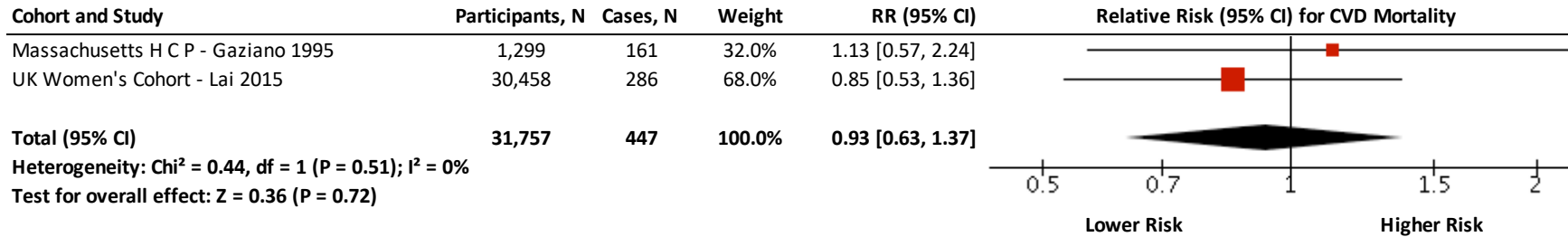


Figure S20. Relation between citrus fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B)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

DRIED FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

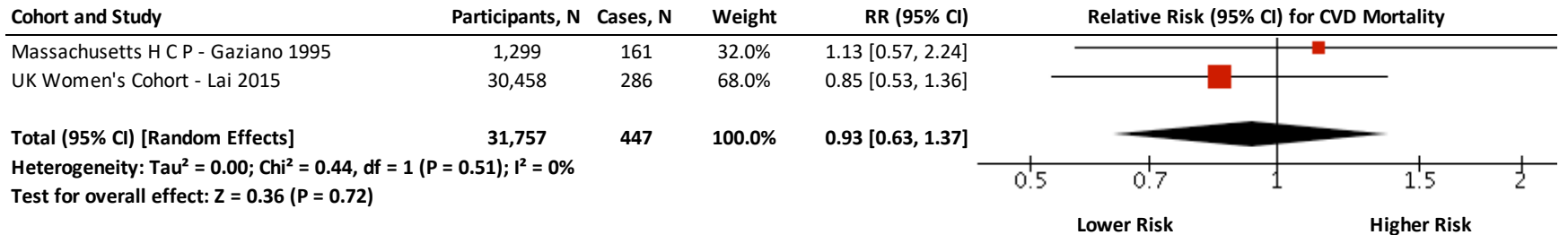


Figure S21. Relation between dried fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

FRUIT JUICE AND CARDIOVASCULAR DISEASE MORTALITY

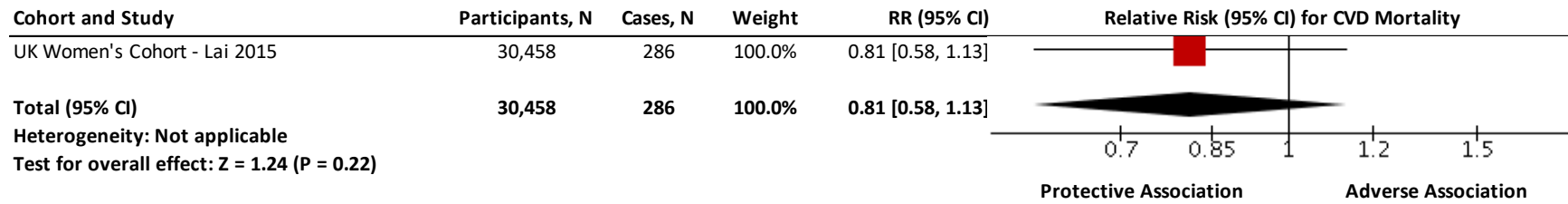
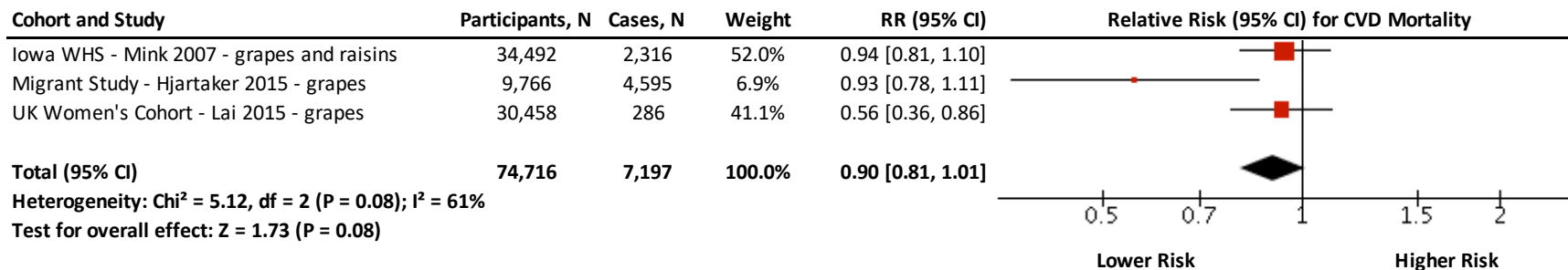


Figure S22. Relation between fruit juice intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed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

GRAPES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

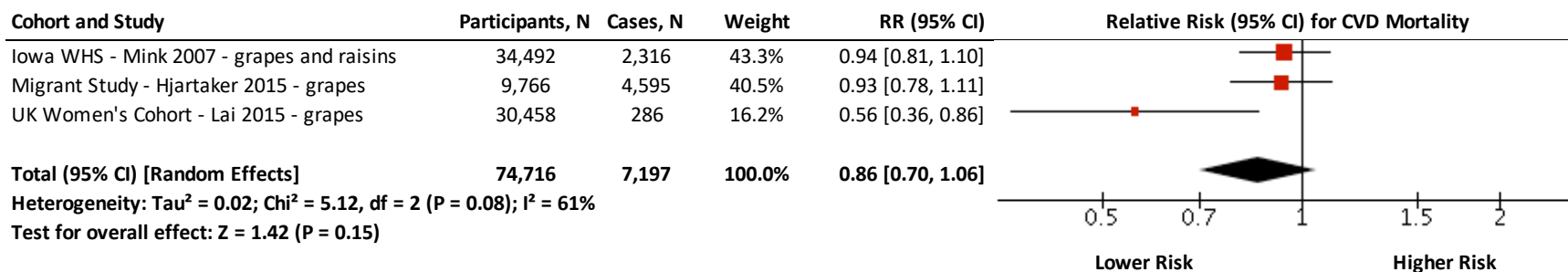
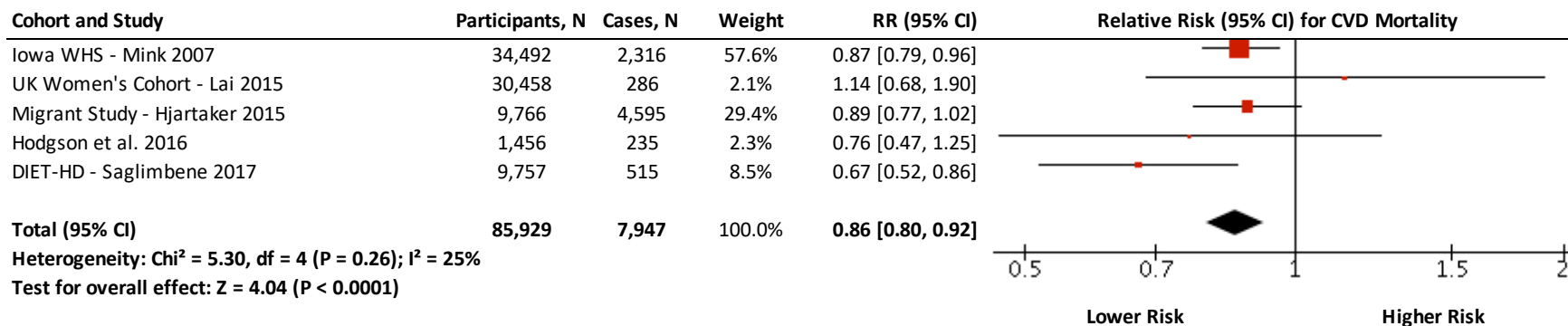


Figure S23. Relation between intake of grapes and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

POMMES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

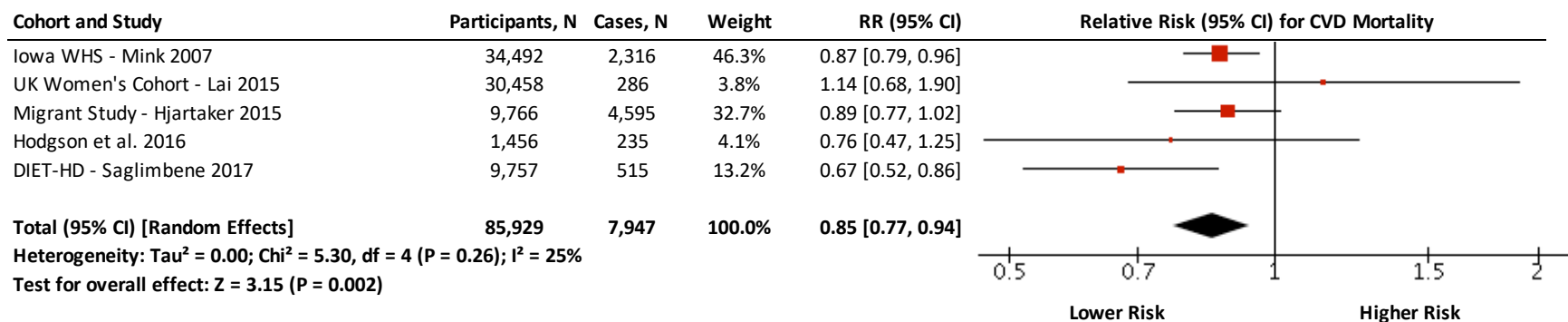


Figure S24. Relation between pomes fruit intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

ALLIUM VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

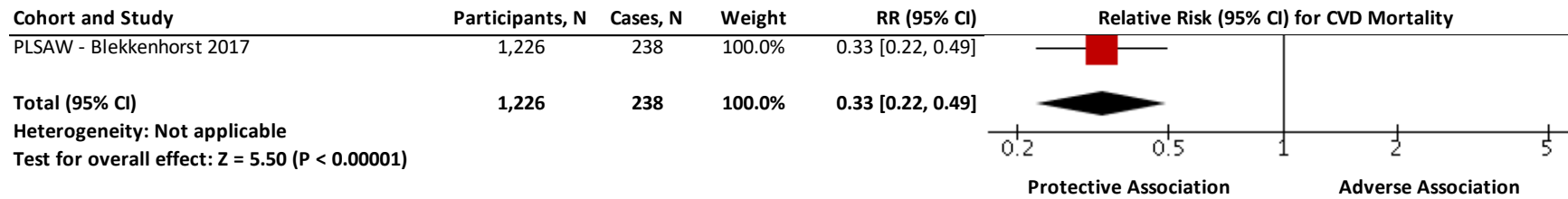


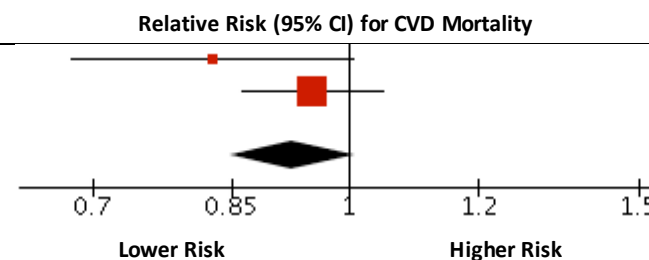
Figure S25. Relation between intake allium vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

CARROTS AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 1.57$, $\text{df} = 1$ ($P = 0.21$); $I^2 = 36\%$
 Test for overall effect: $Z = 1.74$ ($P = 0.08$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 1.57$, $\text{df} = 1$ ($P = 0.21$); $I^2 = 36\%$
 Test for overall effect: $Z = 1.44$ ($P = 0.15$)

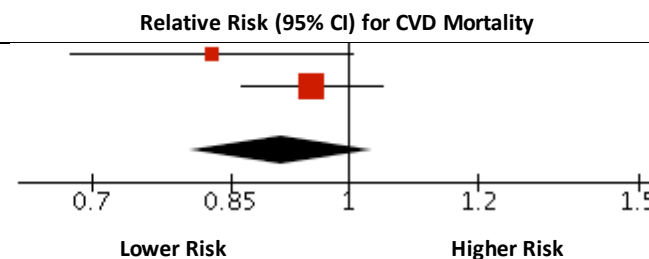


Figure S26. Relation between carrots intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CELERY AND CARDIOVASCULAR DISEASE MORTALITY

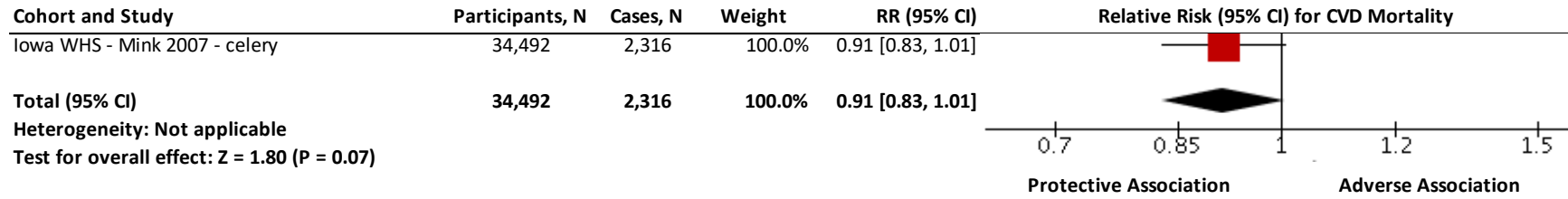


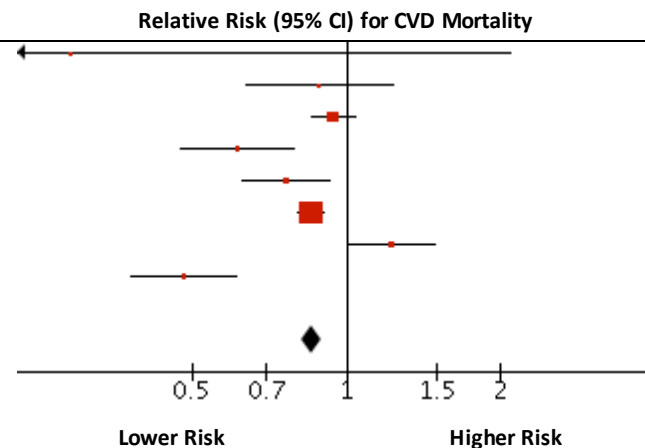
Figure S27. Relation between celery intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CRUCIFEROUS VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 48.35$, $\text{df} = 7$ ($P < 0.00001$); $I^2 = 86\%$
 Test for overall effect: $Z = 6.79$ ($P < 0.00001$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.04$; $\text{Chi}^2 = 48.35$, $\text{df} = 7$ ($P < 0.00001$); $I^2 = 86\%$
 Test for overall effect: $Z = 2.75$ ($P = 0.006$)

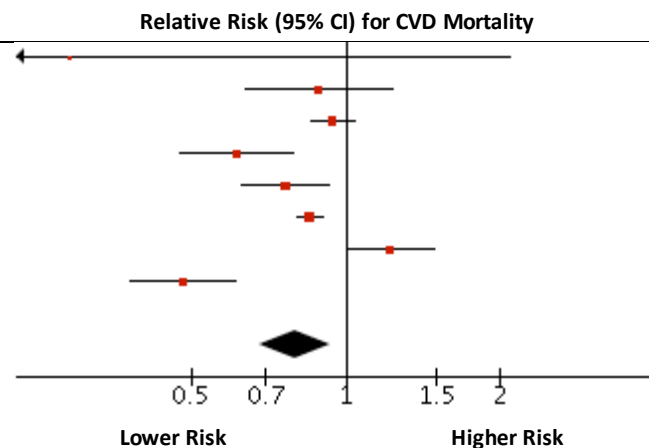
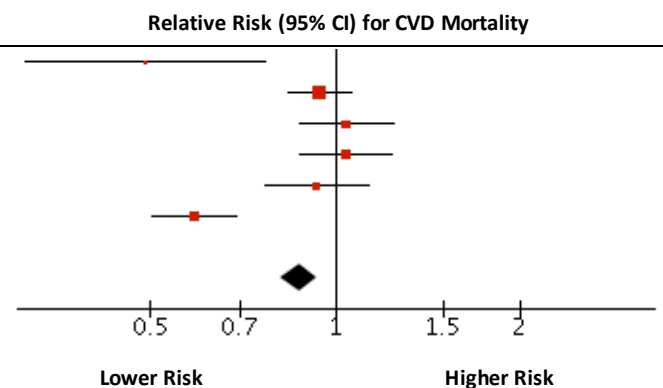


Figure S28. Relation between intake of cruciferous vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

GREEN LEAFY VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	2.3%	0.49 [0.31, 0.77]
Health Food Shoppers - Appleby 2002	10,741	282	34.4%	0.94 [0.84, 1.06]
MONICA Switzerland - Vormund 2015 - M	8,665	751	15.3%	1.04 [0.87, 1.24]
Migrant Study - Hjartaker 2015	9,766	4,595	16.3%	1.04 [0.88, 1.23]
MONICA Switzerland - Vormund 2015 - F	9,196	634	12.4%	0.93 [0.77, 1.13]
PLSAW - Blekkenhorst 2017	1,226	238	19.3%	0.59 [0.50, 0.69]
Total (95% CI)	40,893	6,661	100.0%	0.87 [0.81, 0.94]
Heterogeneity: Chi² = 40.44, df = 5 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 3.86 (P = 0.0001)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Massachusetts Health Care Panel - Gaziano 1995	1,299	161	10.4%	0.49 [0.31, 0.77]
Health Food Shoppers - Appleby 2002	10,741	282	19.0%	0.94 [0.84, 1.06]
Migrant Study - Hjartaker 2015	9,766	4,595	17.8%	1.04 [0.88, 1.23]
MONICA Switzerland - Vormund 2015 - M	8,665	751	17.6%	1.04 [0.87, 1.24]
MONICA Switzerland - Vormund 2015 - F	9,196	634	17.1%	0.93 [0.77, 1.13]
PLSAW - Blekkenhorst 2017	1,226	238	18.1%	0.59 [0.50, 0.69]
Total (95% CI) [Random Effects]	40,893	6,661	100.0%	0.84 [0.68, 1.03]
Heterogeneity: Tau² = 0.06; Chi² = 40.44, df = 5 (P < 0.00001); I² = 88%				
Test for overall effect: Z = 1.68 (P = 0.09)				

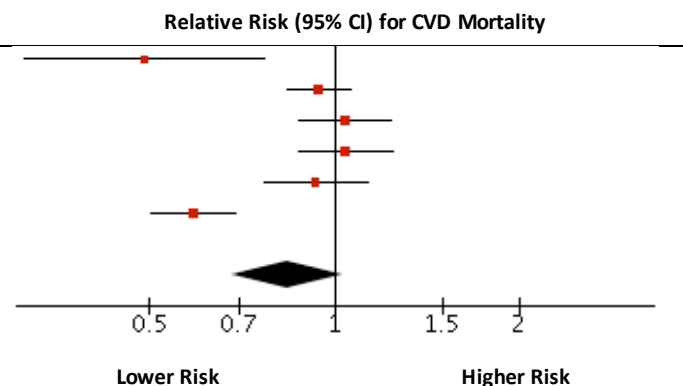
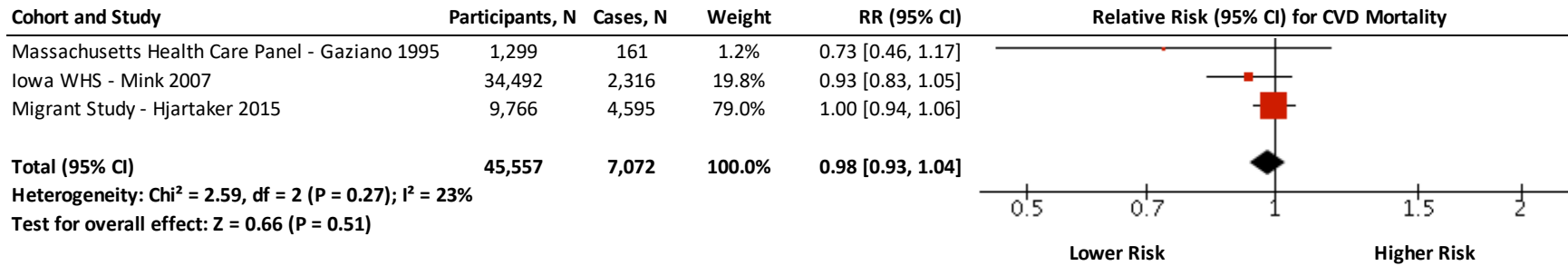


Figure S29. Relation between intake of green leafy vegetables and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

TOMATOES AND CARDIOVASCULAR DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

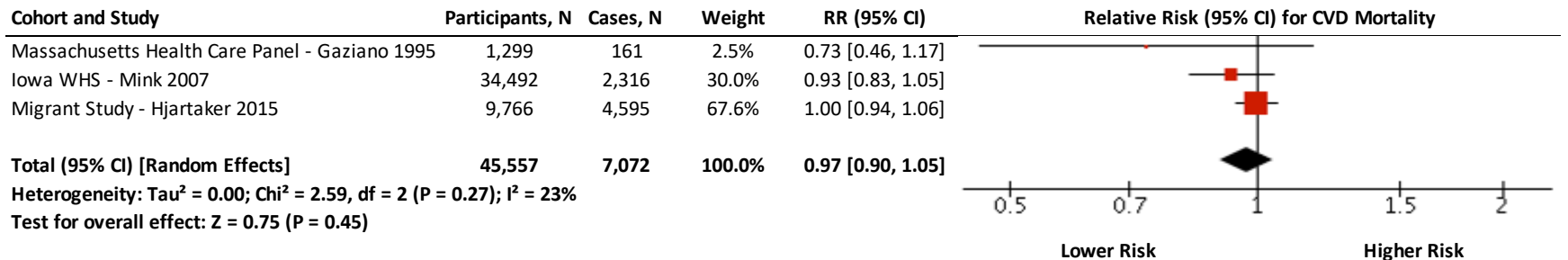
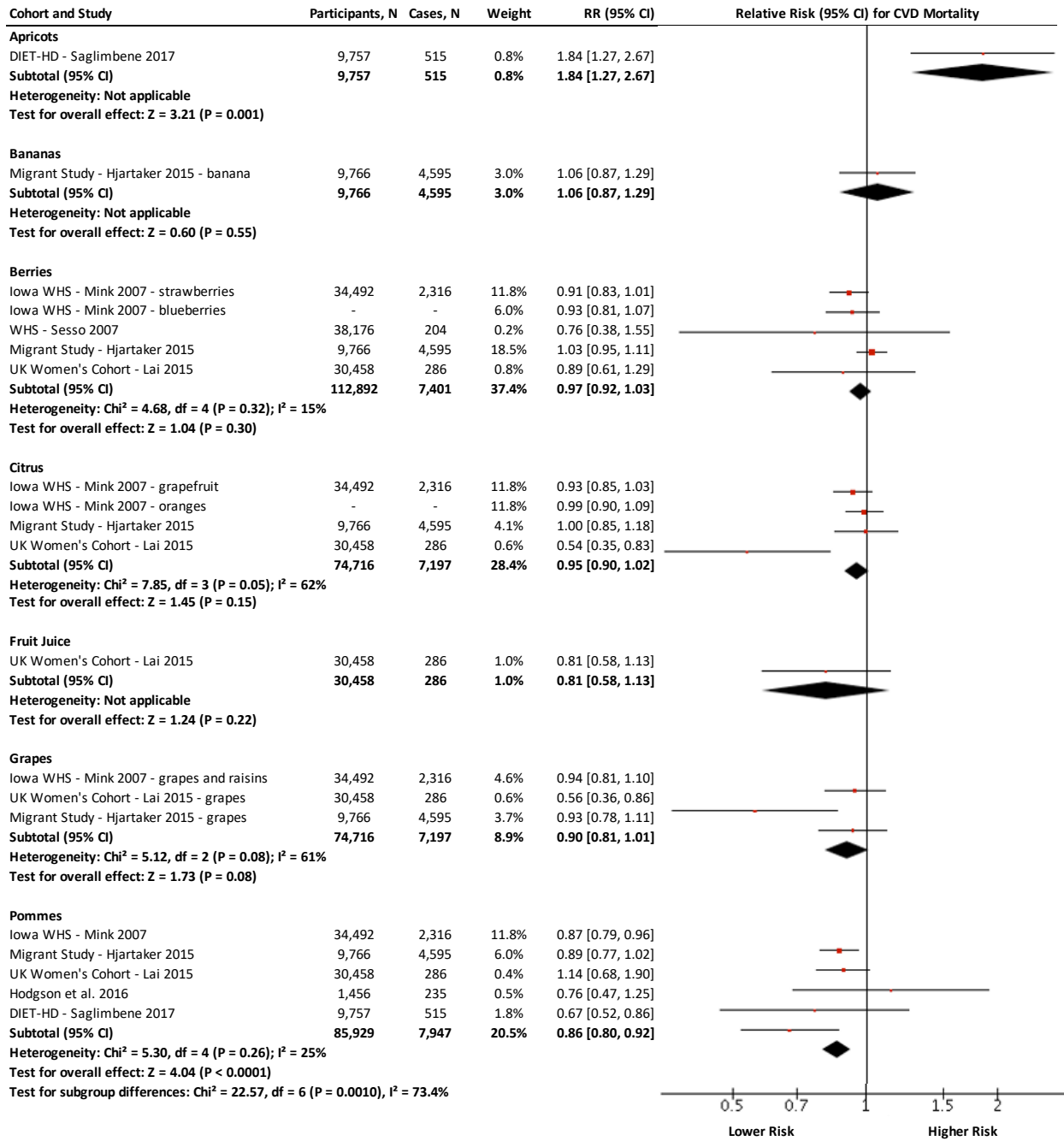


Figure S30. Relation between tomato intake and cardiovascular disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

A. Fixed Effects



B. Random Effects

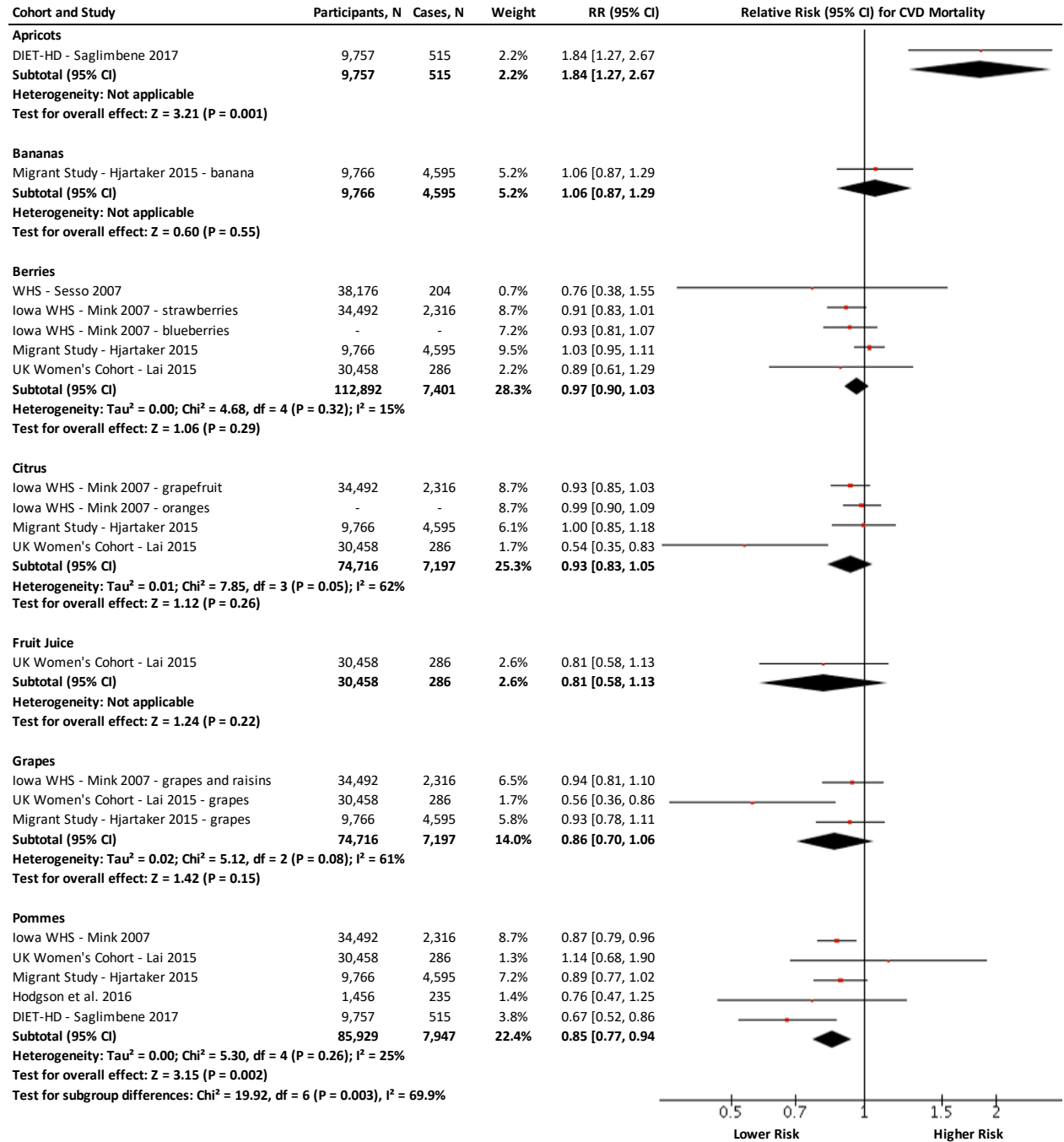
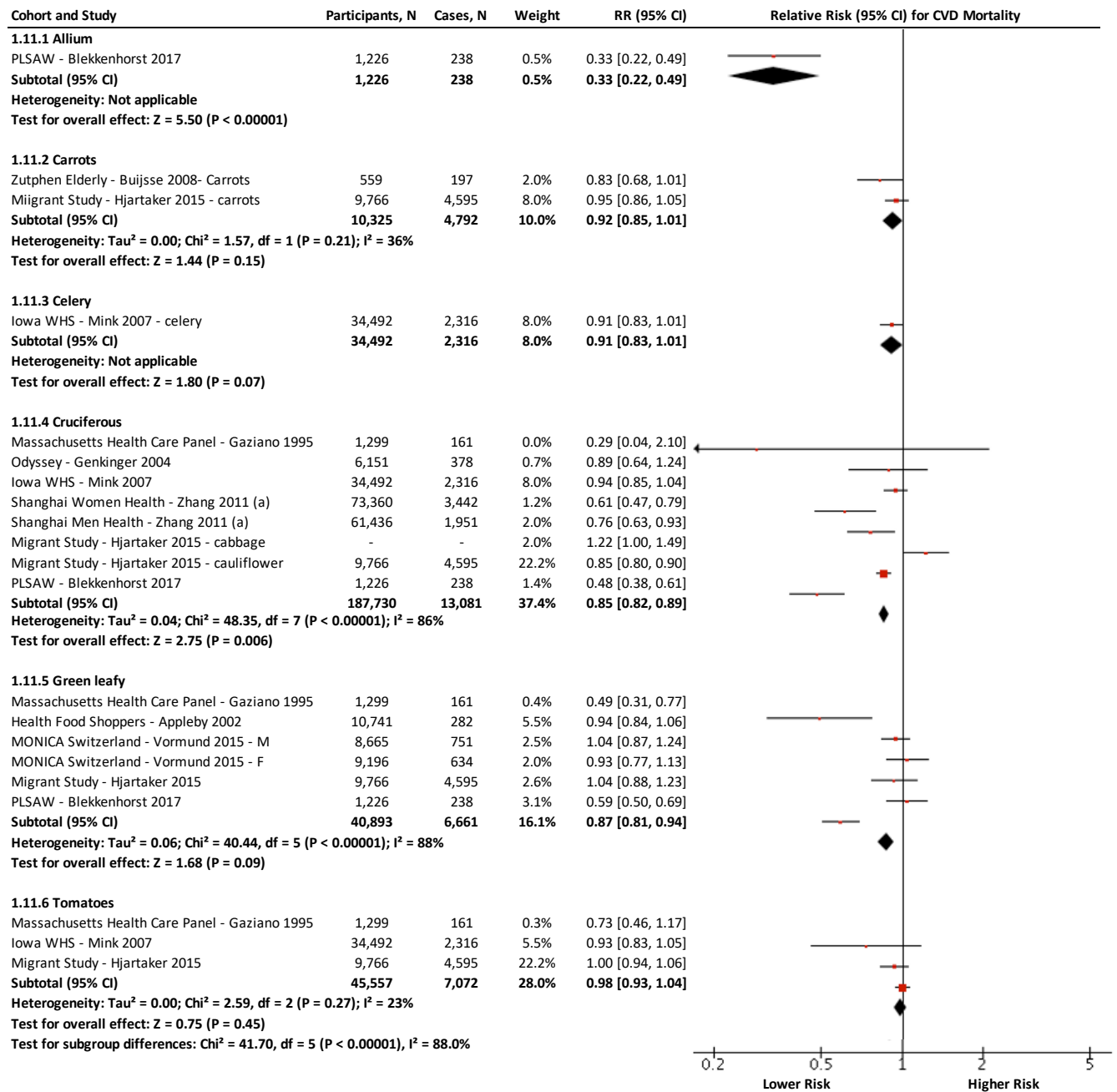


Figure S31. Relation between sources of fruit and CVD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

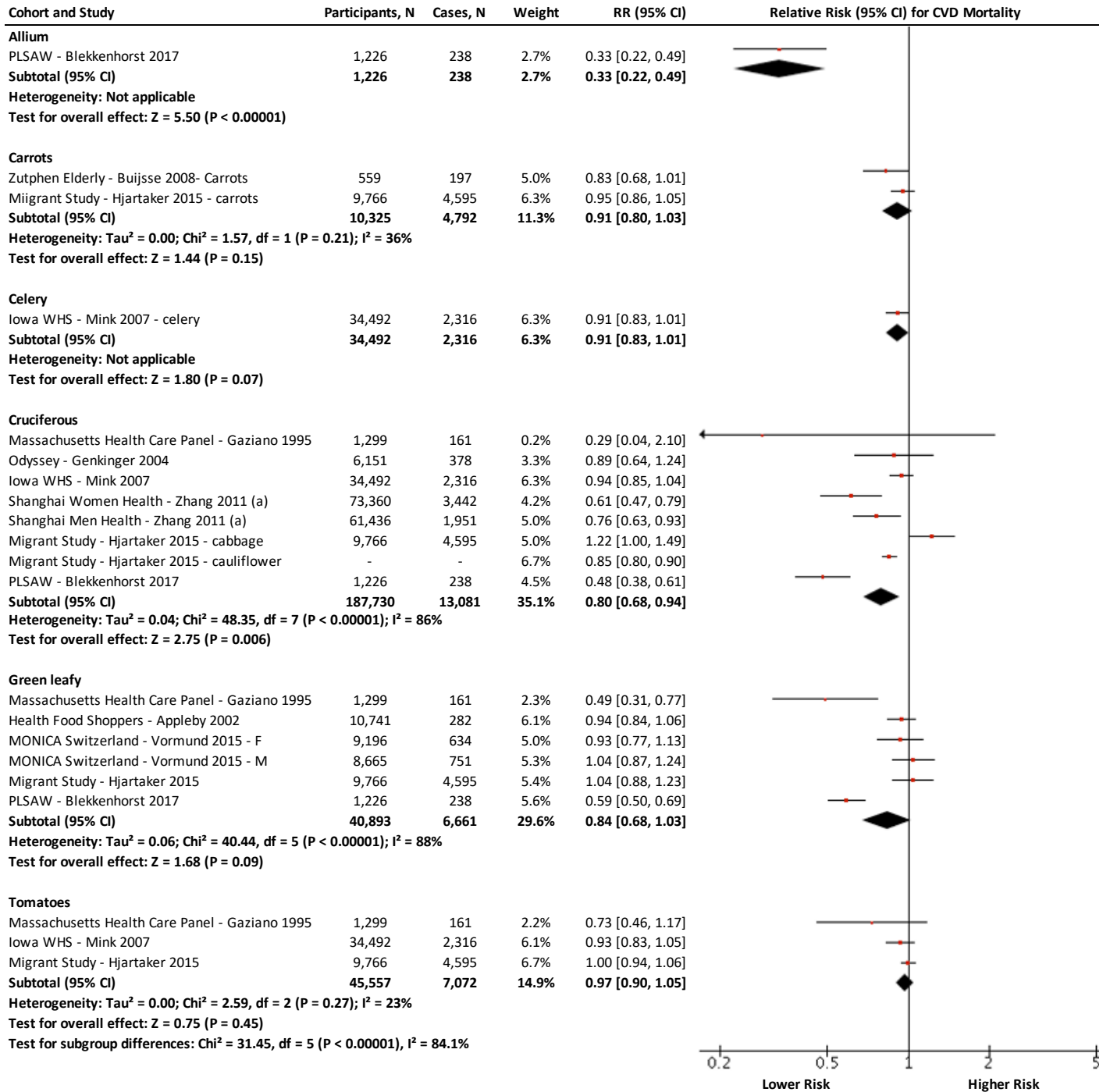


Figure S32. Relation between sources of vegetables and CVD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

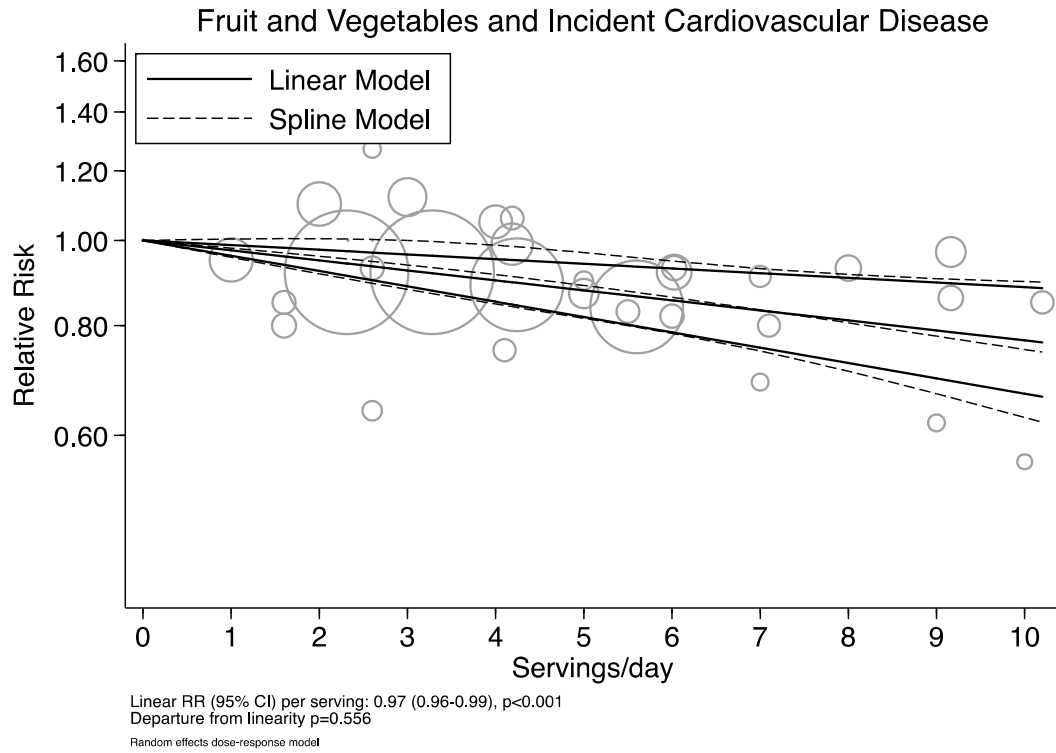


Figure S33. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker method to estimate the covariances of multivariable-adjusted relative risk. Cubic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

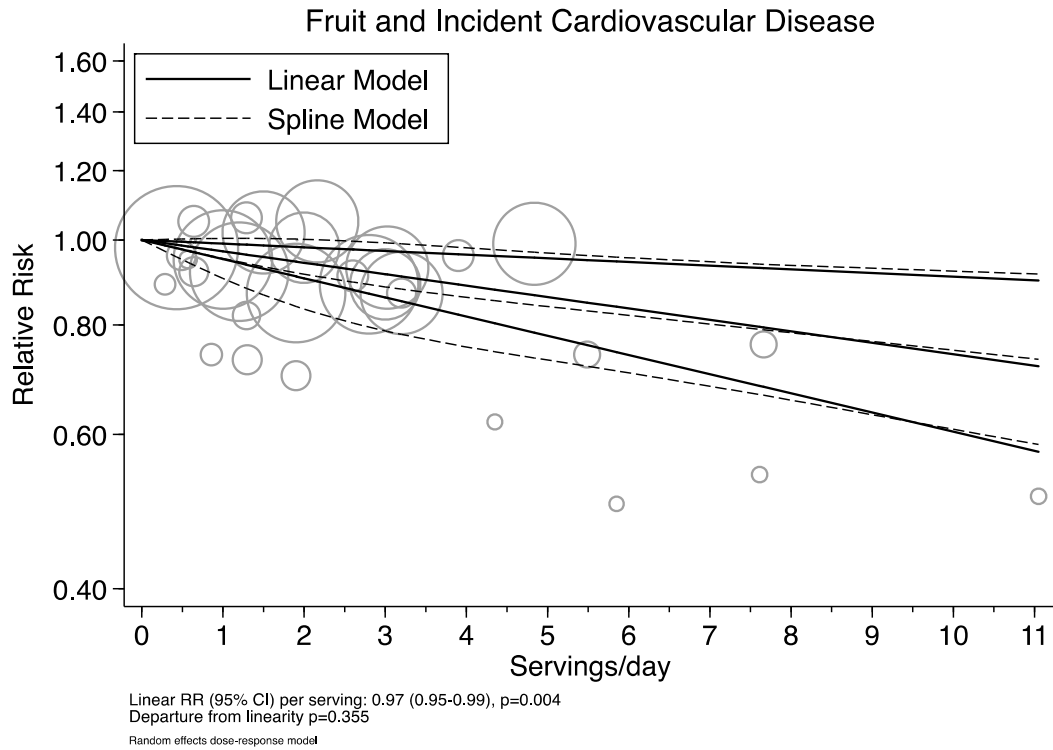


Figure S34. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

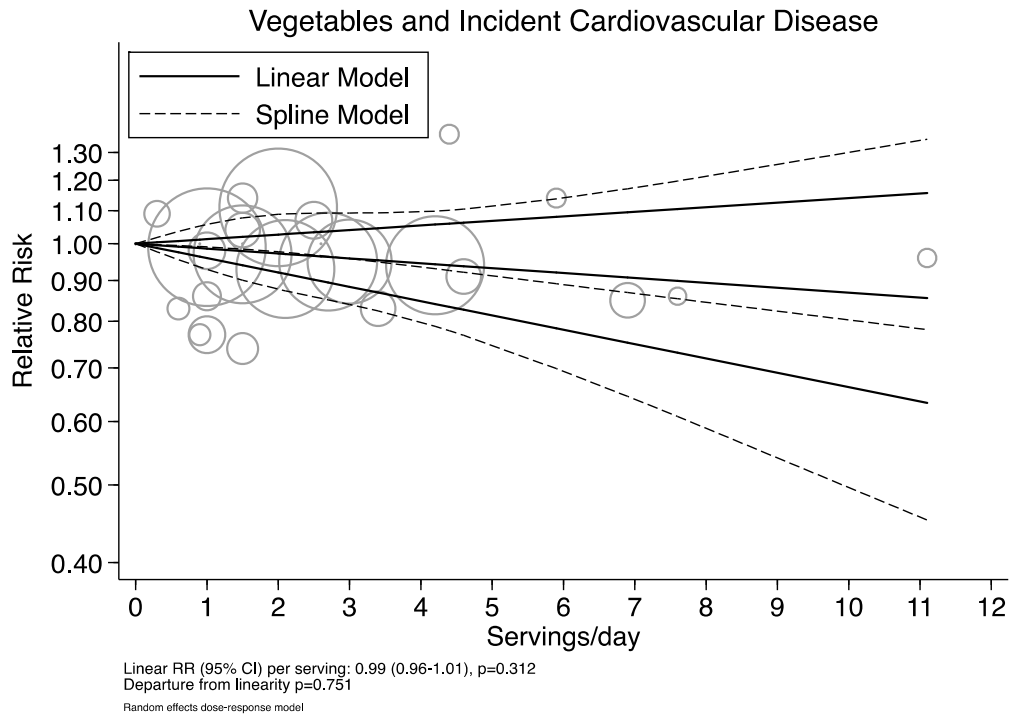


Figure S35. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

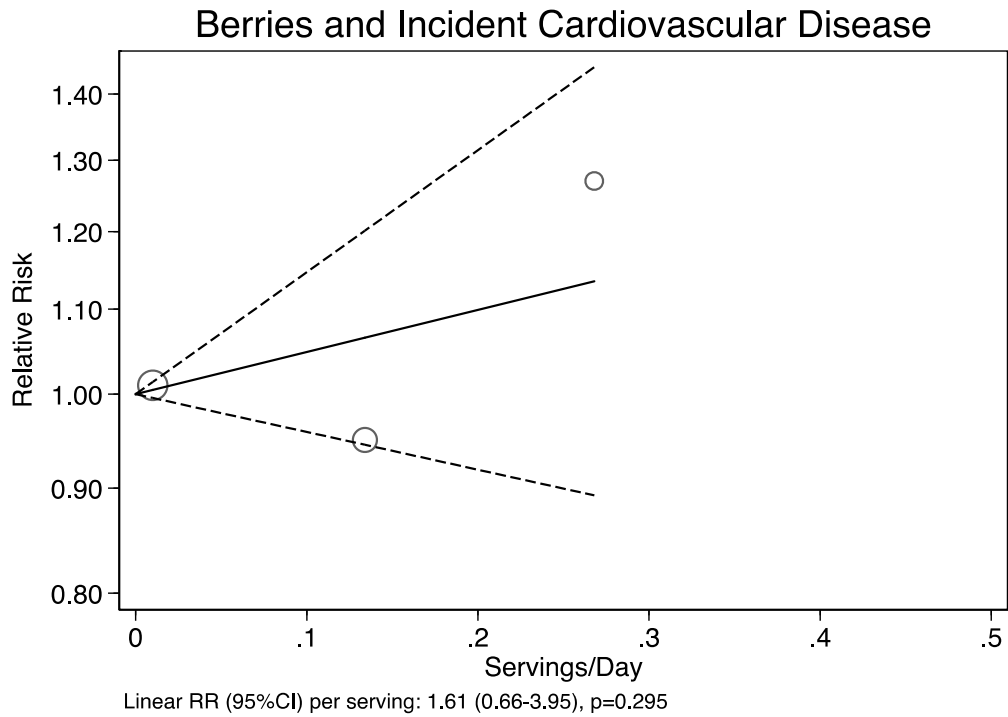


Figure S36. Linear dose-response relation between increasing berries intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

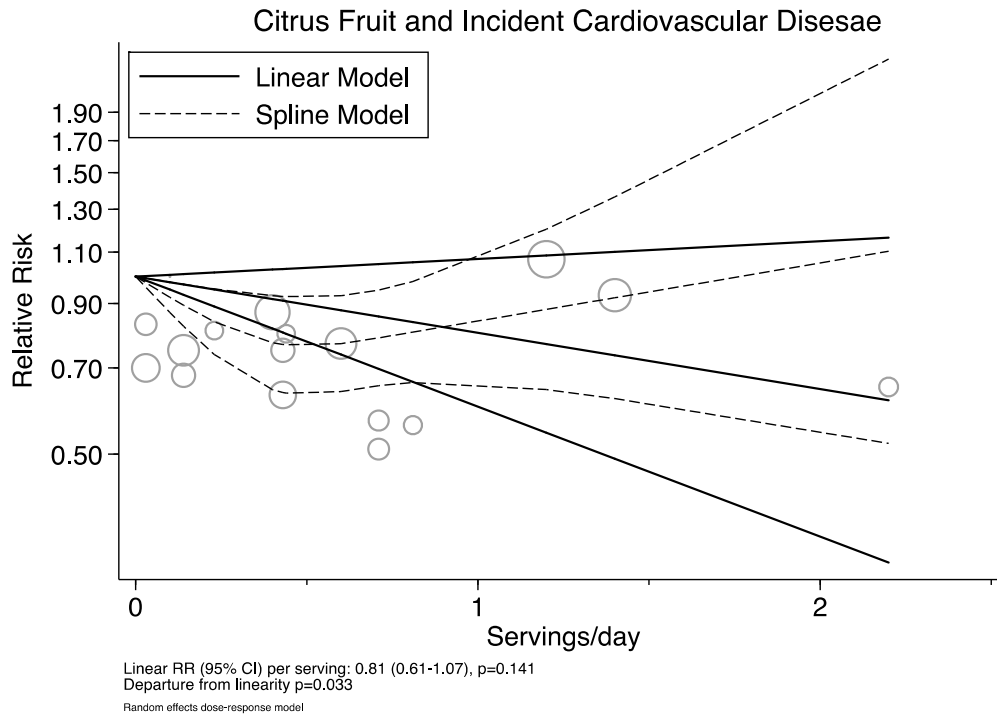


Figure S37. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

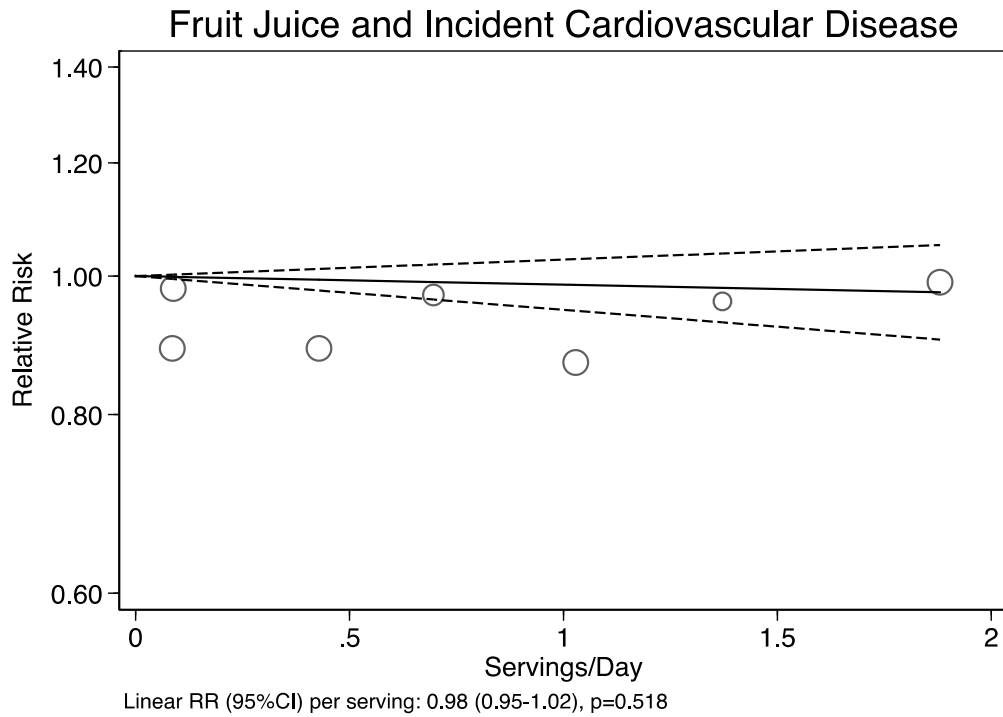


Figure S38. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

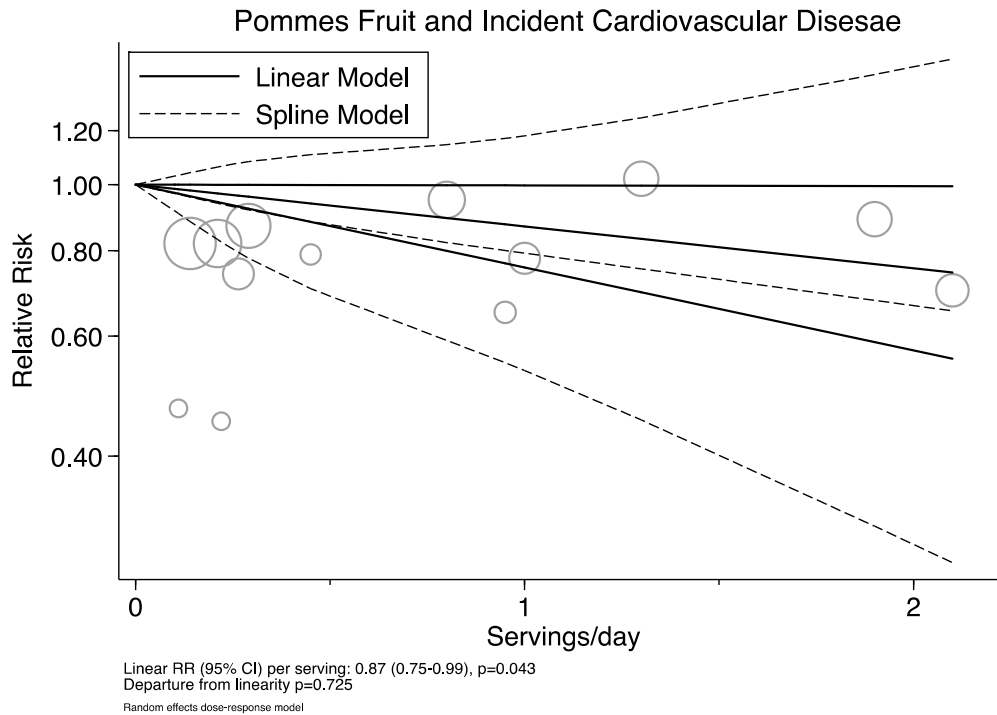


Figure S39. Linear dose-response relation between increasing pommes intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

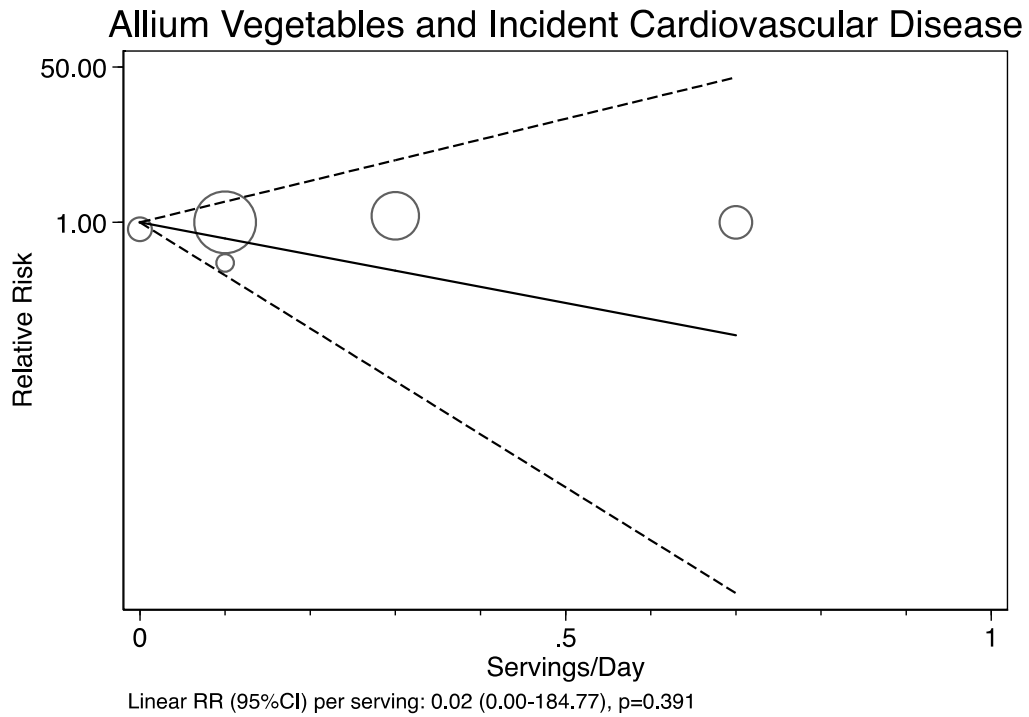


Figure S40. Linear dose-response relation between increasing intake of allium vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

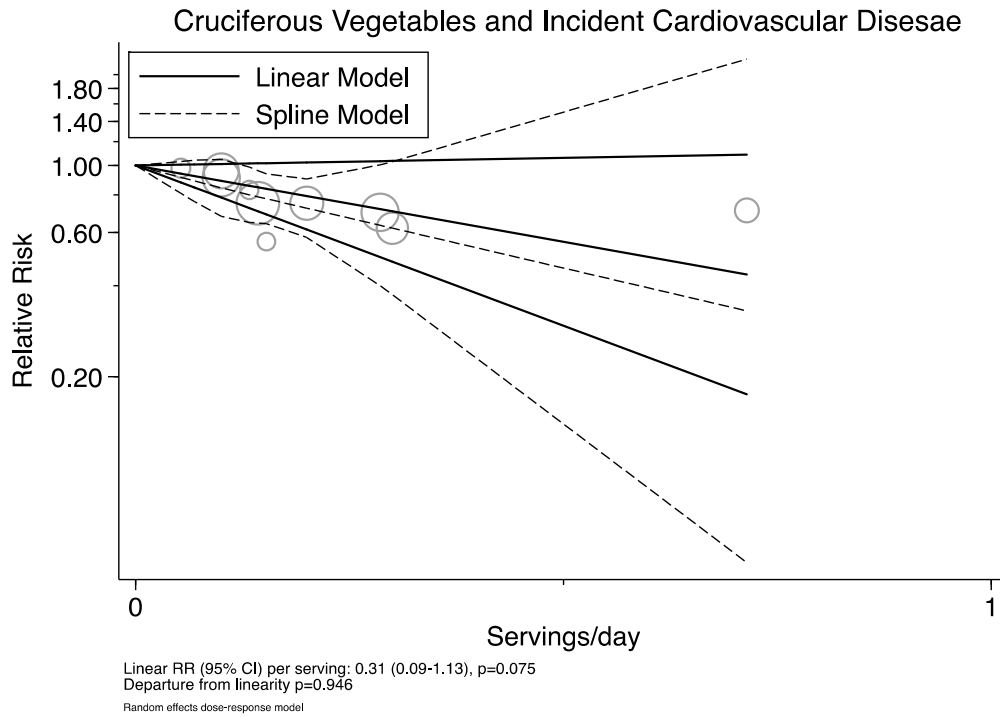


Figure S41. Linear dose-response relation between increasing intake of cruciferous vegetables and incidence of cardiovascular disease y. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

Green Leafy Vegetables and Incident Cardiovascular Diseases

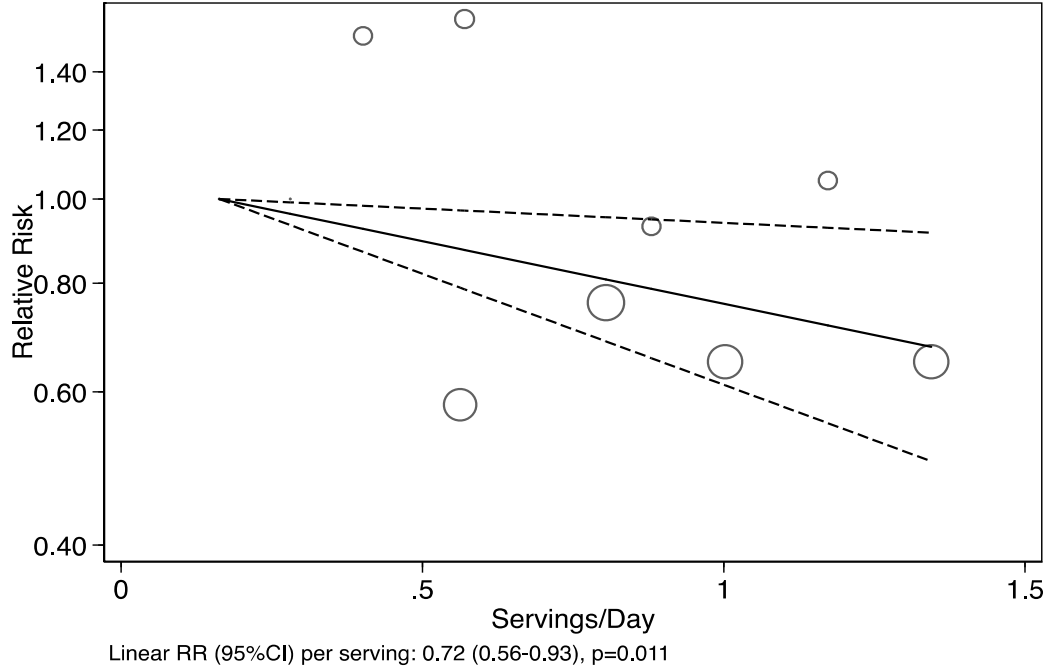


Figure S42. Linear dose-response relation between increasing intake of green leafy vegetables and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

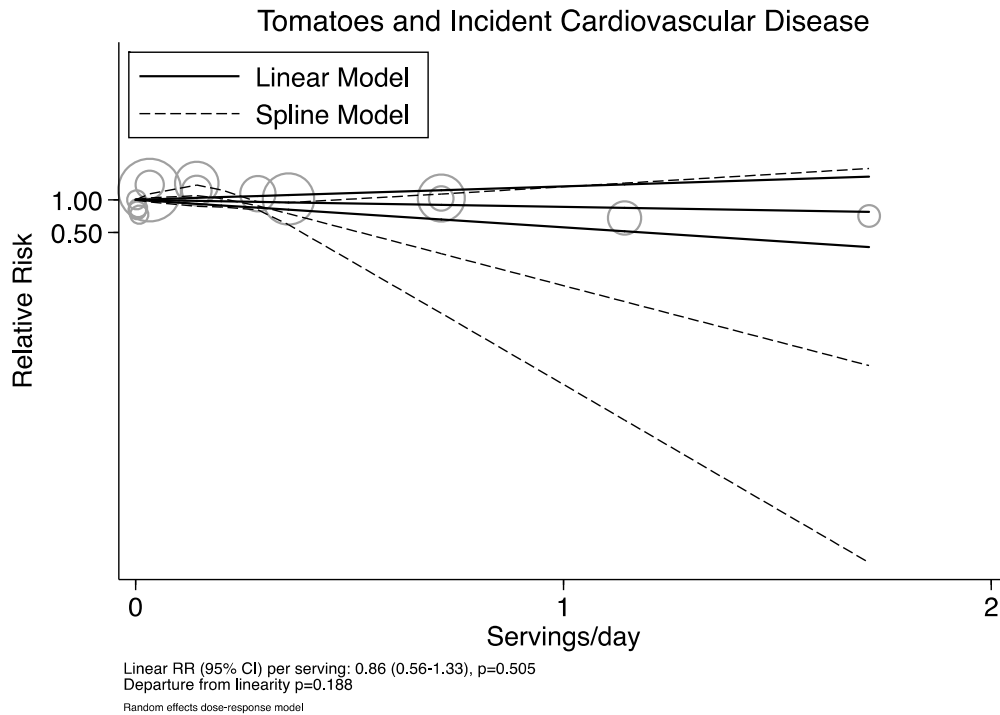


Figure S43. Linear and cubic-spline dose-response relation between increasing tomato intake and incidence of cardiovascular disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

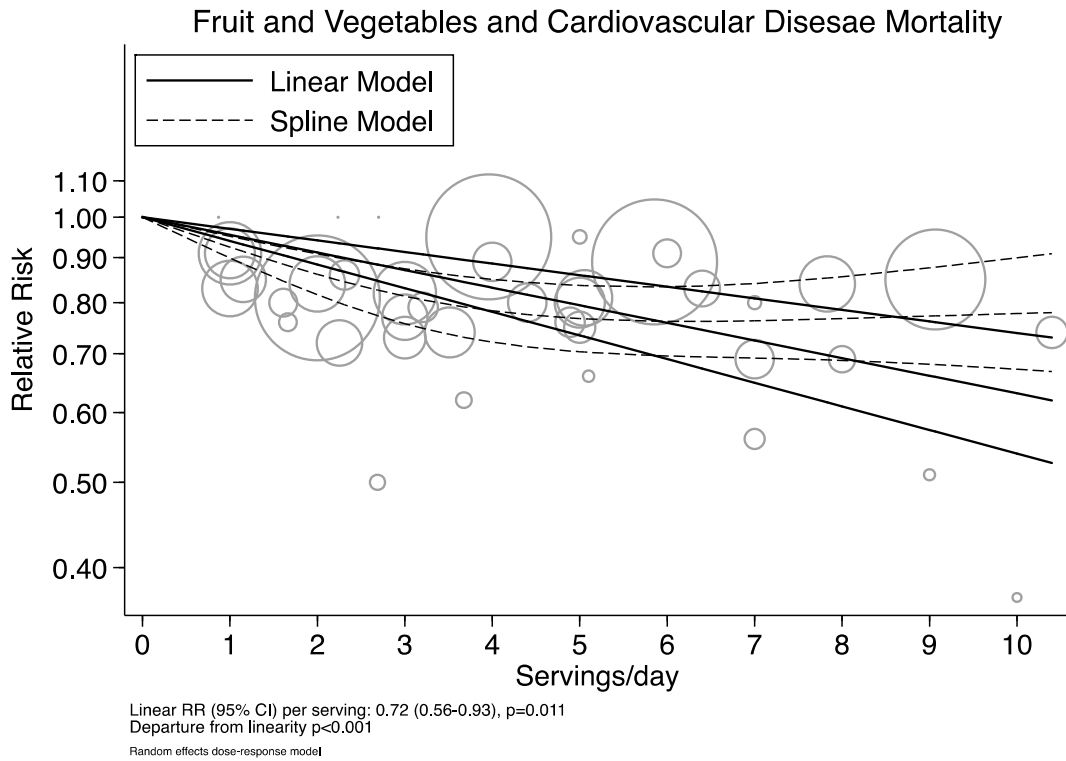


Figure S44. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

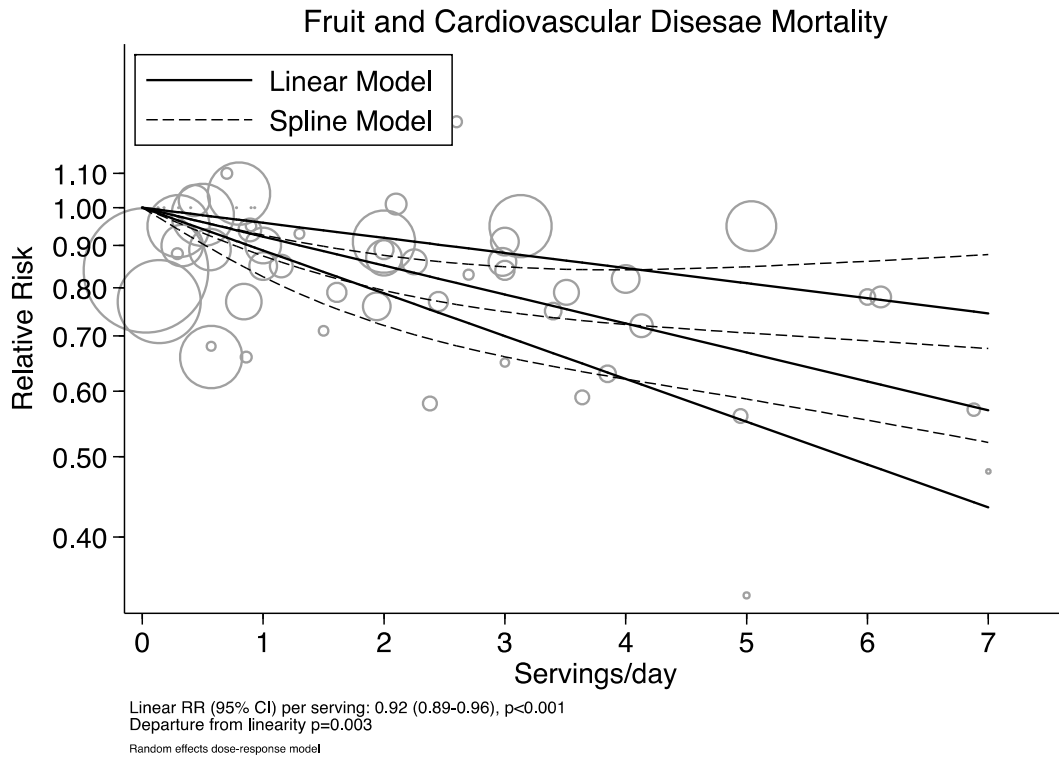


Figure S45. Linear and cubic-spline dose-response relation between increasing fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

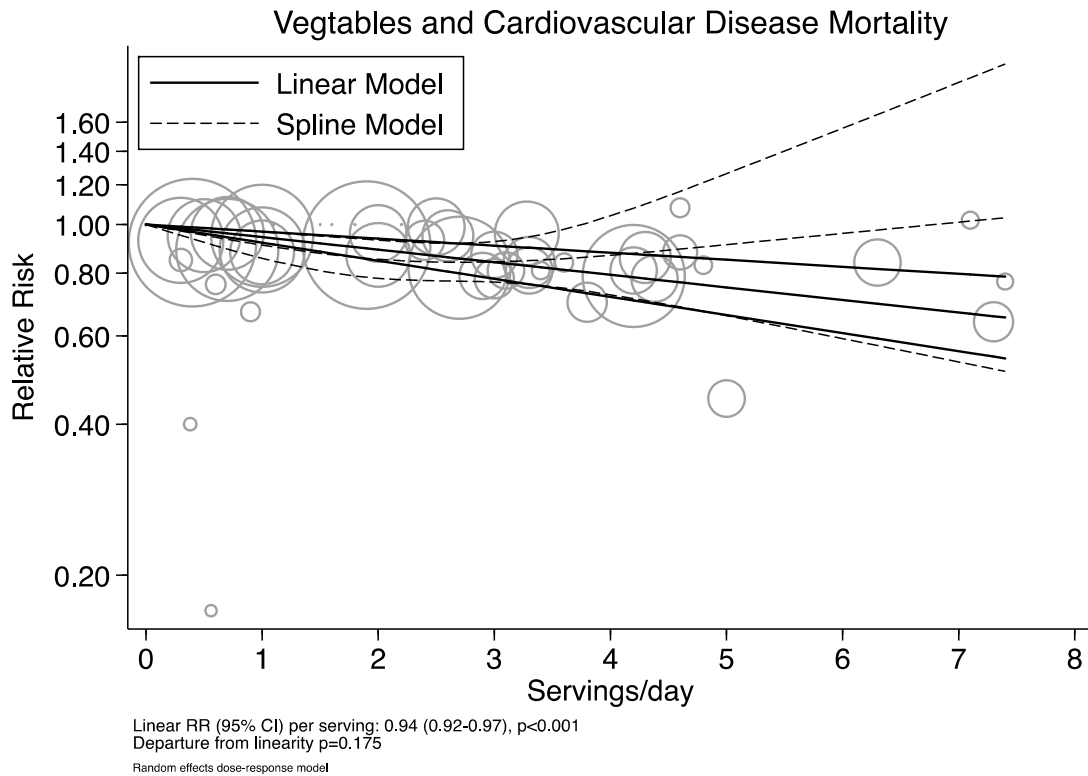


Figure S46. Linear and cubic-spline dose-response relation between increasing intake of vegetables and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

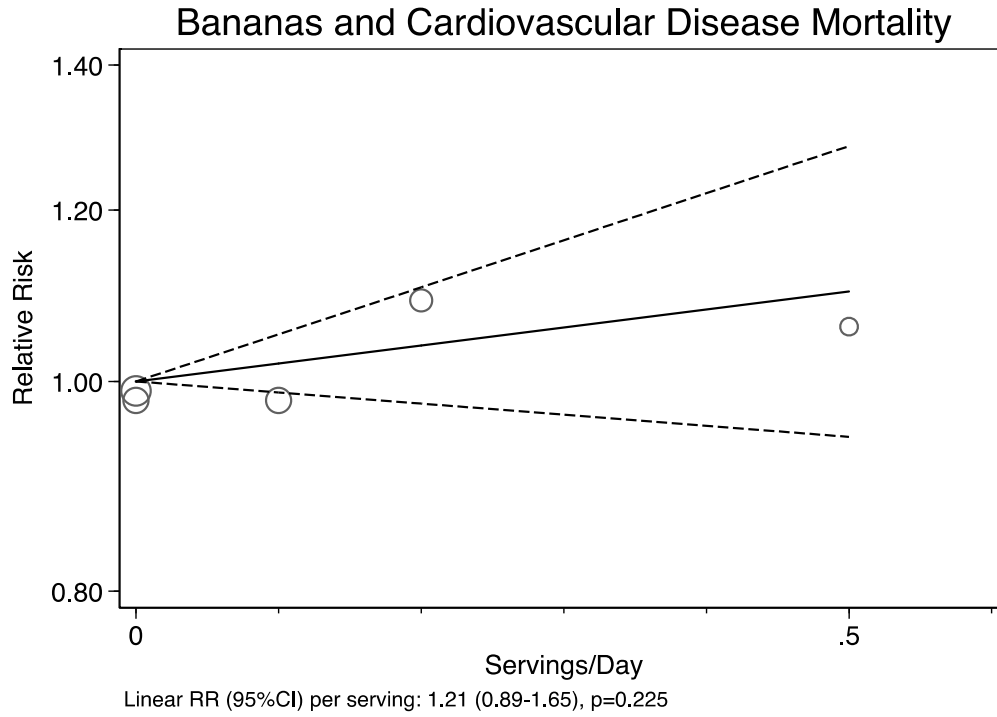


Figure S47. Linear dose-response relation between increasing banana intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

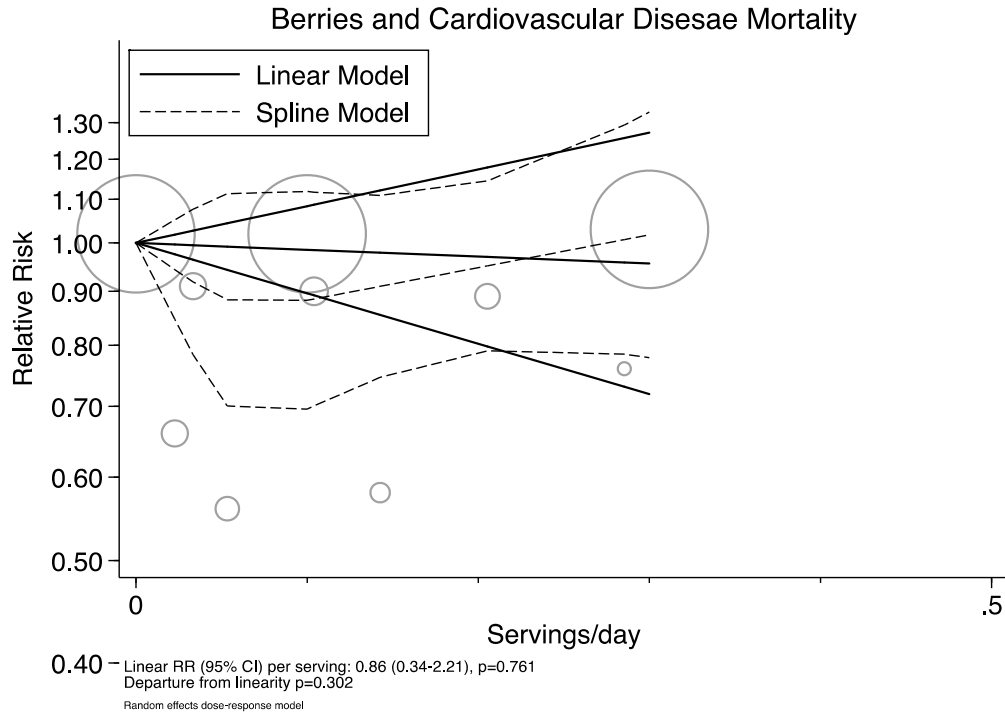


Figure S48. Linear and cubic-spline dose-response relation between increasing berry fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

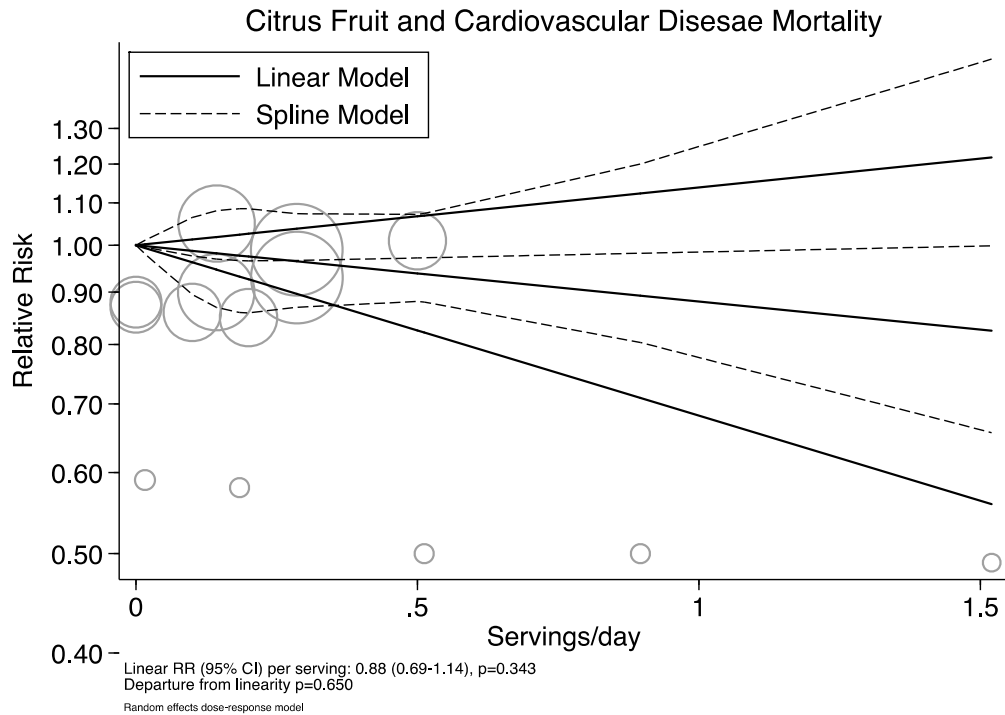


Figure S49. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

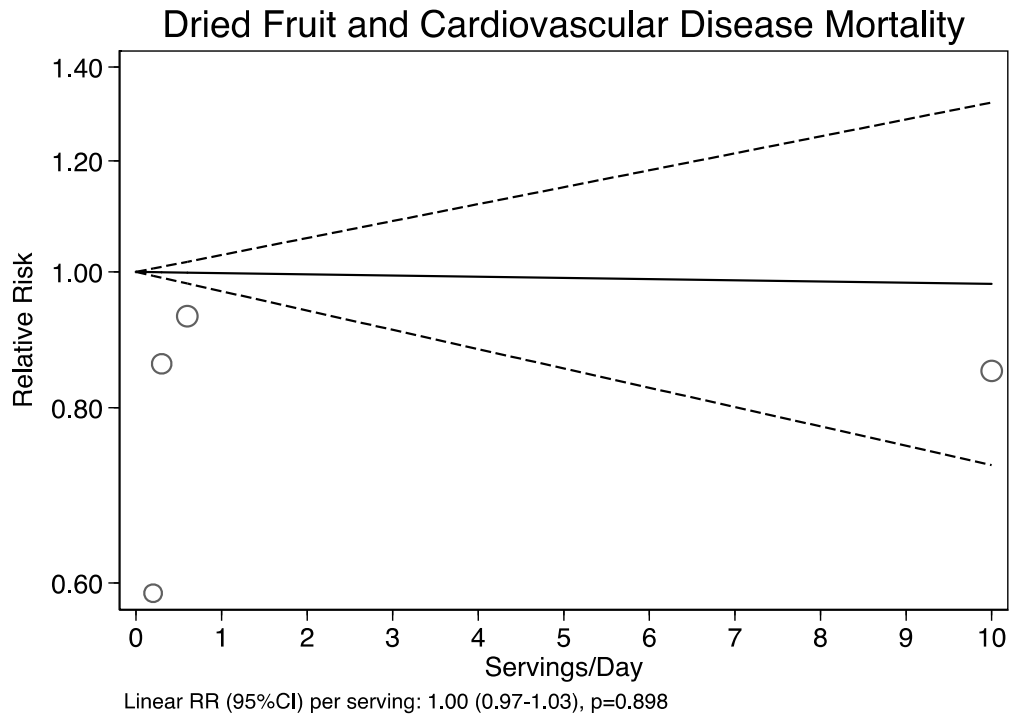


Figure S50. Linear and cubic-spline dose-response relation between increasing dried fruit intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

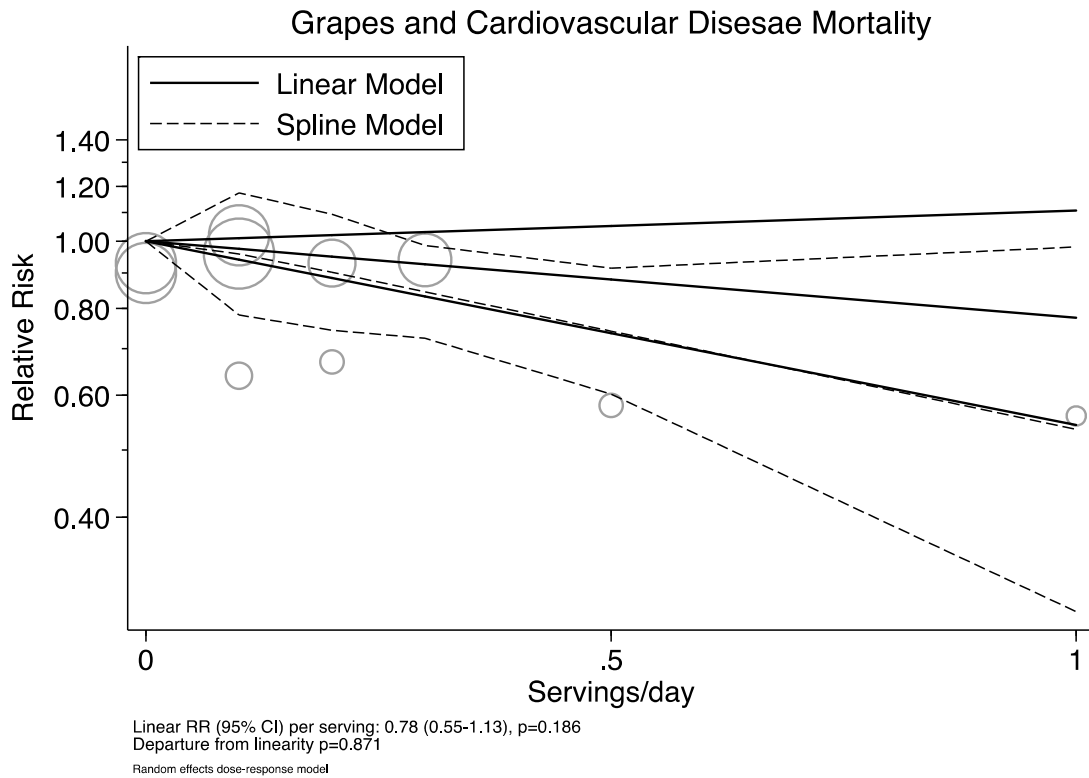


Figure S51. Linear and cubic-spline dose-response relation between increasing grapes intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

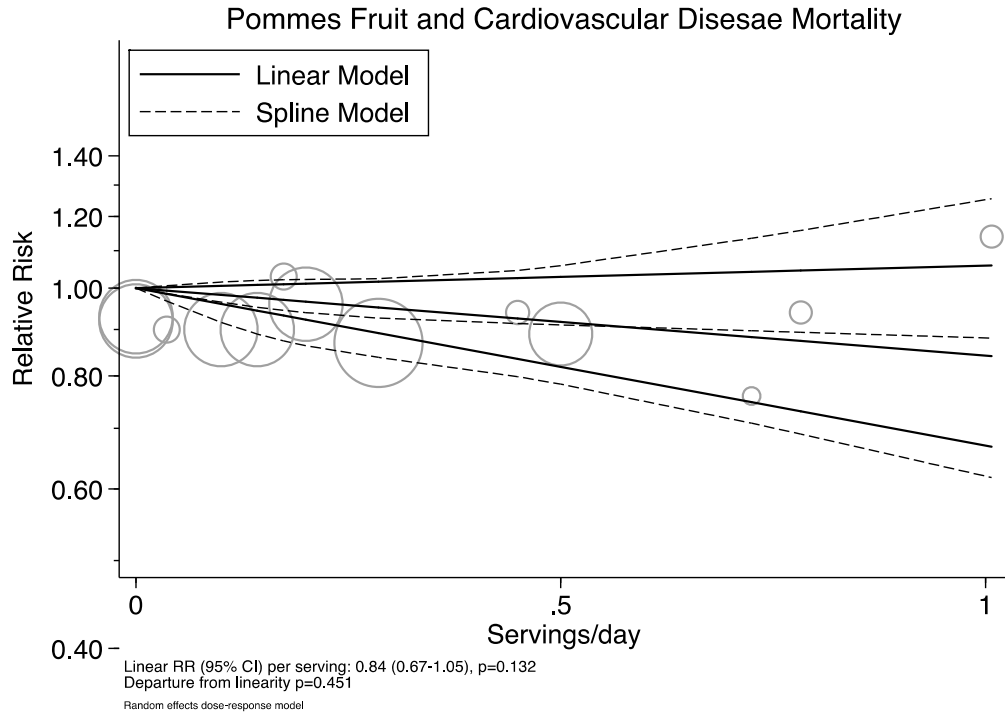


Figure S52. Linear and cubic-spline dose-response relation between increasing pommes intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

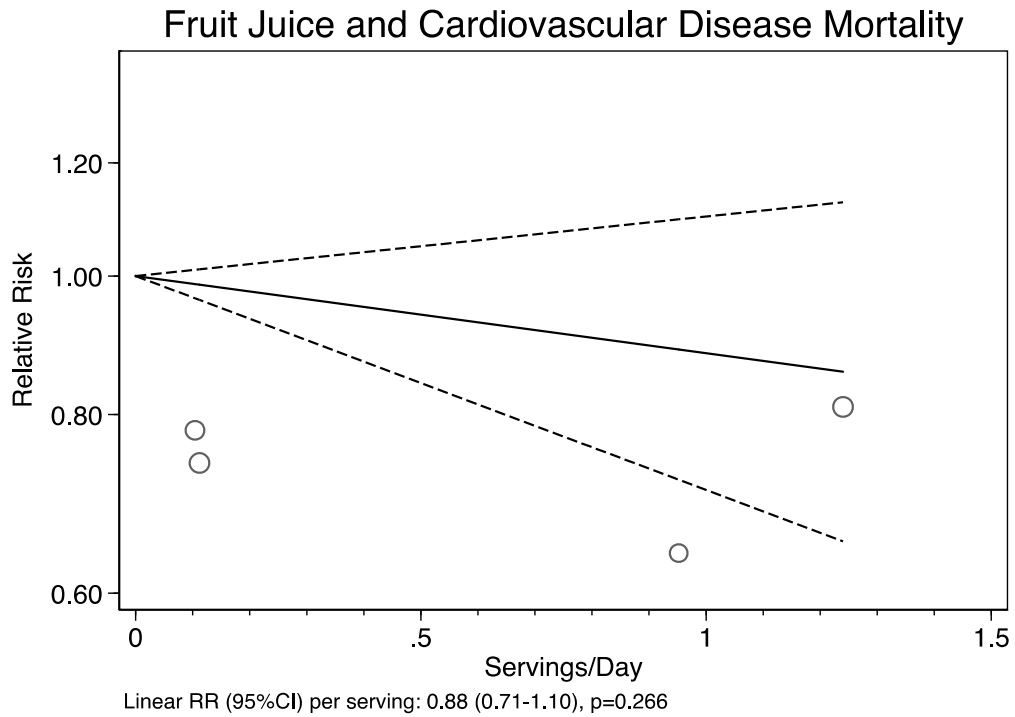


Figure S53. Linear dose-response relation between increasing fruit juice intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

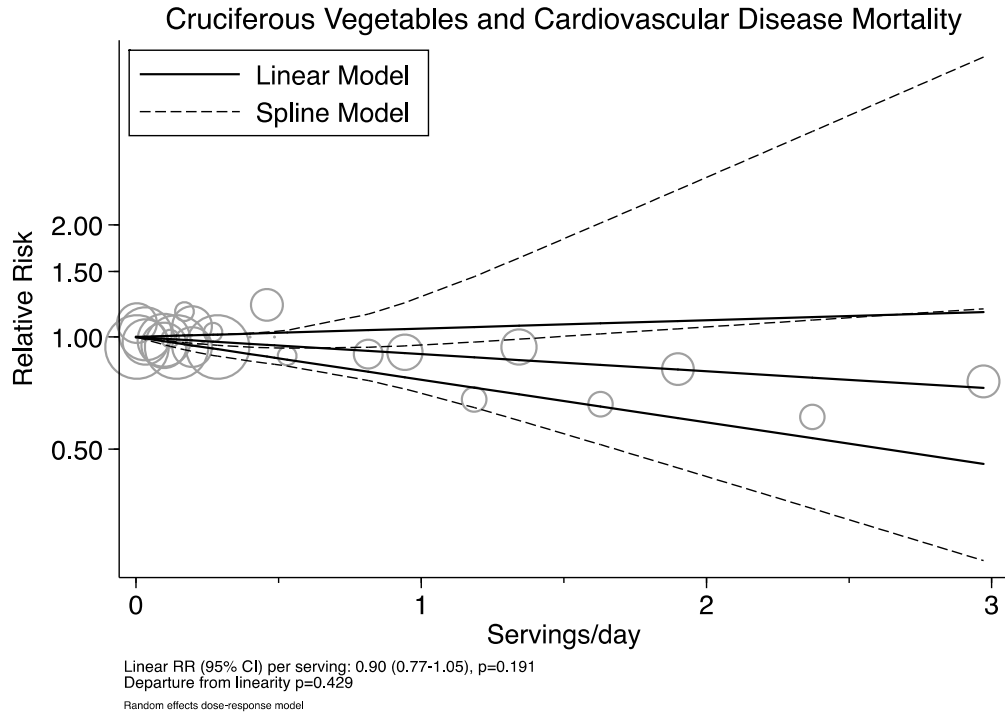


Figure S54. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

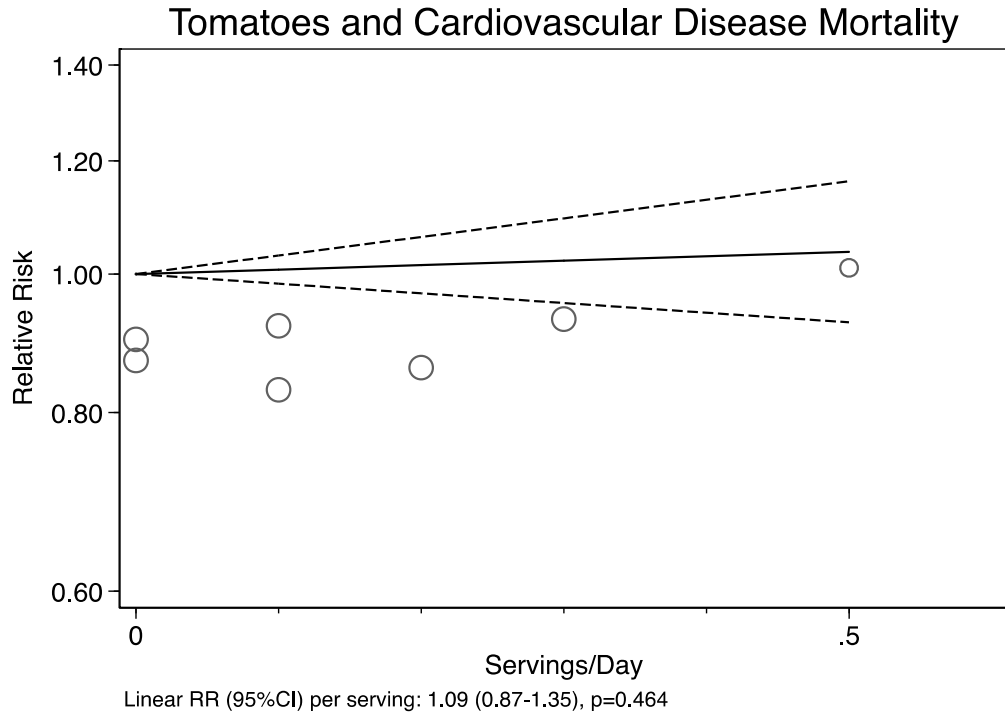
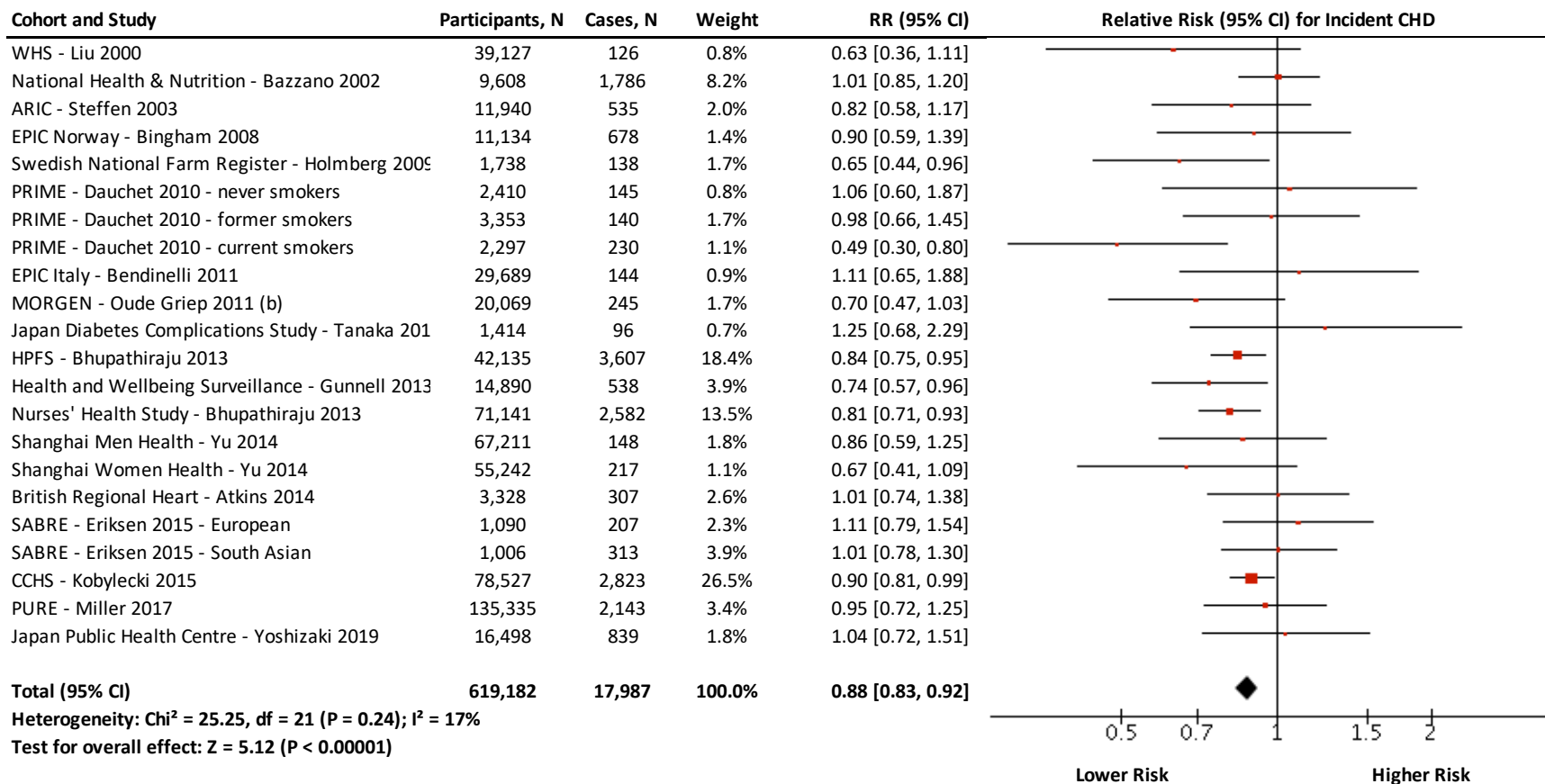


Figure S55. Linear dose-response relation between increasing tomato intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

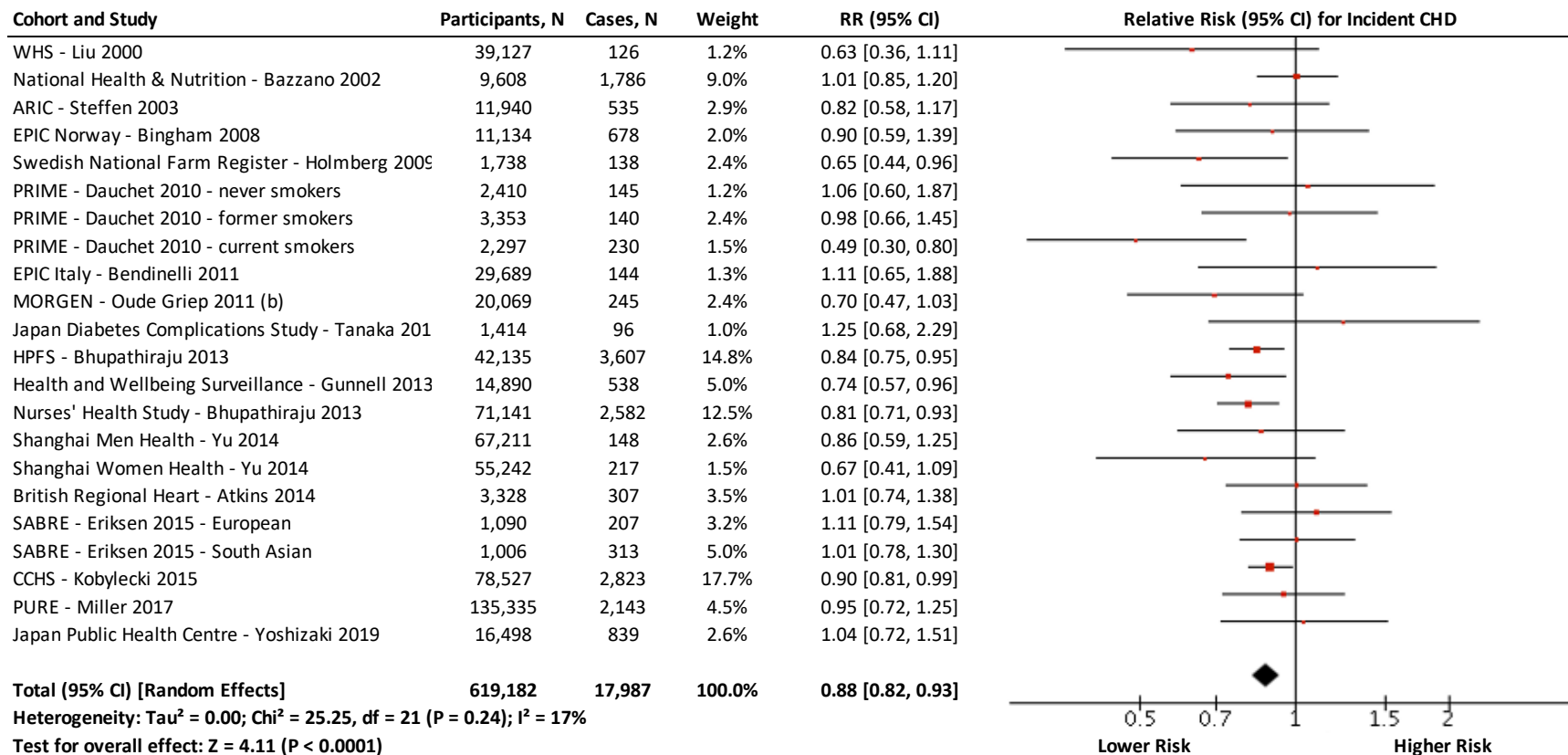
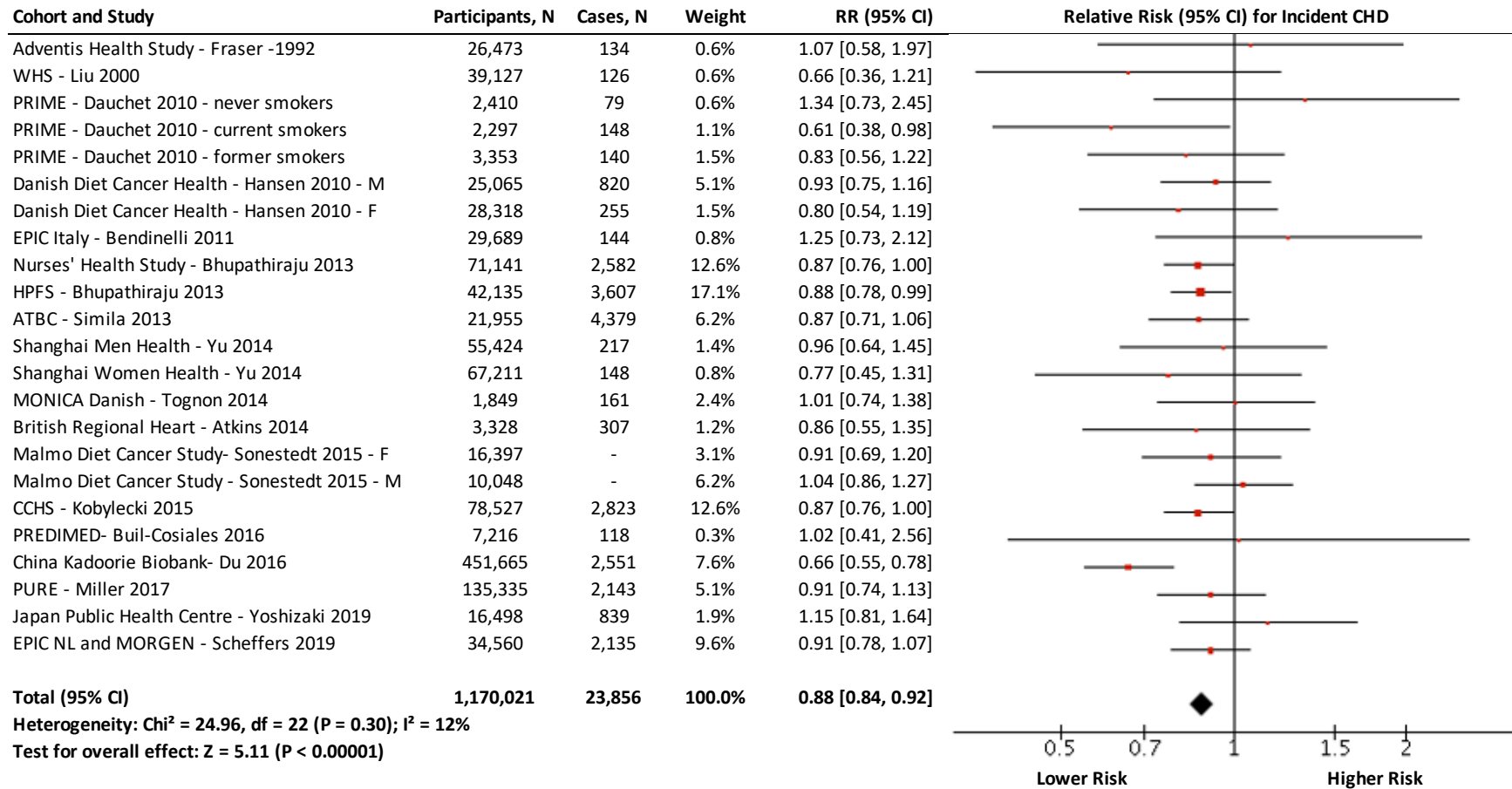


Figure S56. Relation between total fruit and vegetables intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

FRUIT AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

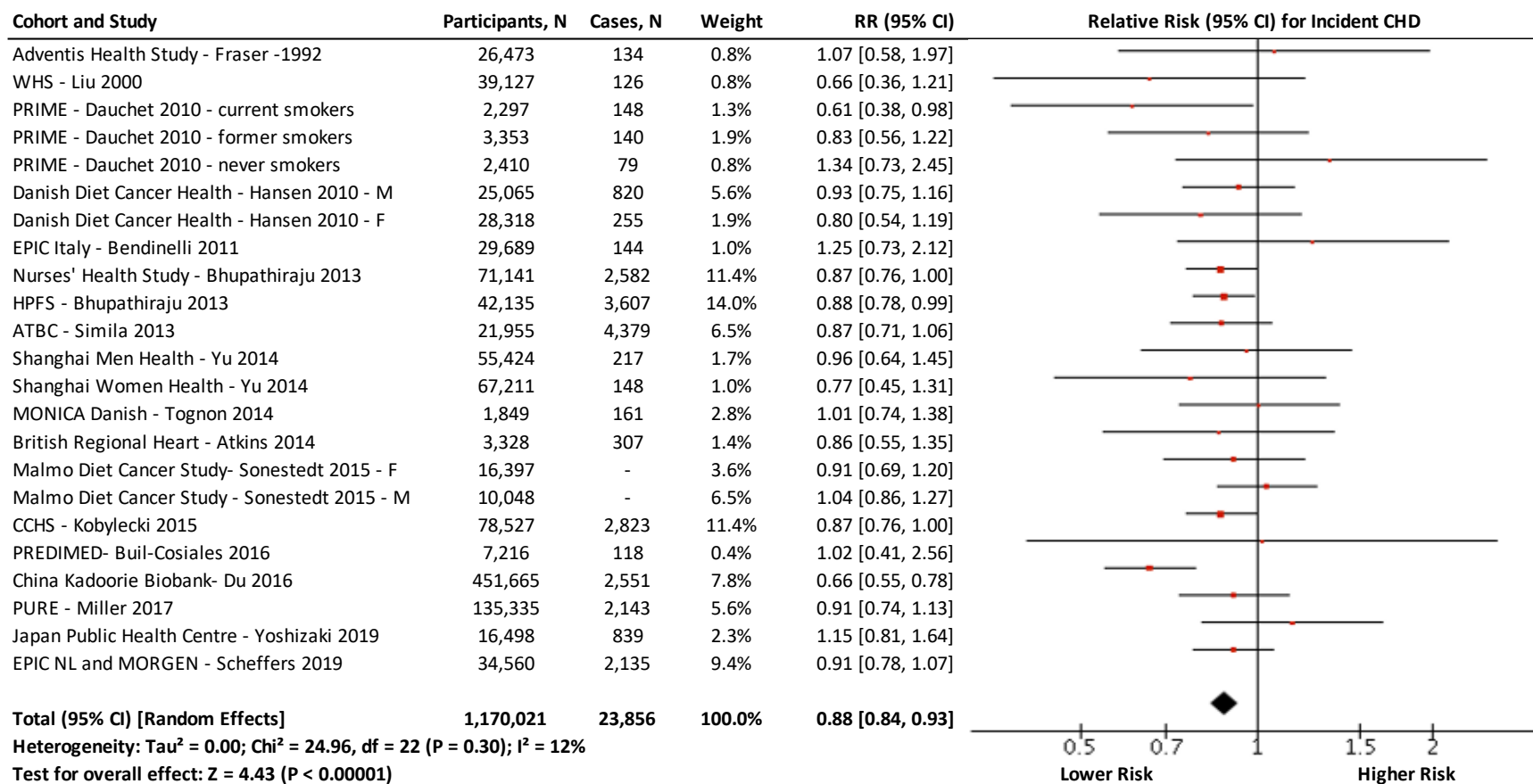
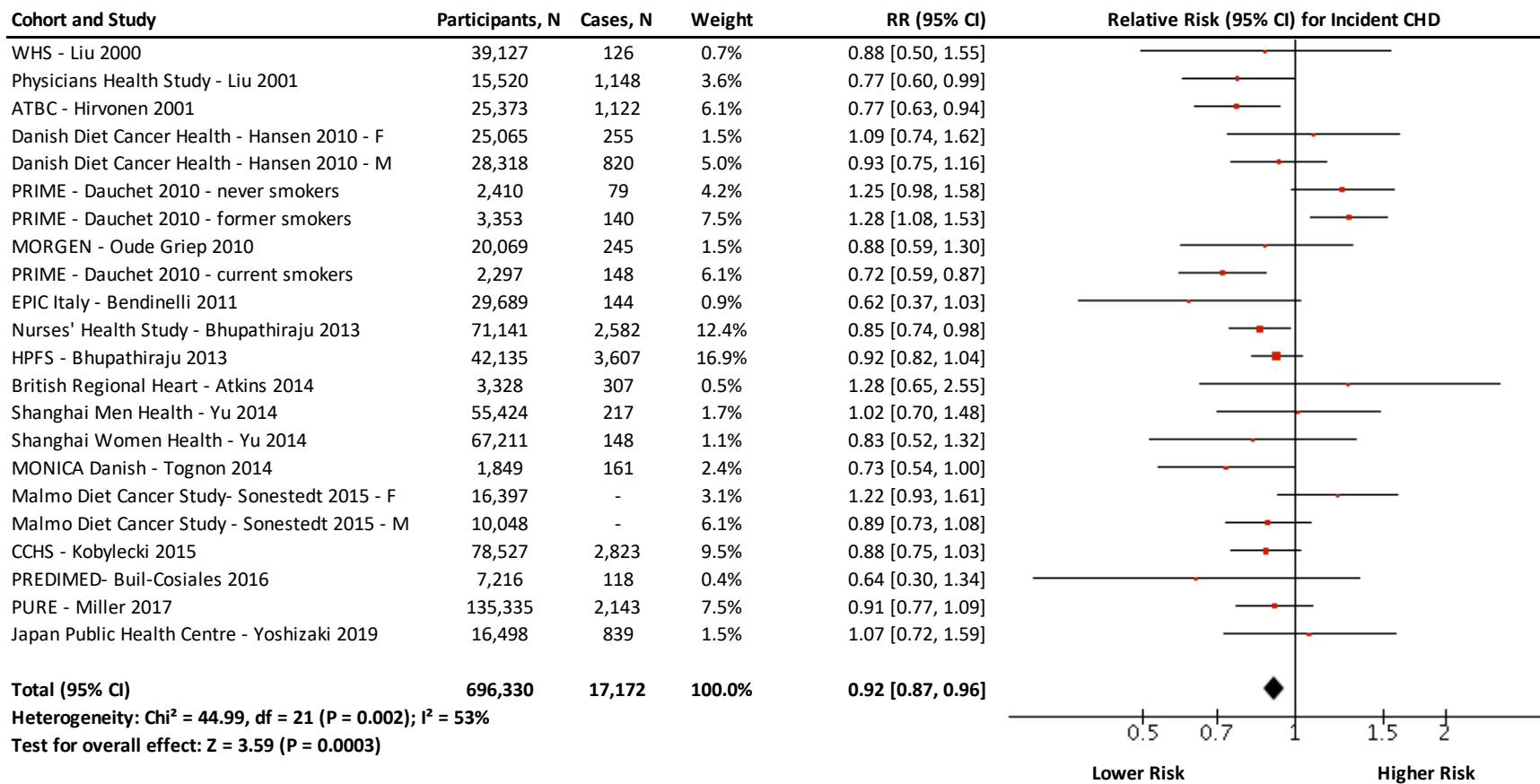


Figure S57. Relation between fruit intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

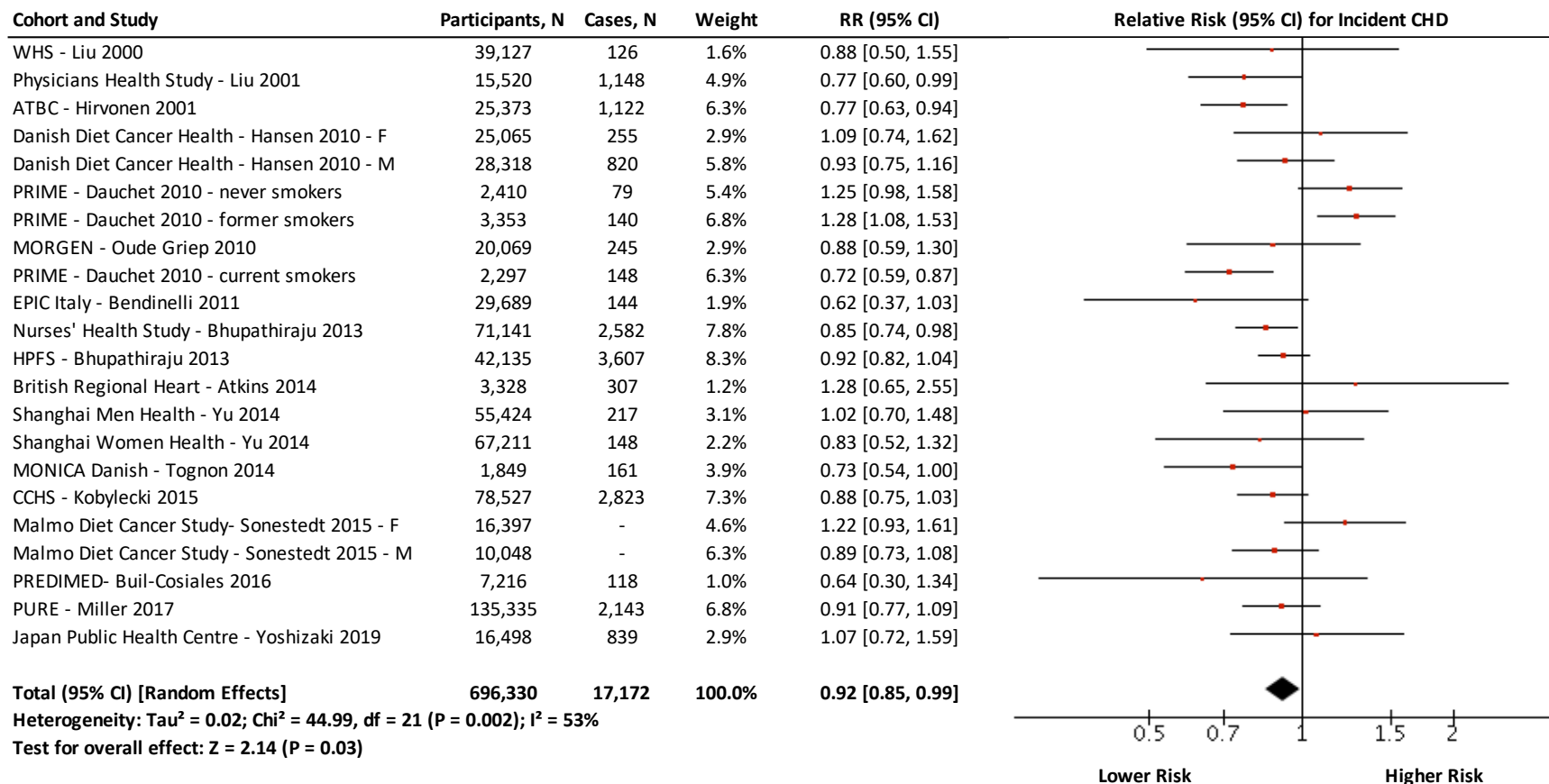
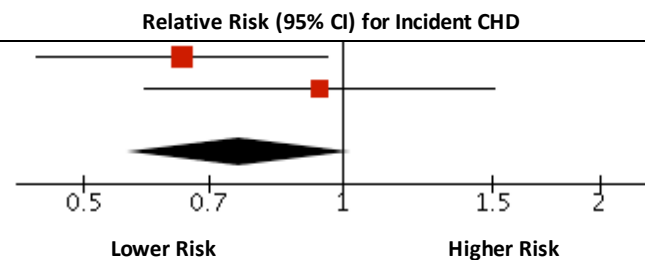


Figure S58. Relation between intake of vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

BANANAS AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Women Health - Yu 2014 - bananas	67,211	148	59.0%	0.94 [0.59, 1.51]
Shanghai Men Health - Yu 2014 - bananas	55,424	217	41.0%	0.65 [0.44, 0.96]
Total (95% CI)	122,635	365	100.0%	0.76 [0.56, 1.02]
Heterogeneity: Chi² = 1.40, df = 1 (P = 0.24); I² = 29%				
Test for overall effect: Z = 1.81 (P = 0.07)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Women Health - Yu 2014 - bananas	67,211	148	43.6%	0.94 [0.59, 1.51]
Shanghai Men Health - Yu 2014 - bananas	55,424	217	56.4%	0.65 [0.44, 0.96]
Total (95% CI) [Random Effects]	122,635	365	100.0%	0.76 [0.53, 1.10]
Heterogeneity: Tau² = 0.02; Chi² = 1.40, df = 1 (P = 0.24); I² = 29%				
Test for overall effect: Z = 1.47 (P = 0.14)				

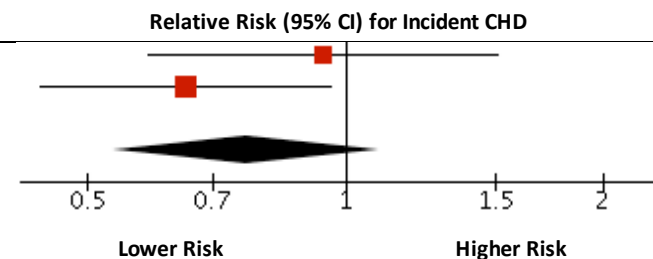


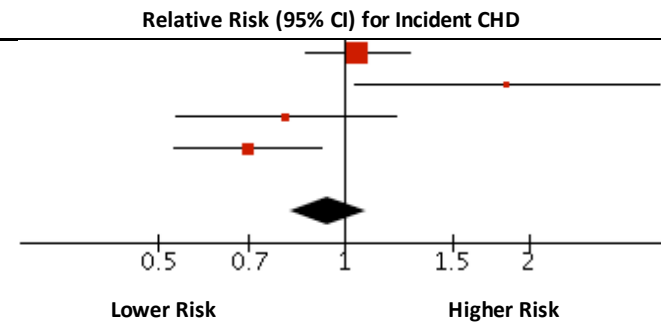
Figure S59. Relation between intake of bananas and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

BERRIES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 11.72$, $\text{df} = 3$ ($P = 0.008$); $I^2 = 74\%$
 Test for overall effect: $Z = 0.81$ ($P = 0.42$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.08$; $\text{Chi}^2 = 11.72$, $\text{df} = 3$ ($P = 0.008$); $I^2 = 74\%$
 Test for overall effect: $Z = 0.20$ ($P = 0.84$)

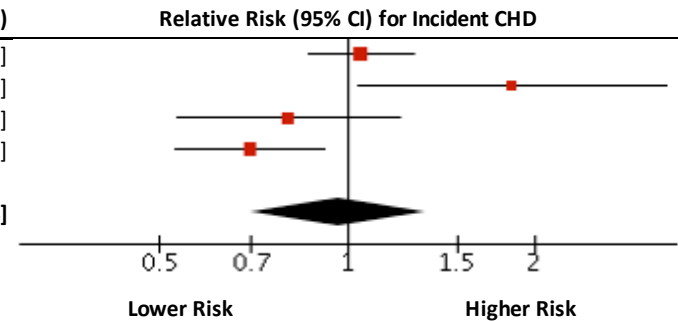
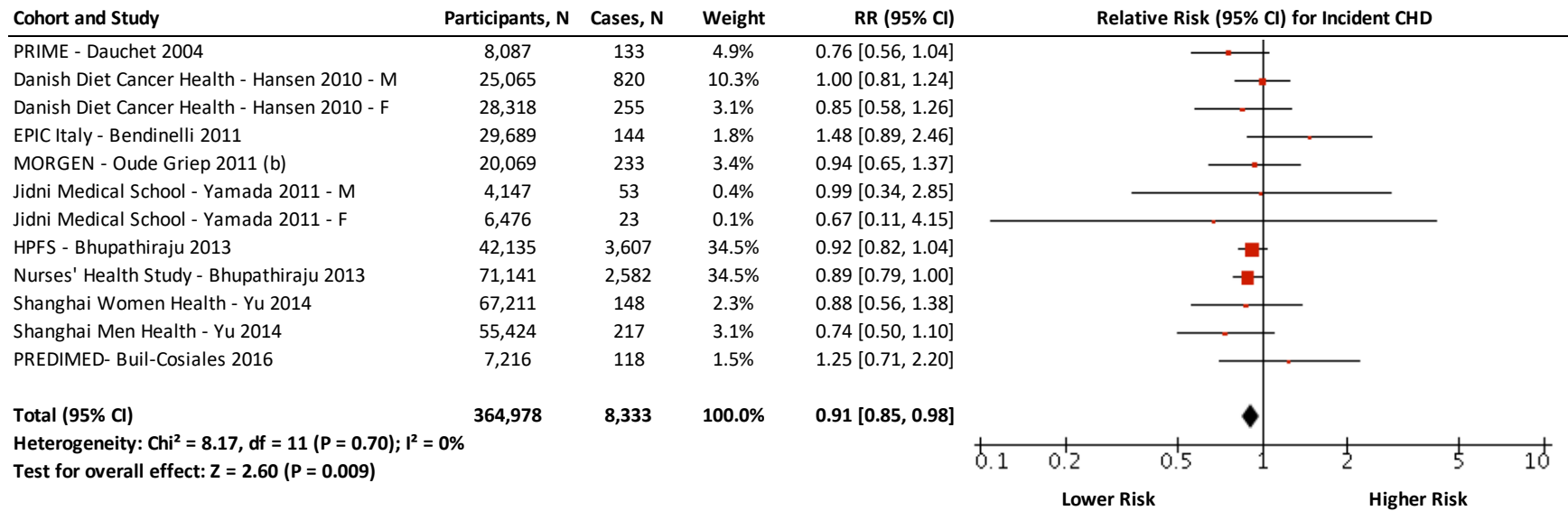


Figure S60. Relation between intake of berries and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CITRUS FRUIT AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects



B. Random Effects

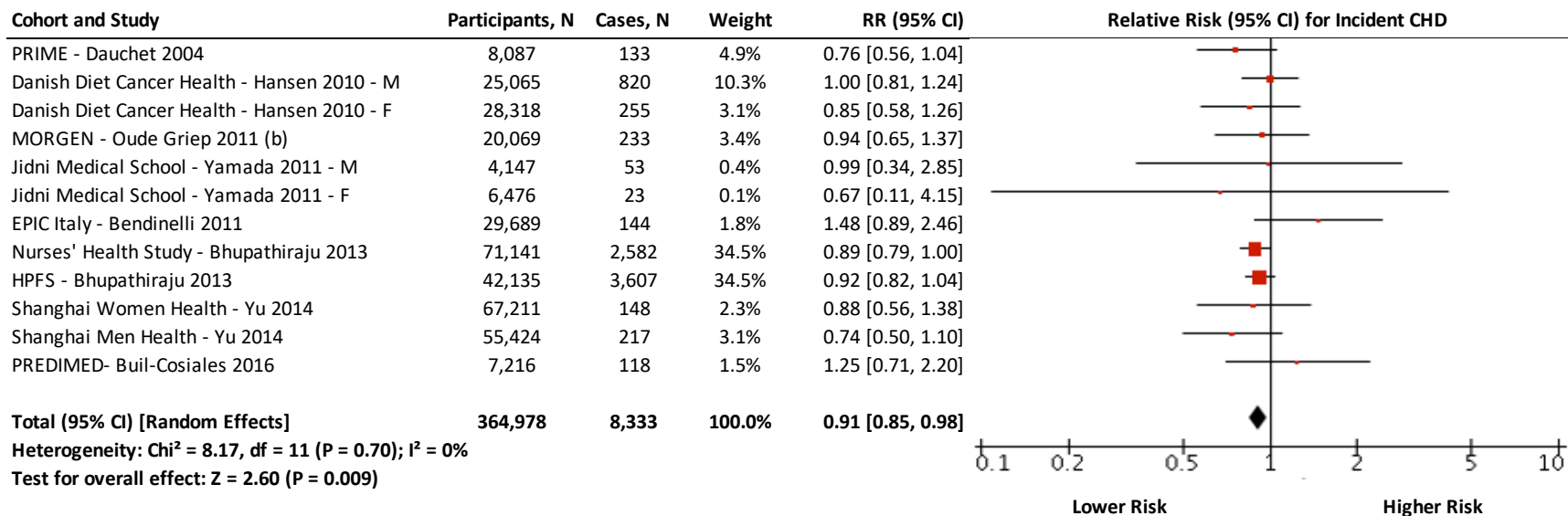


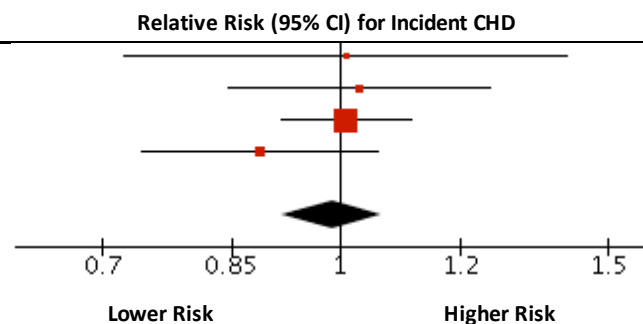
Figure S61. Relation between citrus fruit intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B)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

FRUIT JUICE AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

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

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



B. Random Effects

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

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

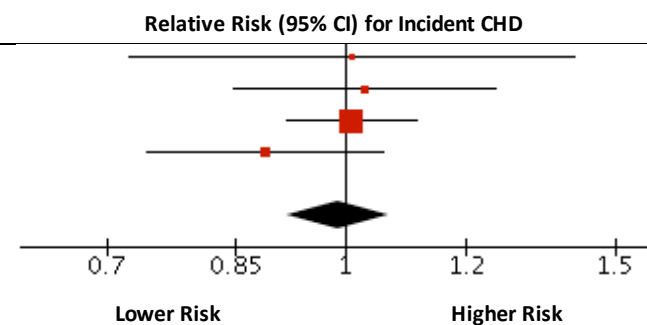


Figure S62. Relation between intake of fruit juice and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B)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

GRAPES AND CORONARY HEART DISEASE INCIDENCE

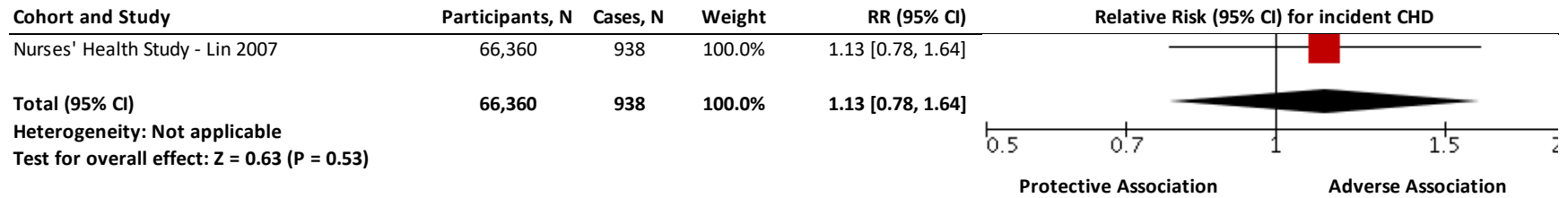


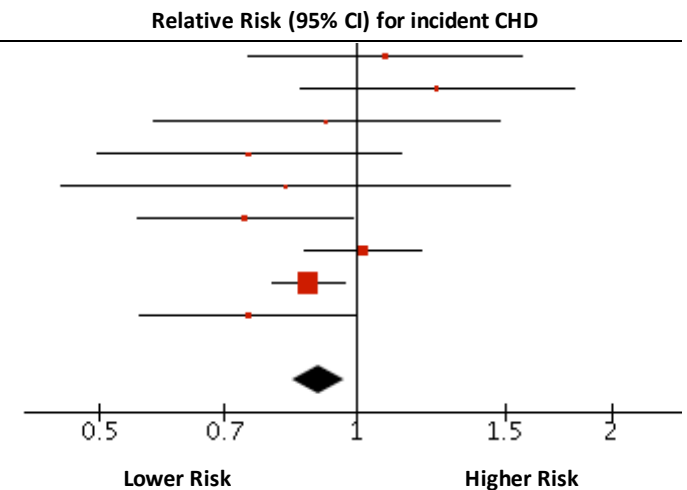
Figure S63. Relation between intake of grapes and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed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

POMMES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 10.63$, $\text{df} = 8$ ($P = 0.22$); $I^2 = 25\%$
 Test for overall effect: $Z = 2.80$ ($P = 0.005$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.01$; $\text{Chi}^2 = 10.63$, $\text{df} = 8$ ($P = 0.22$); $I^2 = 25\%$
 Test for overall effect: $Z = 1.95$ ($P = 0.05$)

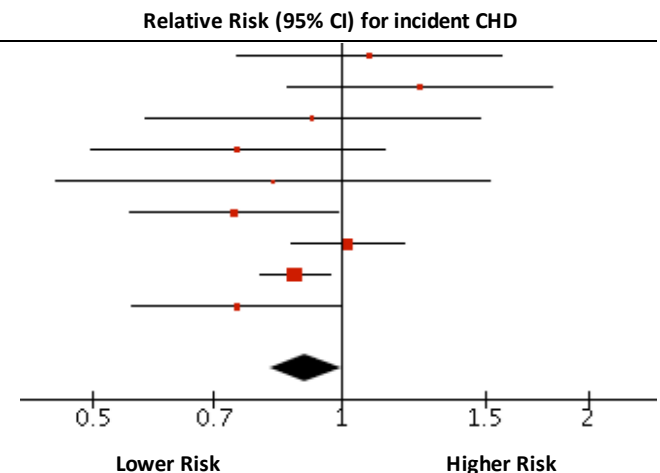
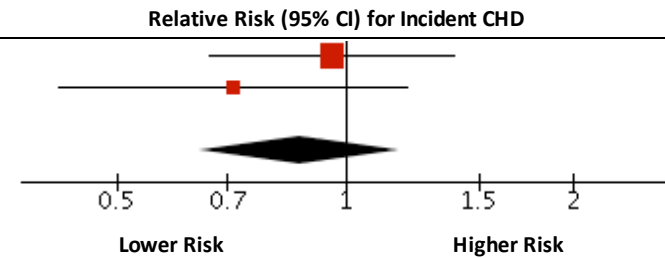


Figure S64. Relation between intake of pomes fruit and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B)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

WATERMELON AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Men Health - Yu 2014 - Watermelon	55,424	217	66.9%	0.96 [0.66, 1.39]
Shanghai Women Health - Yu 2014 - Watermelon	67,211	148	33.1%	0.71 [0.42, 1.21]
Total (95% CI)	122,635	365	100.0%	0.87 [0.64, 1.18]
Heterogeneity: Chi² = 0.83, df = 1 (P = 0.36); I² = 0%				
Test for overall effect: Z = 0.90 (P = 0.37)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Shanghai Men Health - Yu 2014 - Watermelon	55,424	217	66.9%	0.96 [0.66, 1.39]
Shanghai Women Health - Yu 2014 - Watermelon	67,211	148	33.1%	0.71 [0.42, 1.21]
Total (95% CI) [Random Effects]	122,635	365	100.0%	0.87 [0.64, 1.18]
Heterogeneity: Tau² = 0.00; Chi² = 0.83, df = 1 (P = 0.36); I² = 0%				
Test for overall effect: Z = 0.90 (P = 0.37)				

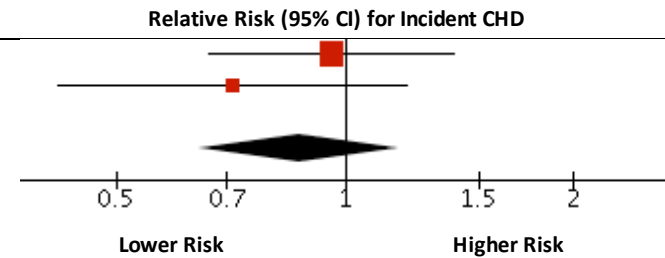
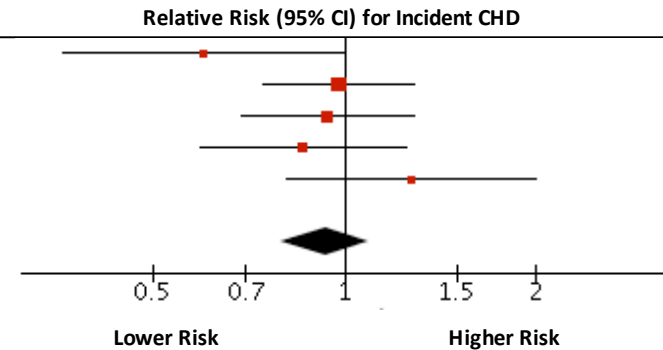


Figure S65. Relation between watermelon intake and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

ALLIUM VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Caerphilly Prospective Study - Hertog 1997	1,900	186	9.8%	0.60 [0.36, 1.00]
Nurses' Health Study - Lin 2007	66,360	938	33.7%	0.98 [0.74, 1.29]
MORGEN - Oude Griep 2011 (b)	20,069	245	25.8%	0.94 [0.69, 1.29]
Shanghai Men Health - Yu 2014	55,424	217	18.3%	0.86 [0.59, 1.25]
Shanghai Women Health - Yu 2014	67,211	148	12.5%	1.27 [0.81, 2.00]
Total (95% CI)	210,964	1,734	100.0%	0.93 [0.80, 1.09]
Heterogeneity: Chi² = 4.99, df = 4 (P = 0.29); I² = 20%				
Test for overall effect: Z = 0.86 (P = 0.39)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
Caerphilly Prospective Study - Hertog 1997	1,900	186	11.3%	0.60 [0.36, 1.00]
Nurses' Health Study - Lin 2007	66,360	938	30.5%	0.98 [0.74, 1.29]
MORGEN - Oude Griep 2011 (b)	20,069	245	25.1%	0.94 [0.69, 1.29]
Shanghai Women Health - Yu 2014	67,211	148	14.0%	1.27 [0.81, 2.00]
Shanghai Men Health - Yu 2014	55,424	217	19.2%	0.86 [0.59, 1.25]
Total (95% CI) [Random Effects]	210,964	1,734	100.0%	0.93 [0.77, 1.11]
Heterogeneity: Tau² = 0.01; Chi² = 4.99, df = 4 (P = 0.29); I² = 20%				
Test for overall effect: Z = 0.80 (P = 0.42)				

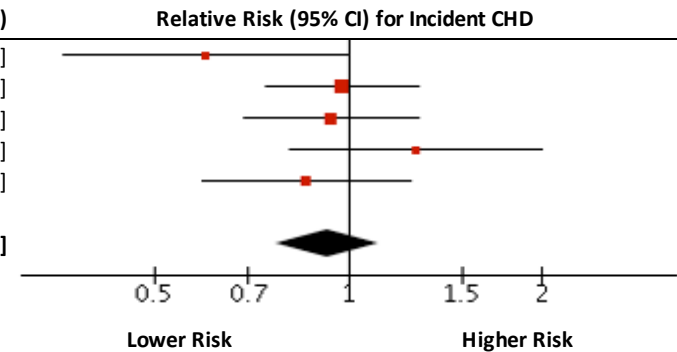


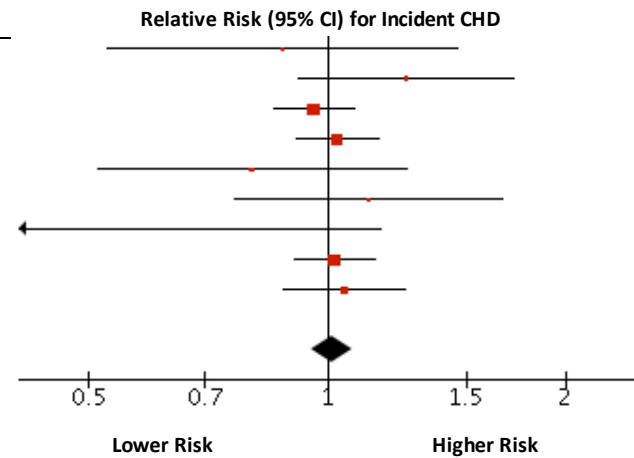
Figure S66. Relation between intake of allium vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

CRUCIFEROUS VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 7.55$, $\text{df} = 8$ ($P = 0.48$); $I^2 = 0\%$
 Test for overall effect: $Z = 0.38$ ($P = 0.71$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 7.55$, $\text{df} = 8$ ($P = 0.48$); $I^2 = 0\%$
 Test for overall effect: $Z = 0.38$ ($P = 0.71$)

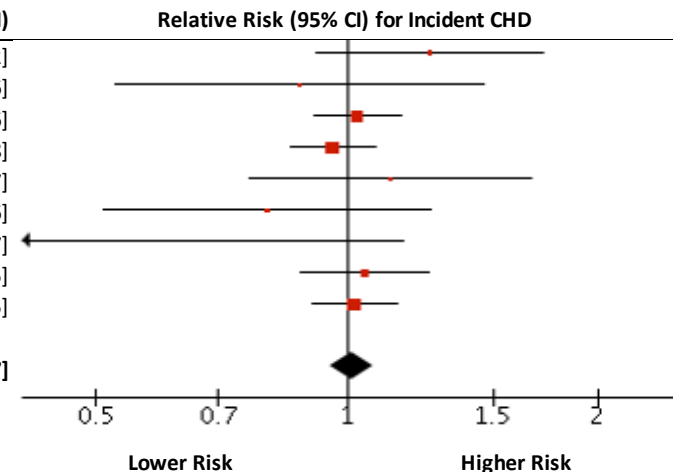
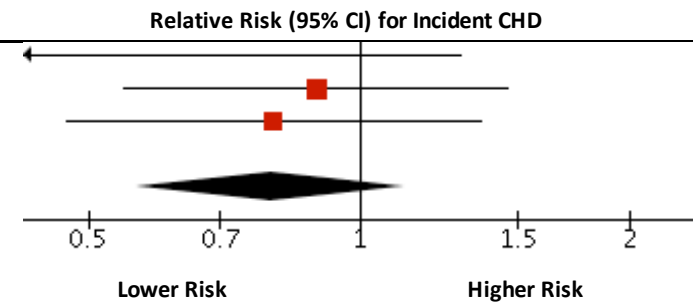


Figure S67. Relation between intake of cruciferous vegetables and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

TOMATOES AND CORONARY HEART DISEASE INCIDENCE

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	201	8.3%	0.39 [0.12, 1.29]
Nurses' Health Study - Lin 2007	66,360	938	49.4%	0.90 [0.55, 1.46]
EPIC Italy - Bendinelli 2011	29,689	144	42.3%	0.80 [0.47, 1.36]
Total (95% CI)	134,494	1,283	100.0%	0.80 [0.57, 1.13]
Heterogeneity: Chi² = 1.59, df = 2 (P = 0.45); I² = 0%				
Test for overall effect: Z = 1.28 (P = 0.20)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
WHS - Sesso 2003 (a)	38,445	201	8.3%	0.39 [0.12, 1.29]
Nurses' Health Study - Lin 2007	66,360	938	49.4%	0.90 [0.55, 1.46]
EPIC Italy - Bendinelli 2011	29,689	144	42.3%	0.80 [0.47, 1.36]
Total (95% CI) [Random Effects]	134,494	1,283	100.0%	0.80 [0.57, 1.13]
Heterogeneity: Tau² = 0.00; Chi² = 1.59, df = 2 (P = 0.45); I² = 0%				
Test for overall effect: Z = 1.28 (P = 0.20)				

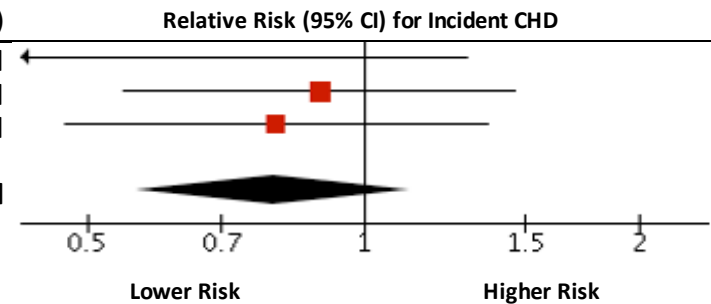
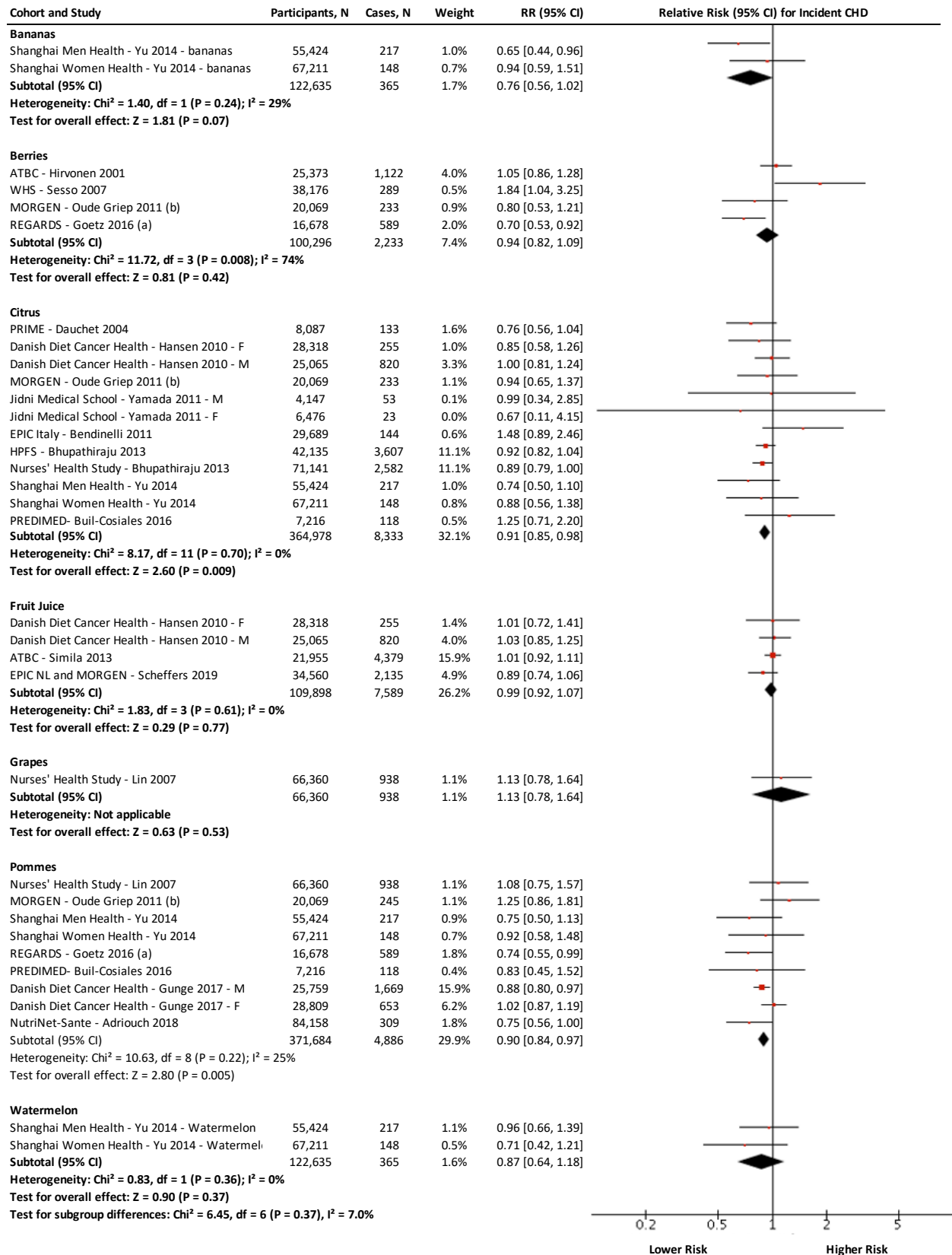


Figure S69. Relation between intake of tomatoes and coronary heart disease incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

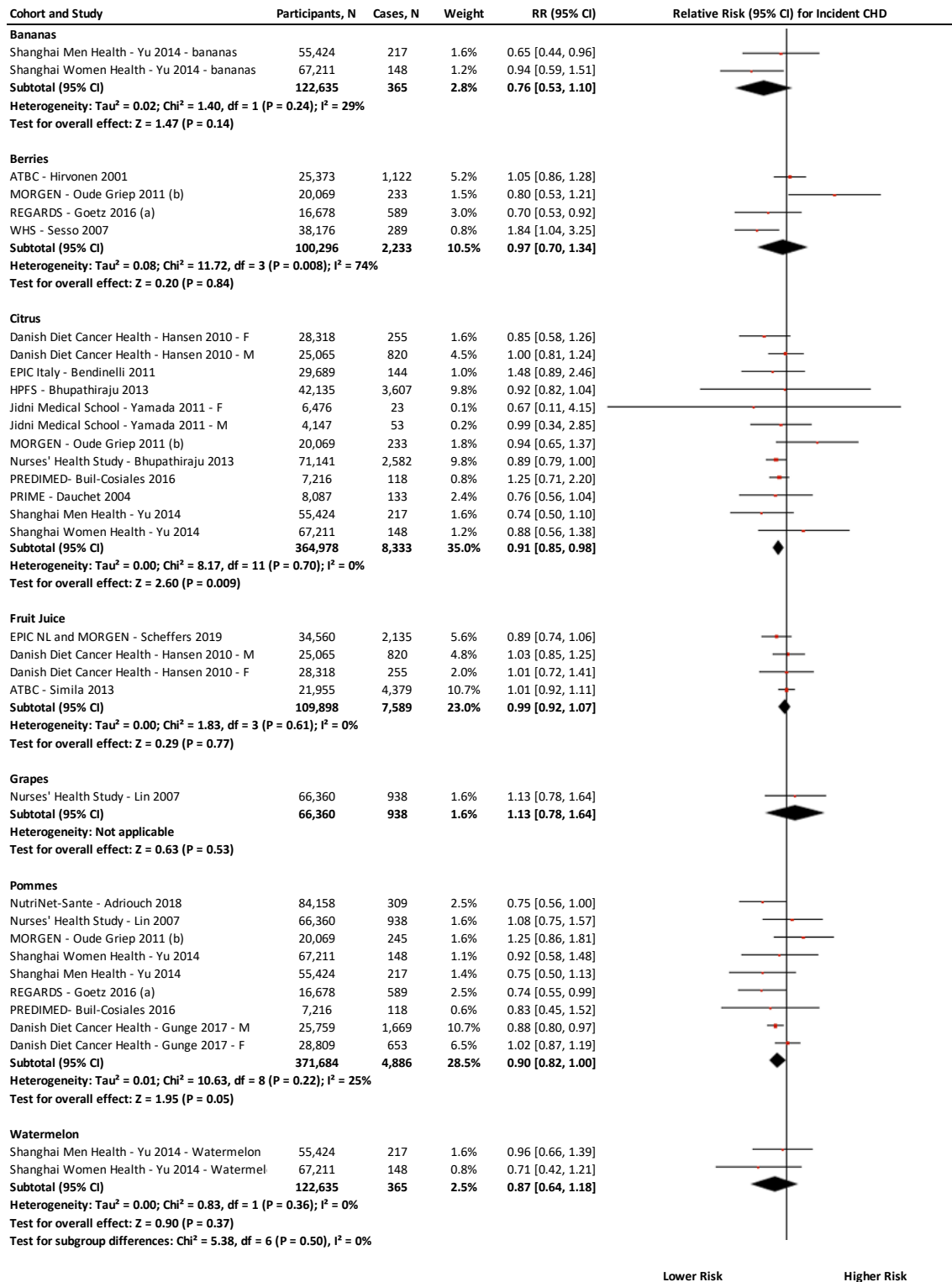
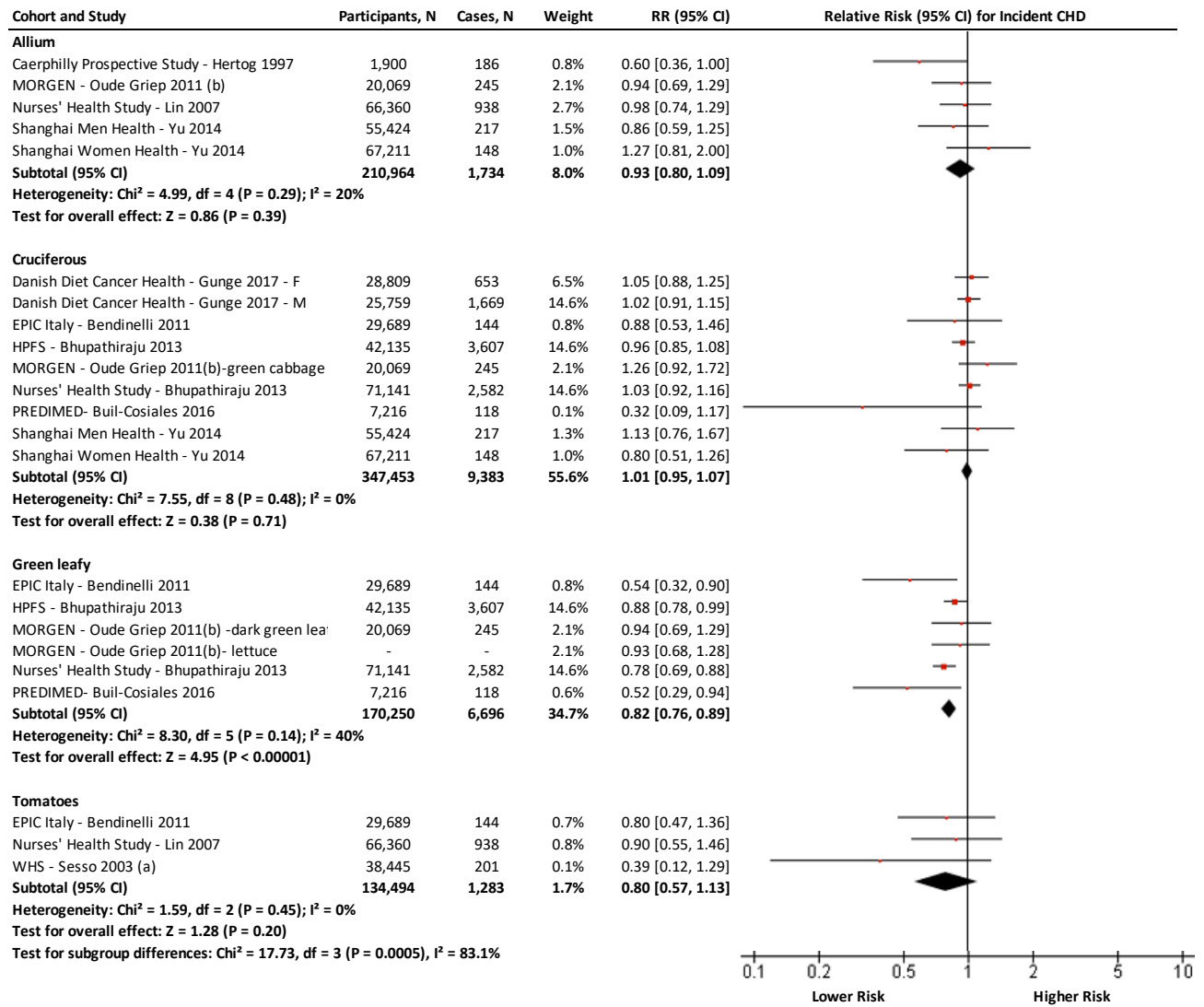


Figure S70. Relation between sources of fruit and CHD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models.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

A. Fixed Effects



B. Random Effects

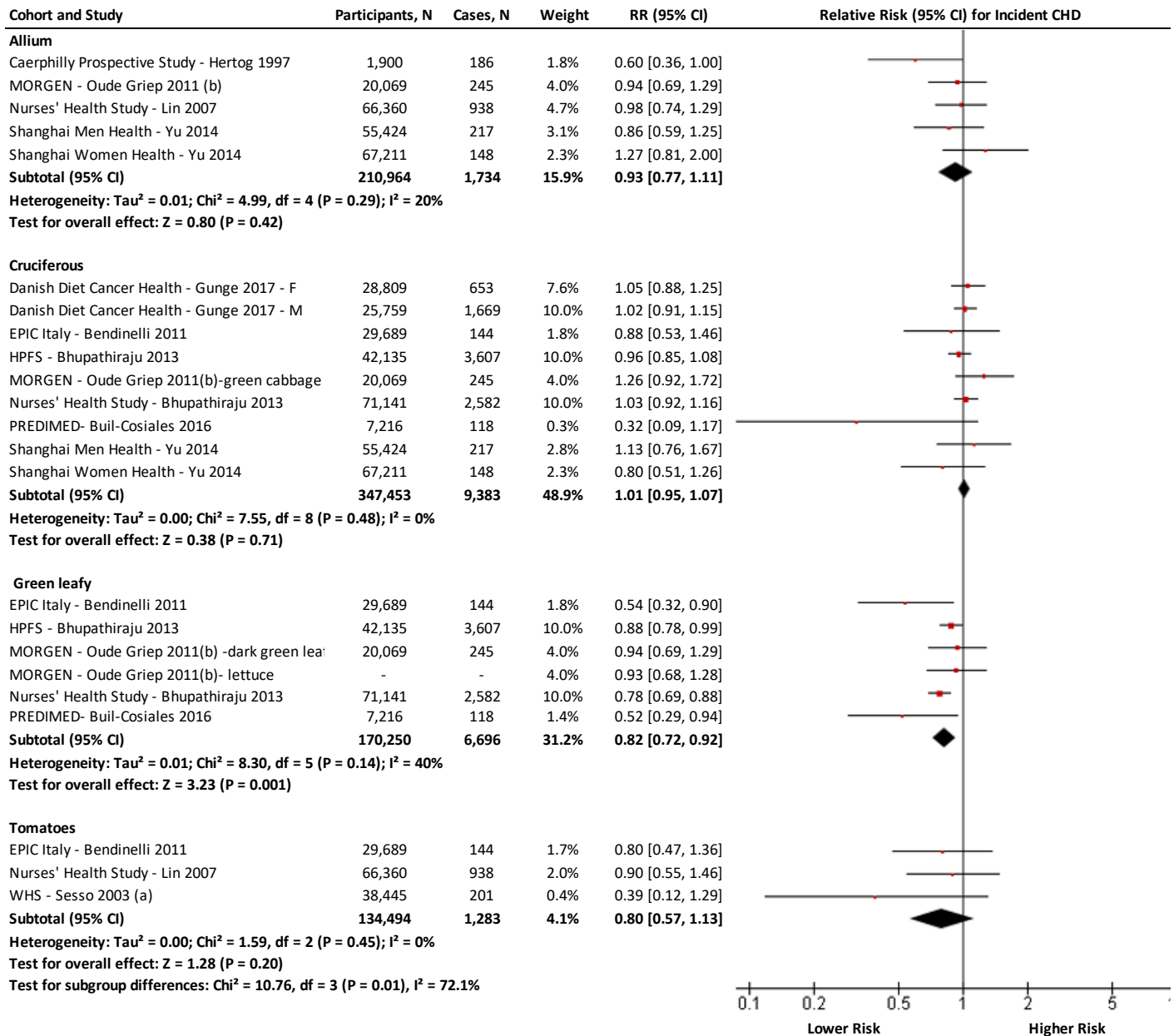


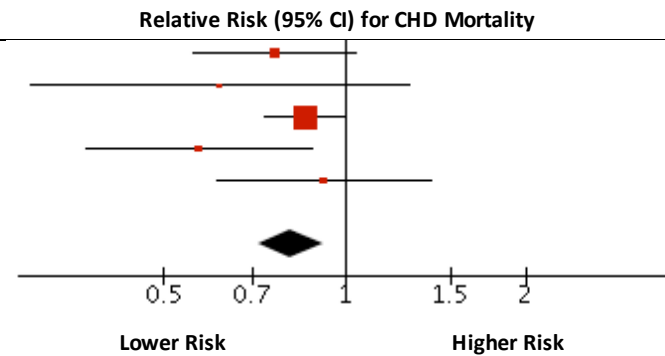
Figure S71. Relation between sources of vegetables and CHD incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 4.15$, $\text{df} = 4$ ($P = 0.39$); $I^2 = 4\%$
 Test for overall effect: $Z = 3.24$ ($P = 0.001$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 4.15$, $\text{df} = 4$ ($P = 0.39$); $I^2 = 4\%$
 Test for overall effect: $Z = 3.15$ ($P = 0.002$)

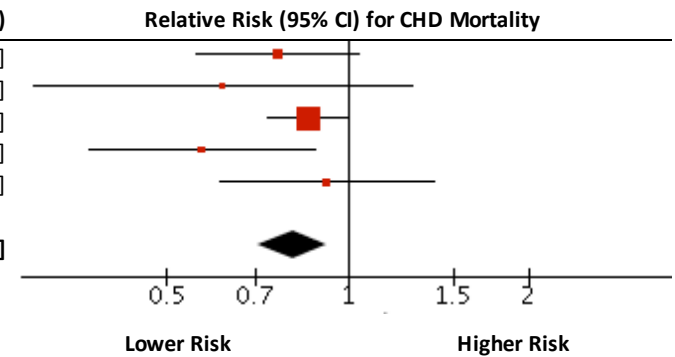
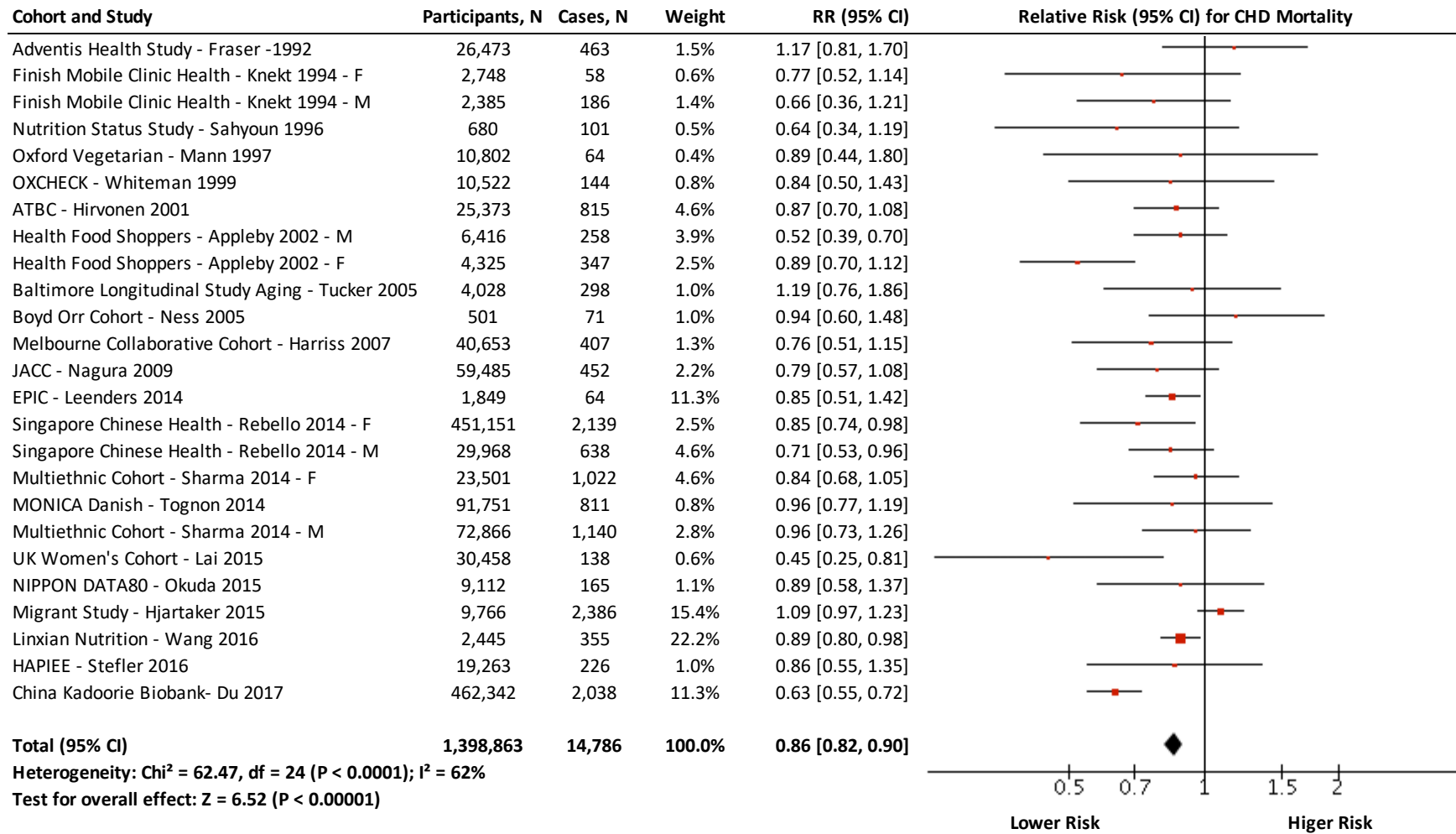


Figure S72. Relation between total fruit and vegetable intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

FRUIT AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

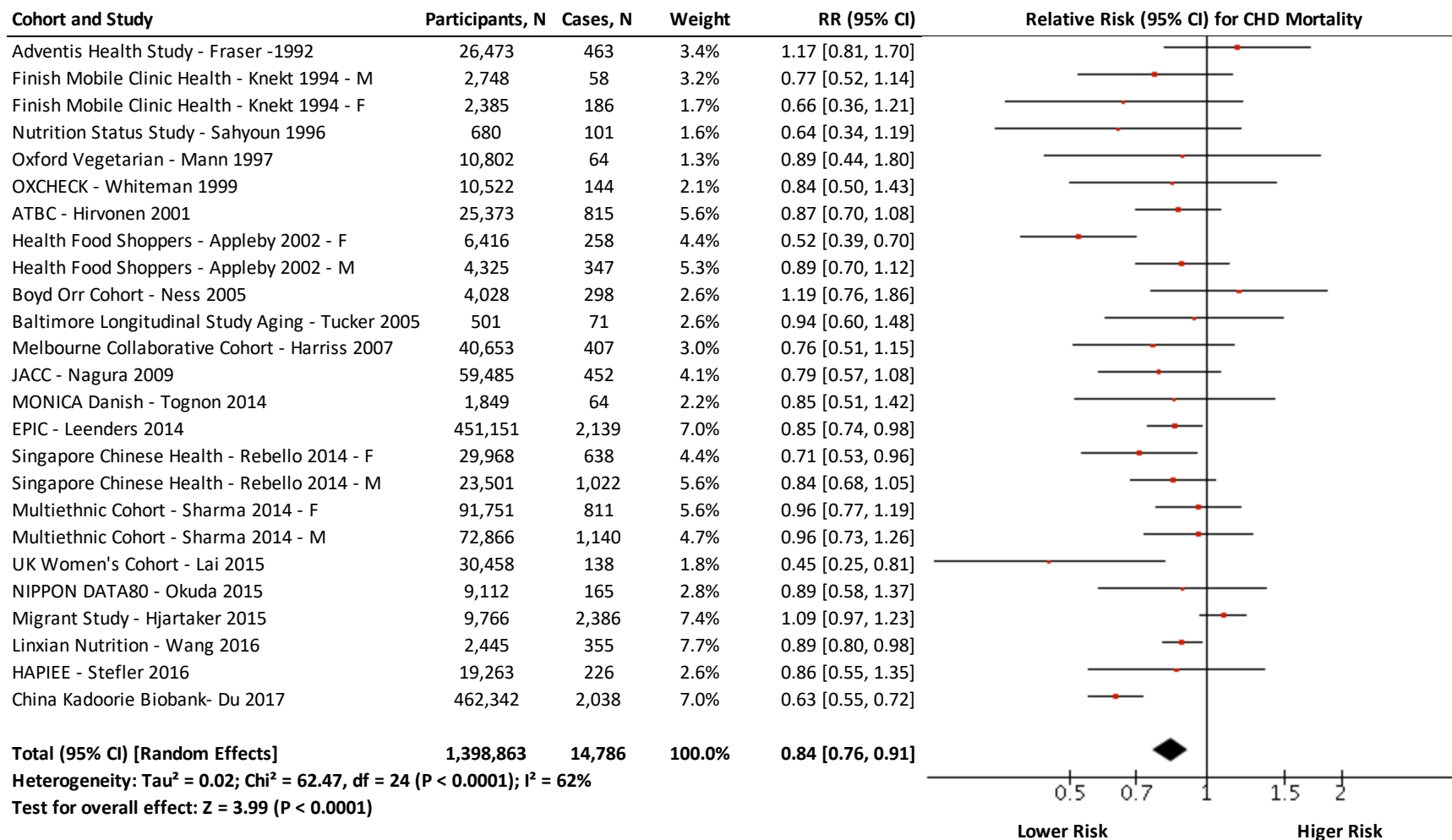
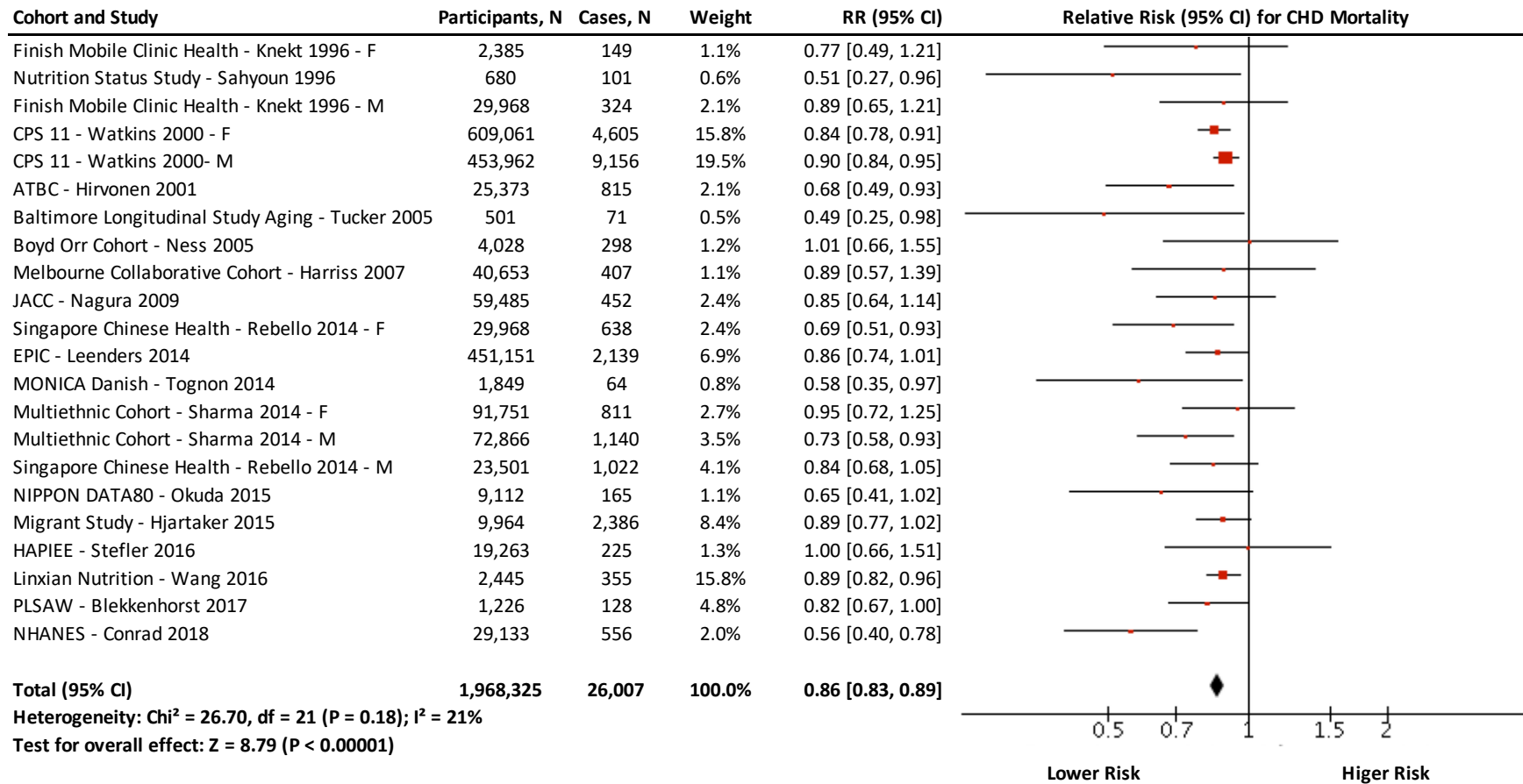


Figure S73. Relation between fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

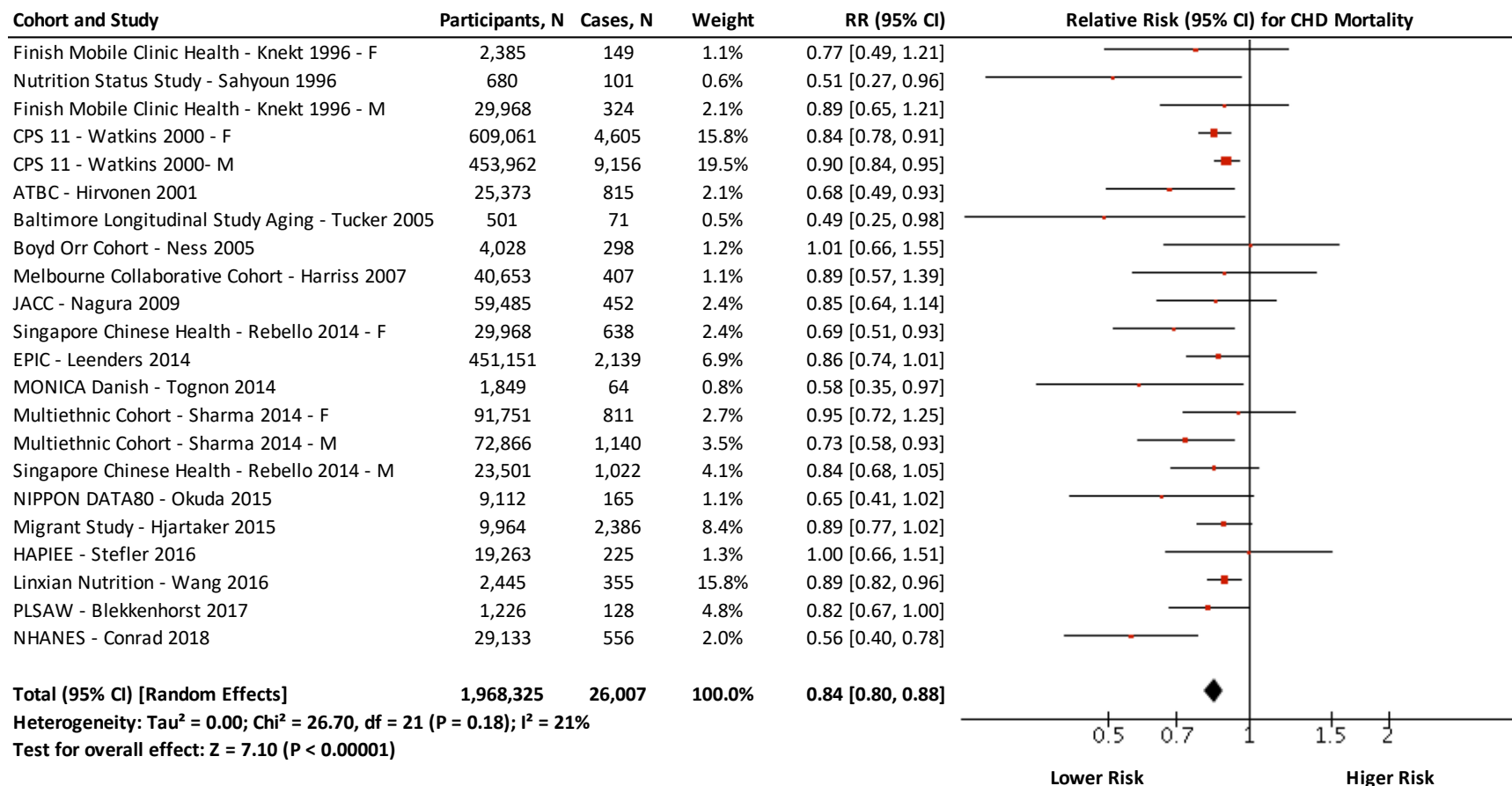


Figure S74. Relation between intake of vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

BANANAS AND CORONARY HEART DISEASE MORTALITY

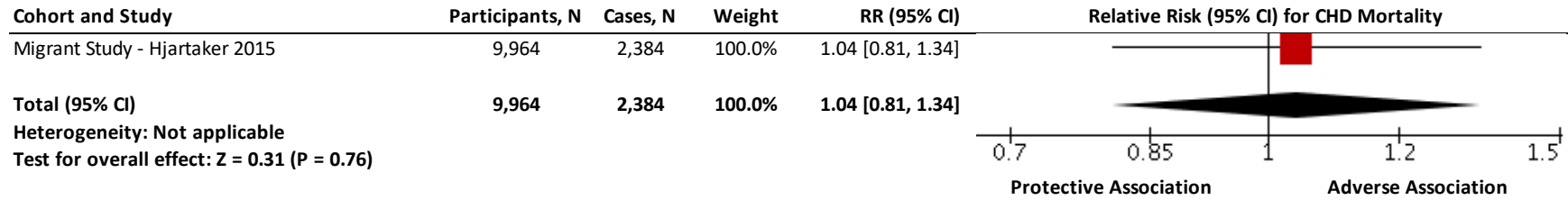
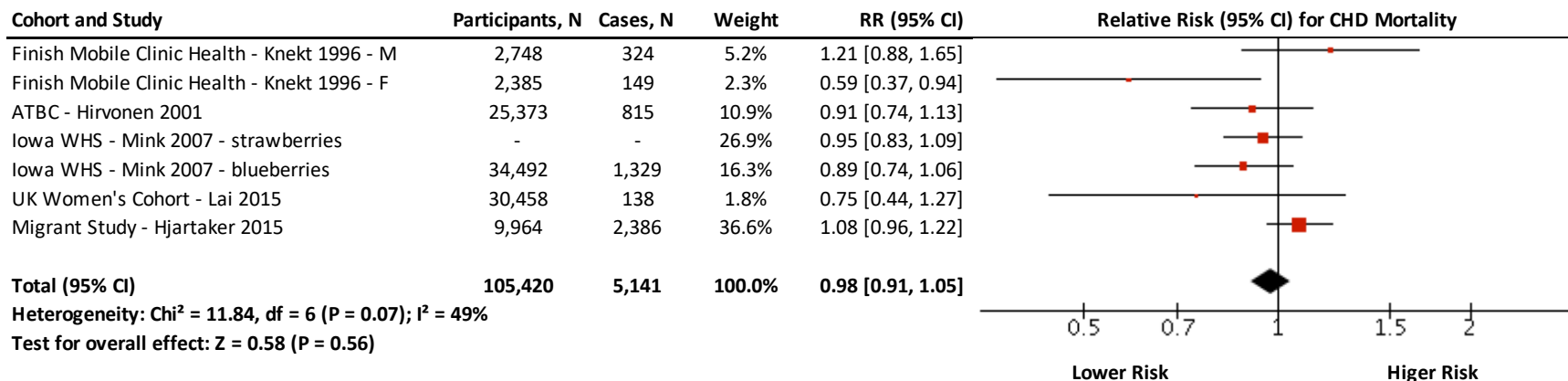


Figure S75. Relation between intake of bananas and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed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

BERRIES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

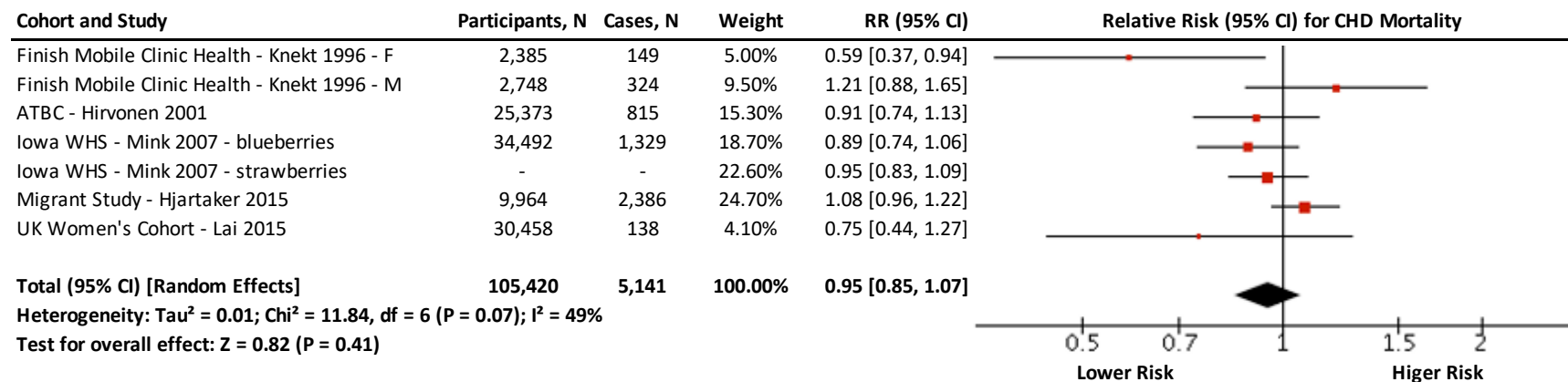
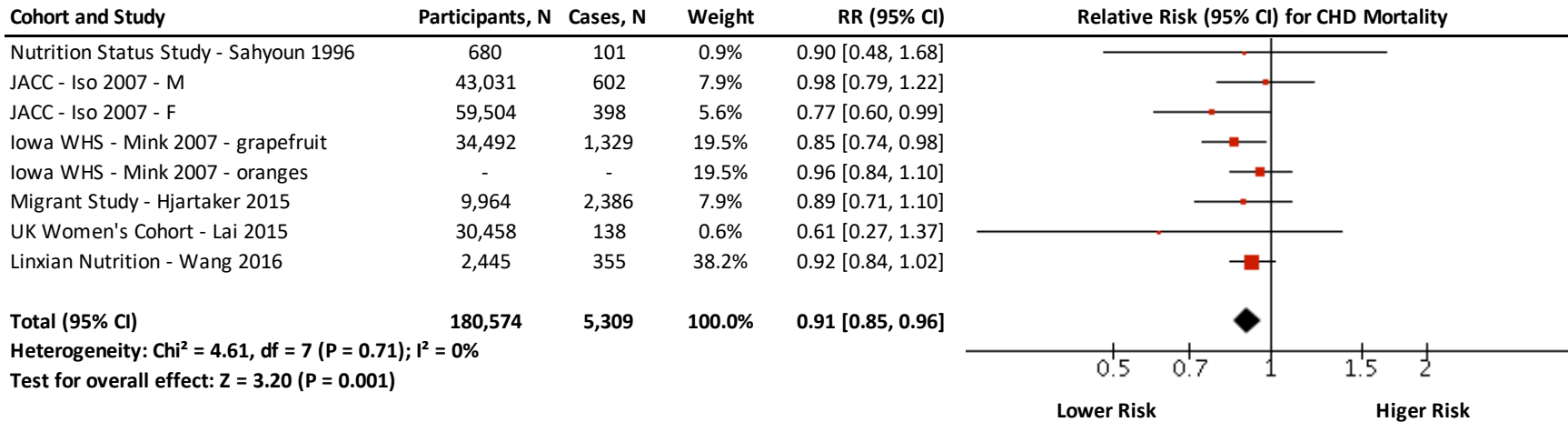


Figure S76. Relation between intake of berries and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CITRUS FRUIT AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

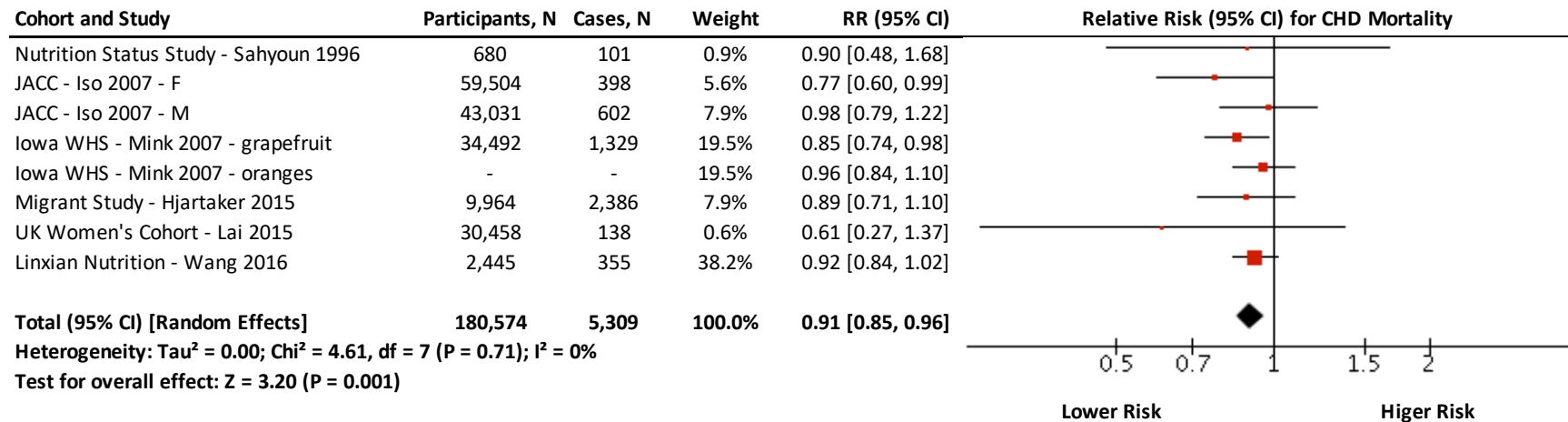


Figure S77. Relation between citrus fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

DRIED FRUIT AND CORONARY HEART DISEASE MORTALITY

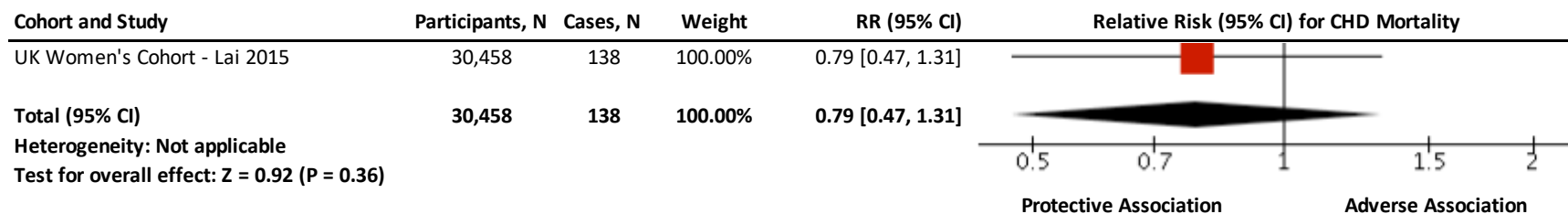
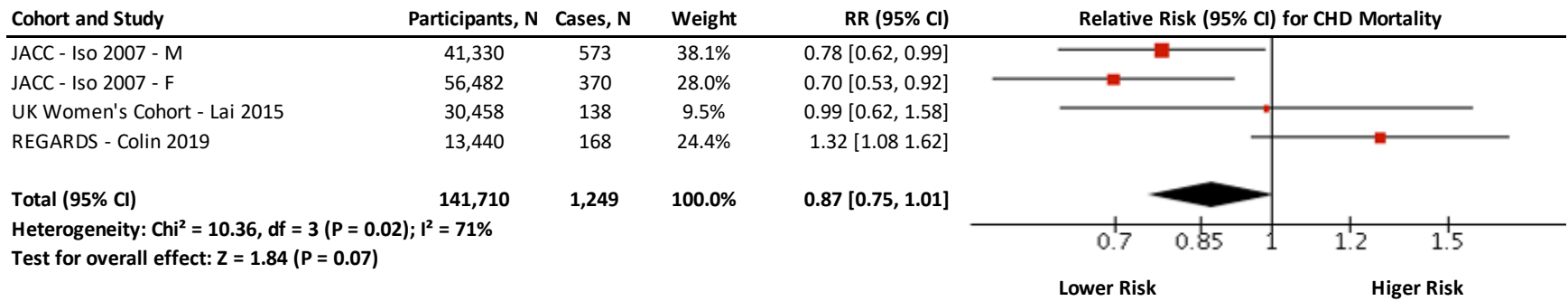


Figure S78. Relation between dried fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

FRUIT JUICE AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

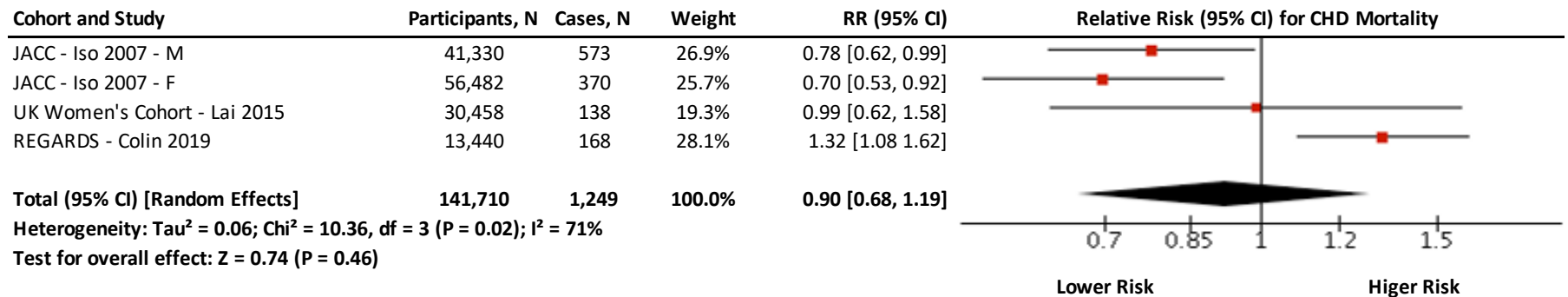


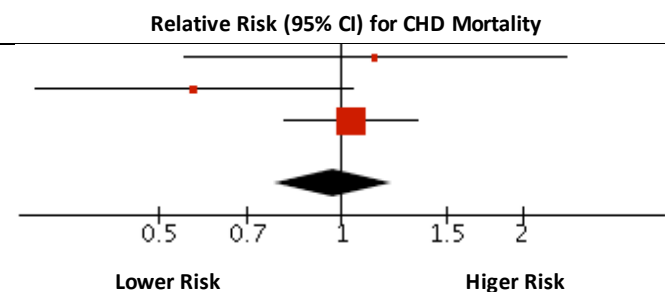
Figure S79. Relation between intake of fruit juice and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B)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

GRAPES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 3.40$, $\text{df} = 2$ ($P = 0.18$); $I^2 = 41\%$
 Test for overall effect: $Z = 0.29$ ($P = 0.77$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.05$; $\text{Chi}^2 = 3.40$, $\text{df} = 2$ ($P = 0.18$); $I^2 = 41\%$
 Test for overall effect: $Z = 0.49$ ($P = 0.63$)

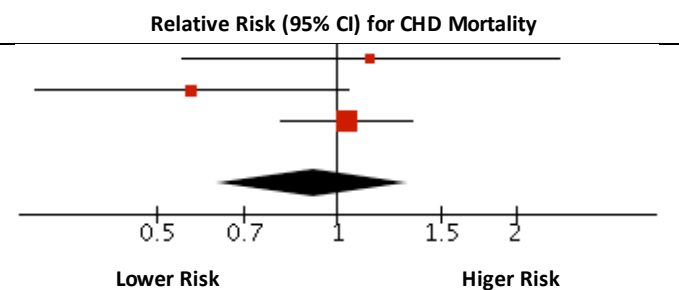


Figure S80. Relation between intake of grapes and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

POMMES AND CORONARY HEART DISEASE MORTALITY

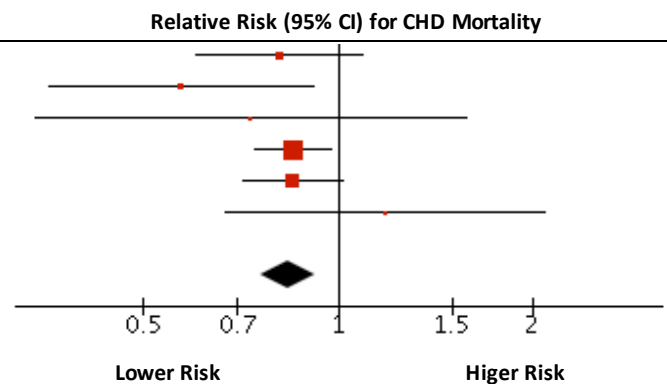
A. Fixed Effects

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

Total (95% CI) 146,407 4,650 100.0% 0.84 [0.76, 0.92]

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

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



B. Random Effects

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

Total (95% CI) [Random Effects] 146,407 4,650 100.0% 0.84 [0.76, 0.92]

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

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

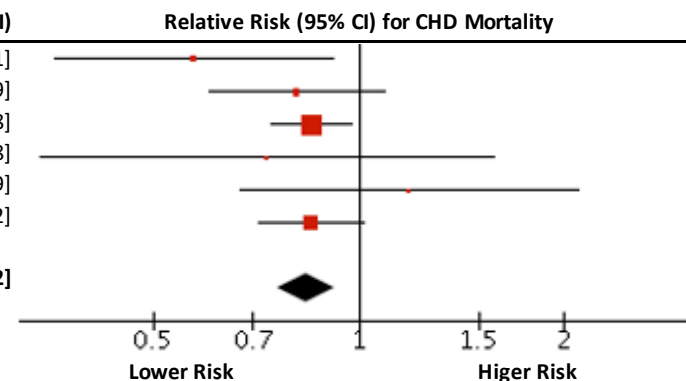
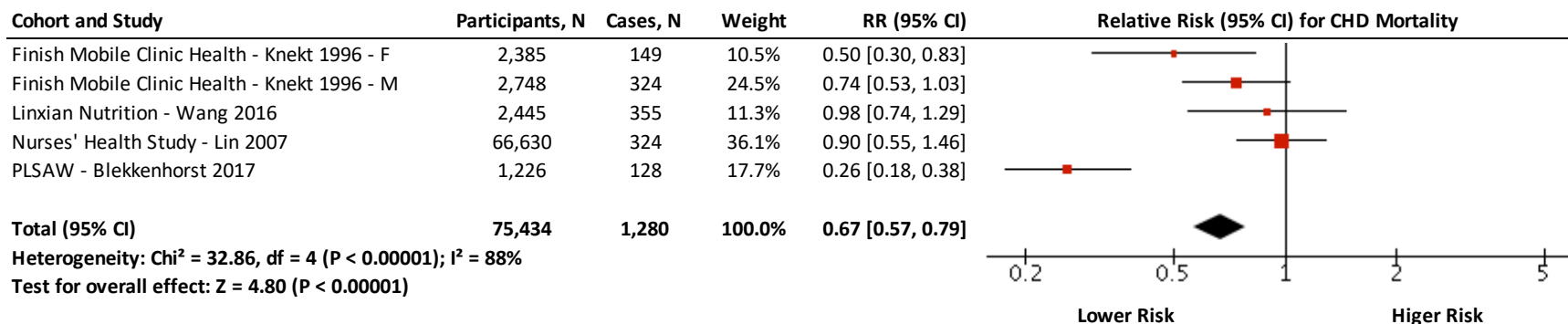


Figure S81. Relation between pommes fruit intake and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

ALLIUM VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

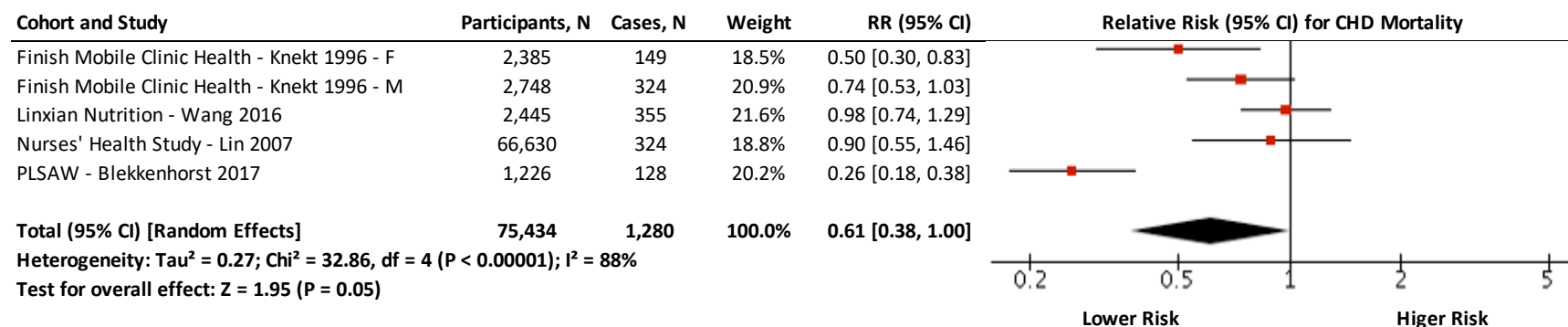
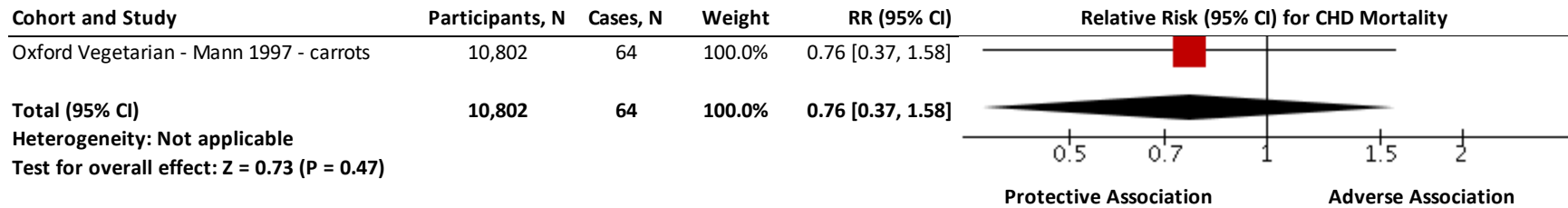


Figure S82. Relation between intake of allium vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

CARROTS AND CORONARY HEART DISEASE MORTALITY



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

CELERY AND CORONARY HEART DISEASE MORTALITY

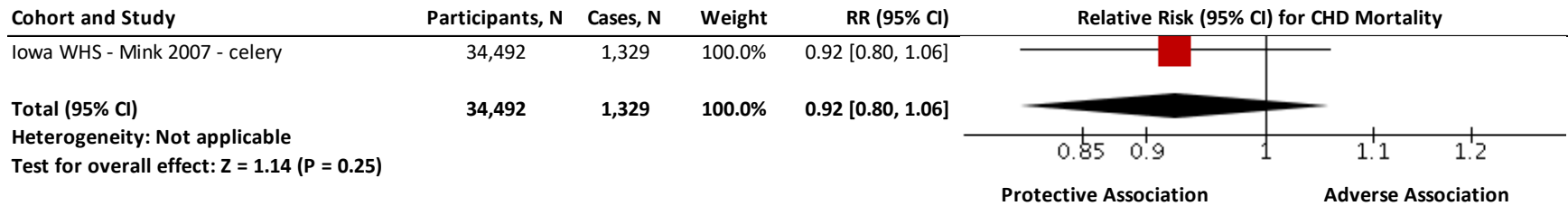
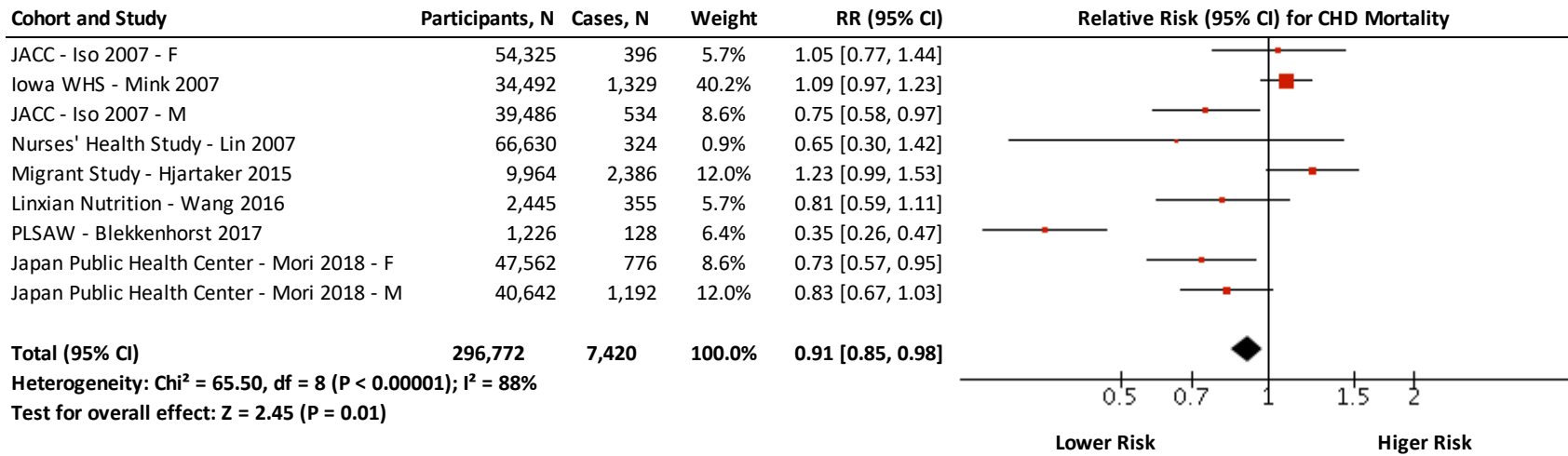


Figure S84. Relation between intake of celery and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed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

CRUCIFEROUS VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

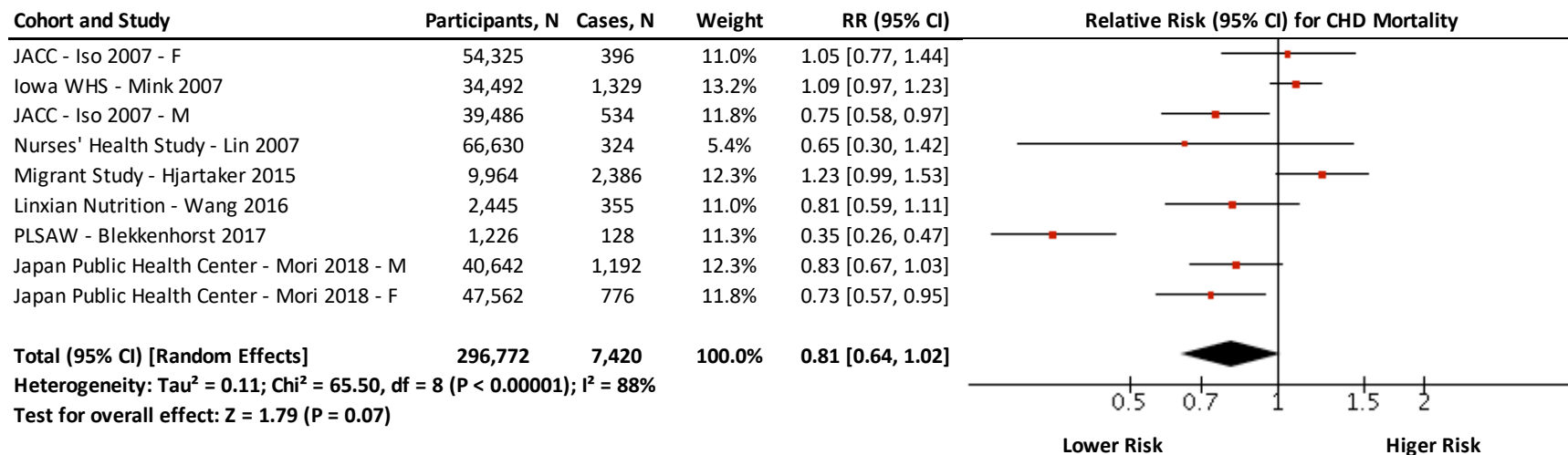
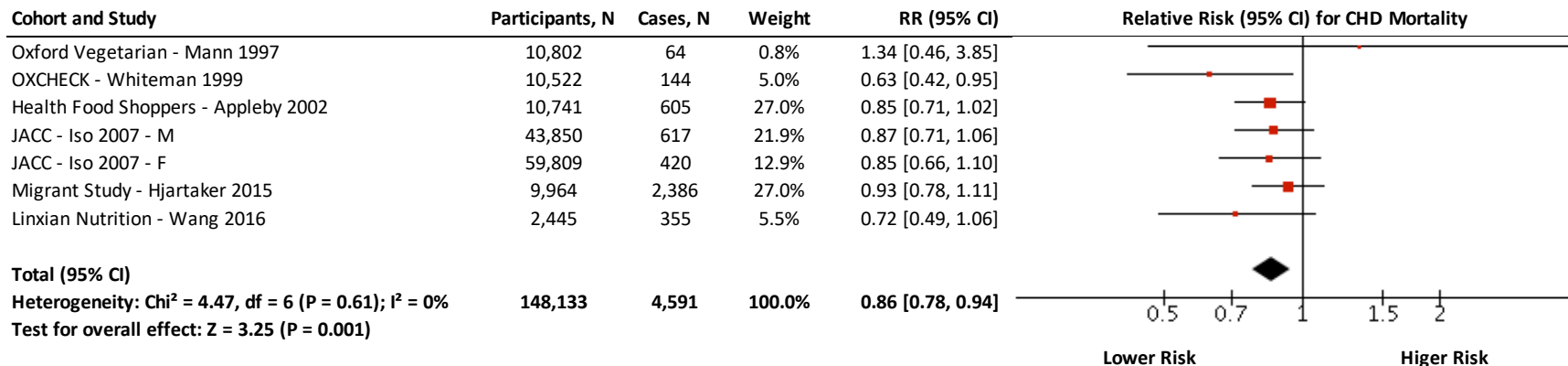


Figure S85. Relation between intake of cruciferous vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

GREEN LEAFY VEGETABLES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects



B. Random Effects

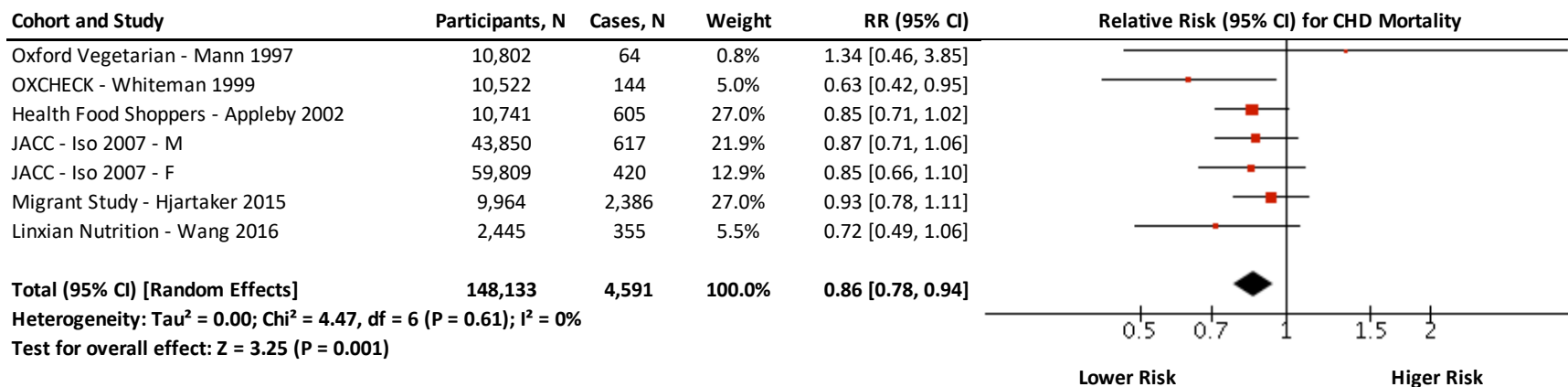
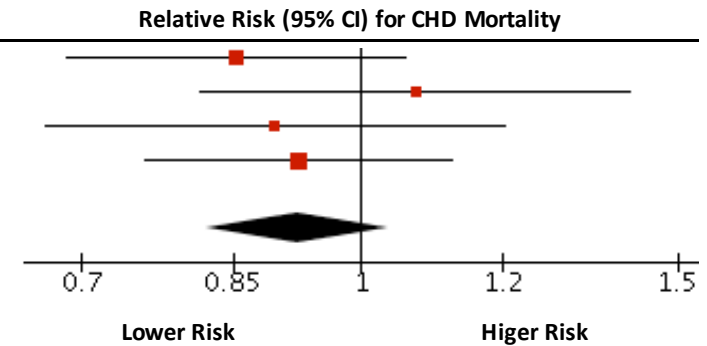


Figure S86. Relation between intake of green leafy vegetables and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects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

TOMATOES AND CORONARY HEART DISEASE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - tomatoes - M	41,547	568	29.7%	0.85 [0.69, 1.06]
JACC - Iso 2007 - tomatoes - F	56,947	379	18.3%	1.07 [0.82, 1.41]
Nurses' Health Study - Lin 2007	66,630	324	16.0%	0.90 [0.67, 1.20]
Migrant Study - Hjartaker 2015	9,964	2,386	36.0%	0.92 [0.76, 1.12]
Total (95% CI)	175,088	3,657	100.0%	0.92 [0.82, 1.04]
Heterogeneity: Chi² = 1.72, df = 3 (P = 0.63); I² = 0%				
Test for overall effect: Z = 1.35 (P = 0.18)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
JACC - Iso 2007 - males - tomatoes	41,547	568	29.7%	0.85 [0.69, 1.06]
Nurses' Health Study - Lin 2007	66,630	324	16.0%	0.90 [0.67, 1.20]
JACC - Iso 2007 - tomatoes - F	56,947	379	18.3%	1.07 [0.82, 1.41]
Migrant Study - Hjartaker 2015	9,964	2,386	36.0%	0.92 [0.76, 1.12]
Total (95% CI) [Random Effects]	175,088	3,657	100.0%	0.92 [0.82, 1.04]
Heterogeneity: Tau² = 0.00; Chi² = 1.72, df = 3 (P = 0.63); I² = 0%				
Test for overall effect: Z = 1.35 (P = 0.18)				

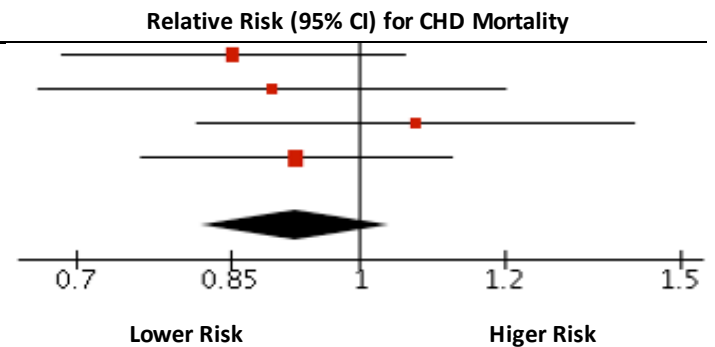
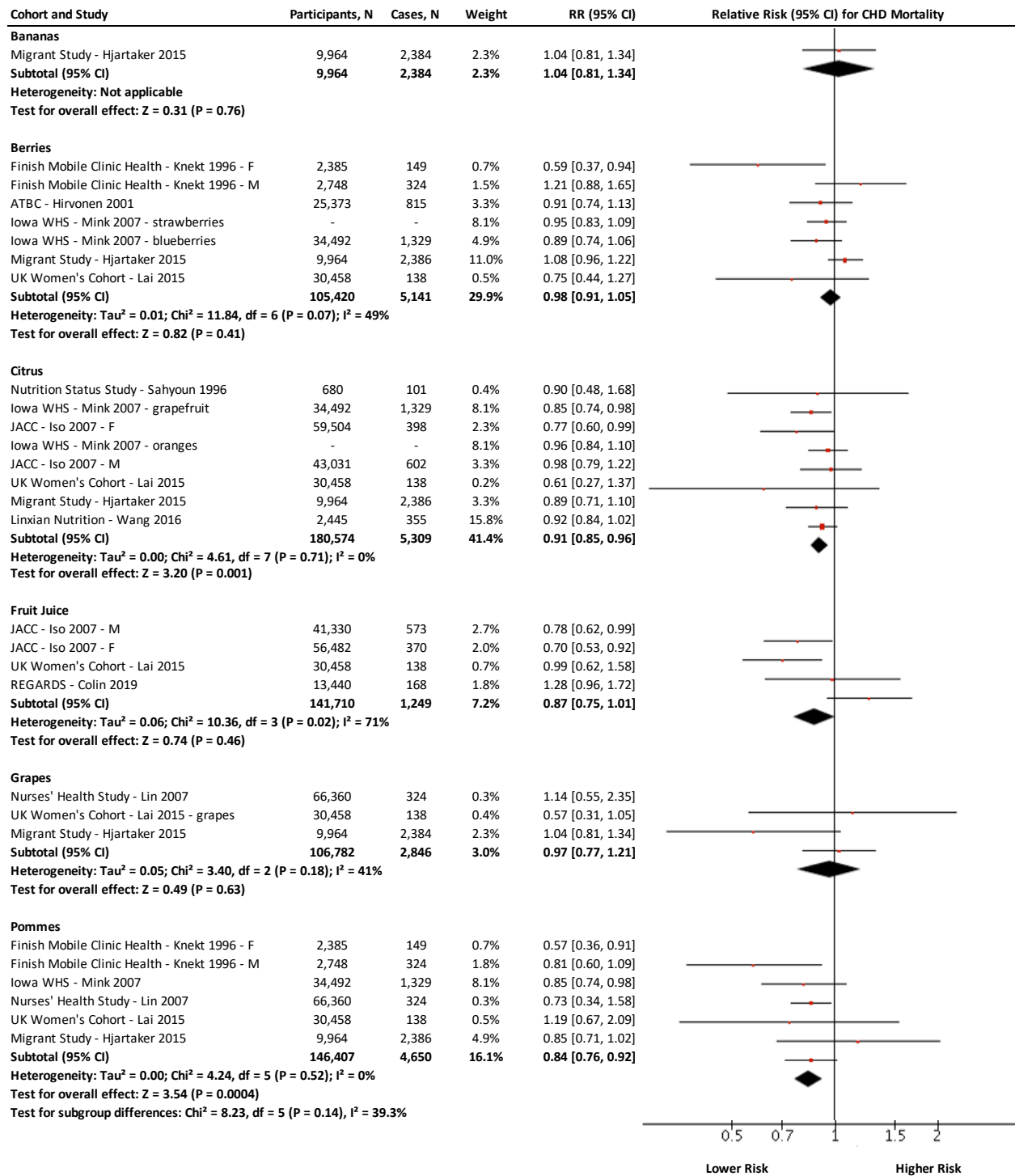


Figure S87. Relation between intake of tomatoes and coronary heart disease mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

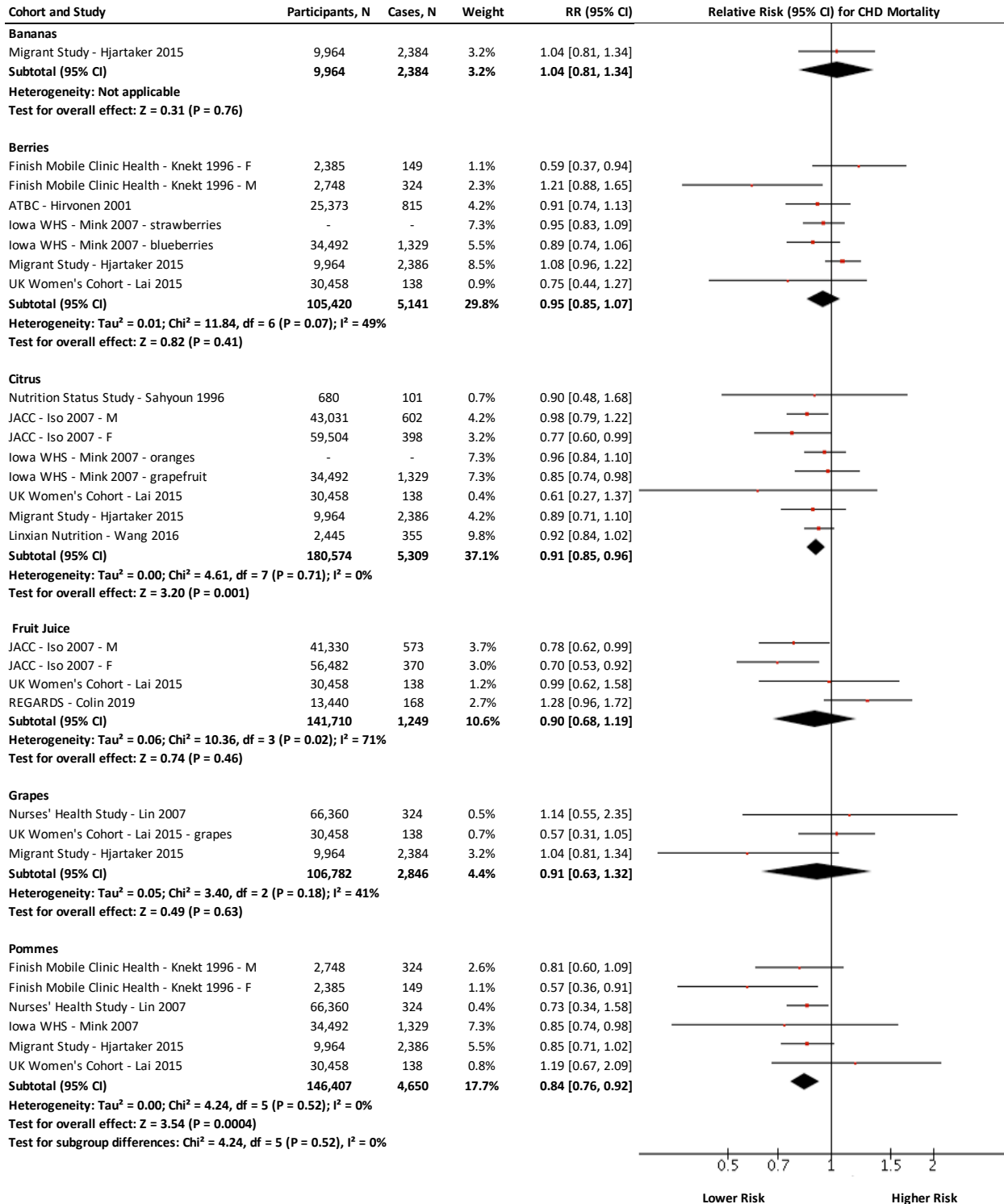
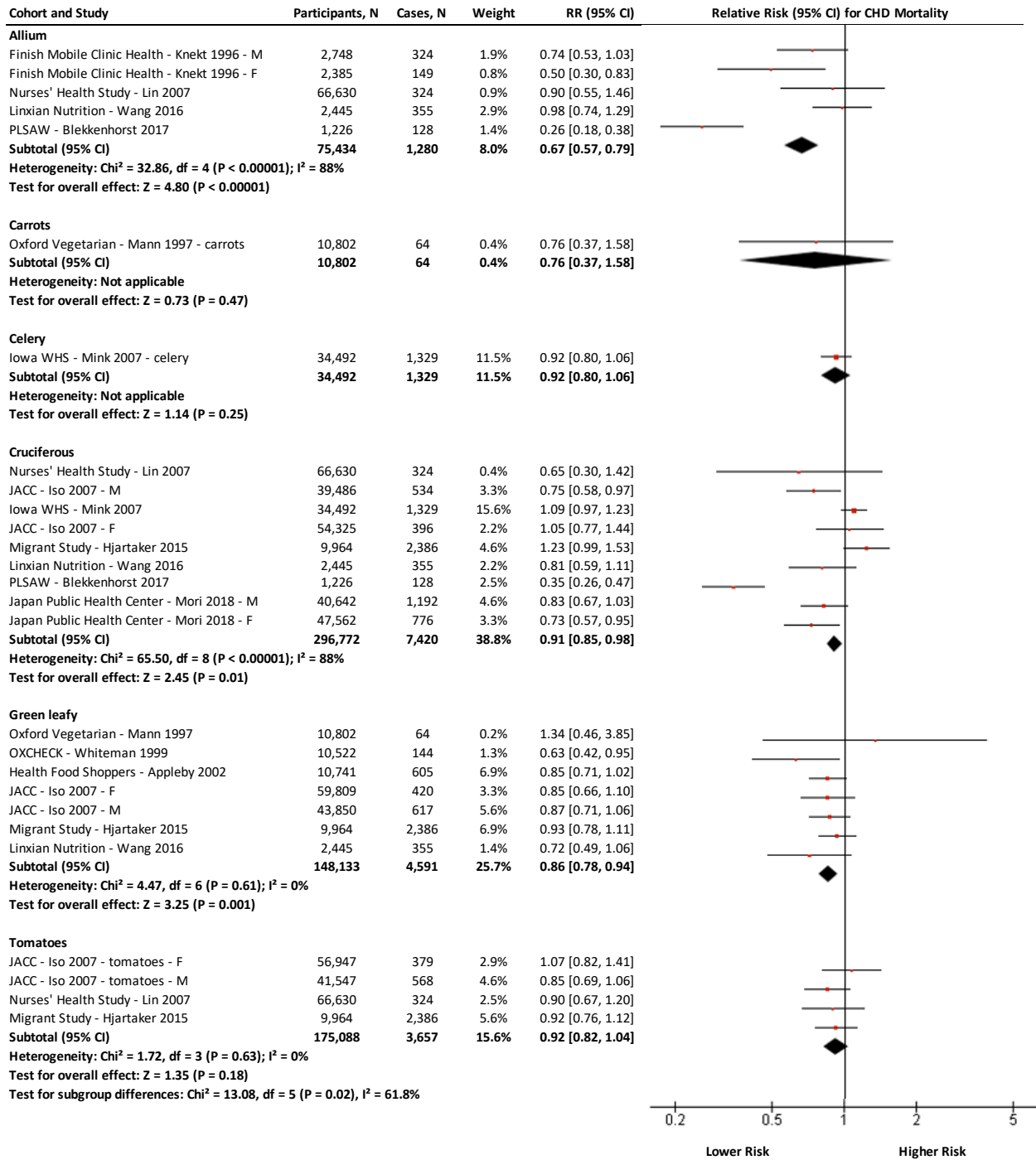


Figure S88. Relation between sources of fruit and CHD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

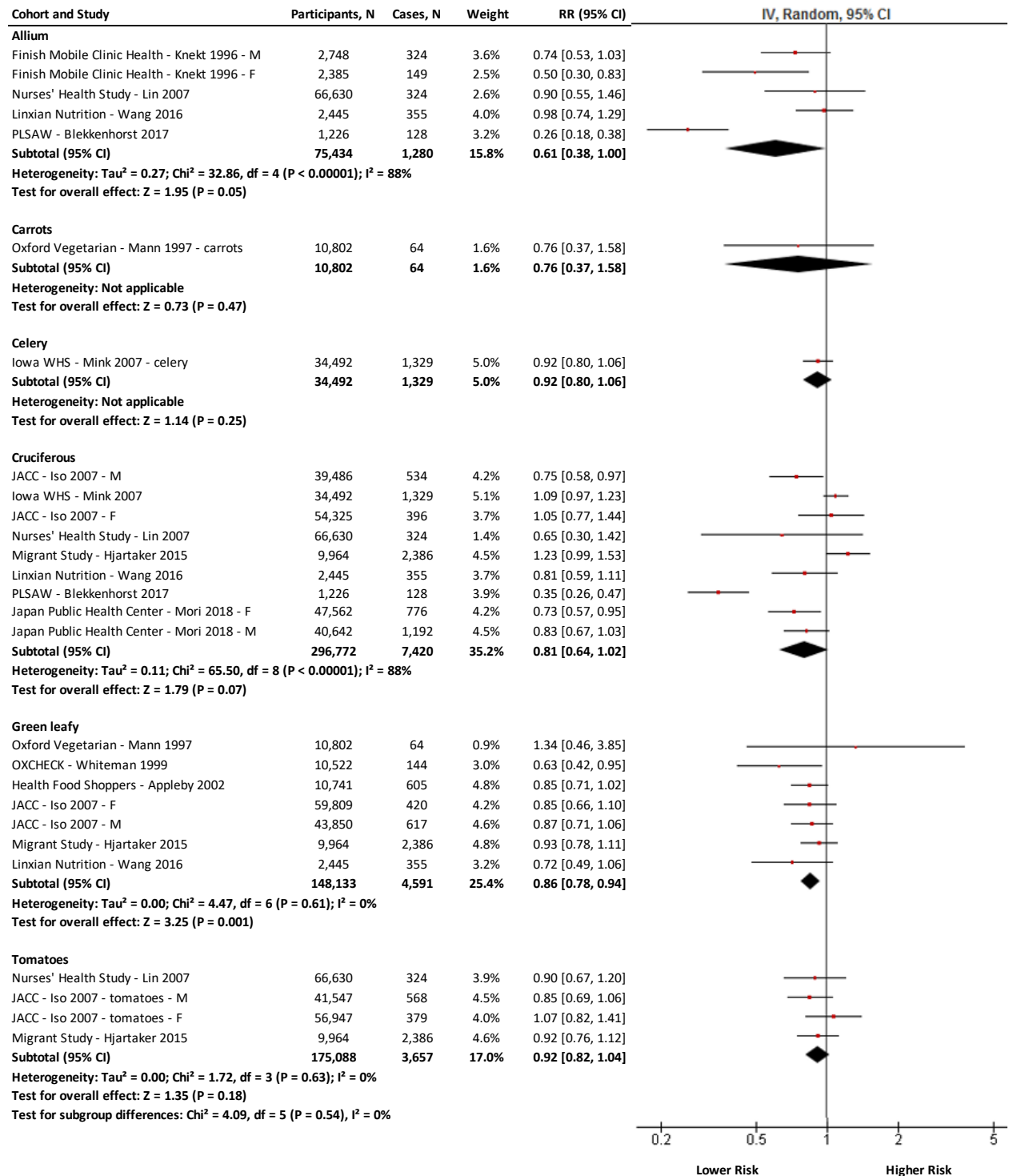


Figure S89. Relation between sources of vegetables and CHD mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

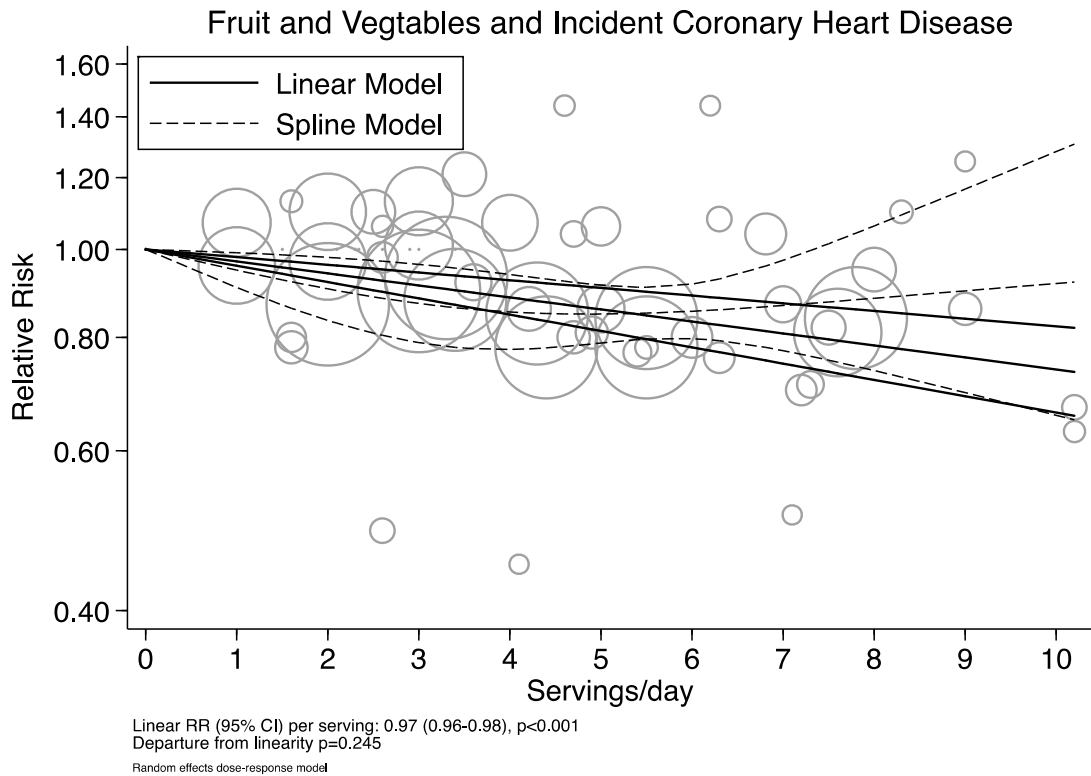


Figure S90. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

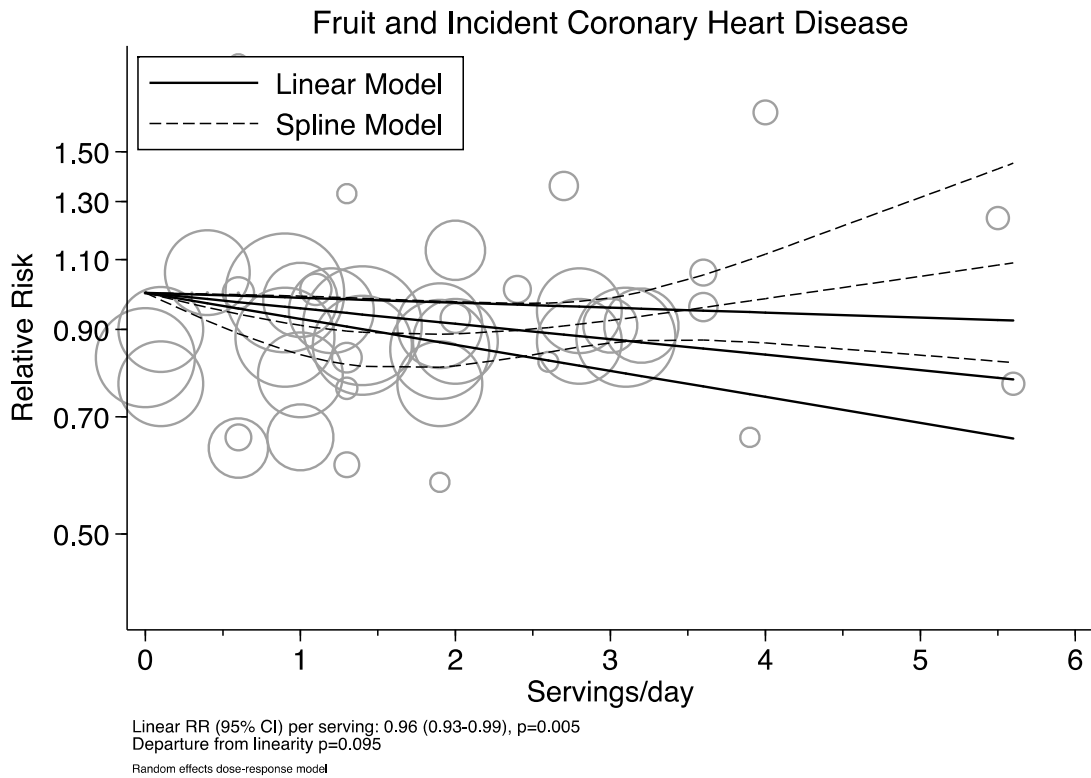


Figure S91. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

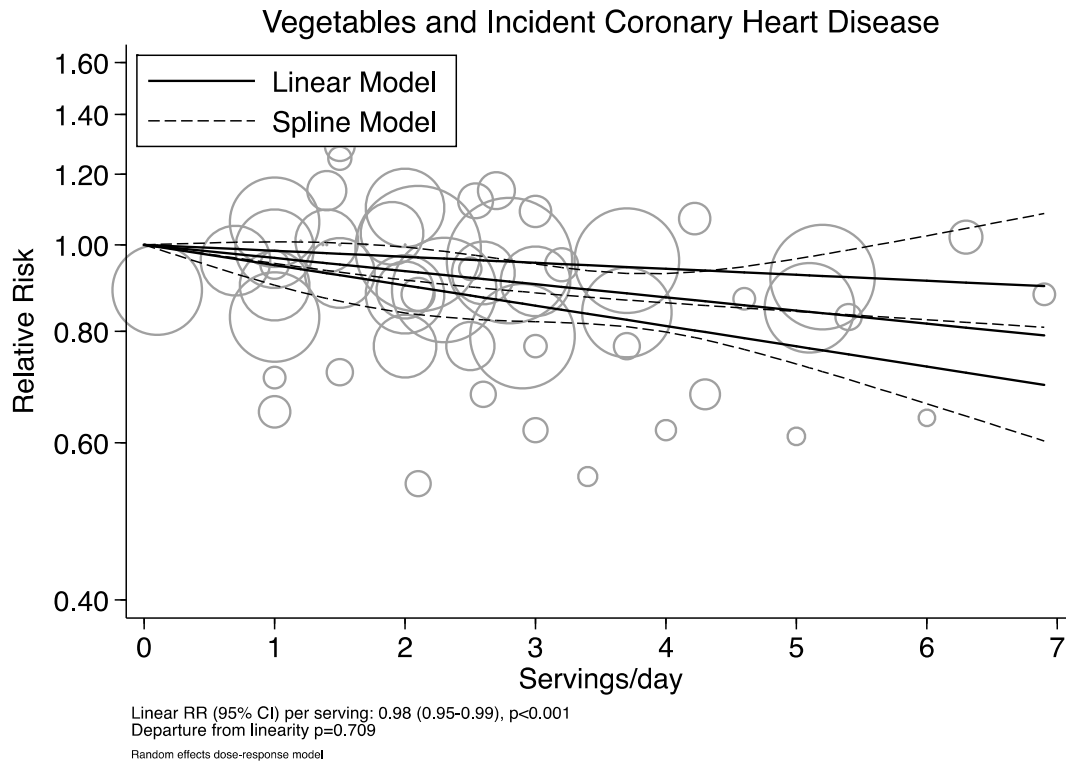


Figure S92. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

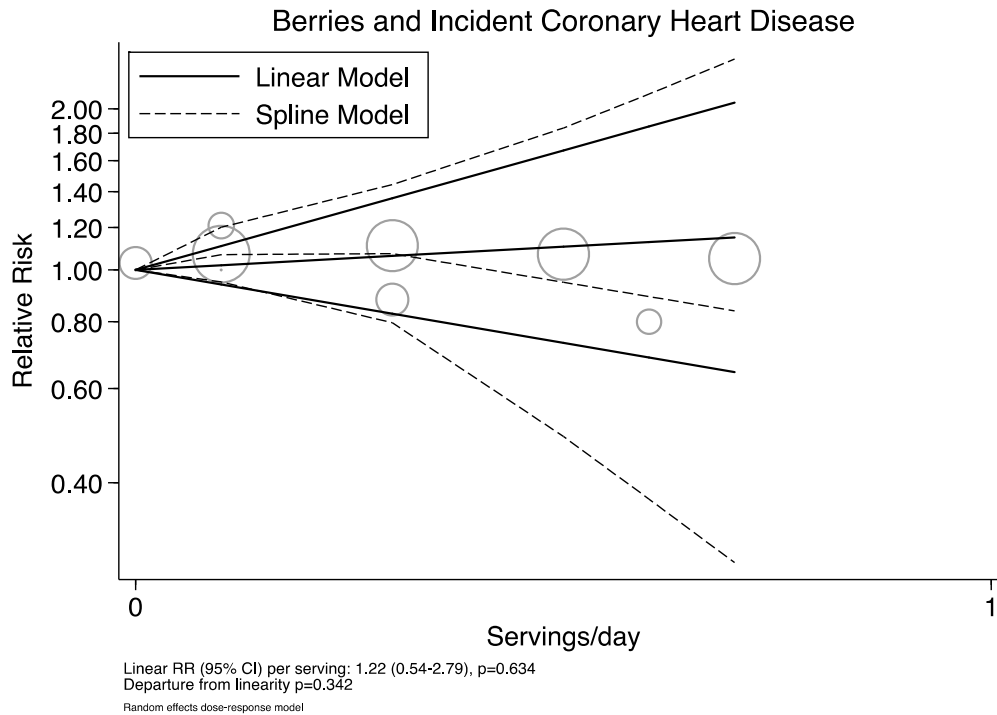


Figure S93. Linear and cubic-spline dose-response relation between increasing berries intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

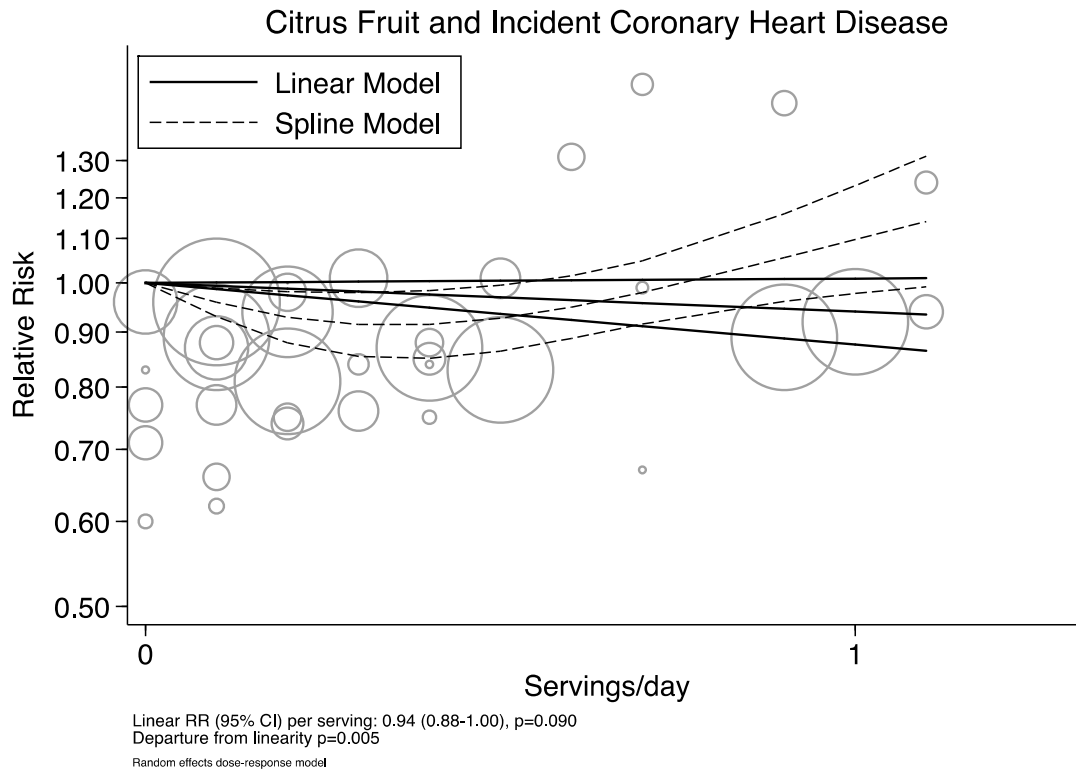


Figure S94. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

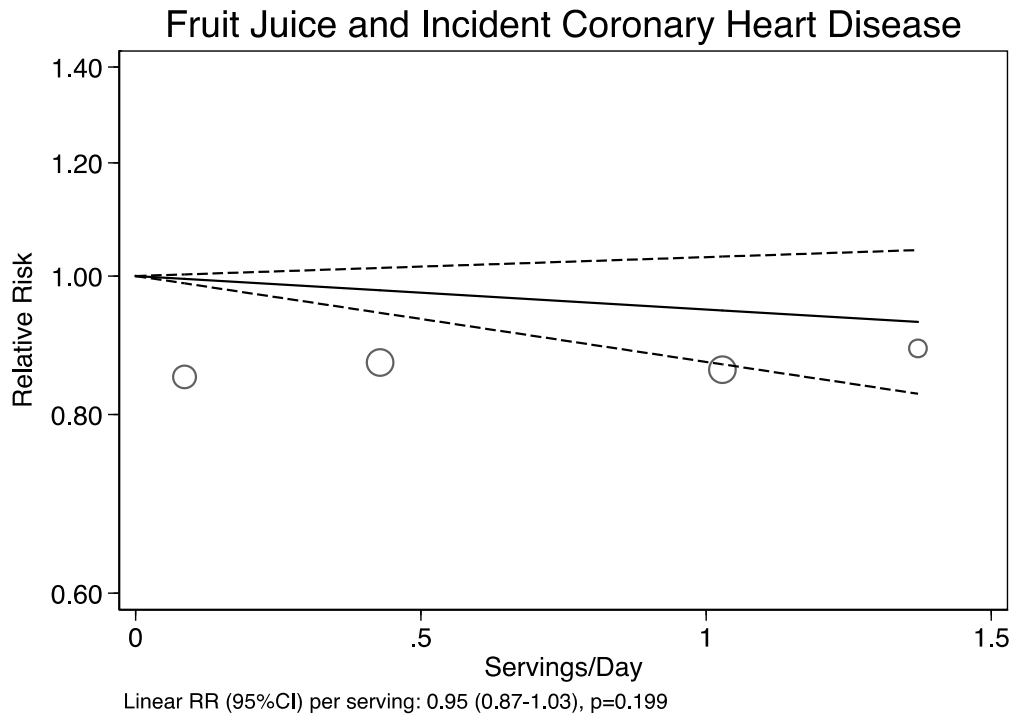


Figure S95. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

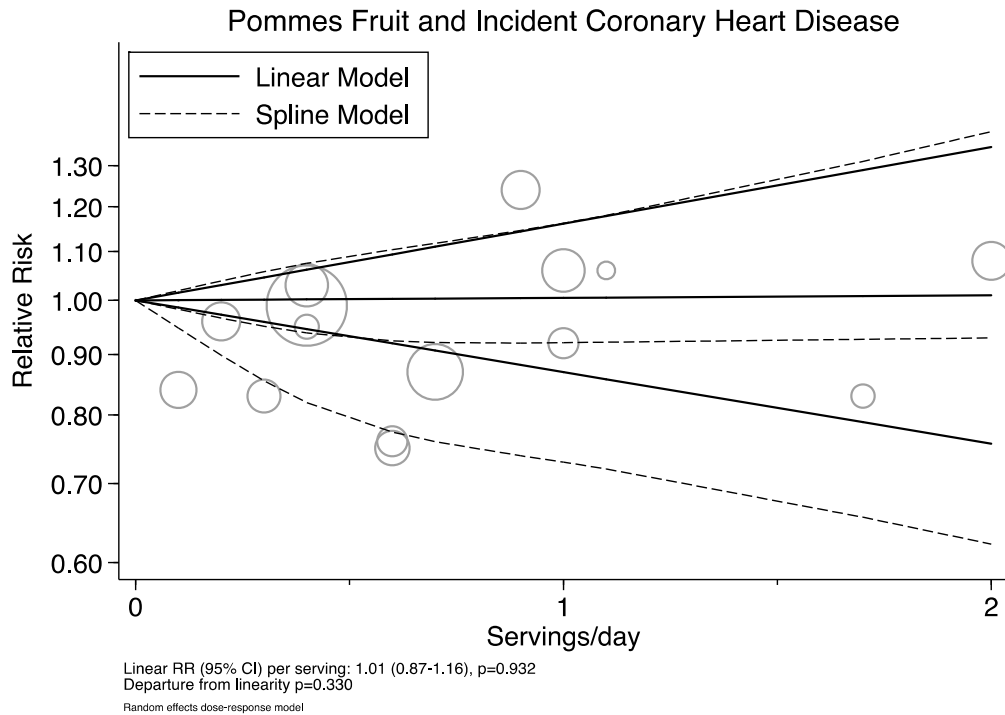


Figure S96. Linear and cubic-spline dose-response relation between increasing pommes intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

Watermelon and Incident Coronary Heart Disease

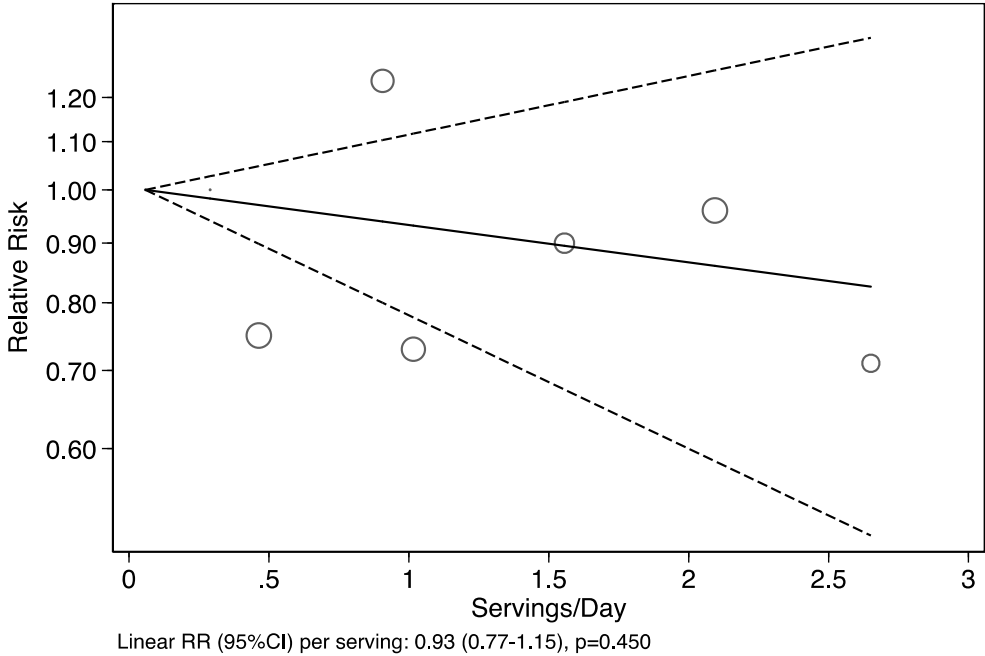


Figure S97. Linear dose-response relation between increasing watermelon intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

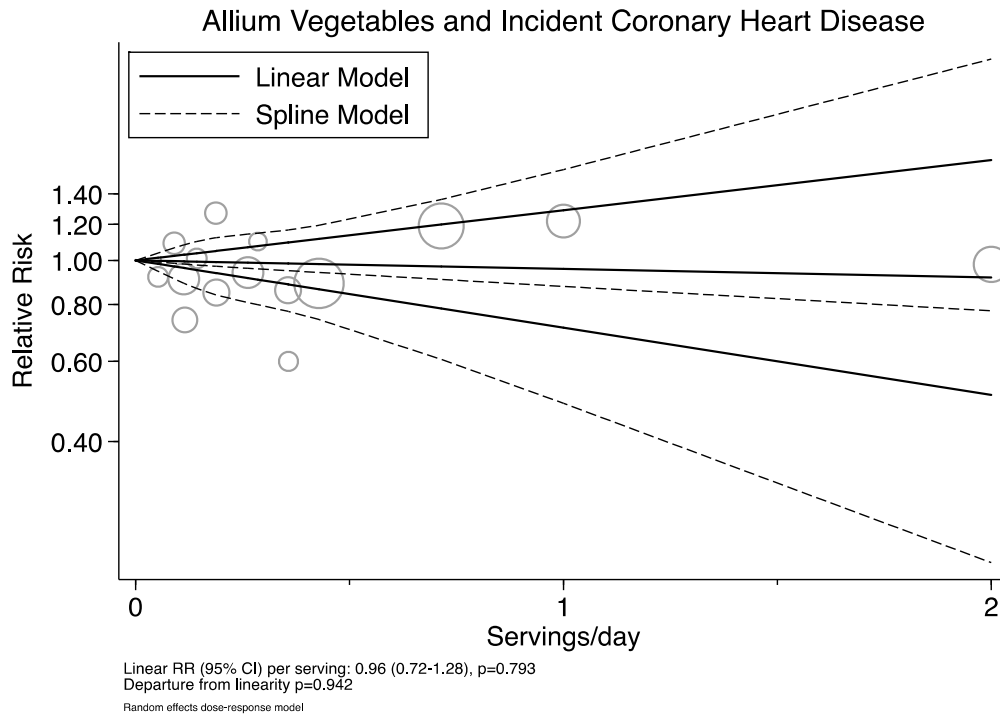


Figure S98. Linear and cubic-spline dose-response relation between increasing intake of allium vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

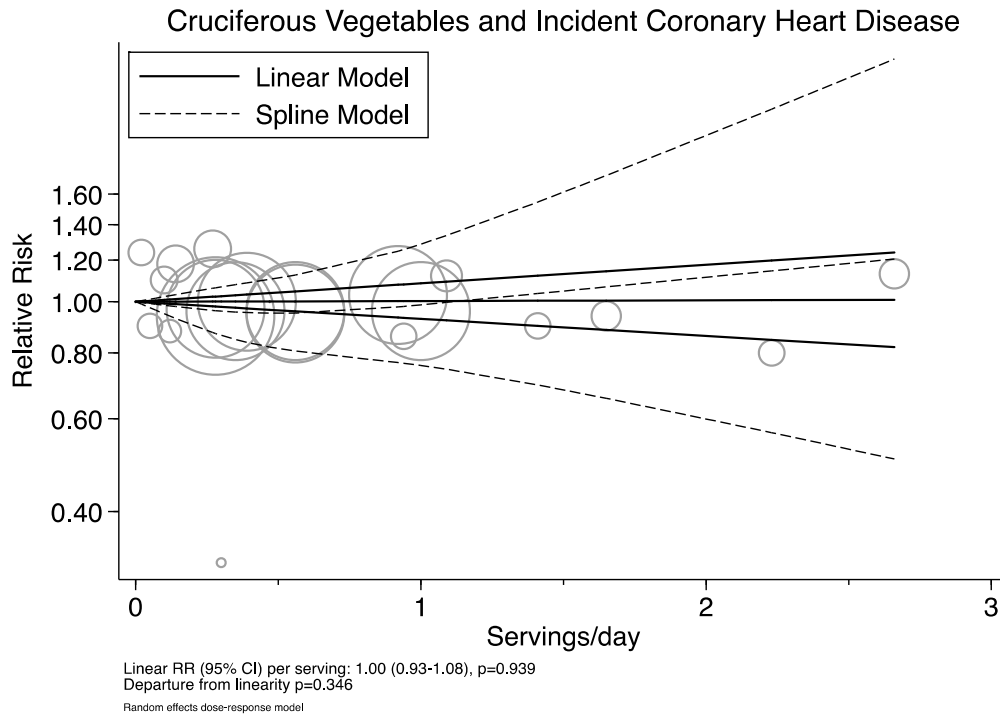


Figure S99. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

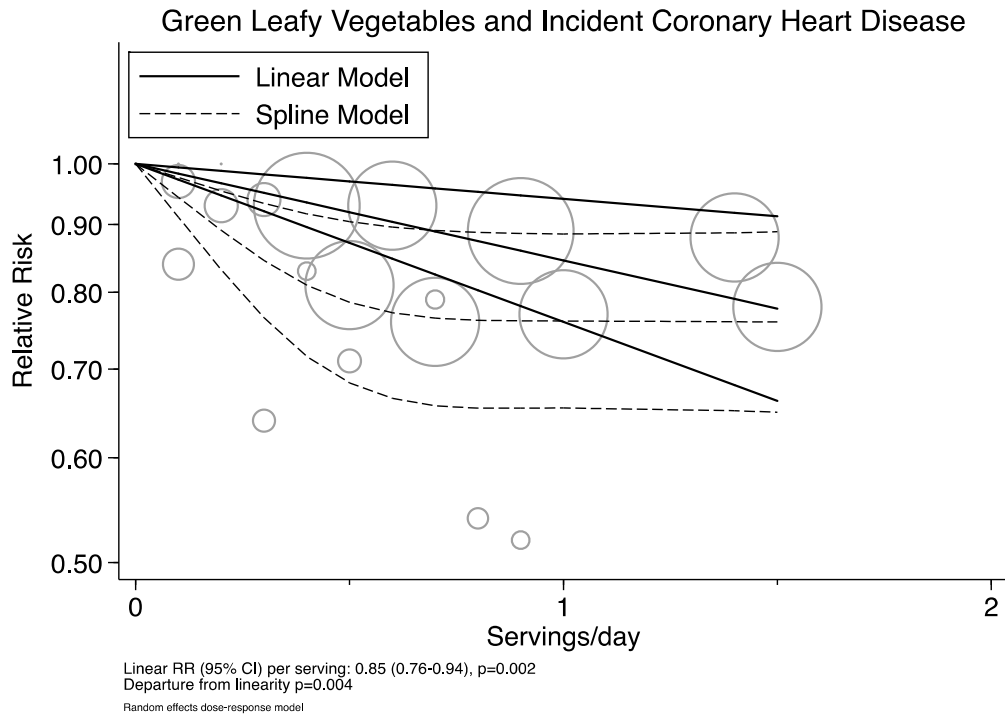


Figure S100. Linear and cubic-spline dose-response relation between increasing intake of green leafy vegetables and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

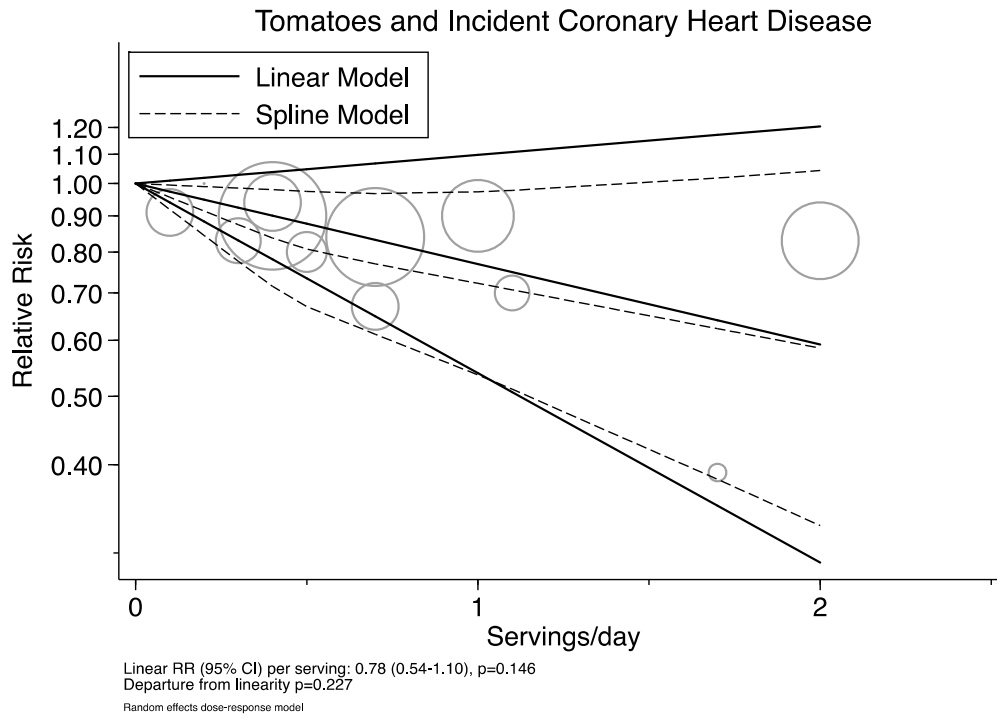


Figure S101. Linear and cubic-spline dose-response relation between increasing tomato intake and incidence of coronary heart disease. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

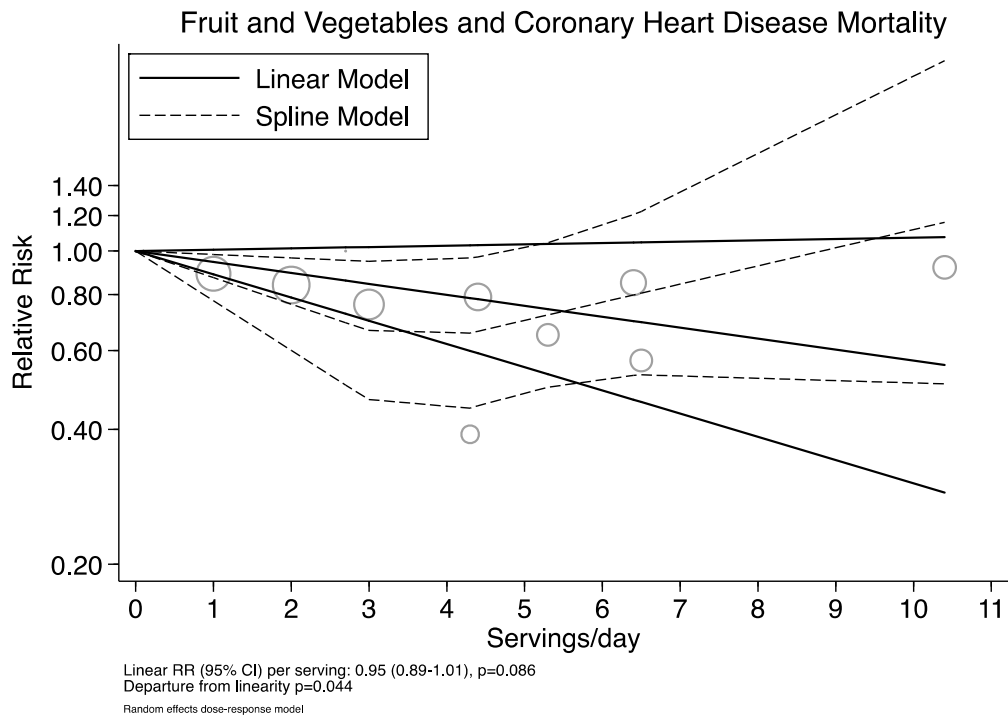


Figure S102. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

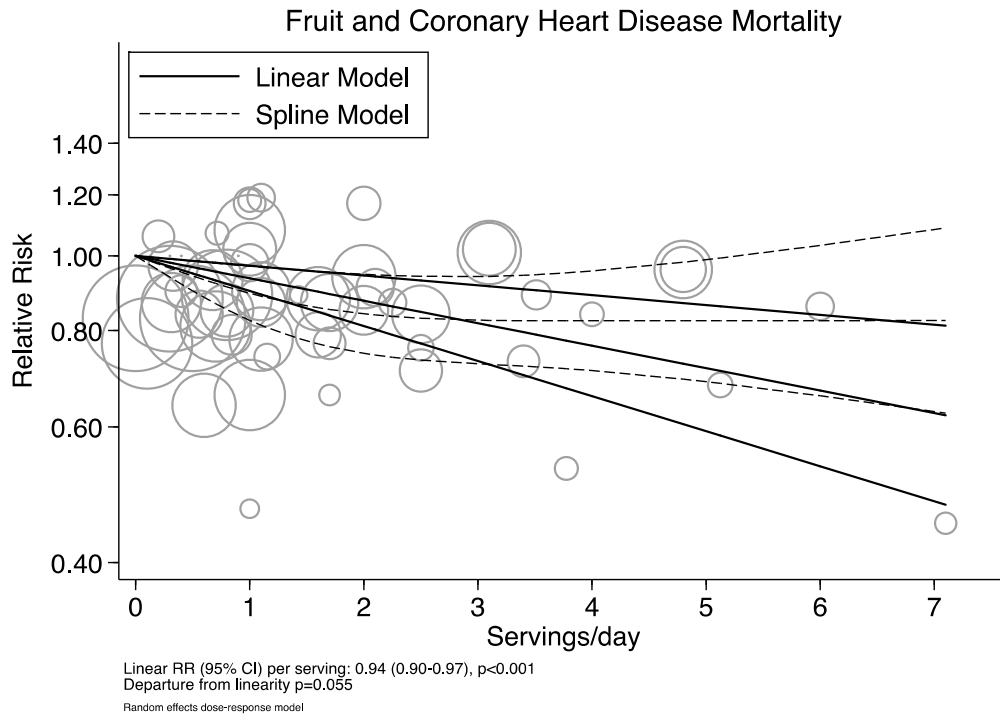


Figure S103. Linear and cubic-spline dose-response relation between increasing fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

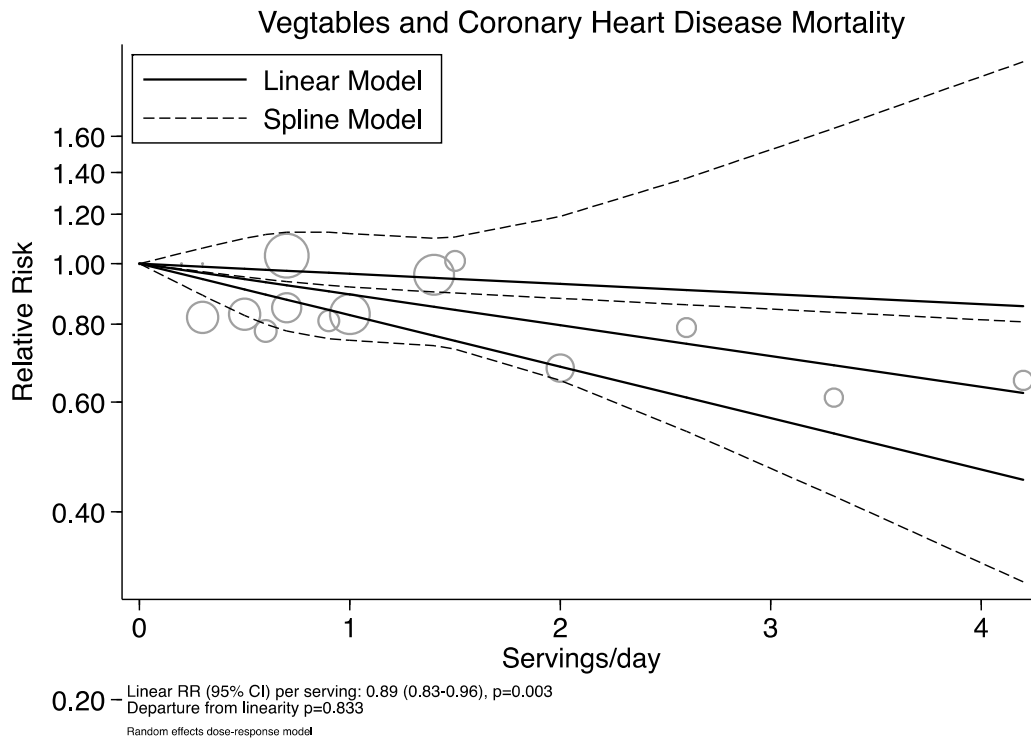


Figure S104. Linear and cubic-spline dose-response relation between increasing intake of vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

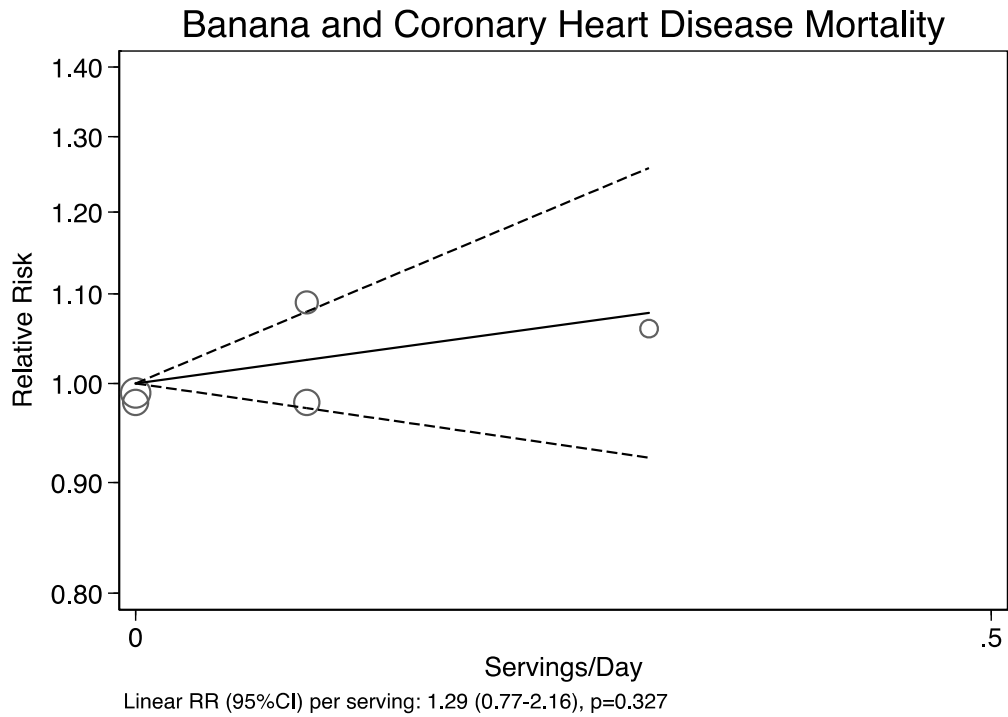


Figure S105. Linear dose-response relation between increasing banana intake and cardiovascular disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

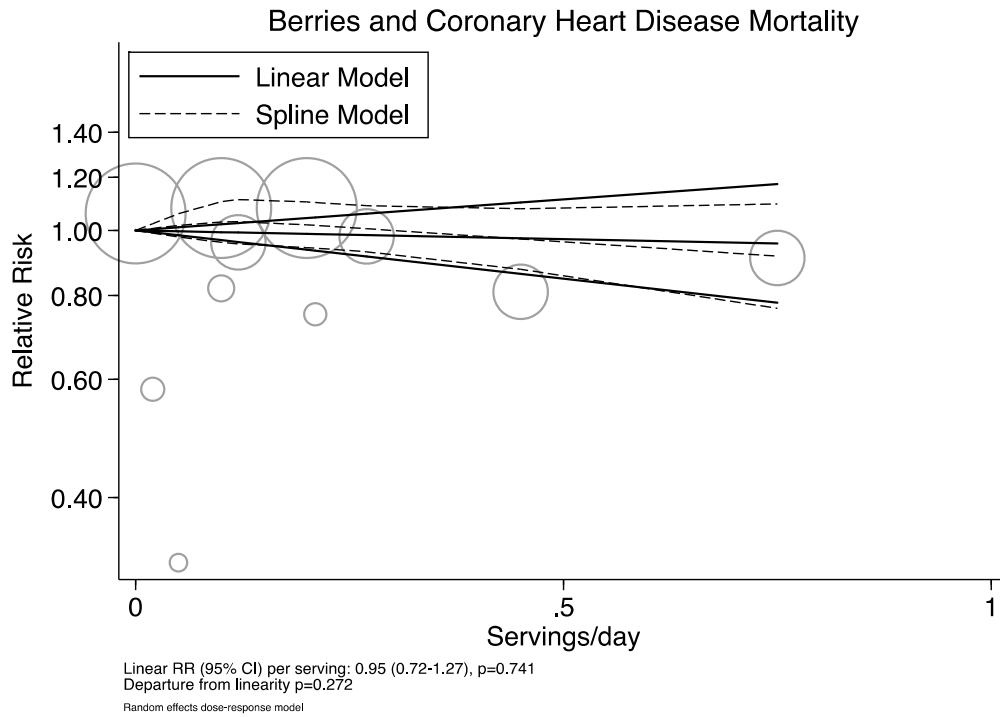


Figure S106. Linear dose-response relation between increasing berries intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

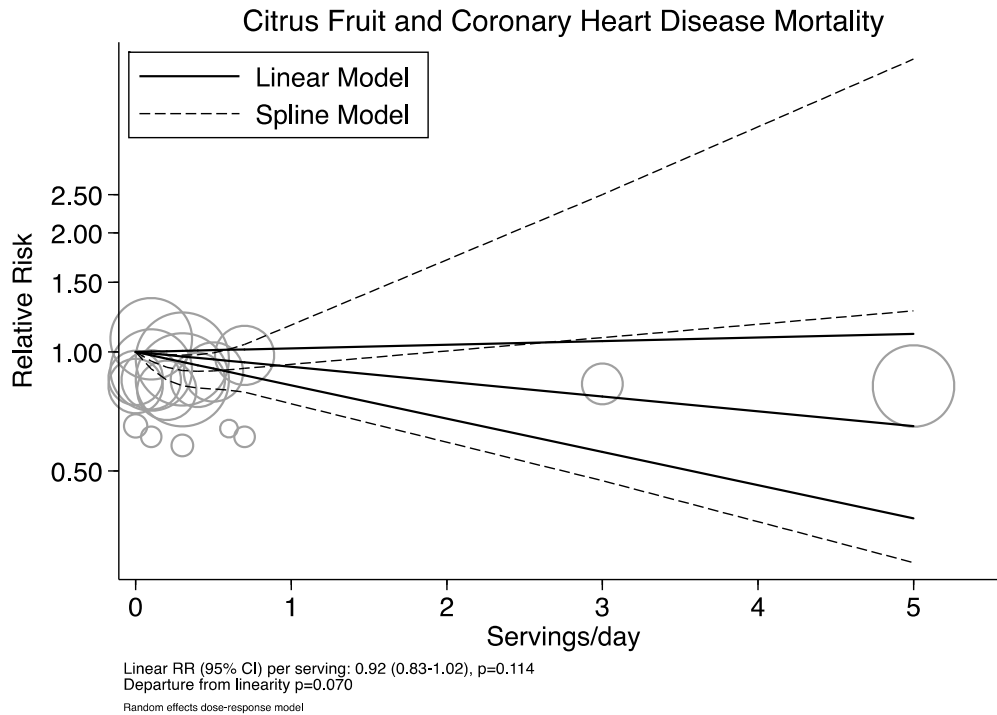


Figure S107. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

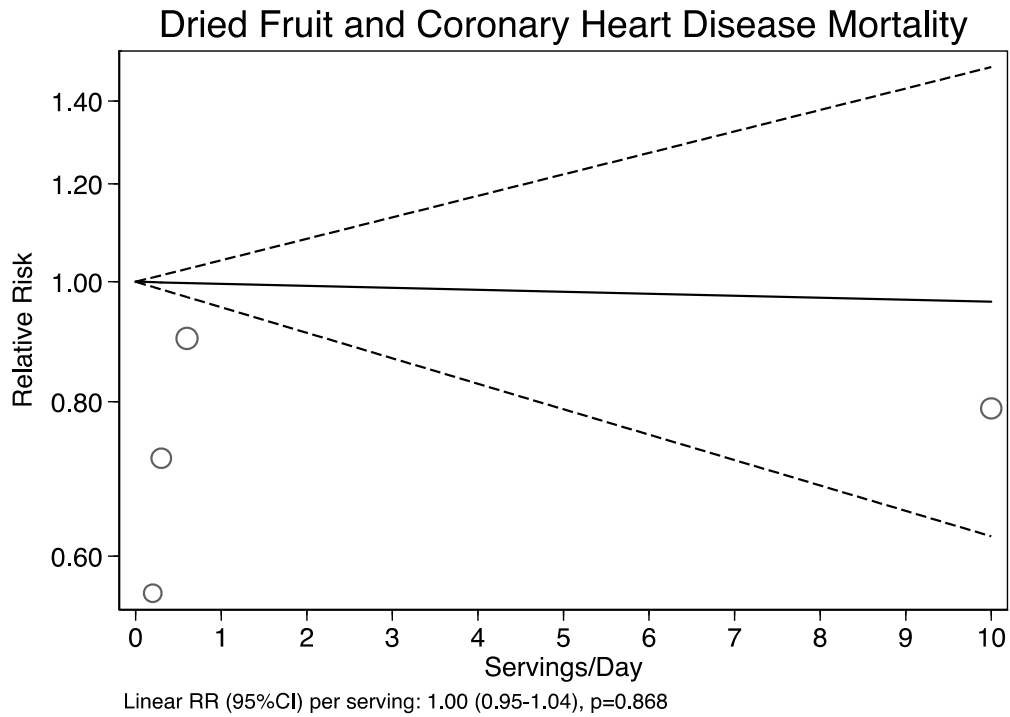


Figure S108. Linear and cubic-spline dose-response relation between increasing dried fruit intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

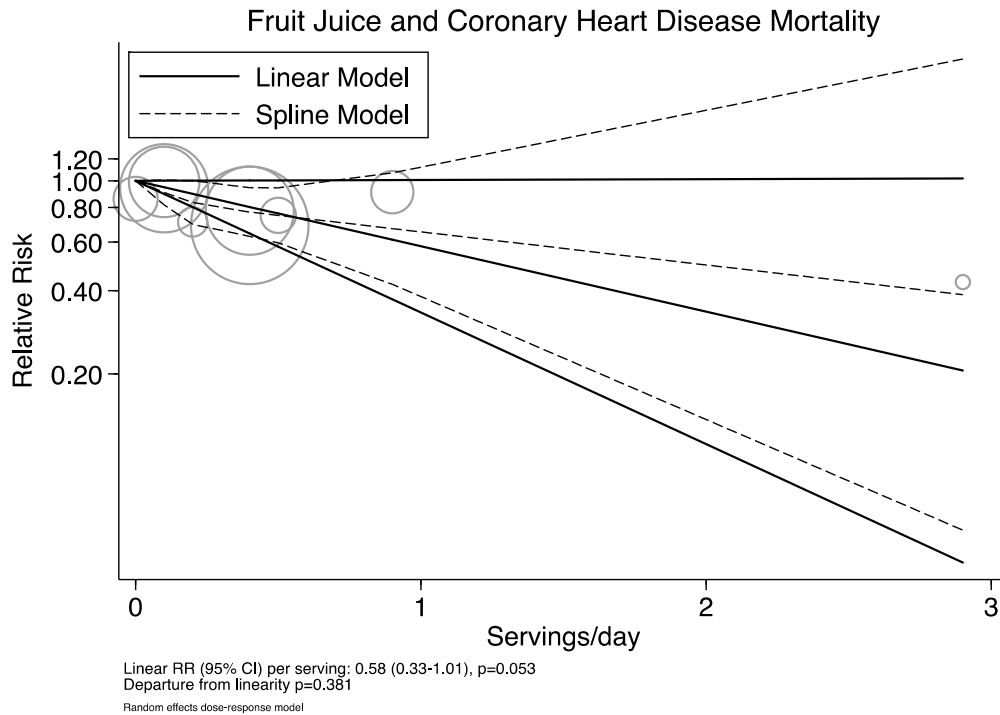


Figure S109. Linear and cubic-spline dose-response relation between increasing fruit juice intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

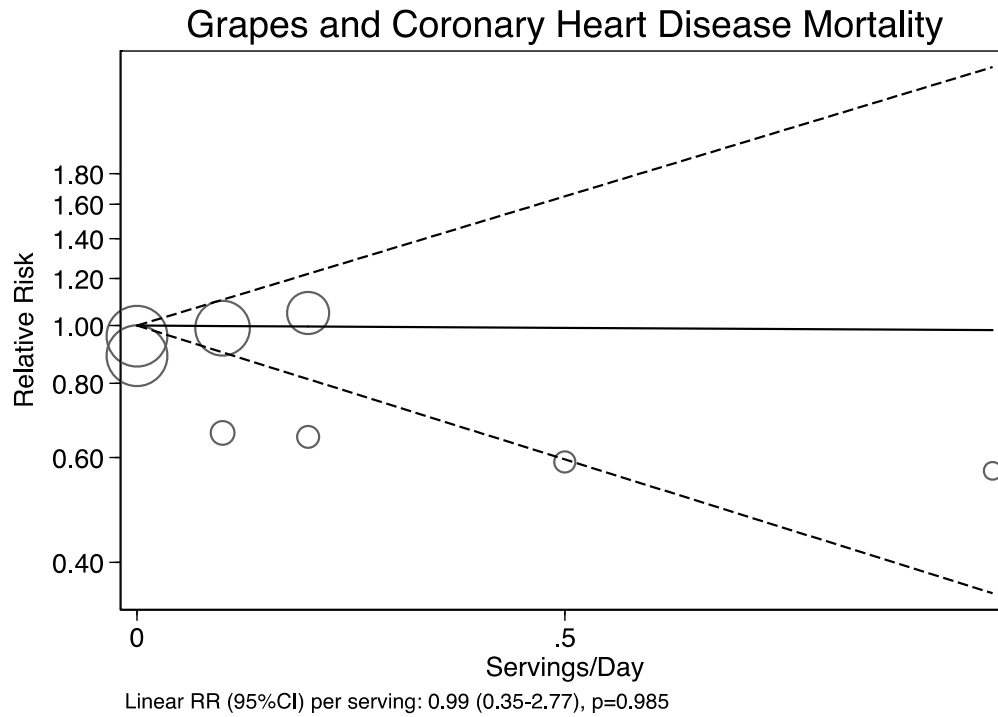


Figure S110. Linear dose-response relation between increasing grape intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

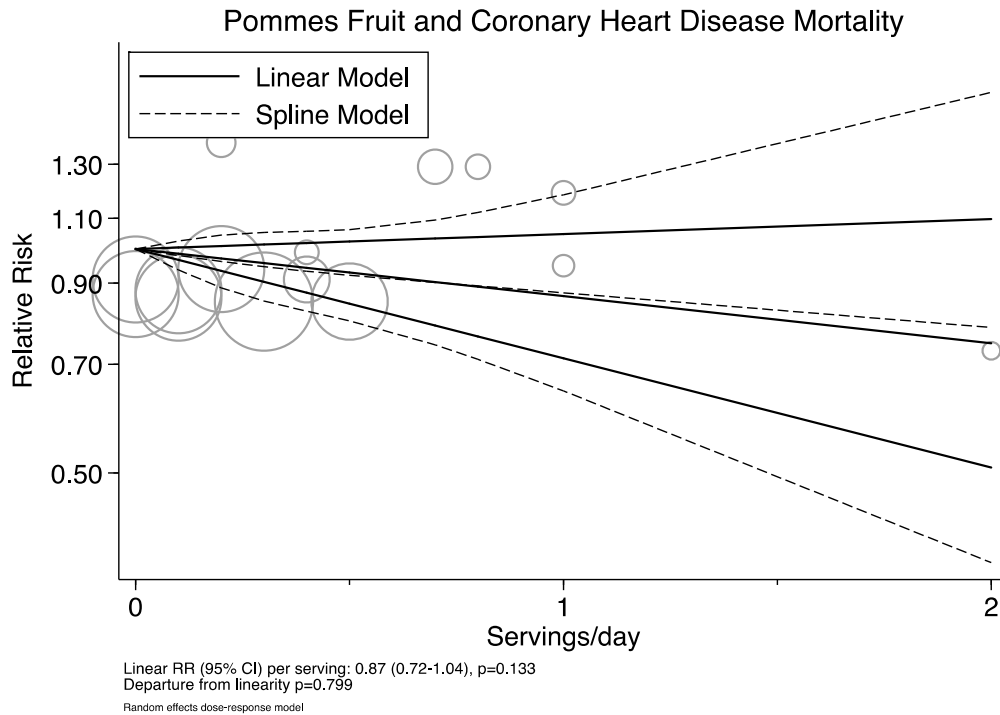


Figure S111. Linear and cubic-spline dose-response relation between increasing pommes intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

Allium Vegetables and Coronary Heart Disease Mortality

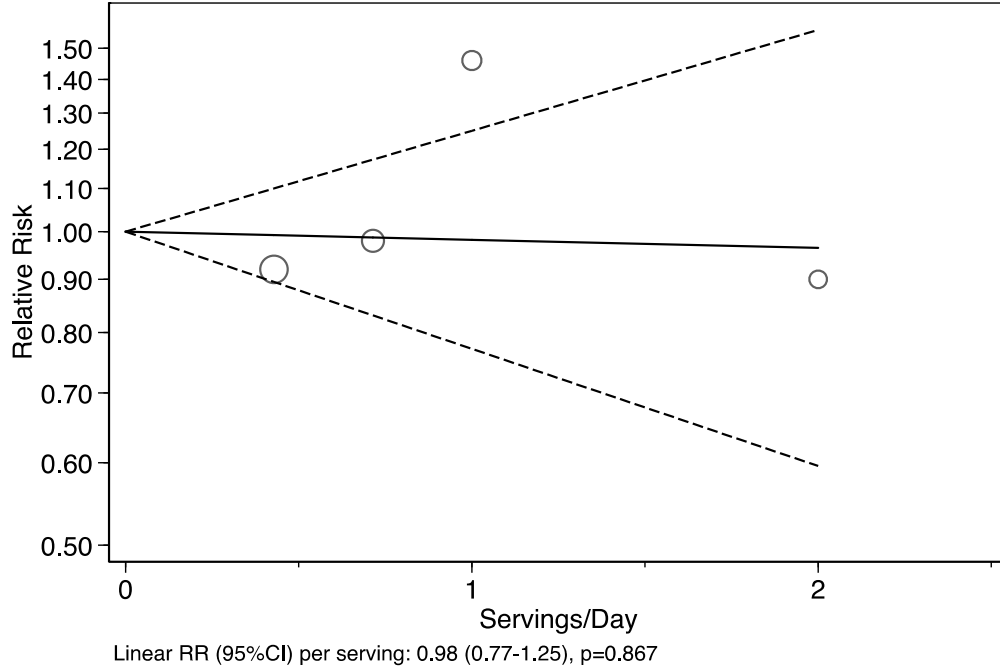


Figure S112. Linear dose-response relation between increasing intake of allium vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

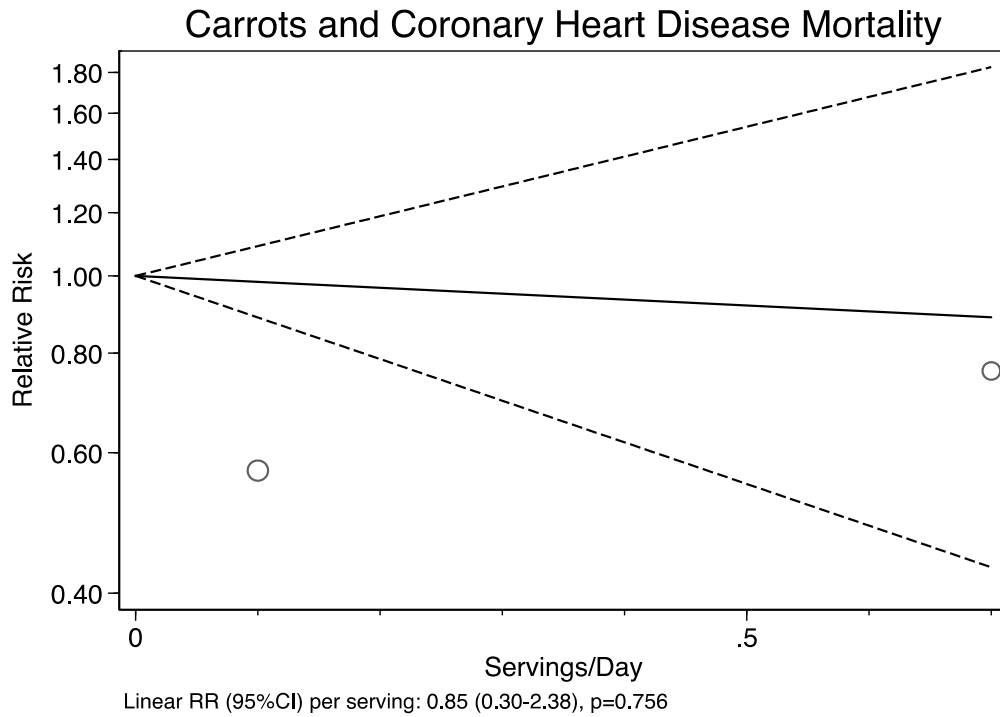


Figure S113. Linear dose-response relation between increasing intake of carrots and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

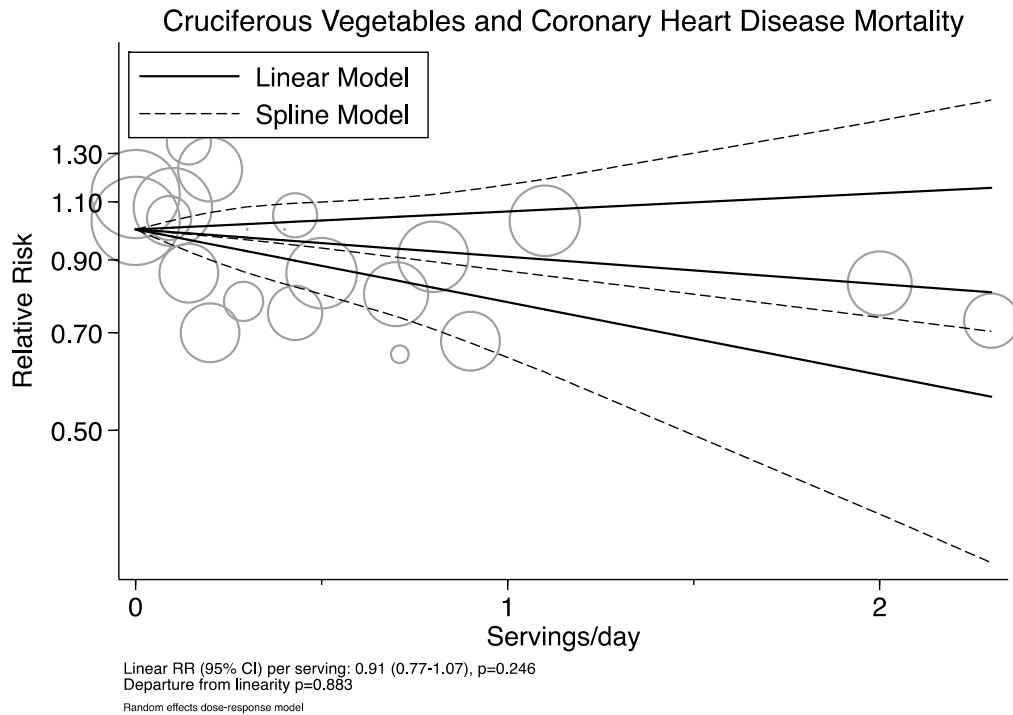


Figure S114. Linear and cubic-spline dose-response relation between increasing intake of cruciferous vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

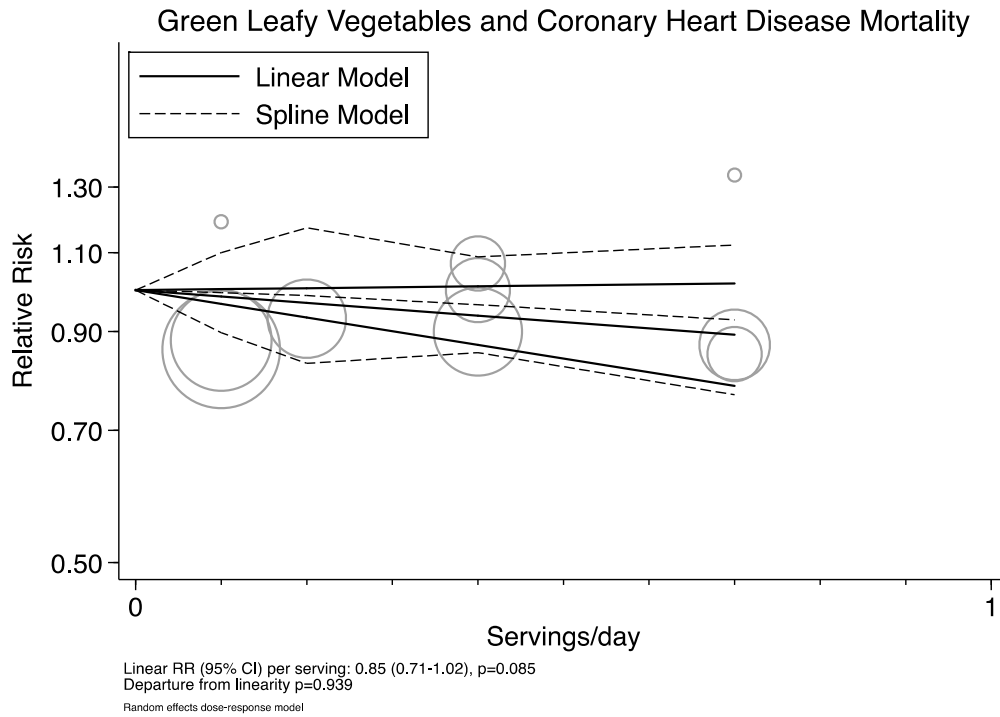


Figure S115. Linear dose-response relation between increasing intake of green leafy vegetables and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

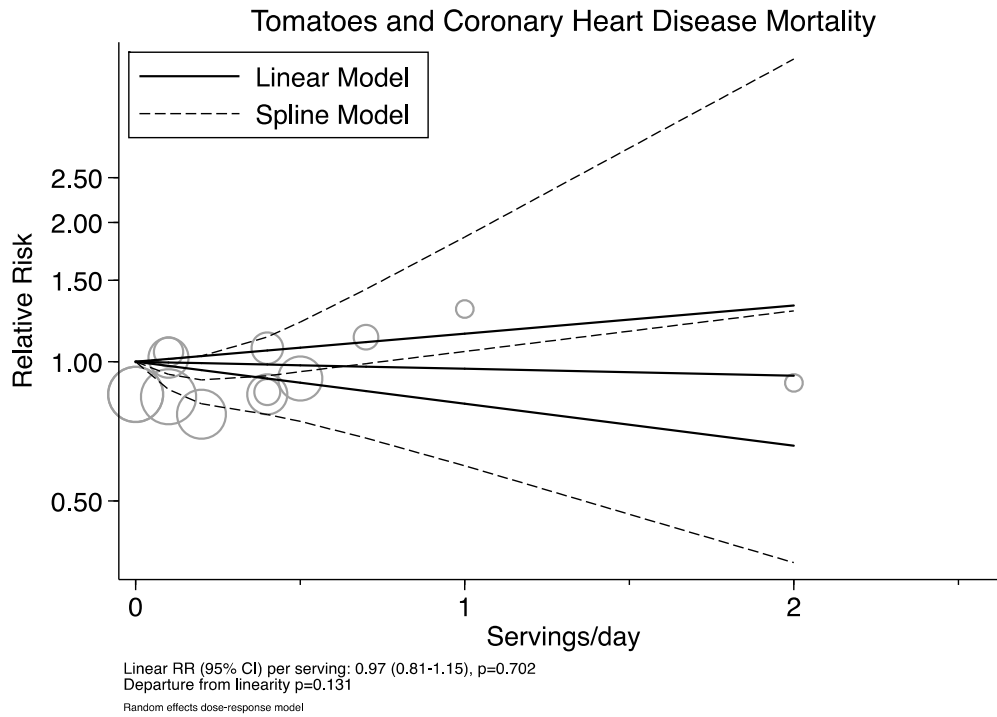
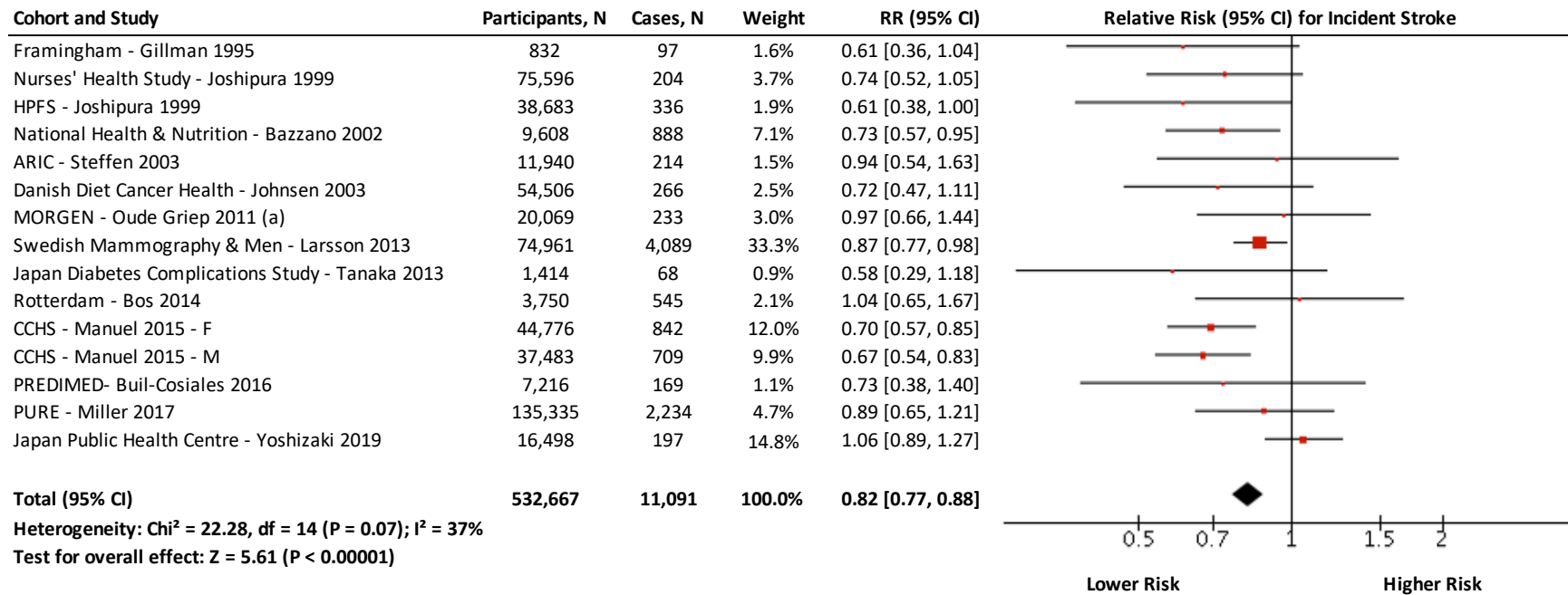


Figure S116. Linear dose-response relation between increasing tomato intake and coronary heart disease mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

TOTAL FRUIT AND VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

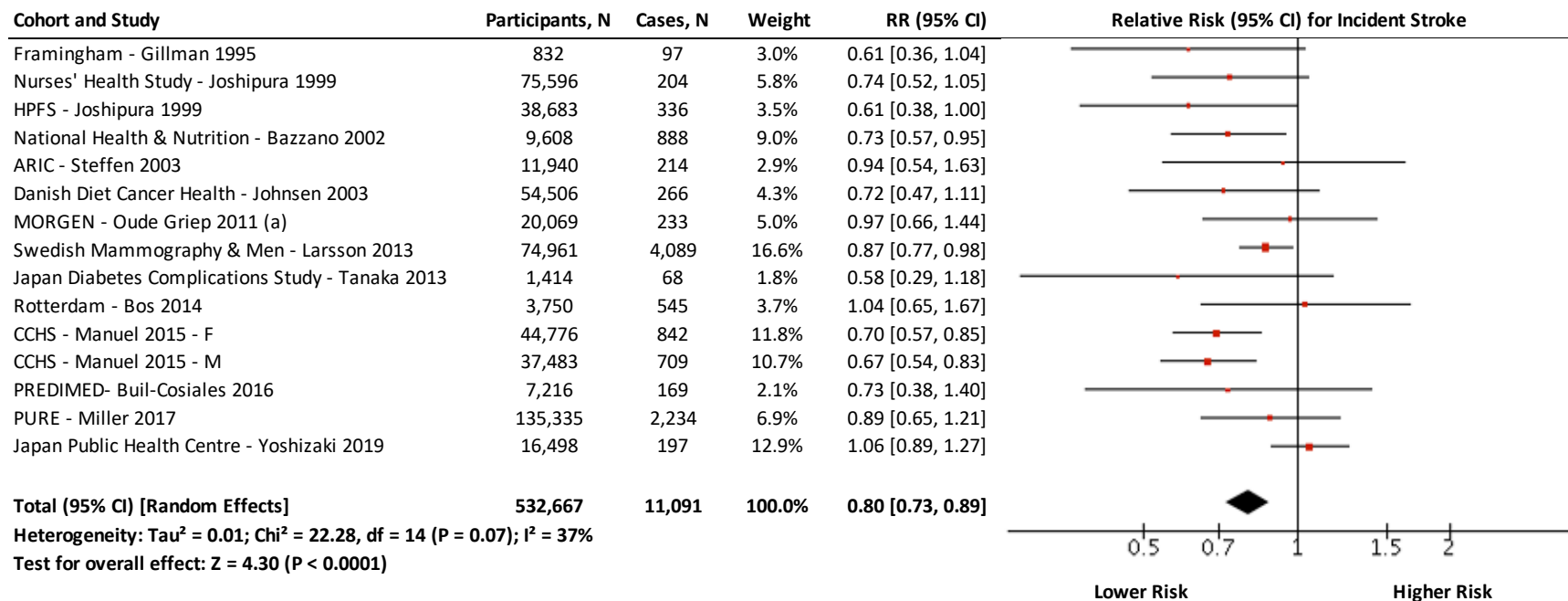
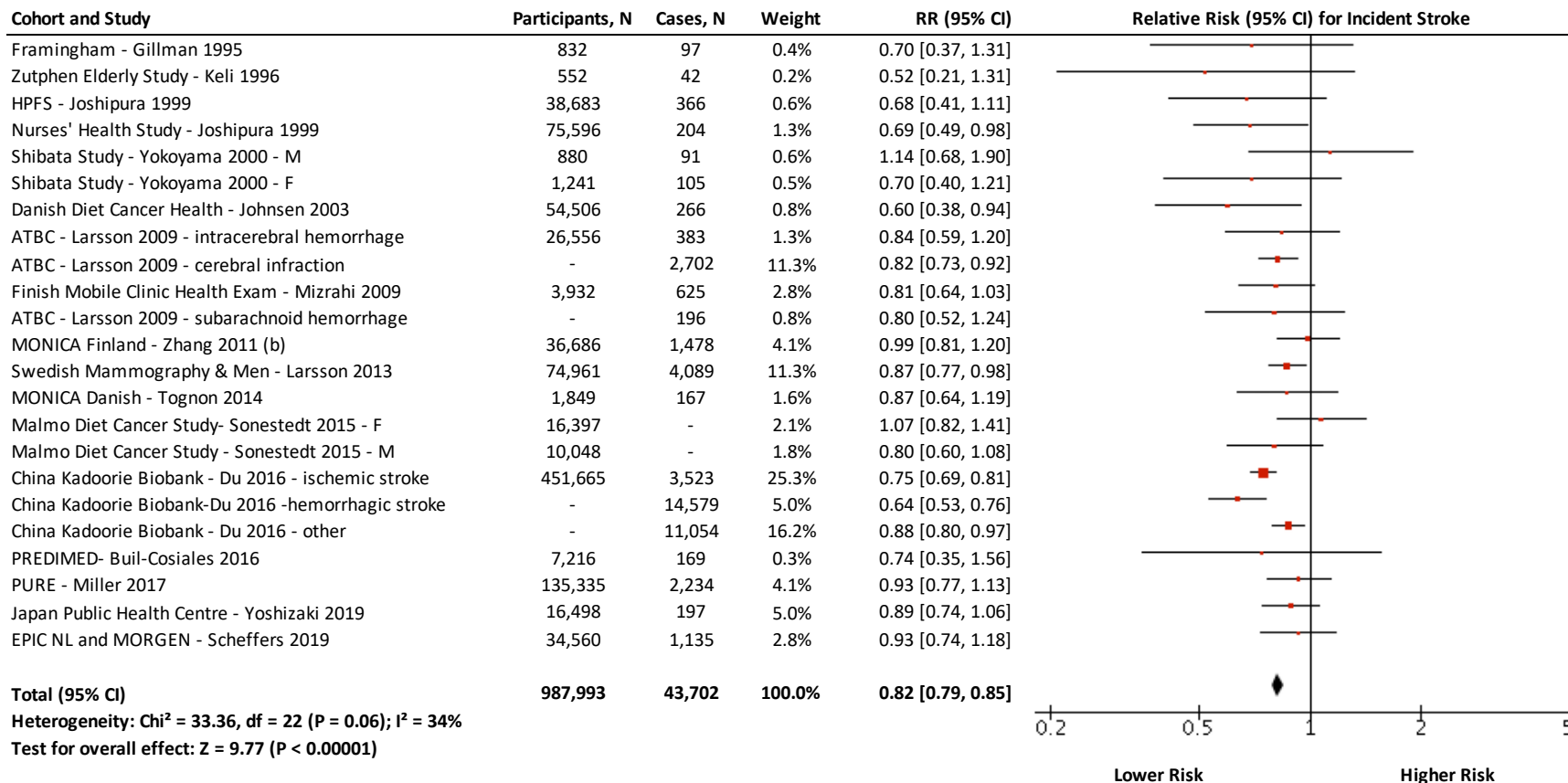


Figure S117. Relation between total fruit and vegetables intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

FRUIT AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

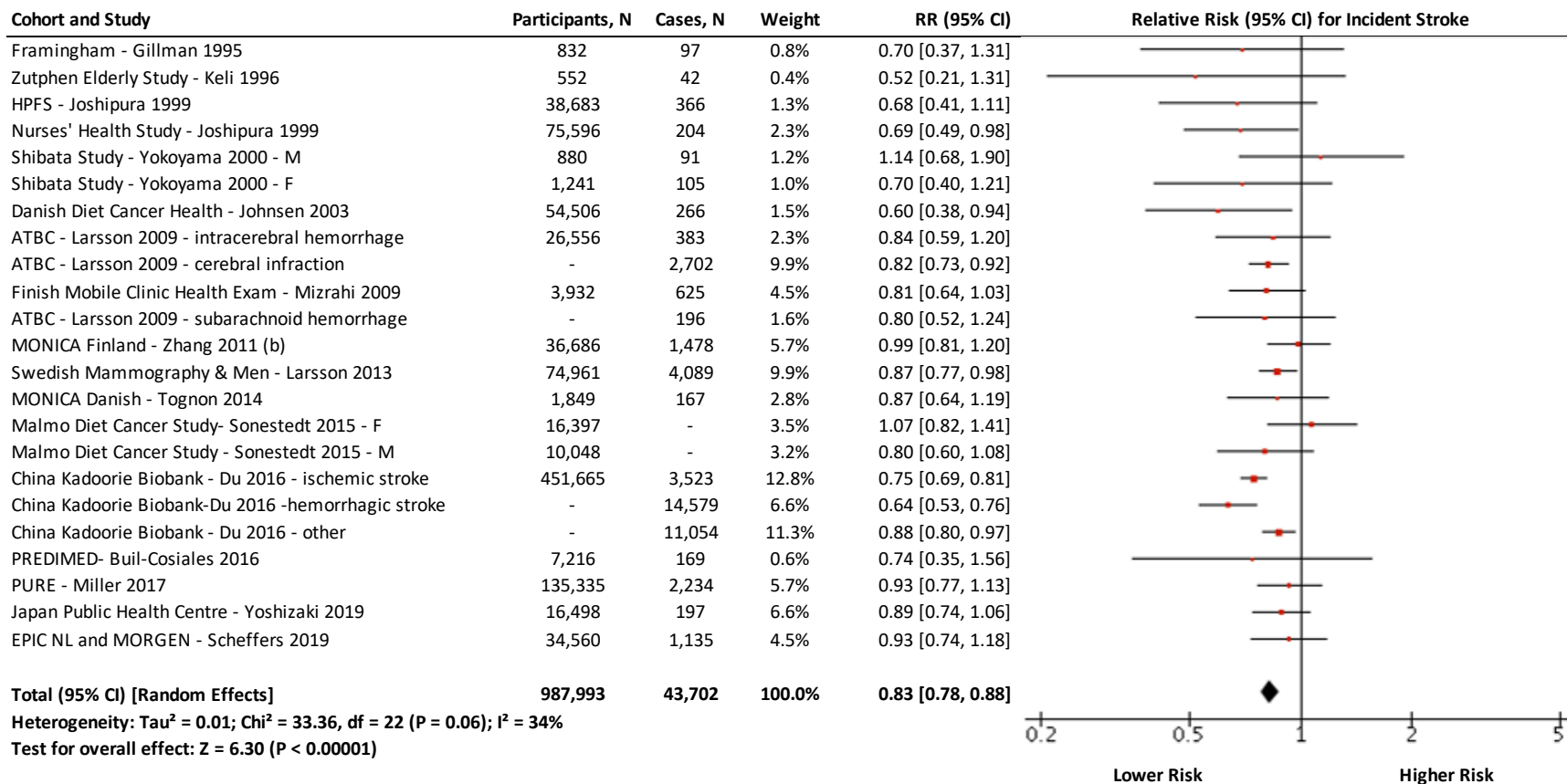
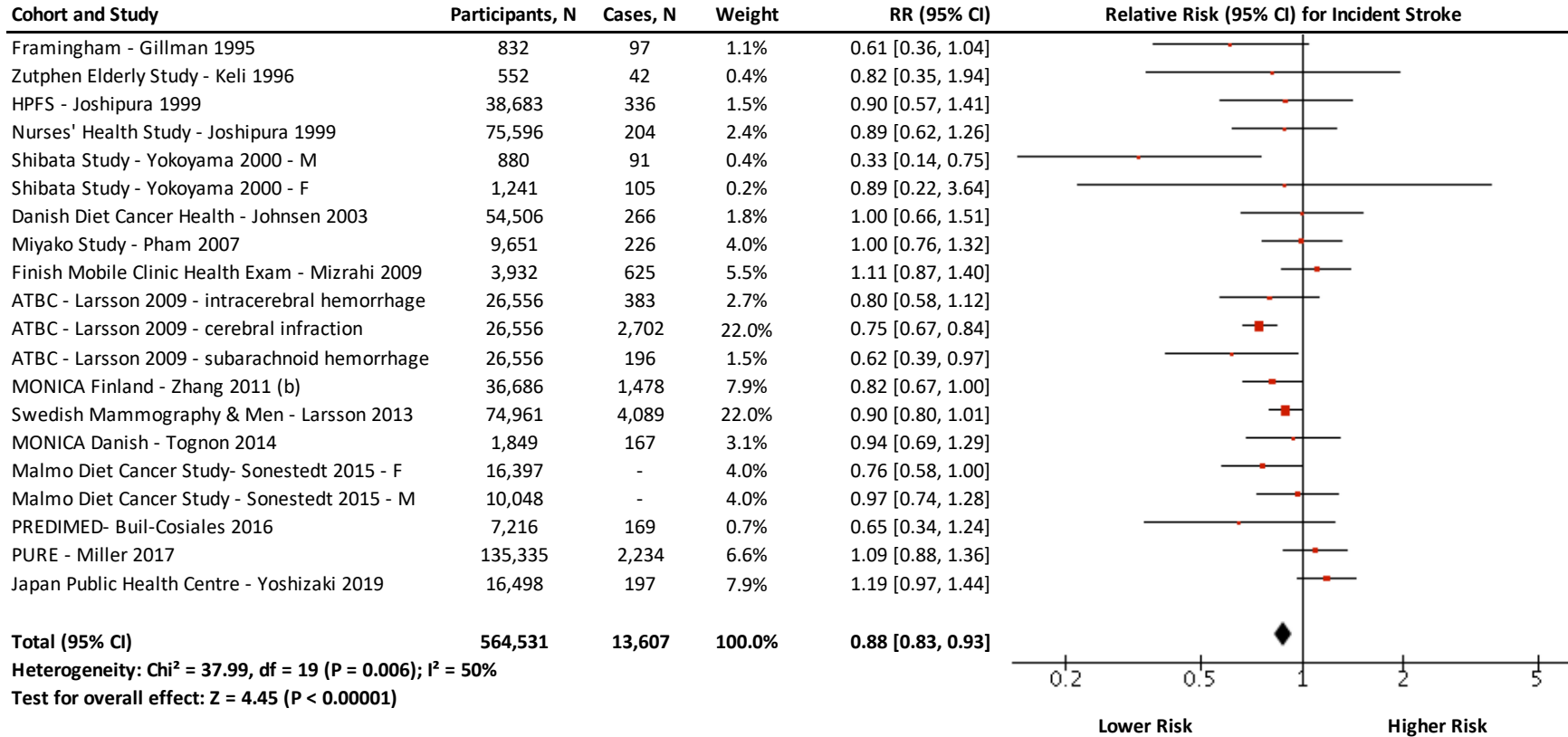


Figure S118. Relation between fruit intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models.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

VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

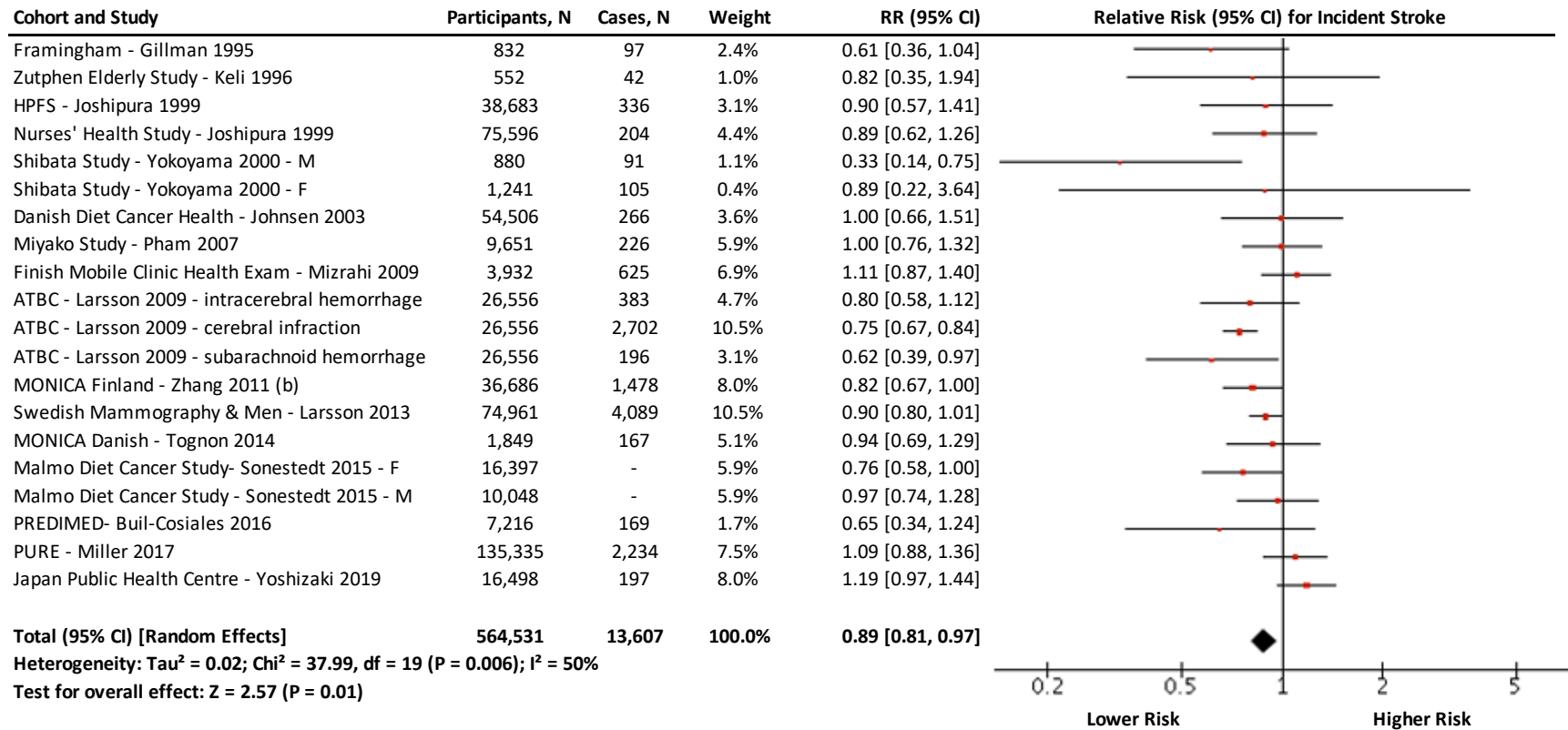


Figure S119. Relation between intake of vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

BERRIES AND STROKE INCIDENCE

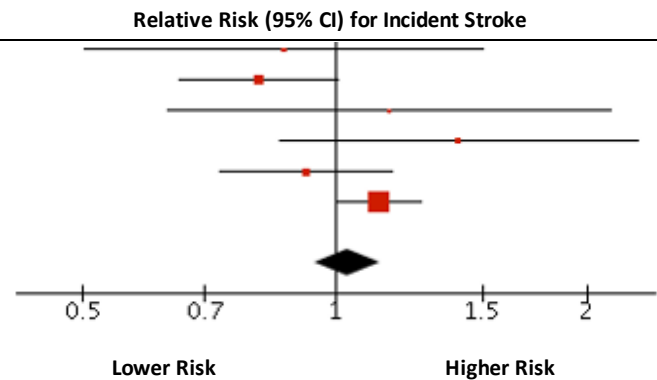
A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2000 - intracerebral hemorrhage	26,593	95	2.7%	0.81 [0.65, 1.01]
ATBC - Hirvonen 2000 - cerebral infarction	-	736	17.6%	1.16 [0.63, 2.13]
ATBC - Hirvonen 2000 - subarachnoid hemorrhage	-	83	2.2%	0.87 [0.50, 1.50]
WHS - Sesso 2007	38,176	339	3.4%	1.40 [0.86, 2.29]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	14.8%	0.92 [0.73, 1.17]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	59.2%	1.13 [1.00, 1.27]

Total (95% CI) **143,662** **5,967** **100.0%** **1.03 [0.94, 1.13]**

Heterogeneity: Chi² = 9.90, df = 5 (P = 0.08); I² = 50%

Test for overall effect: Z = 0.72 (P = 0.47)



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
ATBC - Hirvonen 2000 - cerebral infarction	26,593	736	23.40%	0.81 [0.65, 1.01]
ATBC - Hirvonen 2000 - subarachnoid hemorrhage	-	83	6.10%	1.16 [0.63, 2.13]
ATBC - Hirvonen 2000 - intracerebral hemorrhage	-	95	7.20%	0.87 [0.50, 1.50]
WHS - Sesso 2007	38,176	339	8.70%	1.40 [0.86, 2.29]
Finish Mobile Clinic Health Exam - Mizrahi 2009	3,932	625	21.70%	0.92 [0.73, 1.17]
Swedish Mammography & Men - Larsson 2013	74,961	4,089	32.80%	1.13 [1.00, 1.27]

Total (95% CI) [Random Effects] **143,662** **5,967** **100.0%** **1.00 [0.85, 1.18]**

Heterogeneity: Tau² = 0.02; Chi² = 9.90, df = 5 (P = 0.08); I² = 50%

Test for overall effect: Z = 0.02 (P = 0.99)

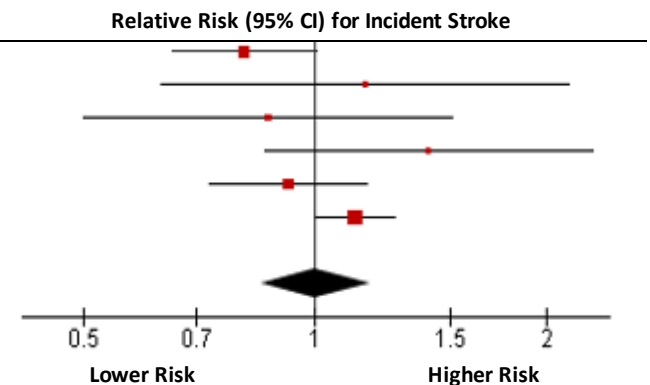


Figure S120. Relation between intake of berries and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

CITRUS FRUIT AND STROKE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 16.43$, $\text{df} = 8$ ($P = 0.04$); $I^2 = 51\%$
 Test for overall effect: $Z = 3.59$ ($P = 0.0003$)

B. Random Effects

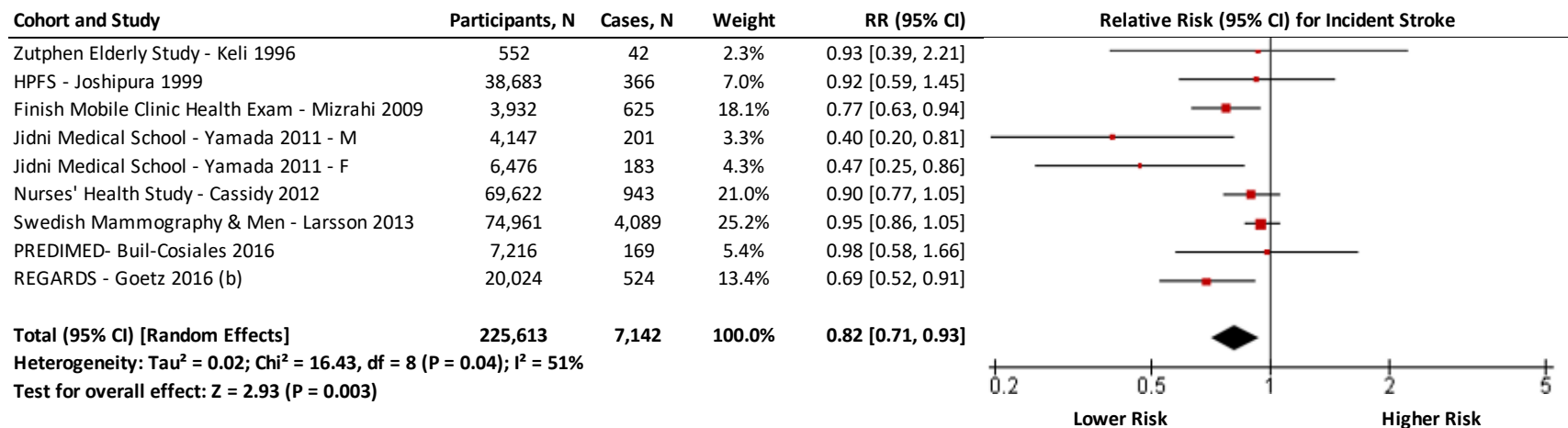
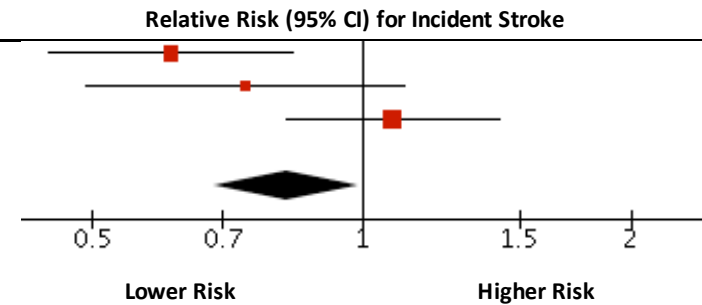


Figure S121. Relation between citrus fruit intake and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

FRUIT JUICE AND STROKE INCIDENCE

A. Fixed Effects

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



B. Random Effects

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

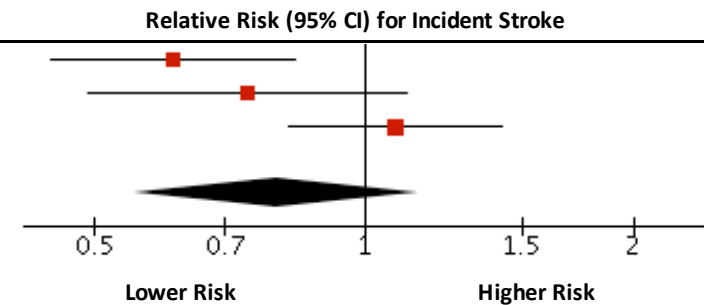
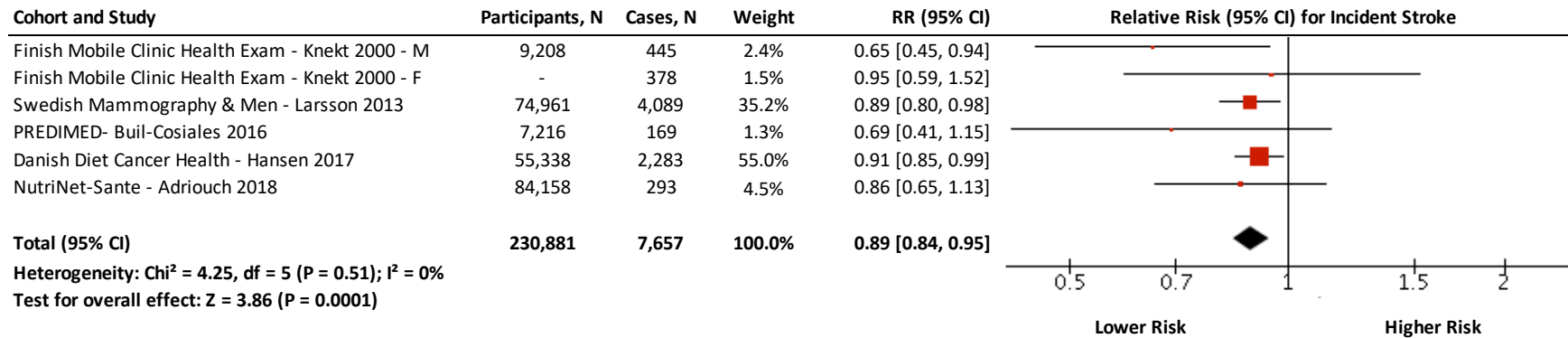


Figure S122. Relation between intake of fruit juice and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

POMMES AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

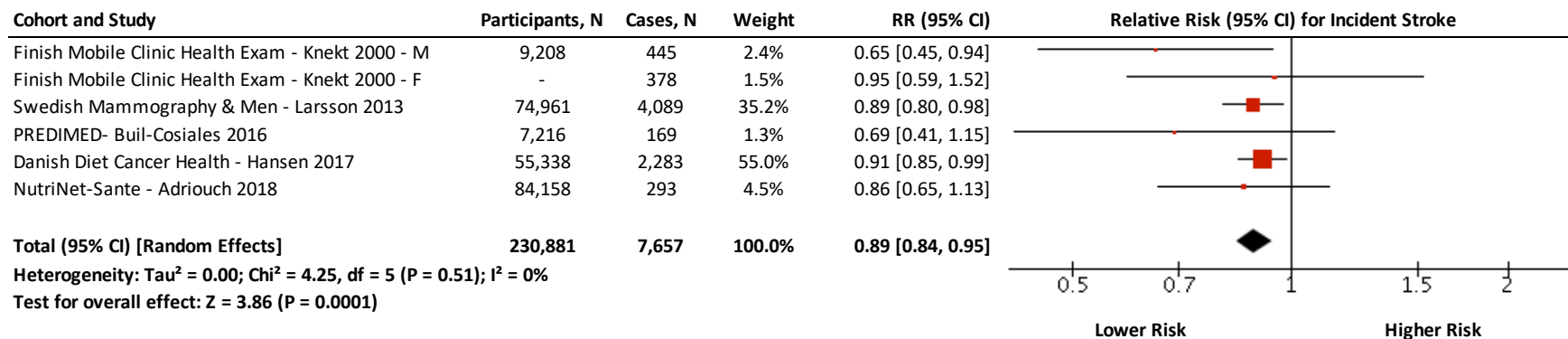
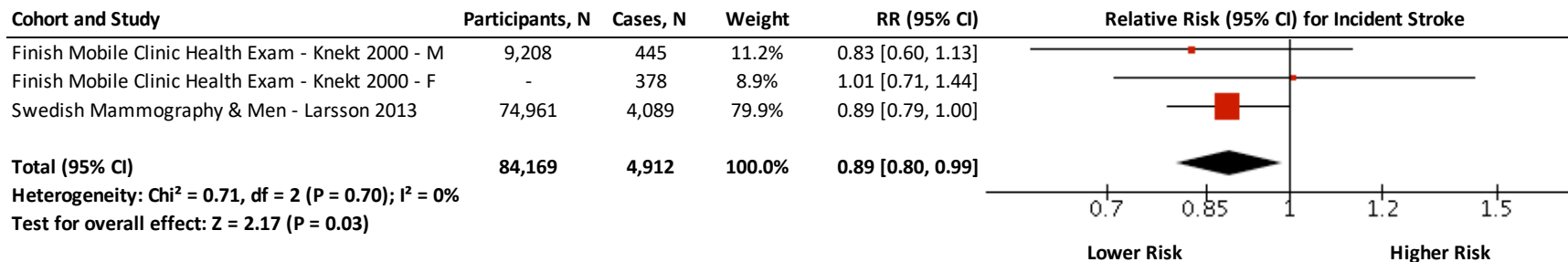


Figure S123. Relation between intake of pomes fruit and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

ALLIUM VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

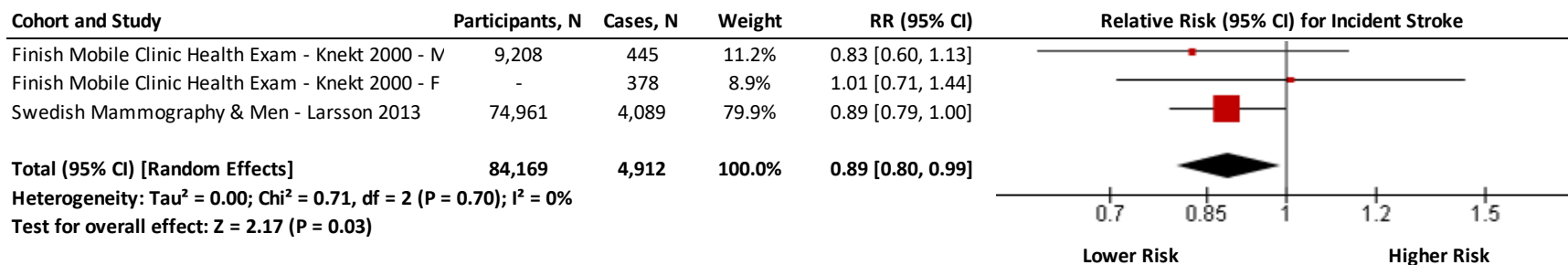
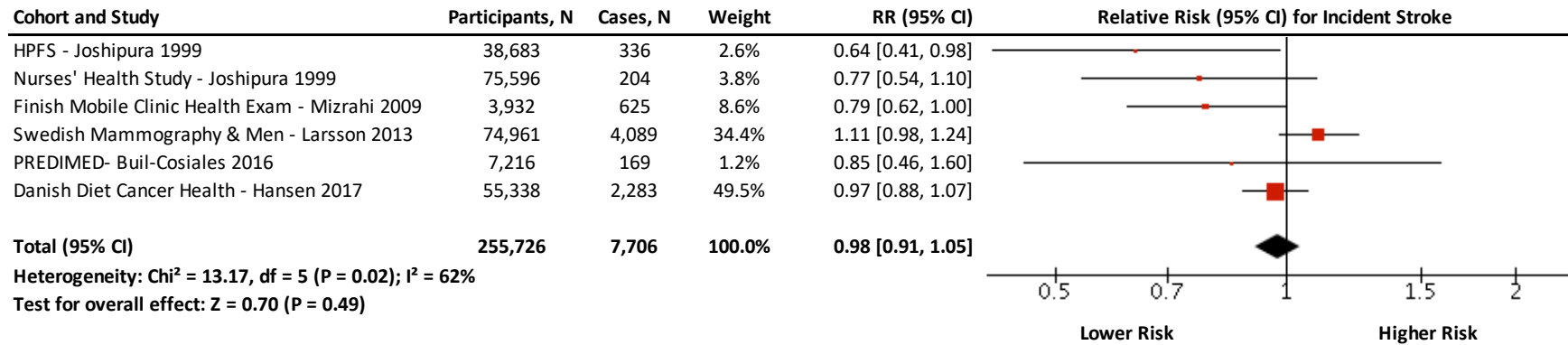


Figure S124. Relation between intake of allium vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CRUCIFEROUS VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects



B. Random Effects

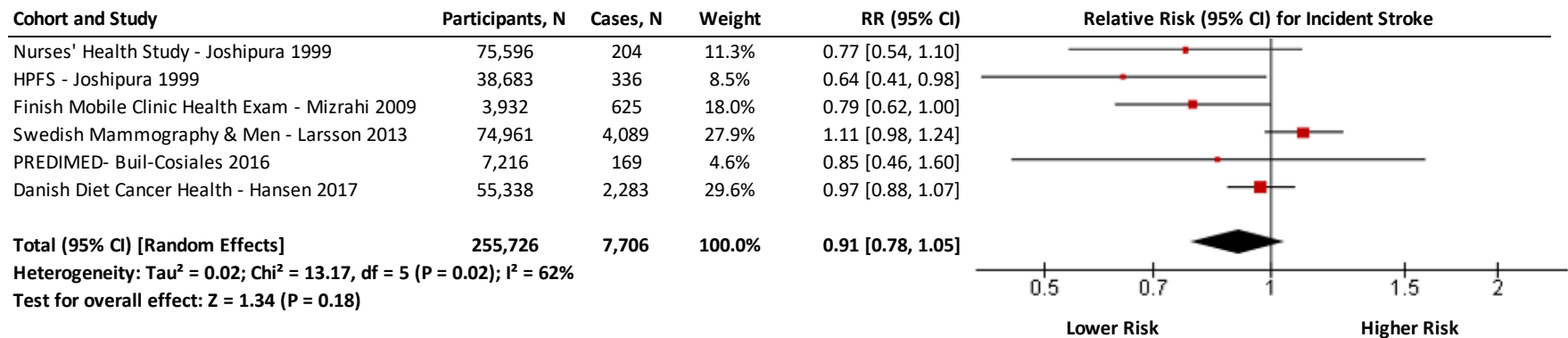


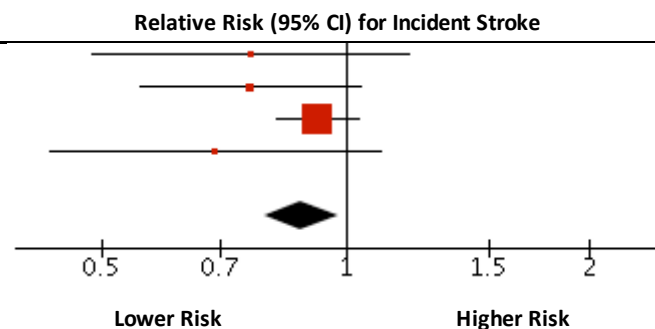
Figure S125. Relation between intake of cruciferous vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

GREEN LEAFY VEGETABLES AND STROKE INCIDENCE

A. Fixed Effects

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

Heterogeneity: $\text{Chi}^2 = 2.82$, $\text{df} = 3$ ($P = 0.42$); $I^2 = 0\%$
 Test for overall effect: $Z = 2.36$ ($P = 0.02$)



B. Random Effects

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

Heterogeneity: $\text{Tau}^2 = 0.00$; $\text{Chi}^2 = 2.82$, $\text{df} = 3$ ($P = 0.42$); $I^2 = 0\%$
 Test for overall effect: $Z = 2.36$ ($P = 0.02$)

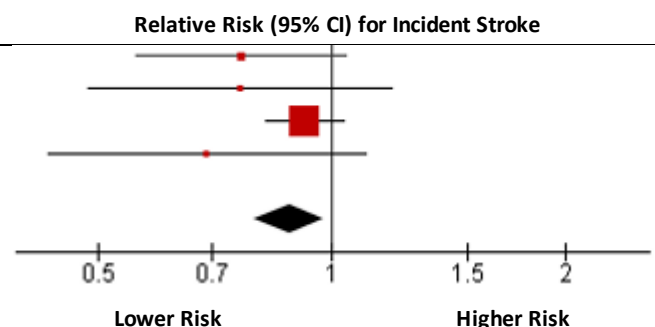


Figure S126. Relation between intake of green leafy vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

TOMATOES AND STROKE INCIDENCE

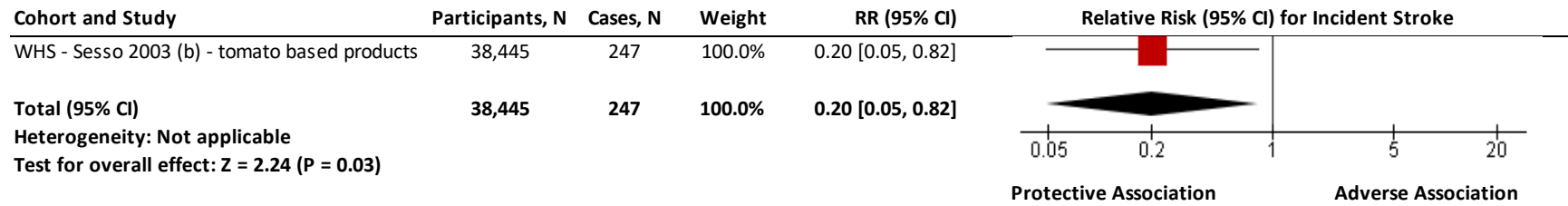
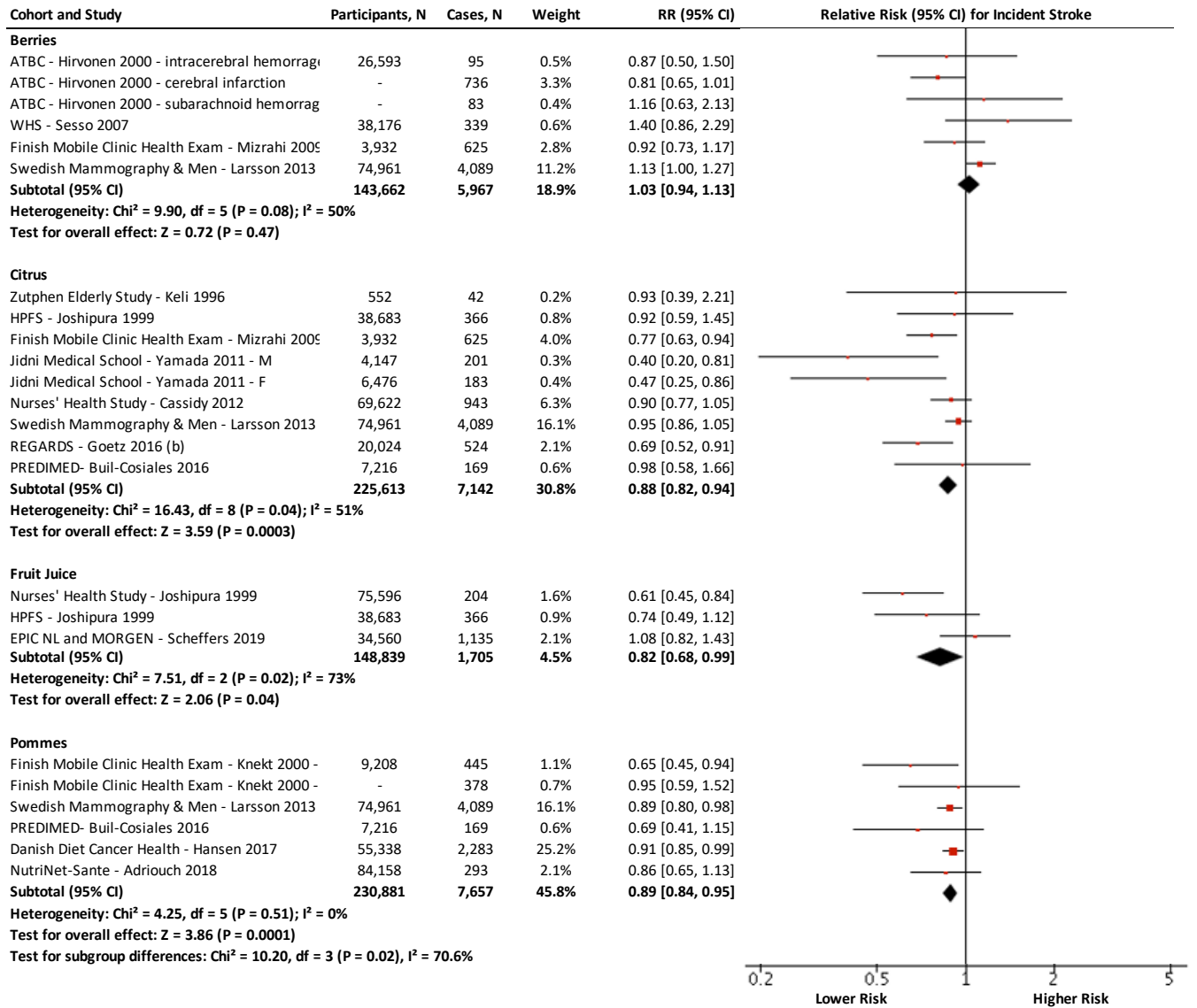


Figure S127. Relation between intake of tomatoes and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the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

A. Fixed Effects



B. Random Effects

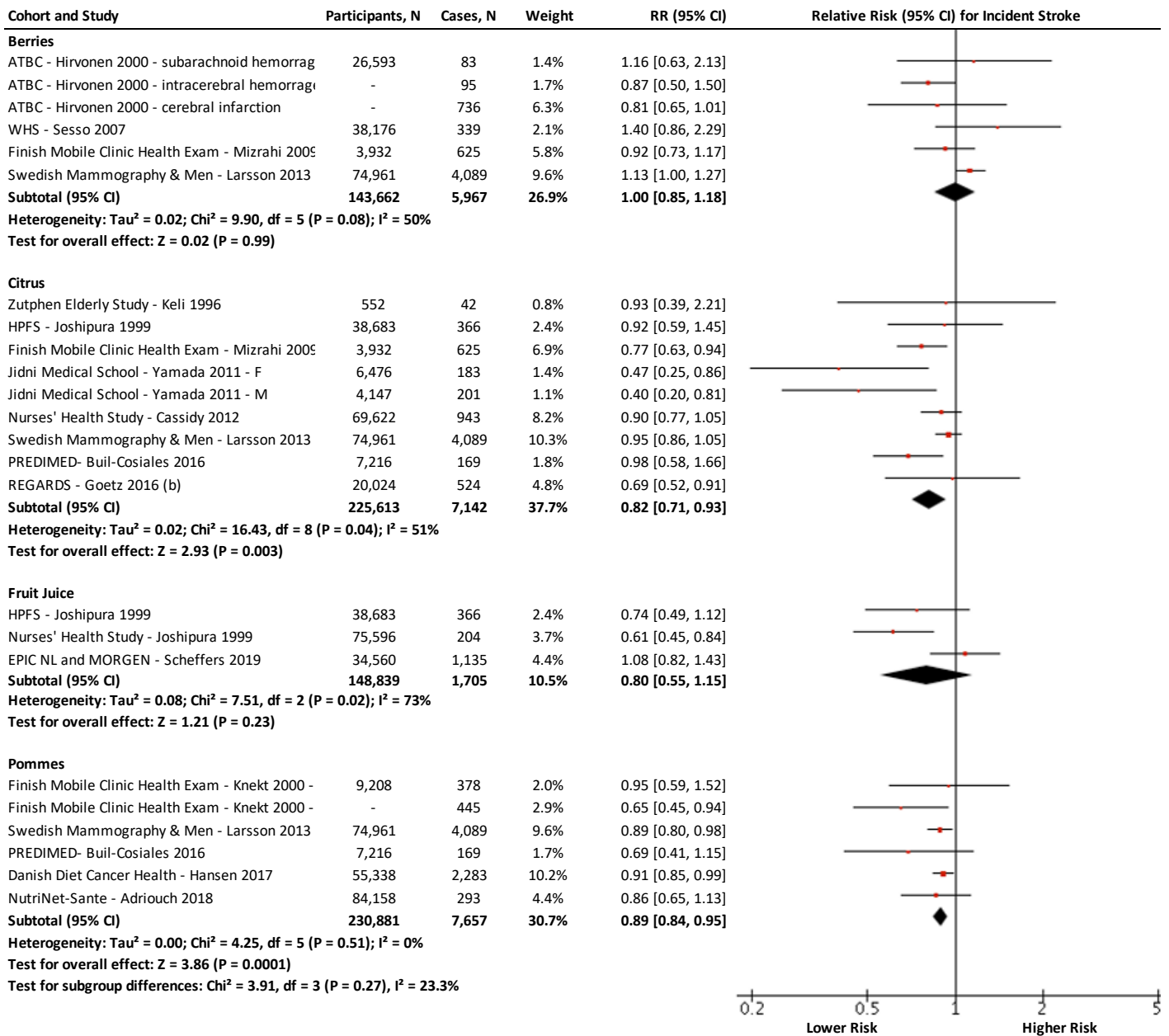
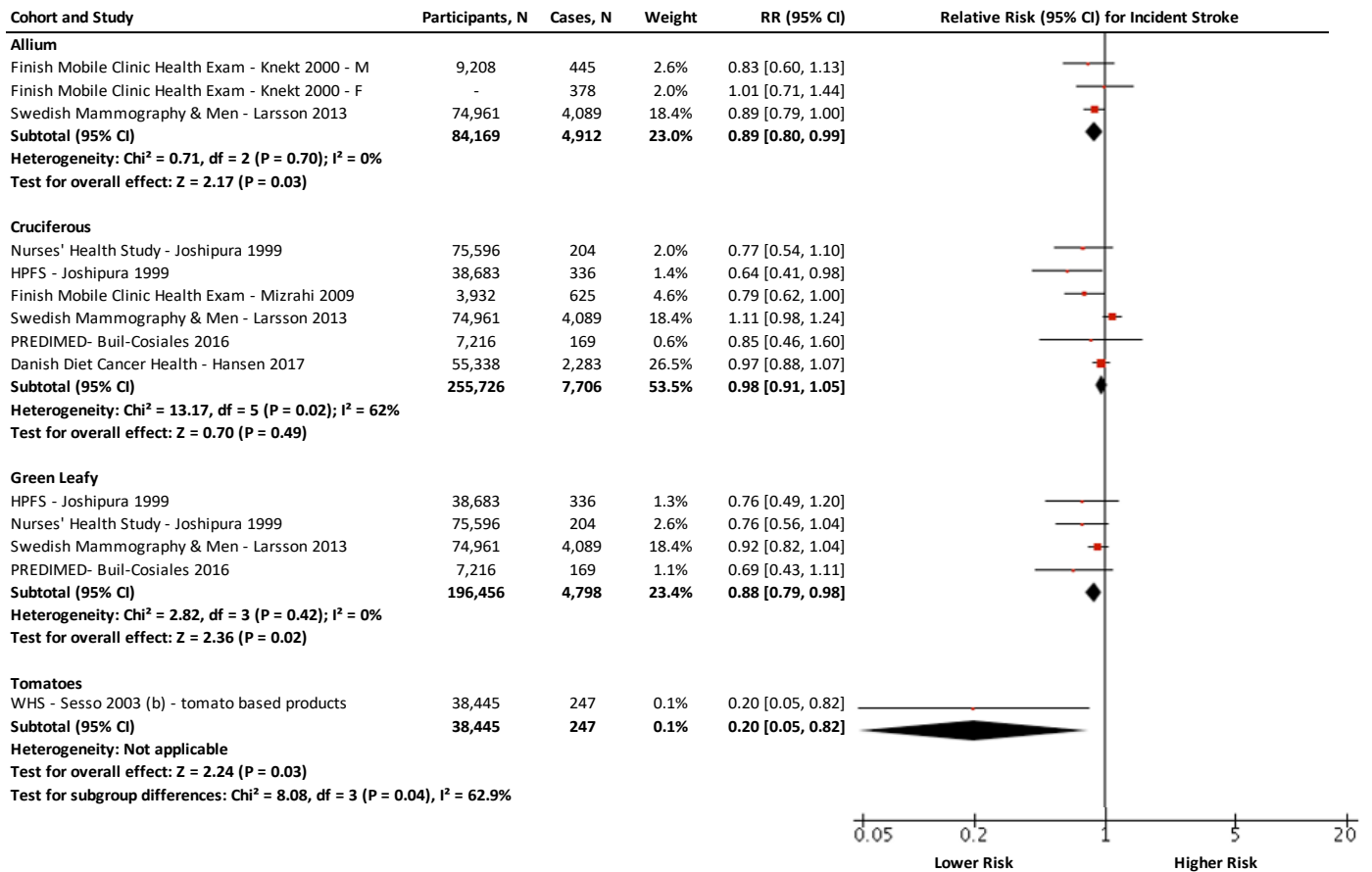


Figure S128. Relation between sources of fruit and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

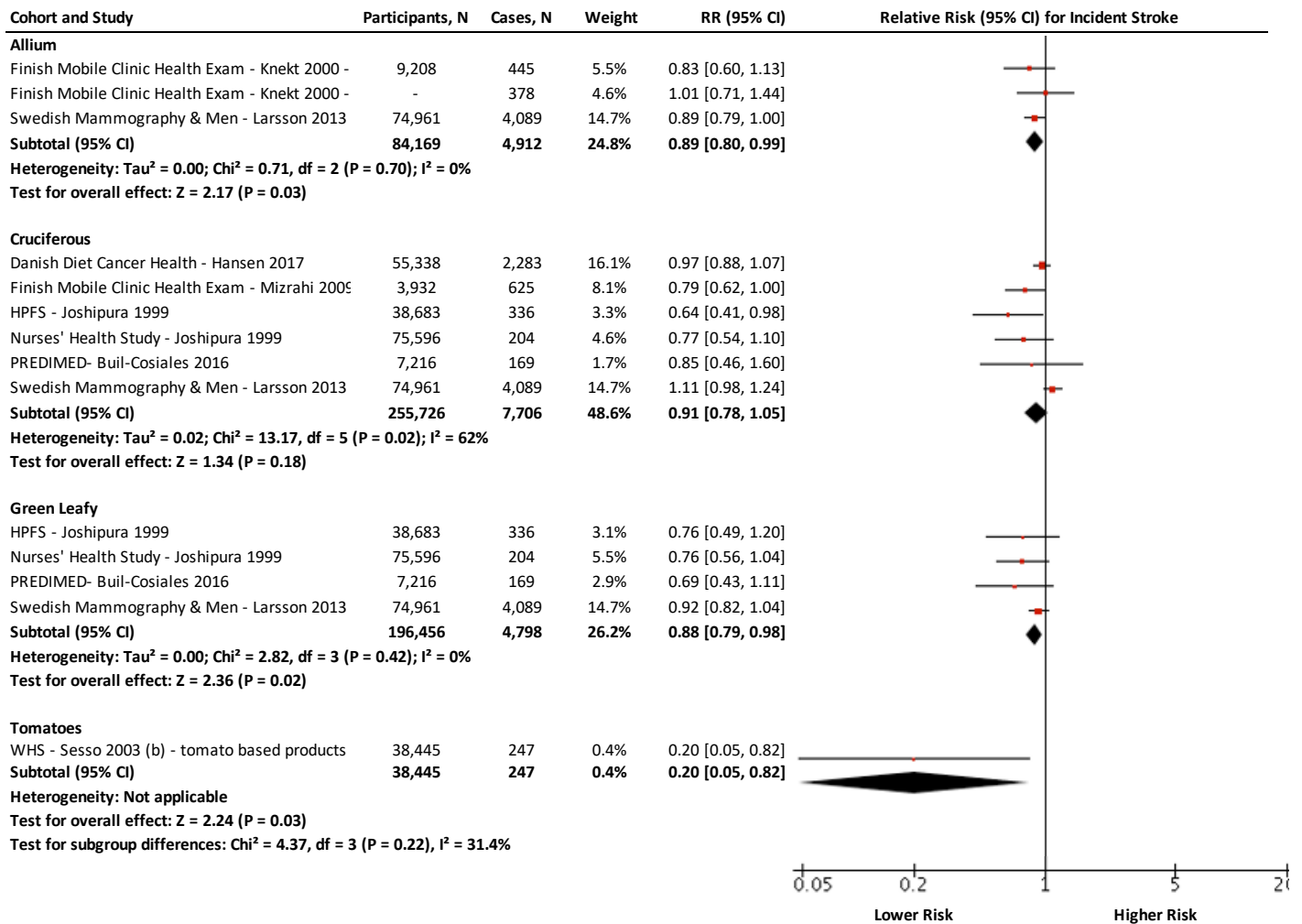
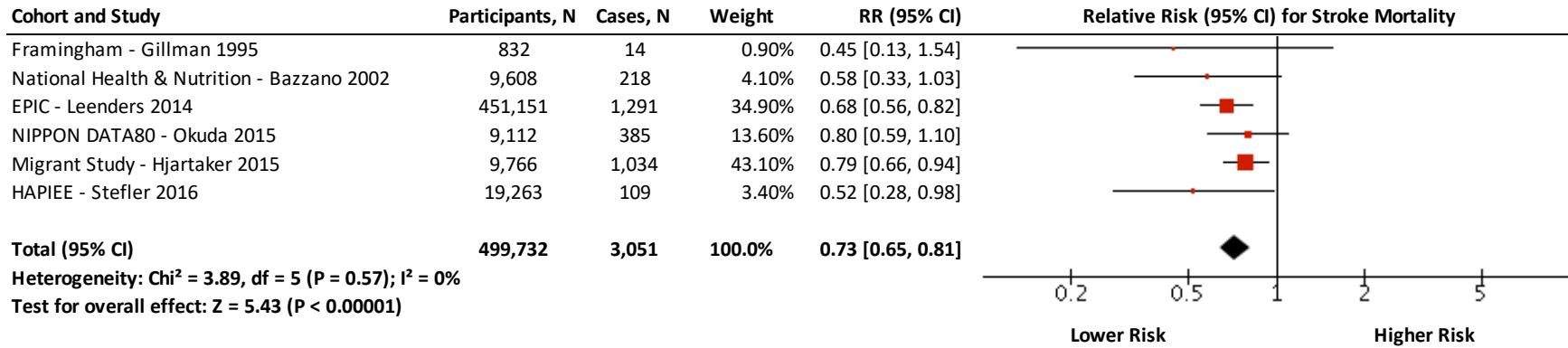


Figure S129. Relation between sources of vegetables and stroke incidence (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

TOTAL FRUIT AND VEGETABLES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

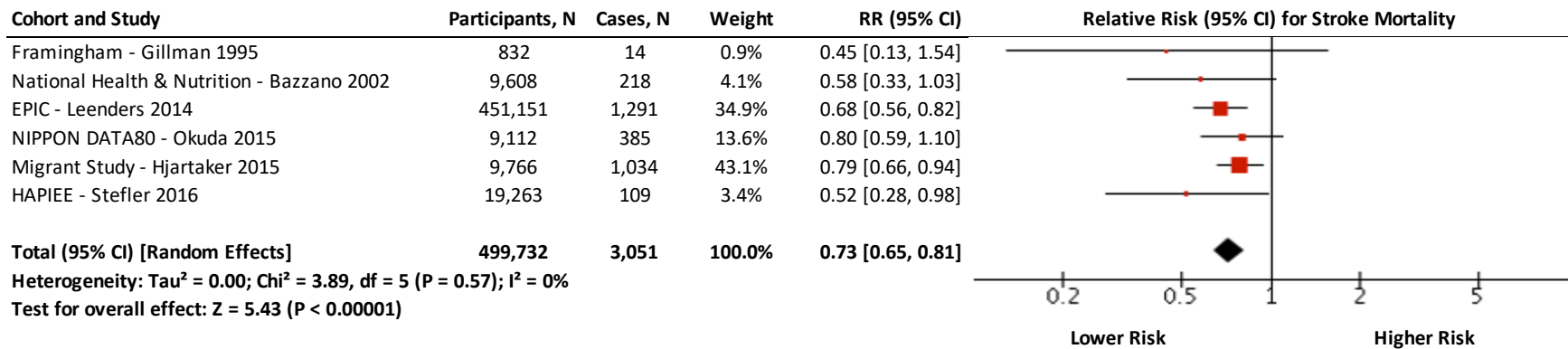
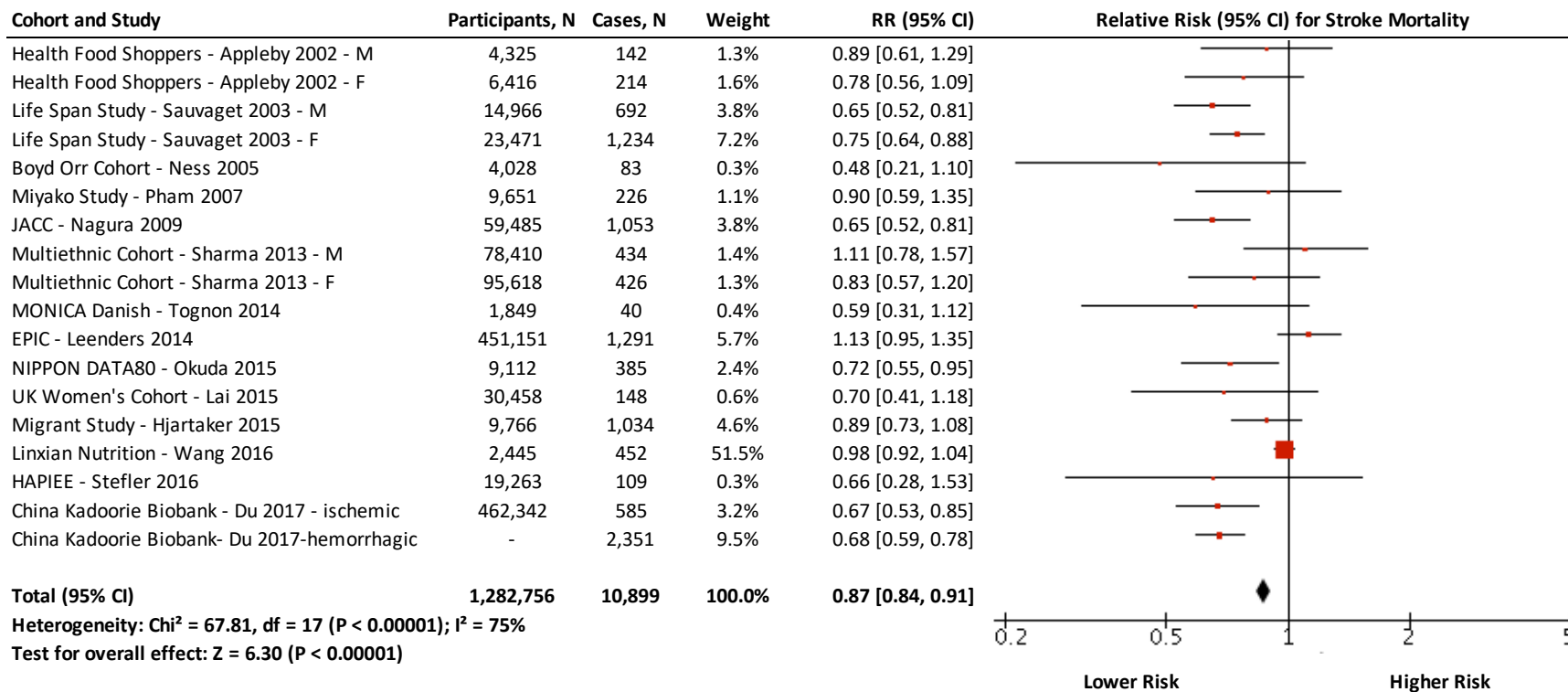


Figure S130. Relation between total fruit and vegetables intake and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

FRUIT AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

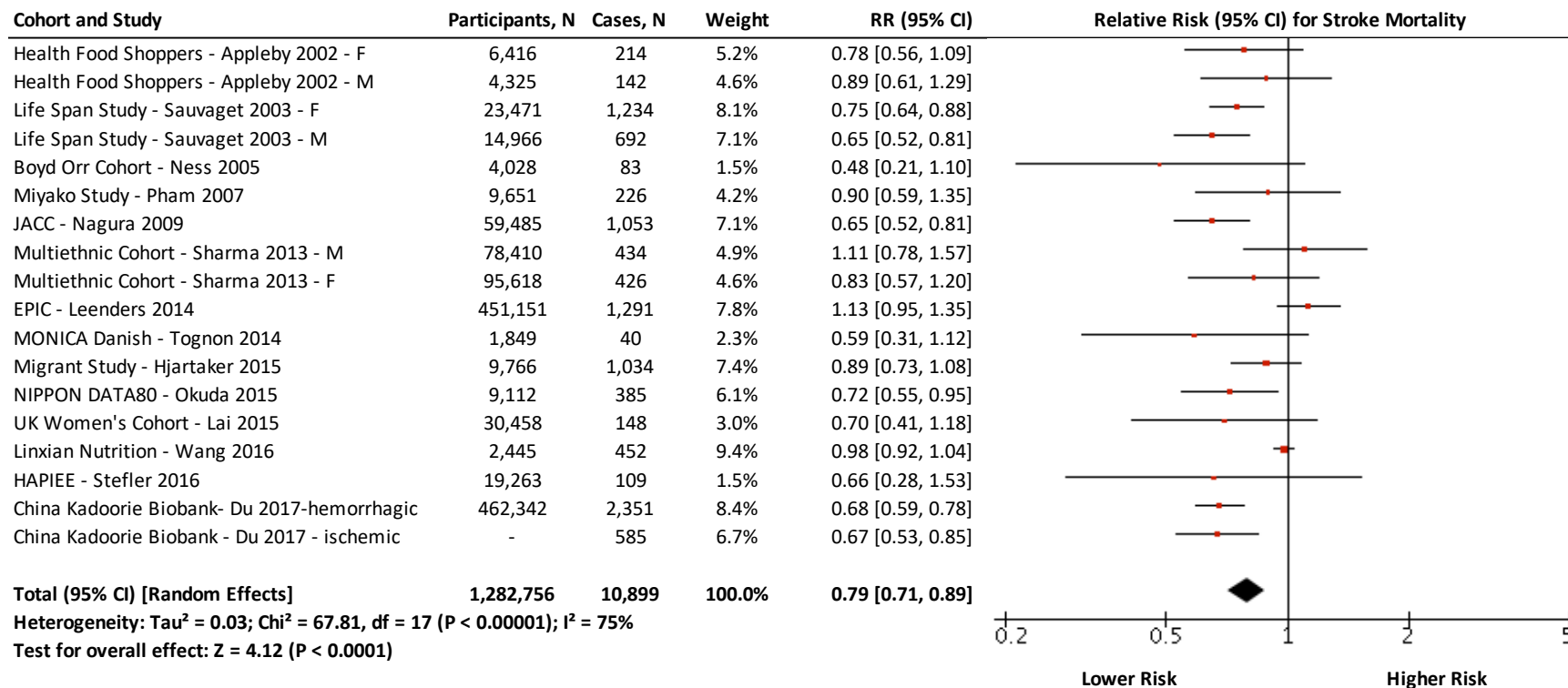
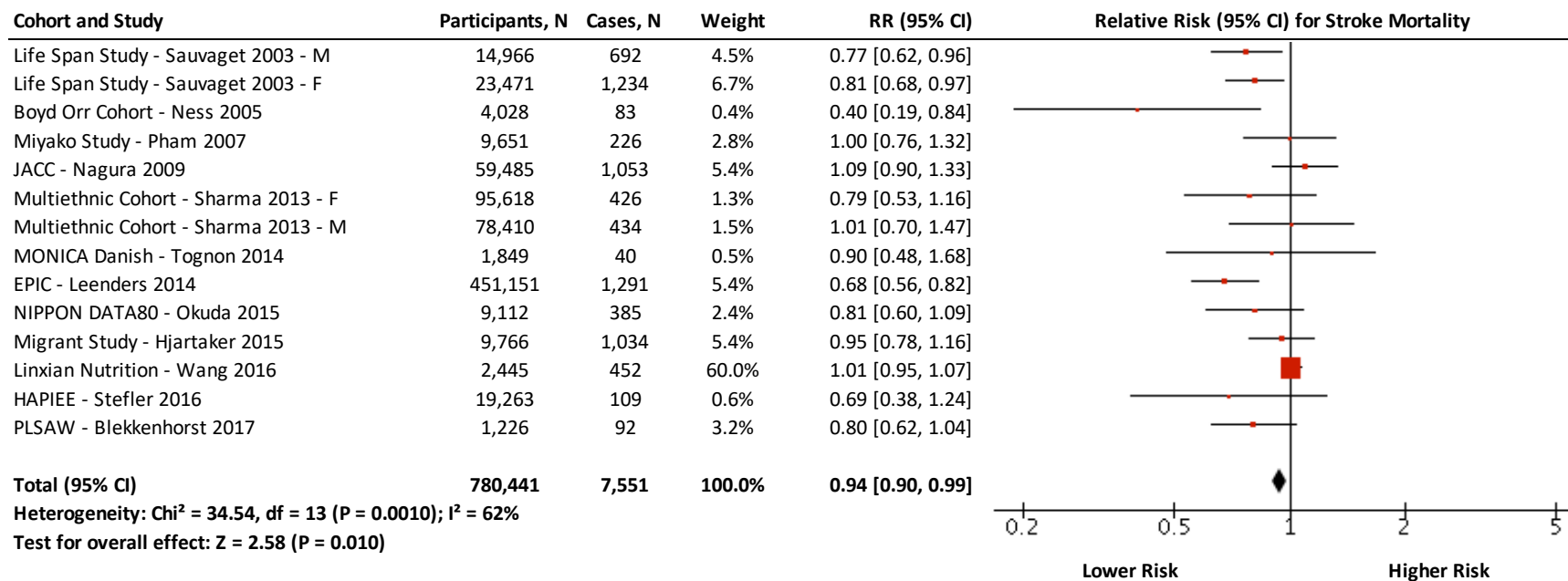


Figure S131. Relation between fruit intake and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

VEGETABLES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

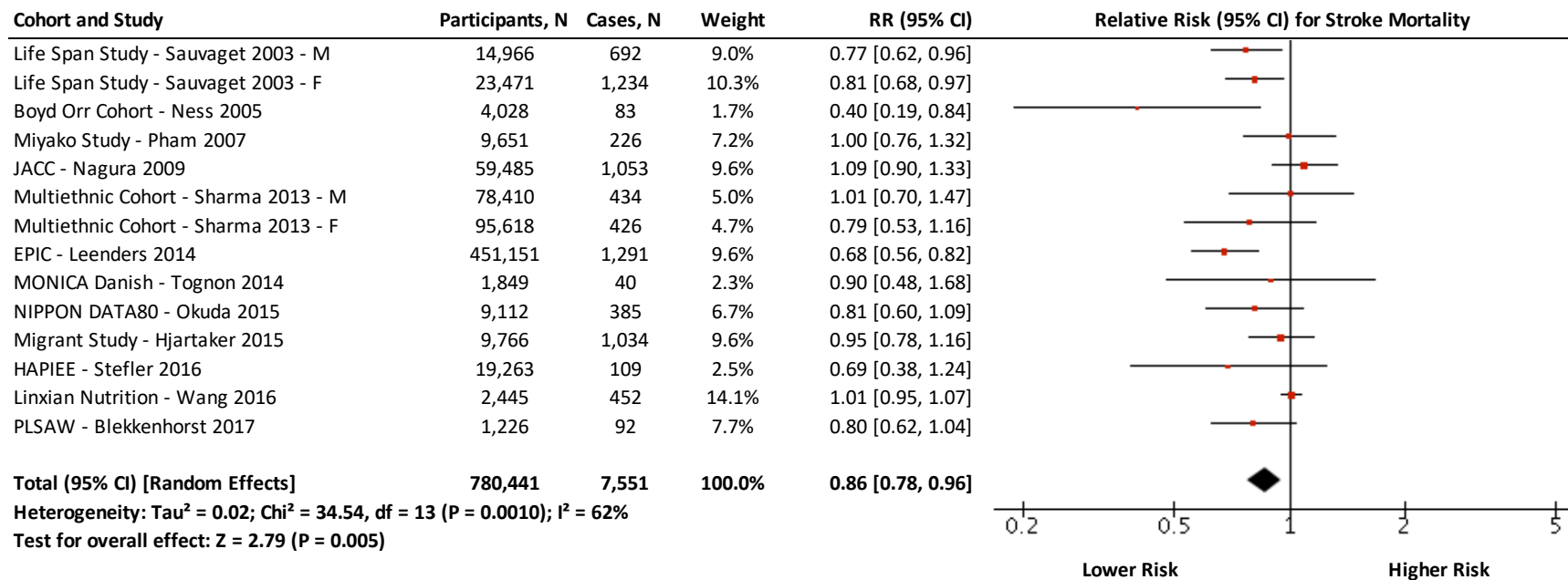


Figure S132. Relation between intake of vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

BANANAS AND STROKE MORTALITY

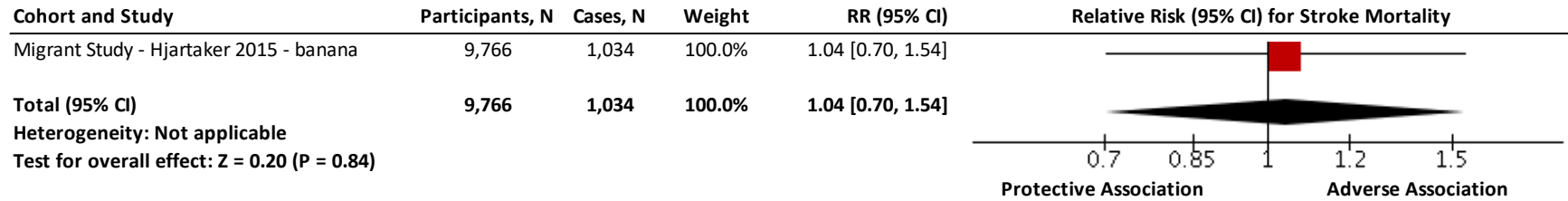
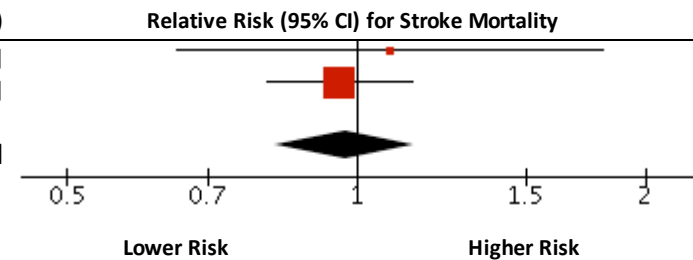


Figure S133. Relation between intake of bananas and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the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

BERRIES AND STROKE MORTALITY

A. Fixed Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015	30,458	148	10.7%	1.08 [0.65, 1.80]
Migrant Study - Hjartaker 2015	9,766	1,034	89.3%	0.96 [0.81, 1.15]
Total (95% CI)	40,224	1,182	100.0%	0.97 [0.82, 1.15]
Heterogeneity: Chi² = 0.19, df = 1 (P = 0.66); I² = 0%				
Test for overall effect: Z = 0.32 (P = 0.75)				



B. Random Effects

Cohort and Study	Participants, N	Cases, N	Weight	RR (95% CI)
UK Women's Cohort - Lai 2015	30,458	148	10.7%	1.08 [0.65, 1.80]
Migrant Study - Hjartaker 2015	9,766	1,034	89.3%	0.96 [0.81, 1.15]
Total (95% CI) [Random Effects]	40,224	1,182	100.0%	0.97 [0.82, 1.15]
Heterogeneity: Tau² = 0.00; Chi² = 0.19, df = 1 (P = 0.66); I² = 0%				
Test for overall effect: Z = 0.32 (P = 0.75)				

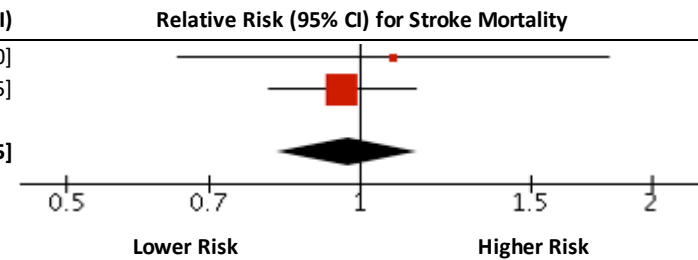
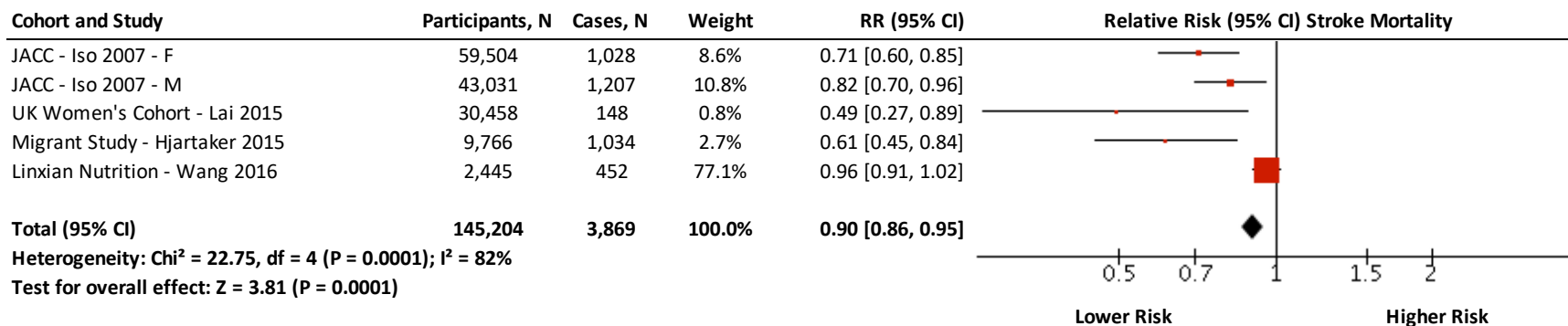


Figure S134. Relation between intake of berries and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of $p < 0.10$, and quantified by I², with values $\geq 50\%$ indicating substantial heterogeneity.

CITRUS FRUIT AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

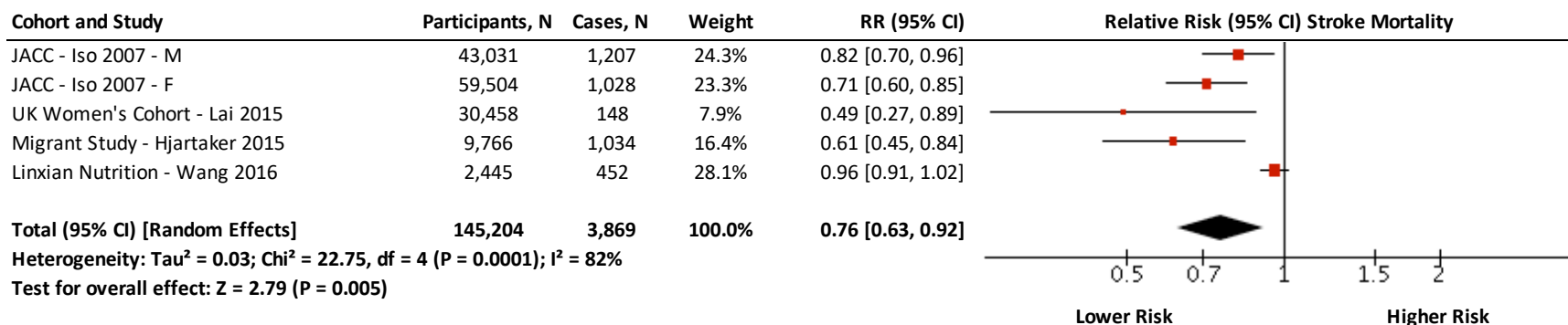


Figure S135. Relation between intake of citrus fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

DRIED FRUIT AND STROKE MORTALITY

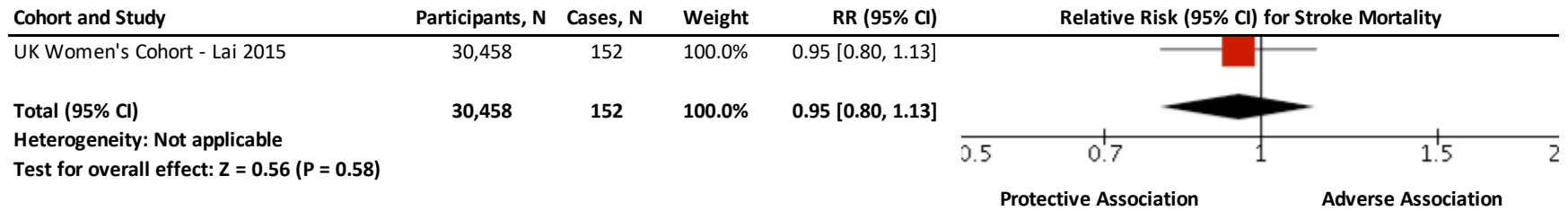
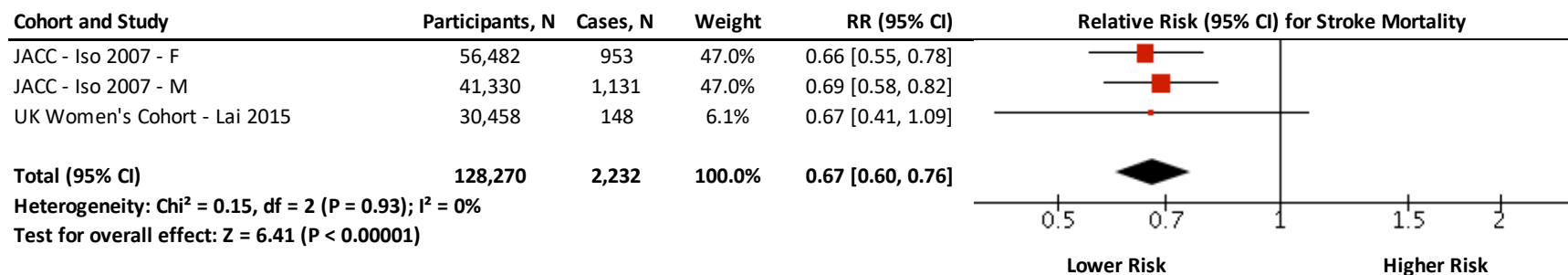


Figure S136. Relation between intake of dried fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

FRUIT JUICE AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

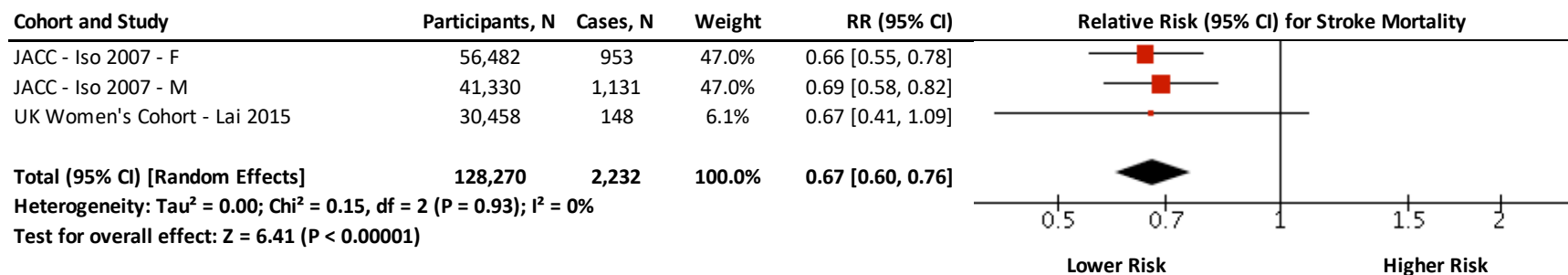
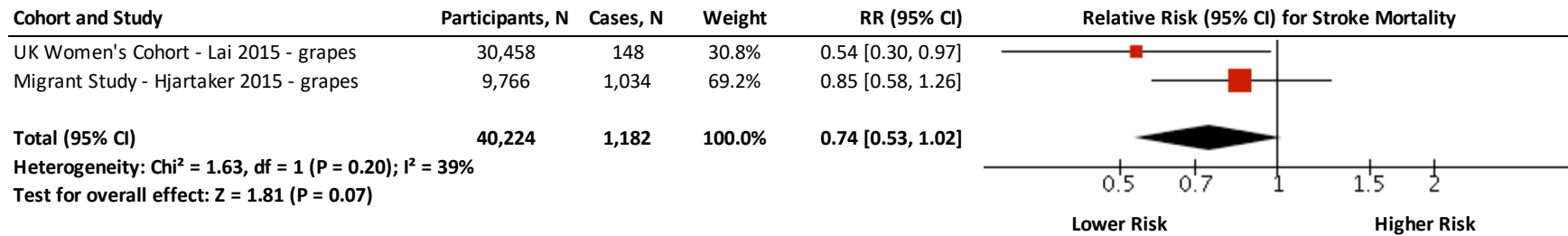


Figure S137. Relation between intake of fruit juice and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

GRAPES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

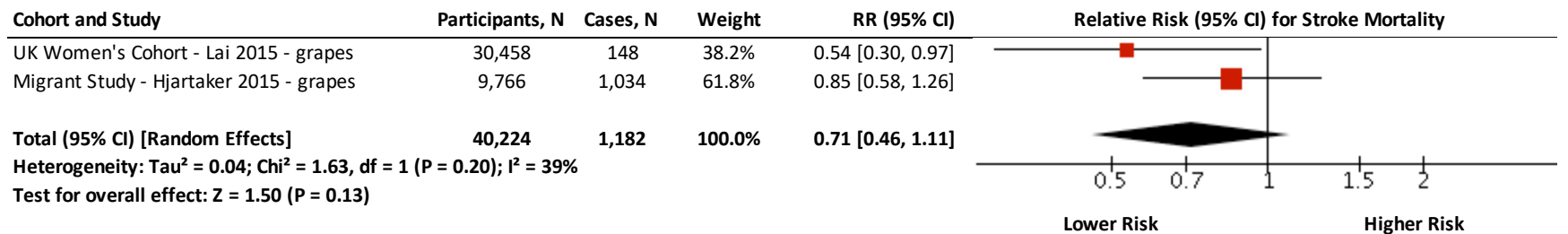
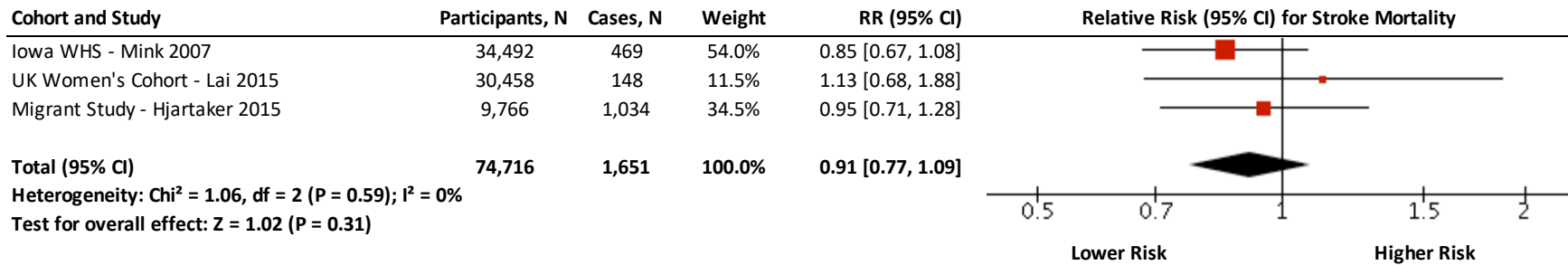


Figure S138. Relation between intake of grapes and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models.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

POMMES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

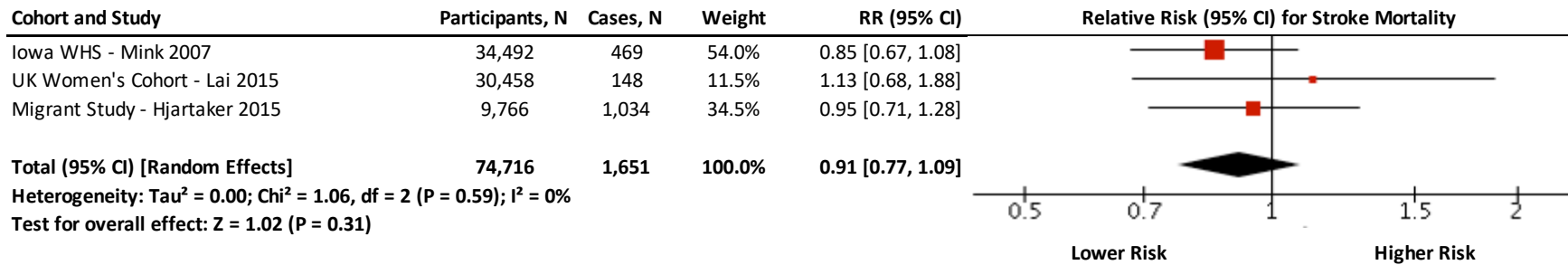
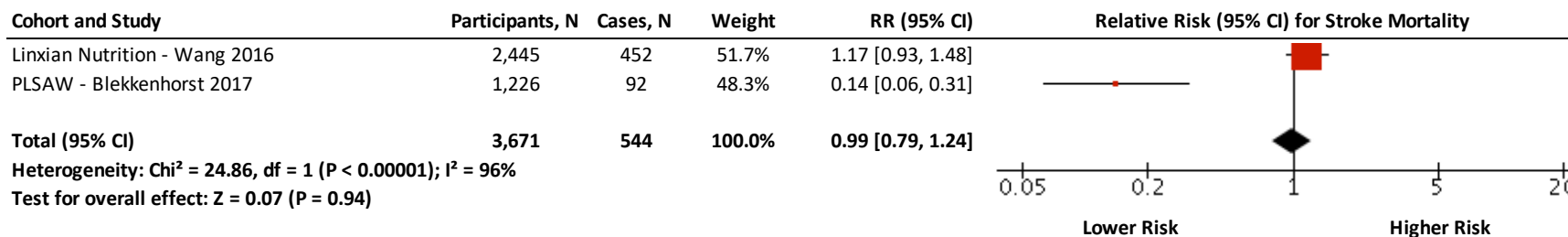


Figure S139. Relation between intake of pomes fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects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

ALLIUM AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

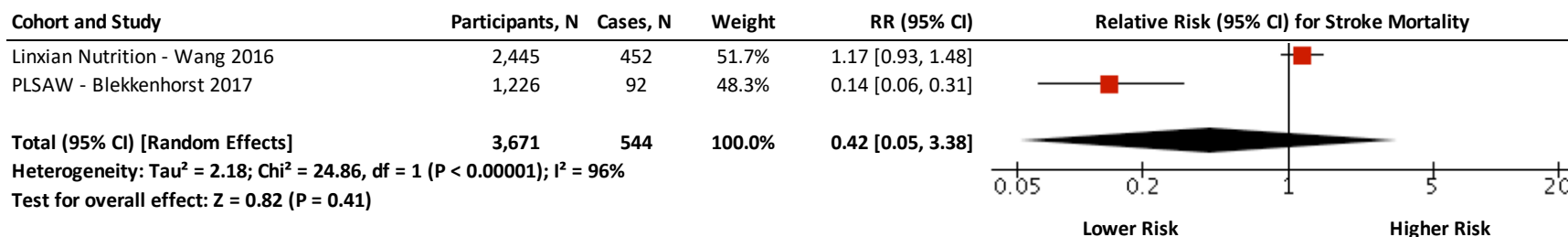


Figure S140. Relation between intake of allium vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

CARROTS AND STROKE MORTALITY

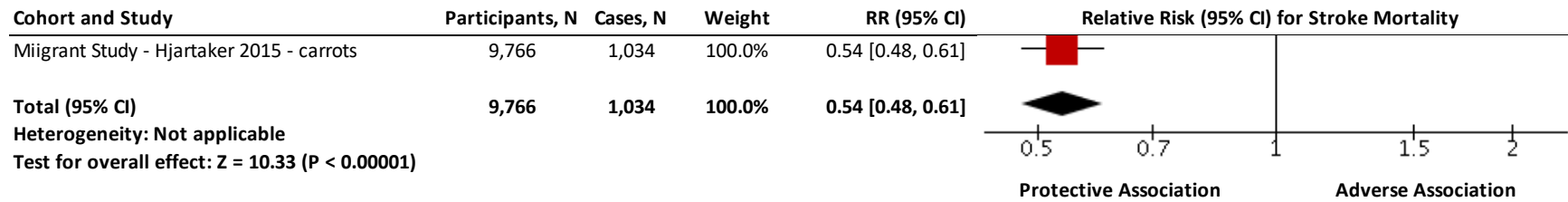
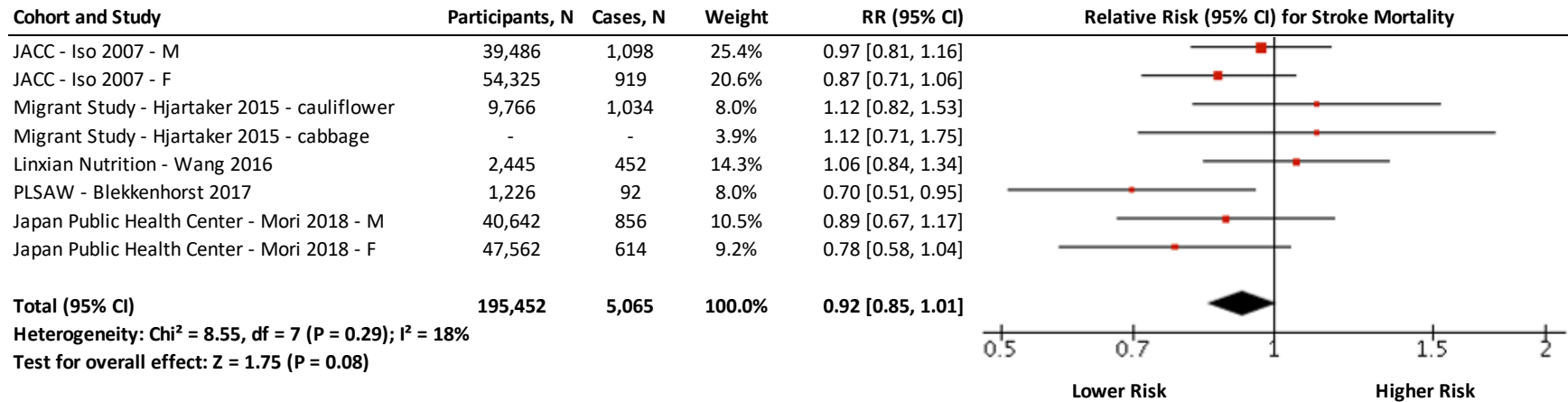


Figure S141. Relation between intake of carrots and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond. Inter-study heterogeneity was assessed using the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

CRUCIFEROUS VEGETABLES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

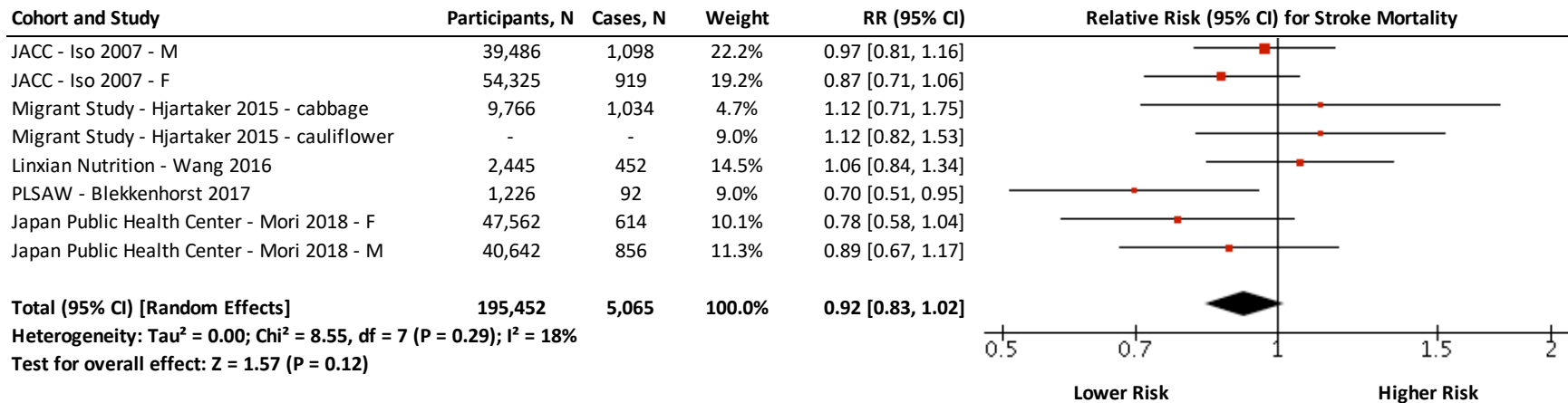
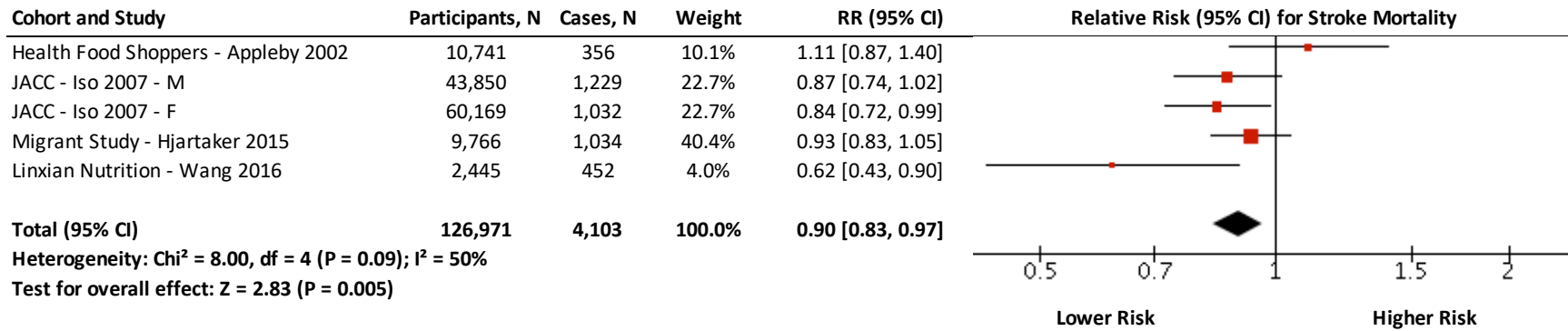


Figure S142. Relation between intake of cruciferous vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

GREEN LEAFY VEGETABLES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

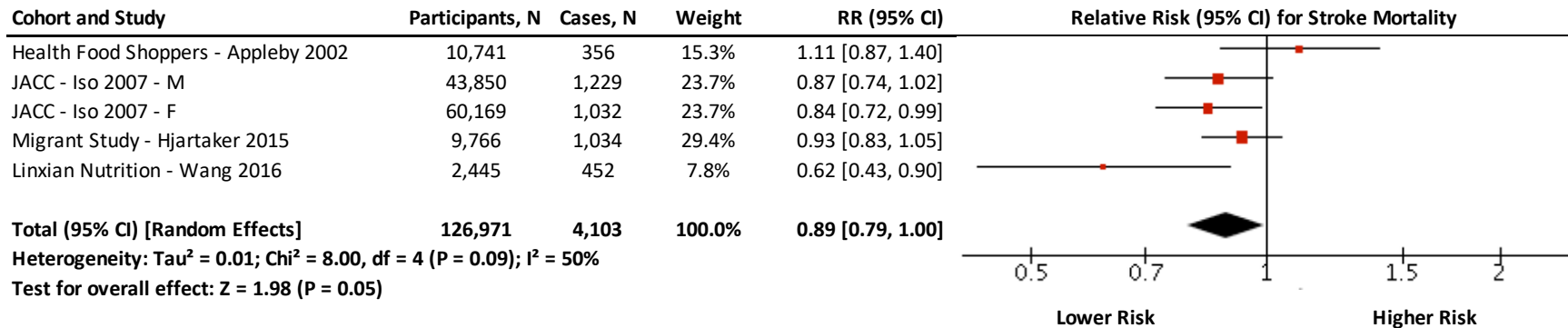
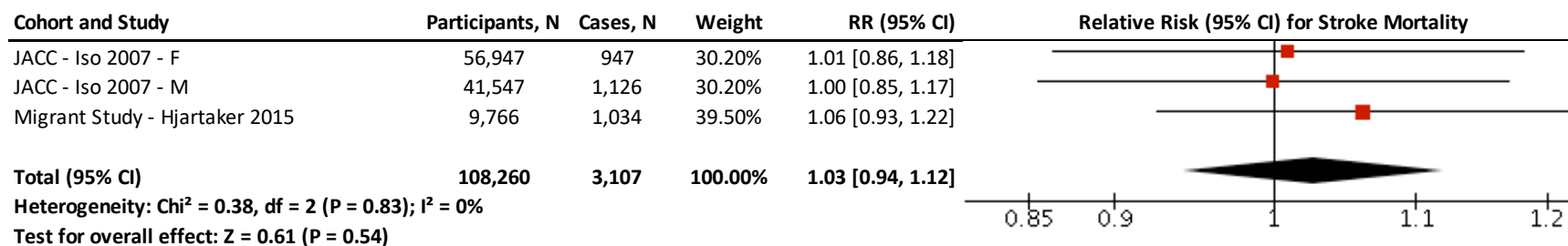


Figure S143. Relation between intake of green leafy vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random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

TOMATOES AND STROKE MORTALITY

A. Fixed Effects



B. Random Effects

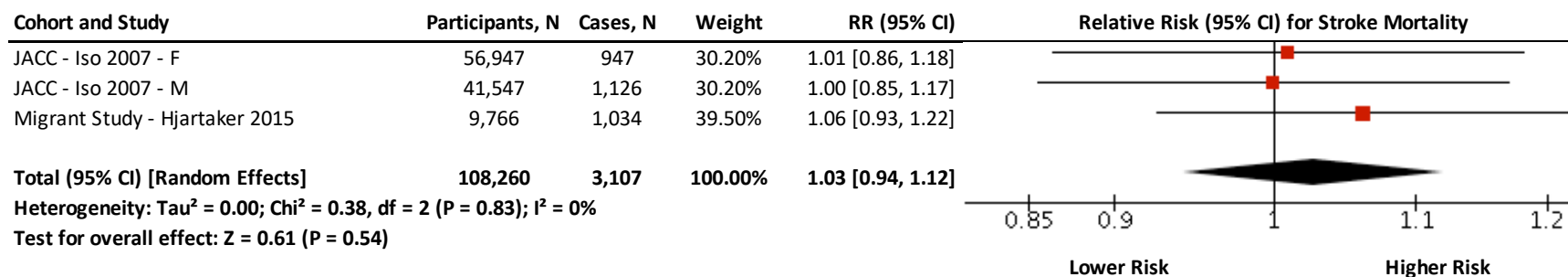
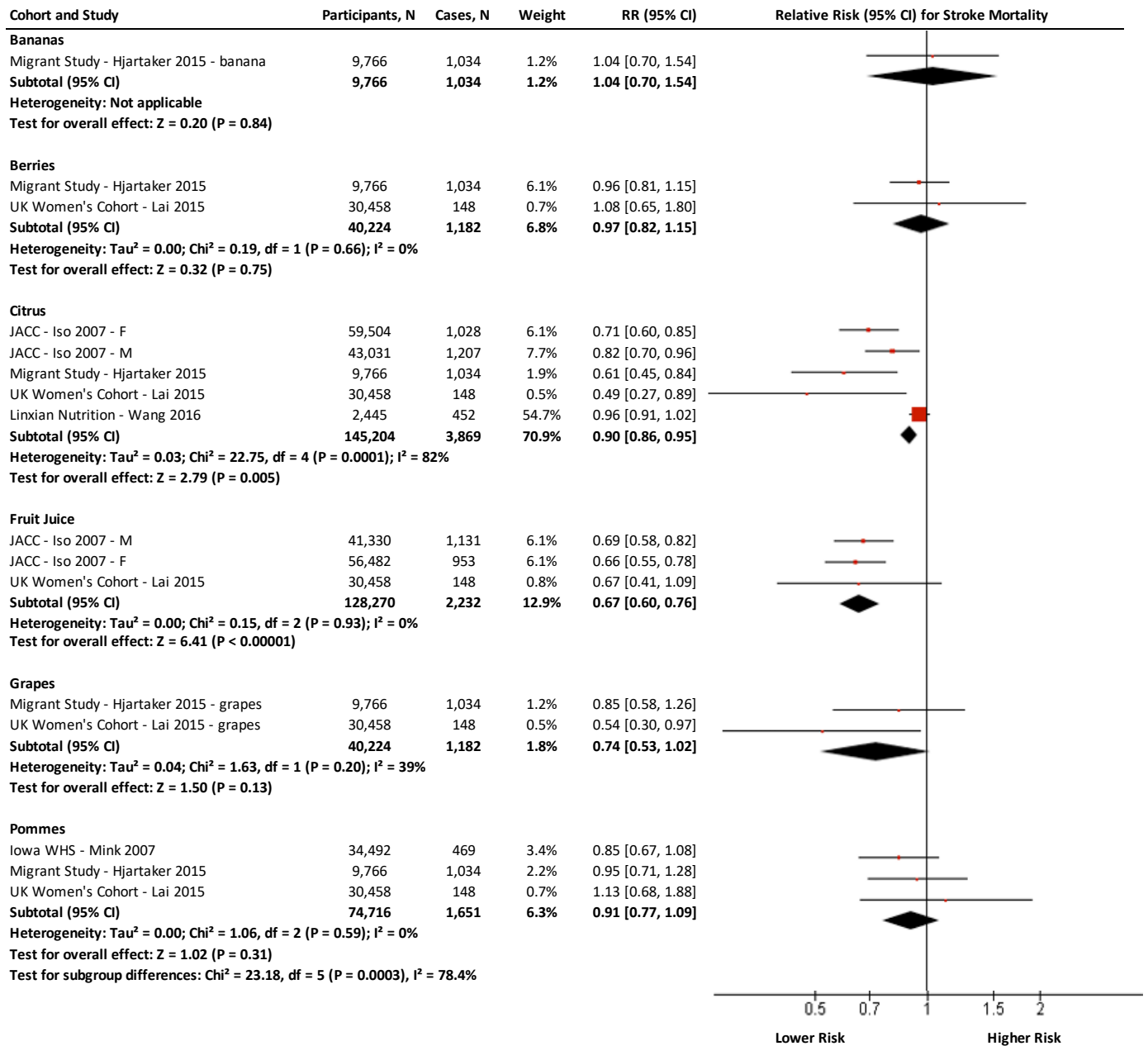


Figure S144. Relation between intake of tomatoes and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi^2) at a significance level of $p < 0.10$, and quantified by I^2 , with values $\geq 50\%$ indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

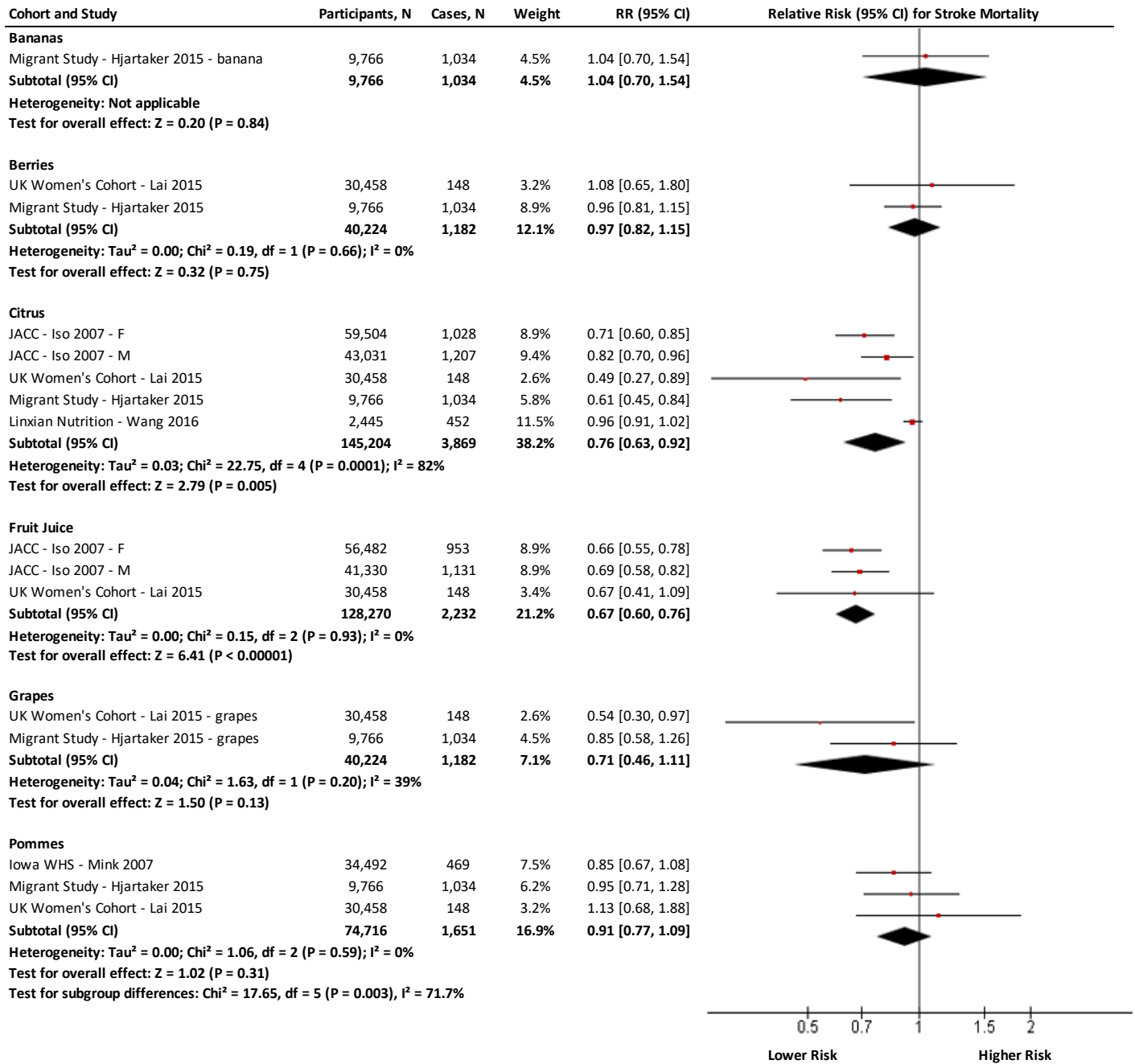
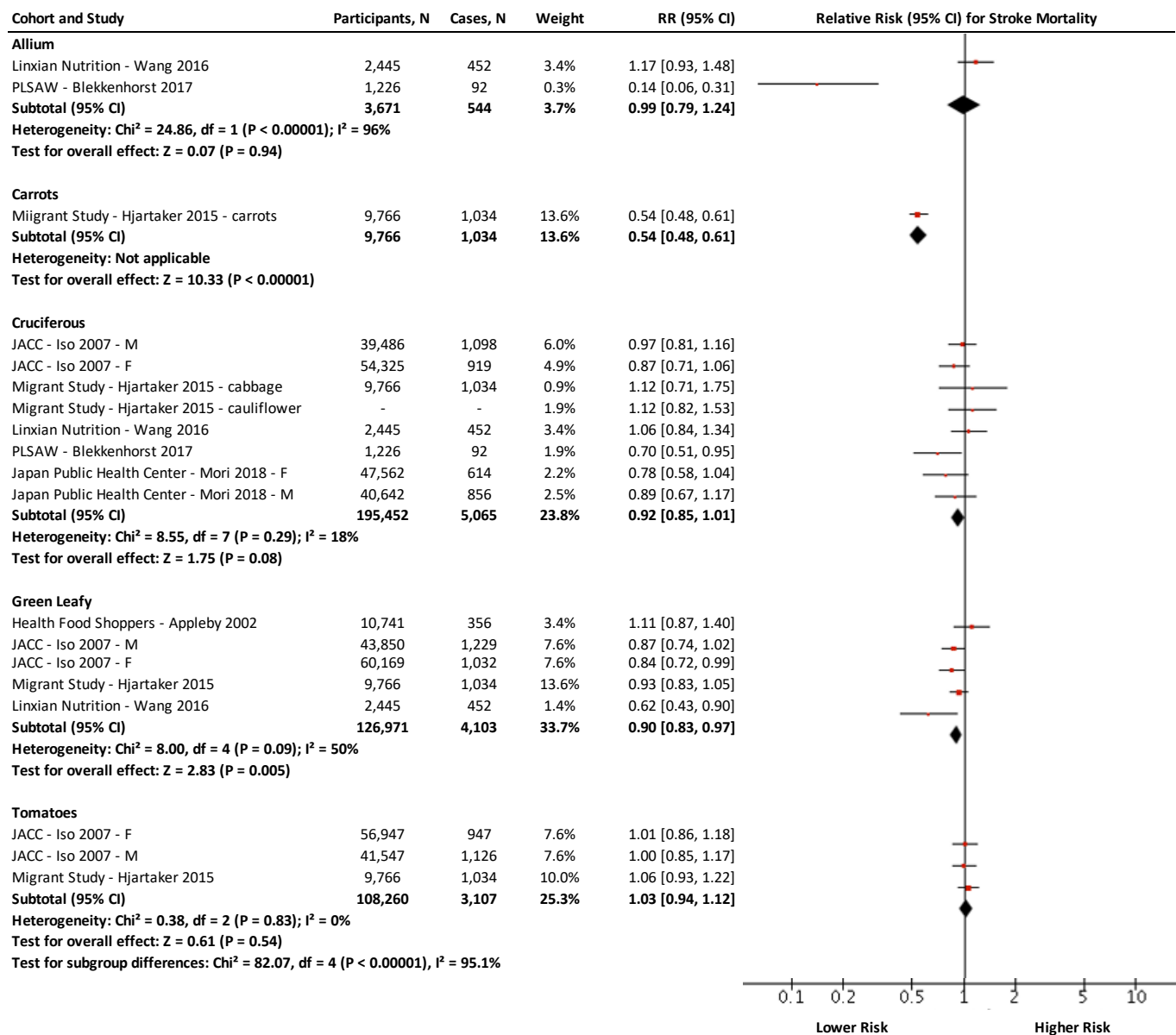


Figure S145. Relation between sources of fruit and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

A. Fixed Effects



B. Random Effects

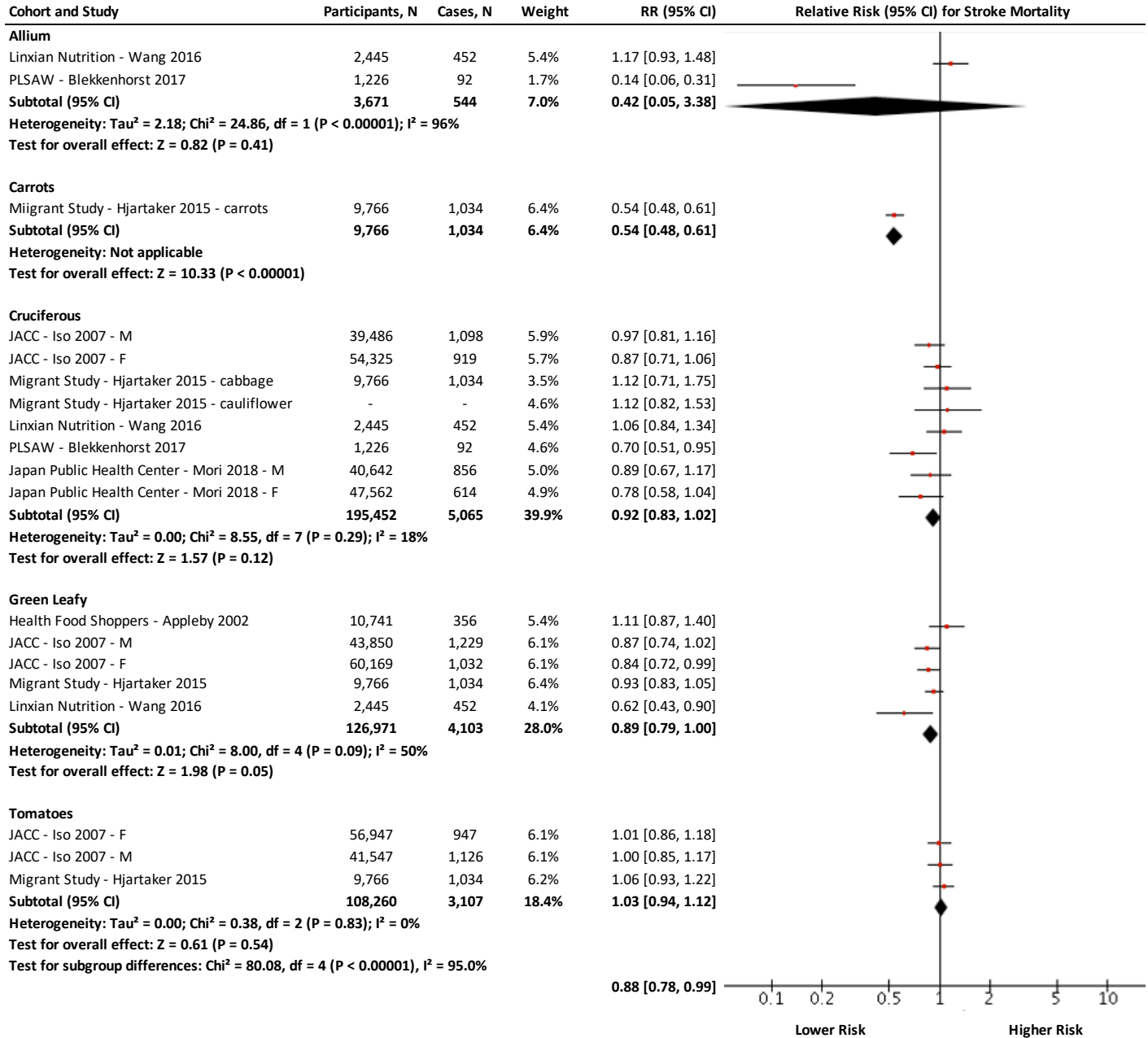


Figure S146. Relation between sources of vegetables and stroke mortality (highest vs. lowest level of intake). All results are presented as relative risk (RR) with 95% confidence intervals (95% CI). Pooled risk estimate is represented by the black diamond using (A) fixed effects and (B) random effects models. Inter-study heterogeneity was assessed using the Cochran Q statistic (Chi²) at a significance level of p<0.10, and quantified by I², with values ≥ 50% indicating substantial heterogeneity.

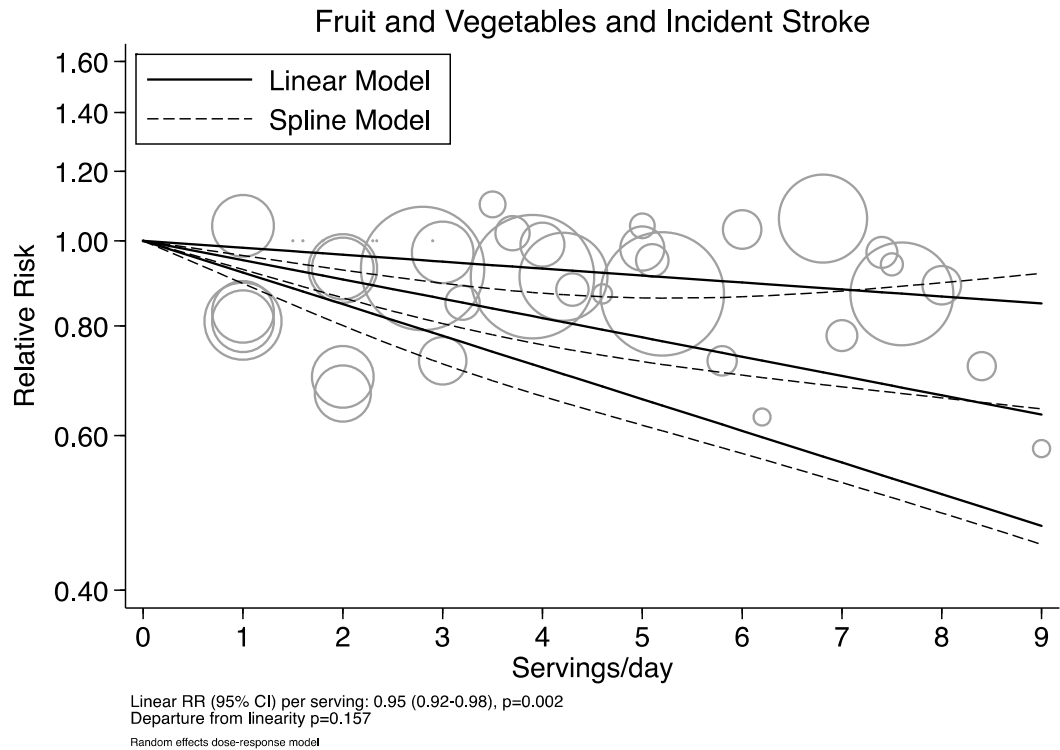


Figure S147. Linear and cubic-spline dose-response relation between increasing fruit and vegetable intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

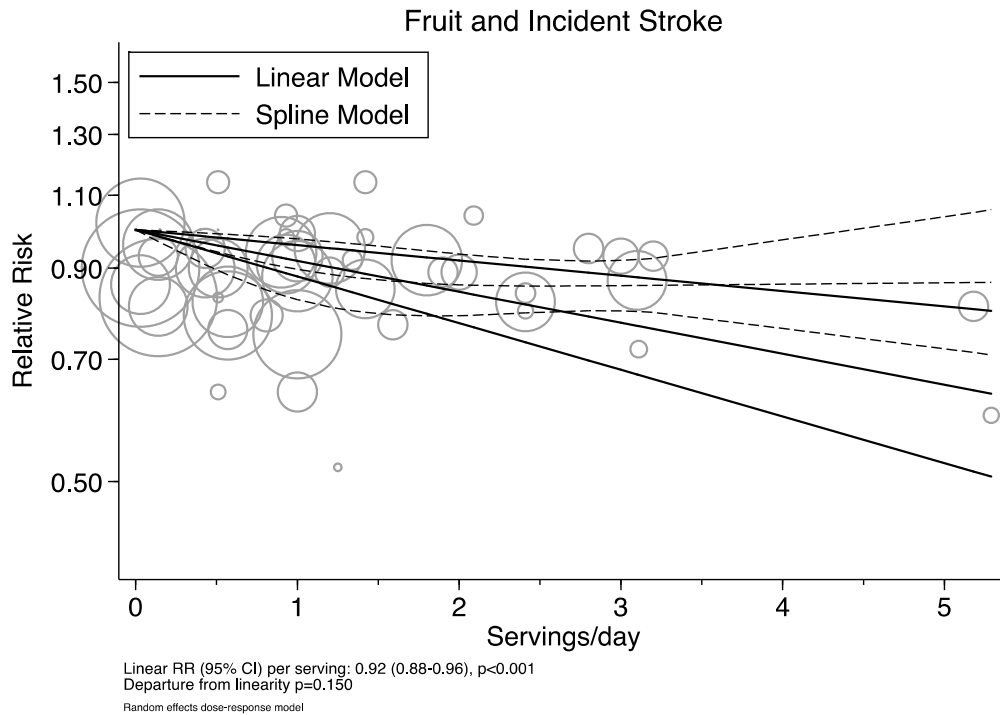


Figure S148. Linear and cubic-spline dose-response relation between increasing fruit intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

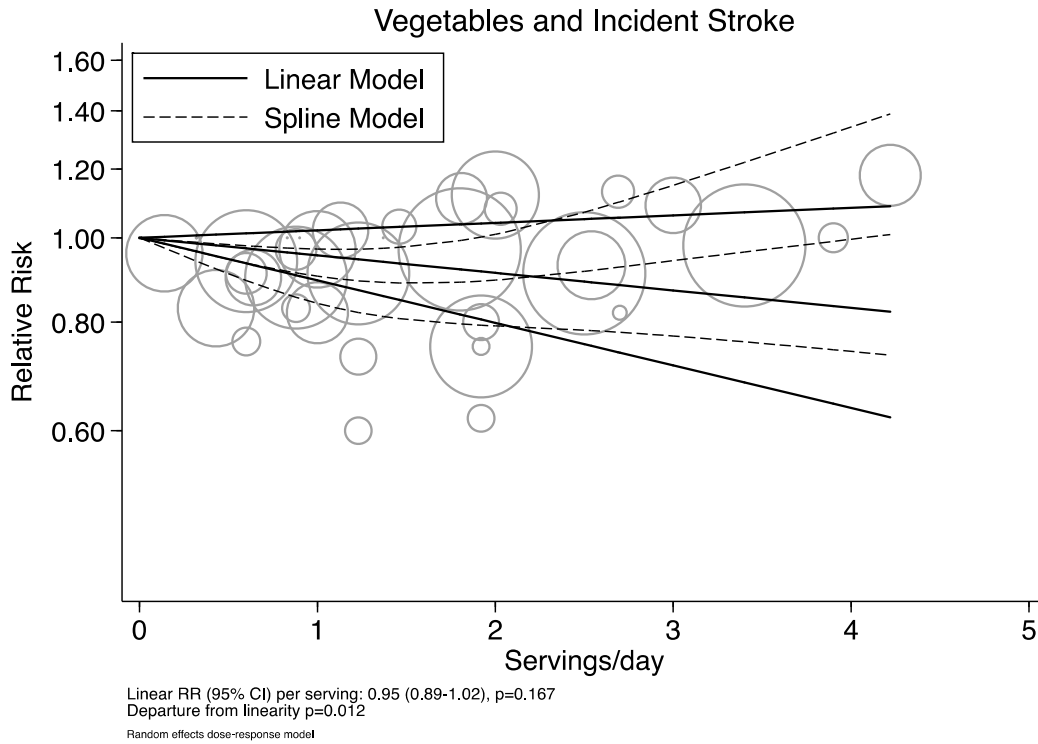


Figure S149. Linear and cubic-spline dose-response relation between increasing intake of vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

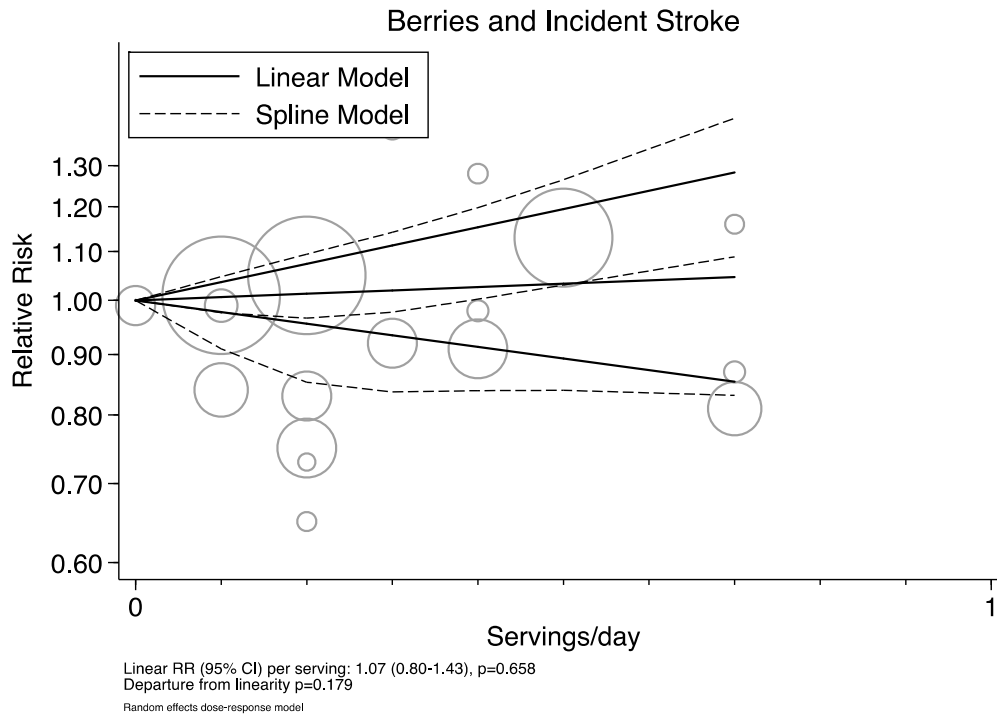


Figure S150. Linear and cubic-spline dose-response relation between increasing berries intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

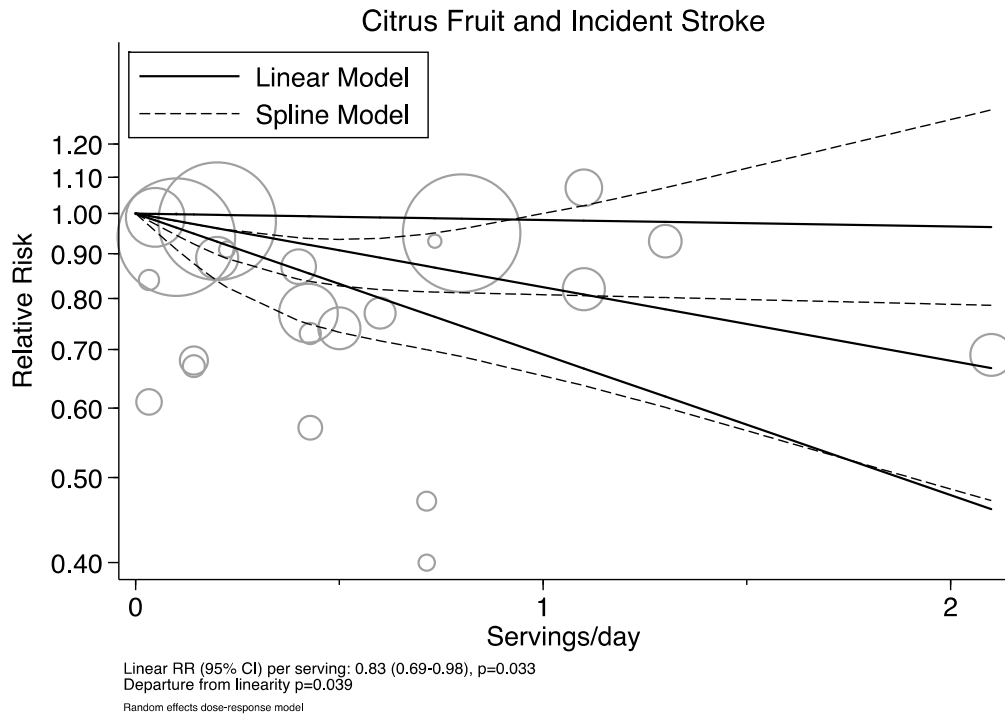


Figure S151. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

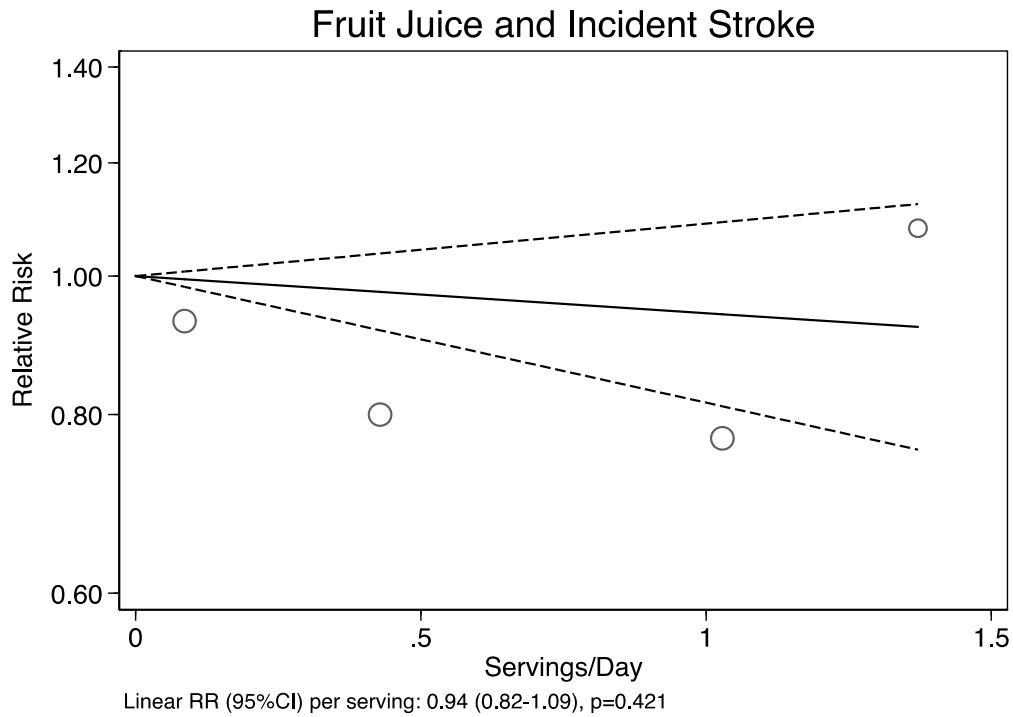


Figure S152. Linear and cubic-spline dose-response relation between increasing fruit juice intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

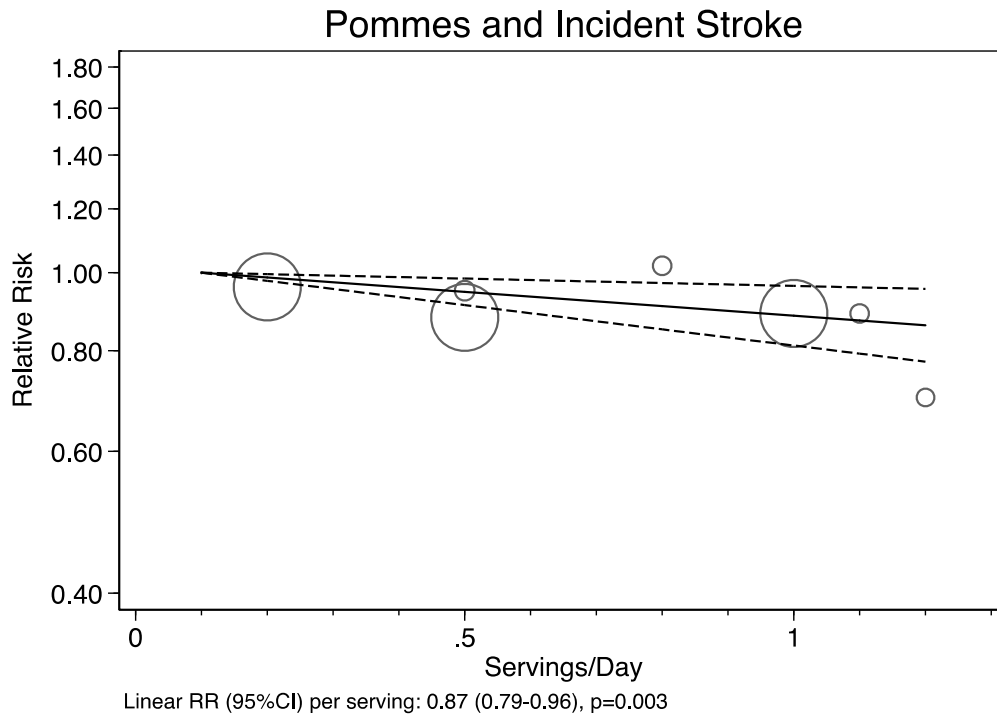


Figure S153. Linear dose-response relation between increasing pommes intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

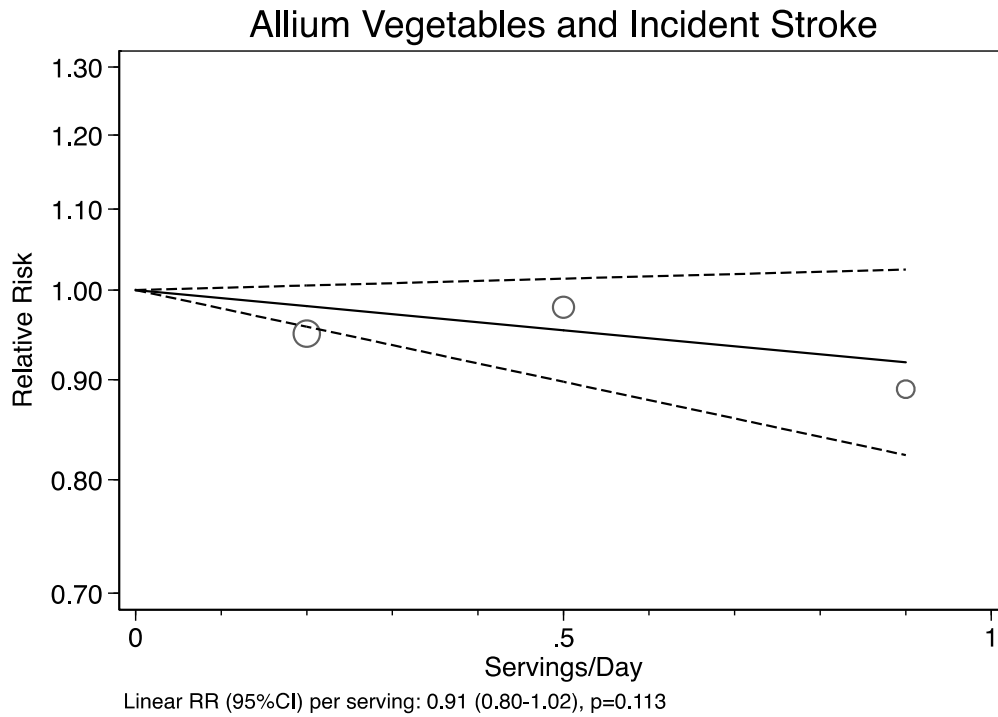


Figure S154 Linear dose-response relation between increasing intake of allium vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

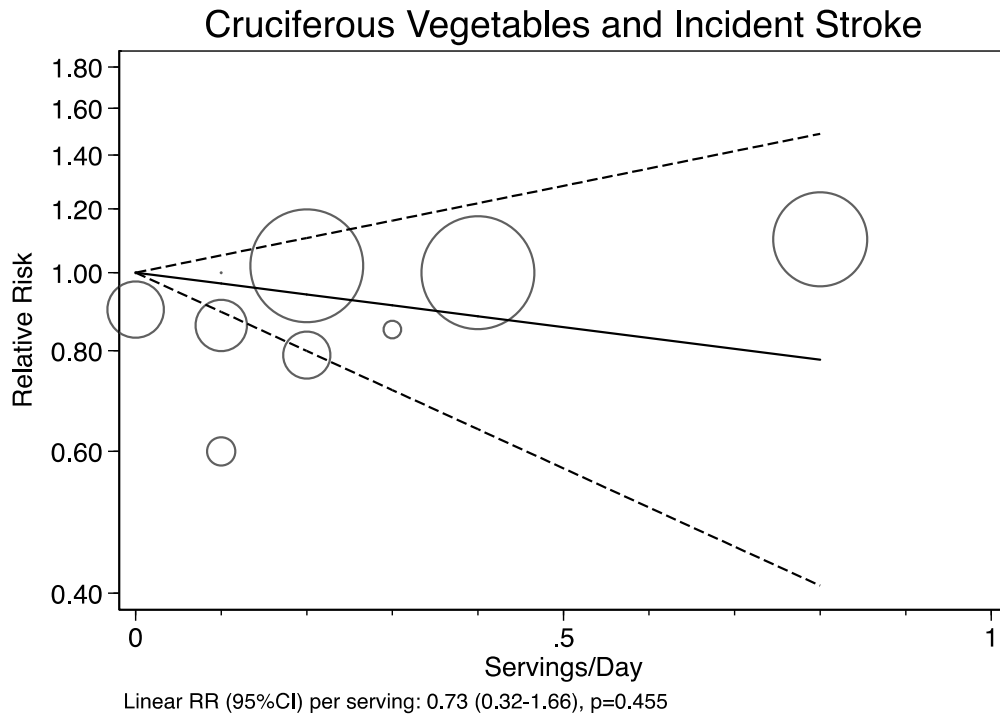


Figure S155. Linear dose-response relation between increasing intake of cruciferous vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

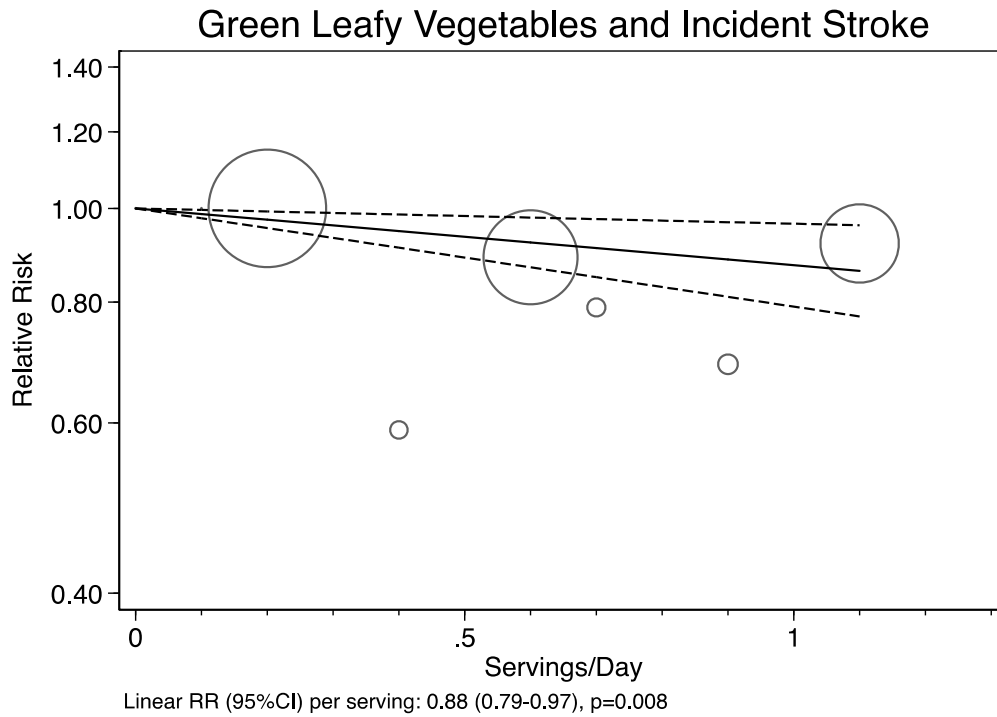


Figure S156. Linear dose-response relation between increasing intake of green leafy vegetables and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

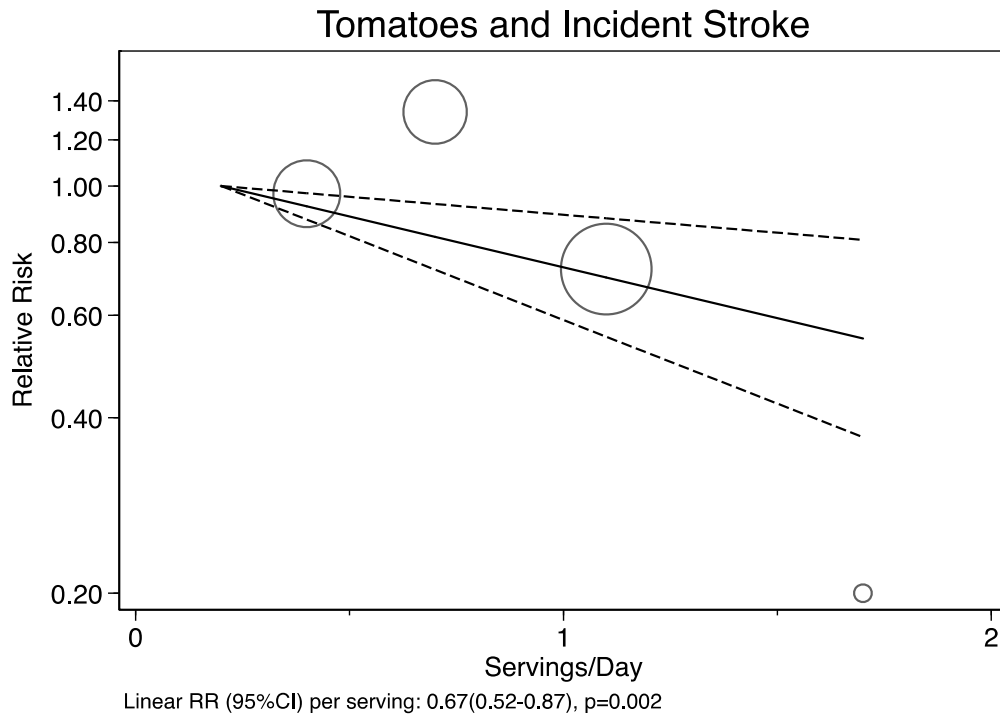


Figure S157. Linear dose-response relation between increasing tomato intake and incidence of stroke. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

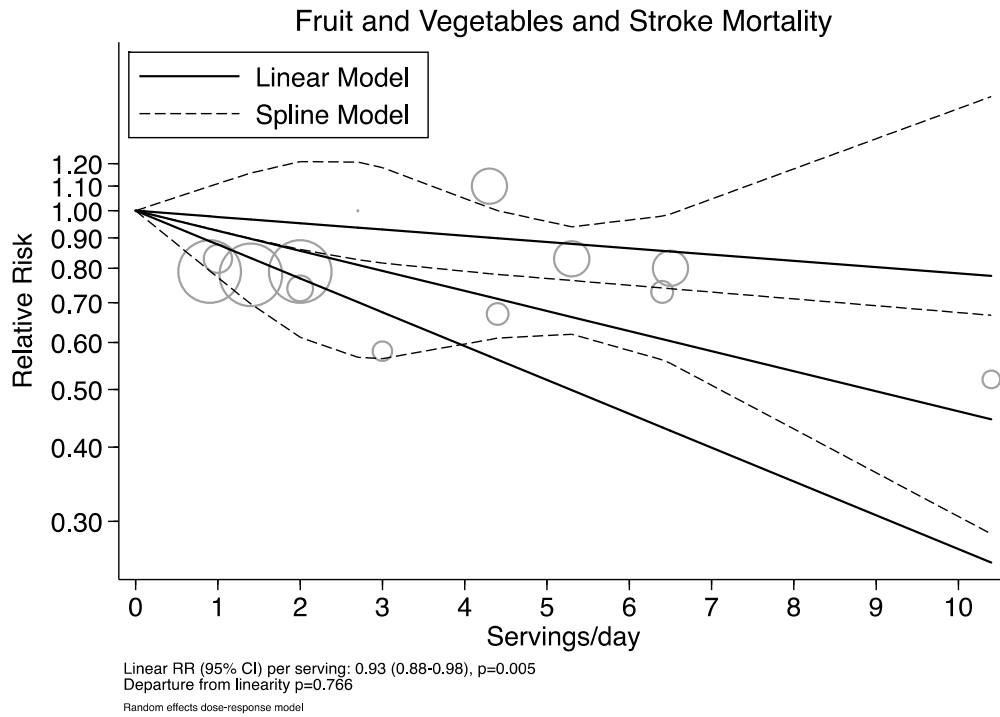


Figure S158. Linear dose-response relation between increasing fruit and vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

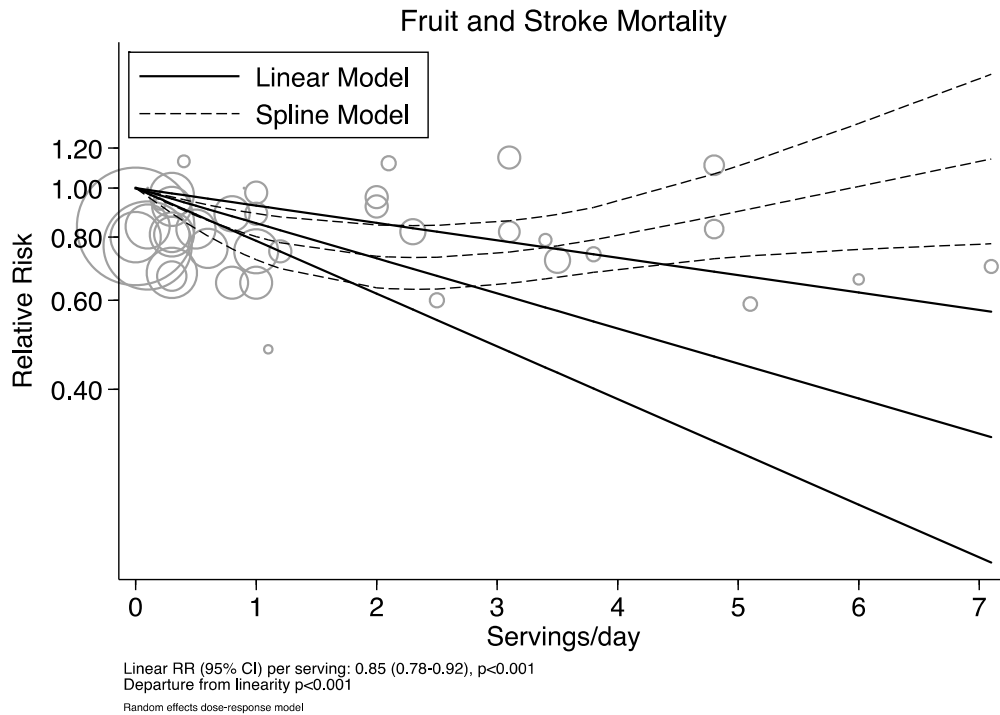


Figure S159. Linear and cubic-spline dose-response relation between increasing fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

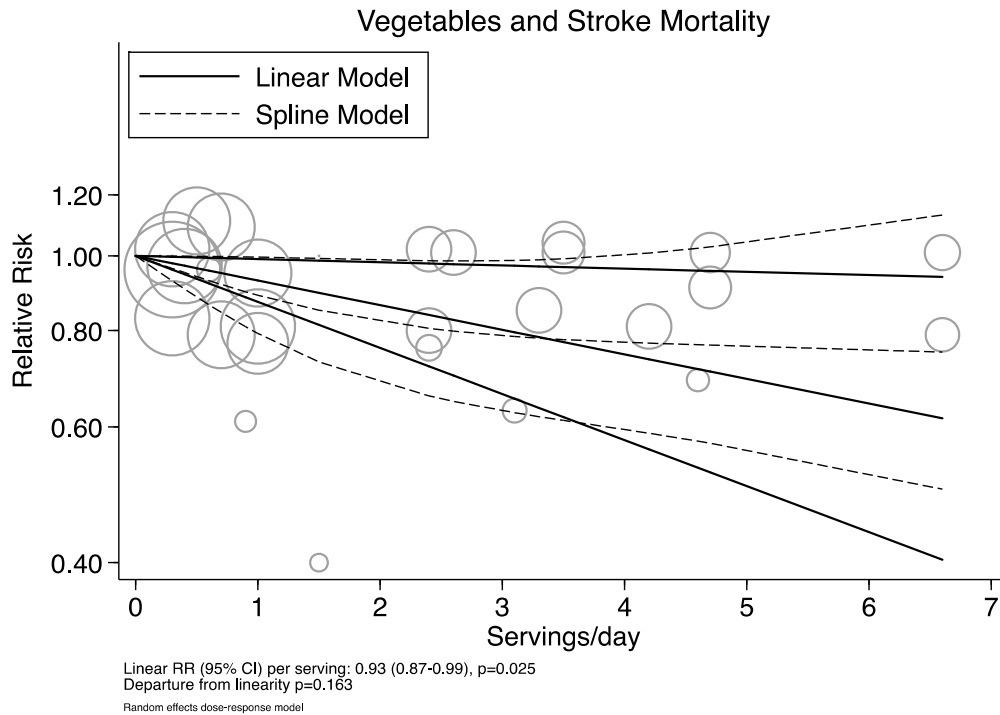


Figure S160. Linear and cubic-spline dose-response relation between increasing intake of vegetables and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

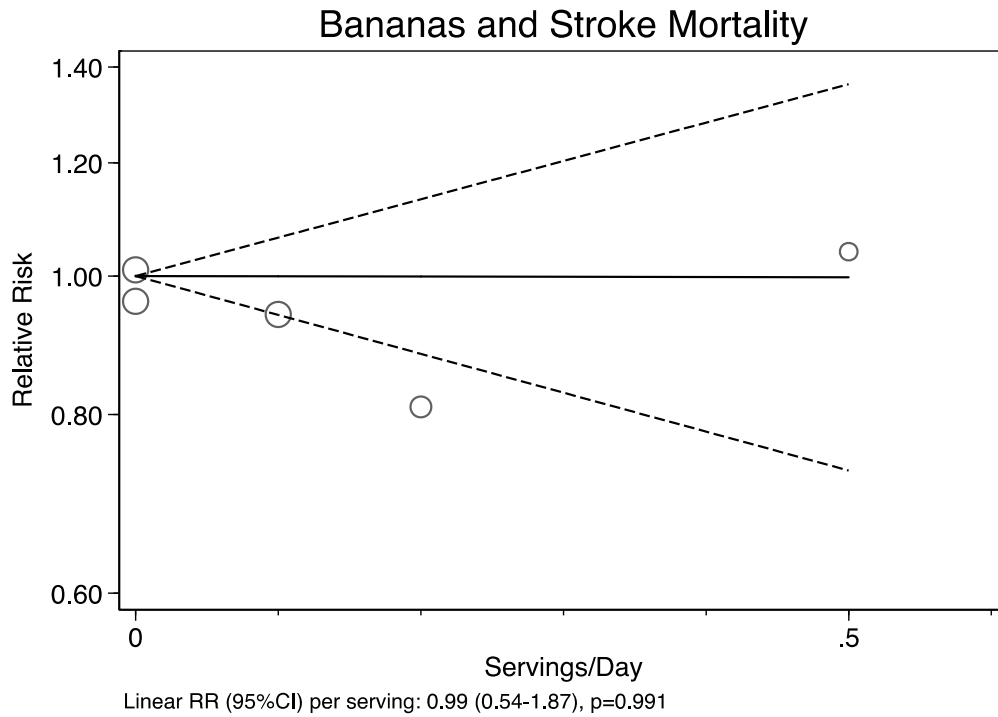


Figure S161 Linear dose-response relation between increasing banana intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

Berries and Stroke Mortality

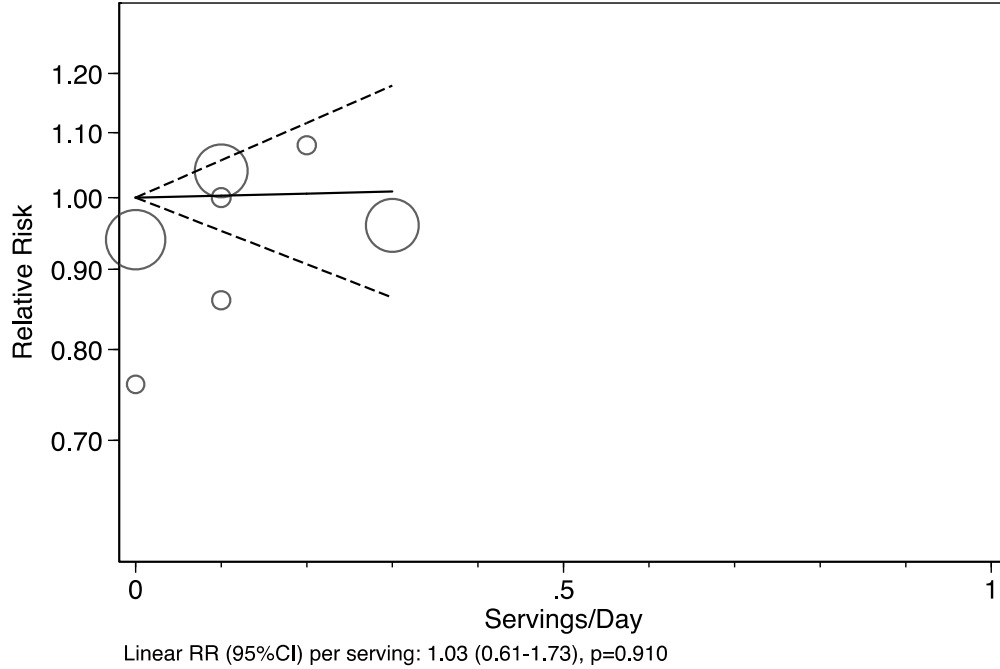


Figure S162. Linear dose-response relation between increasing berries intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

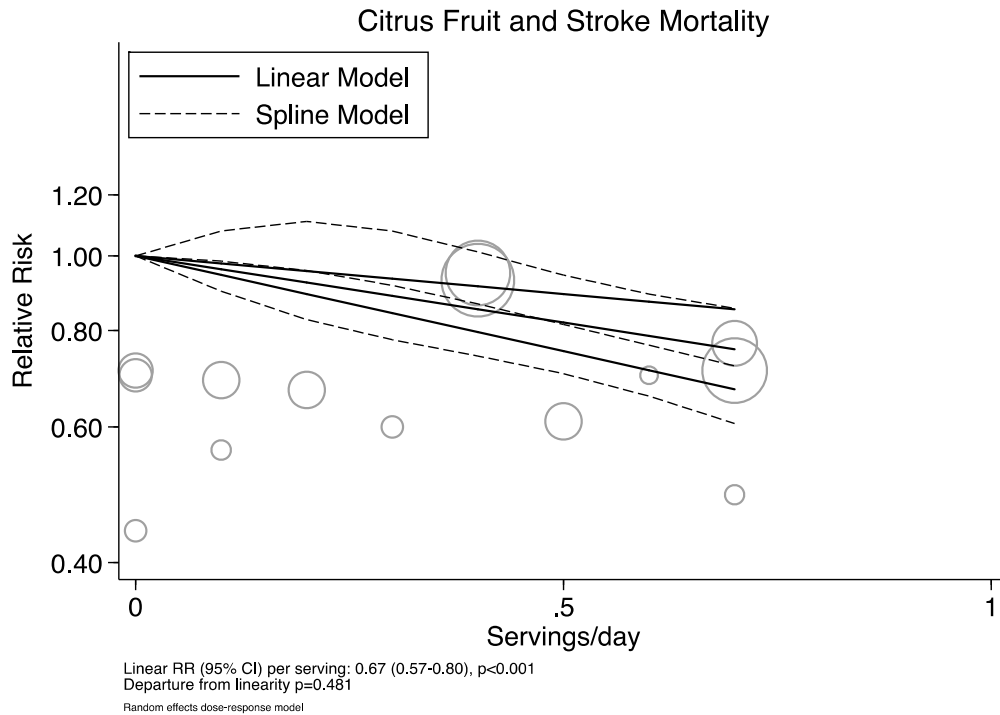


Figure S163. Linear and cubic-spline dose-response relation between increasing citrus fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

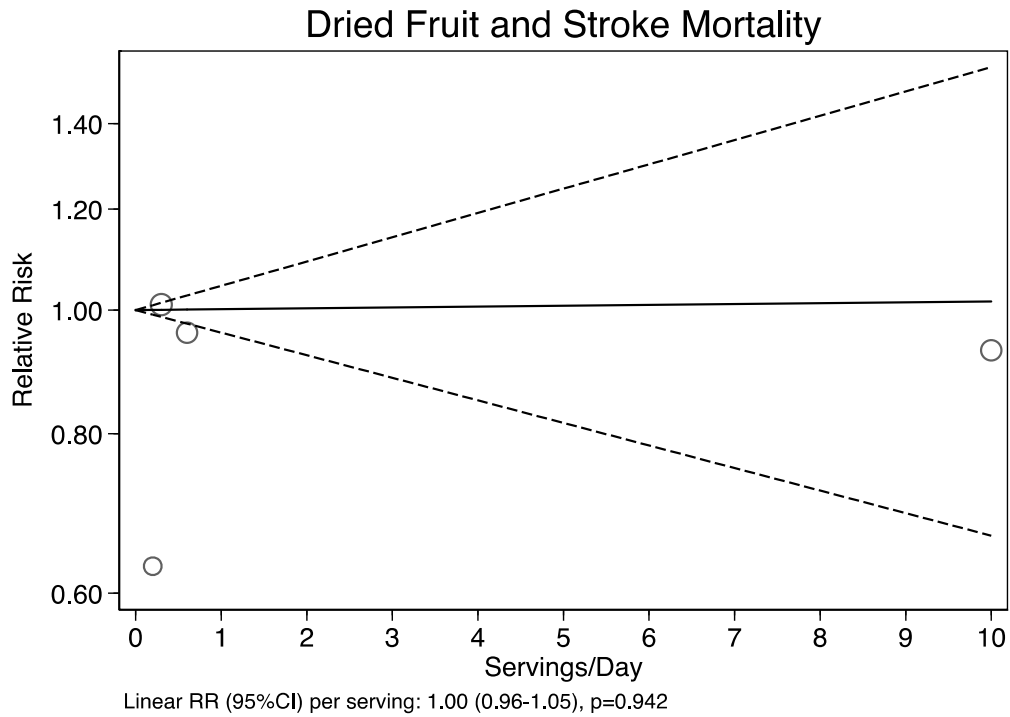


Figure S164. Linear and cubic-spline dose-response relation between increasing dried fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

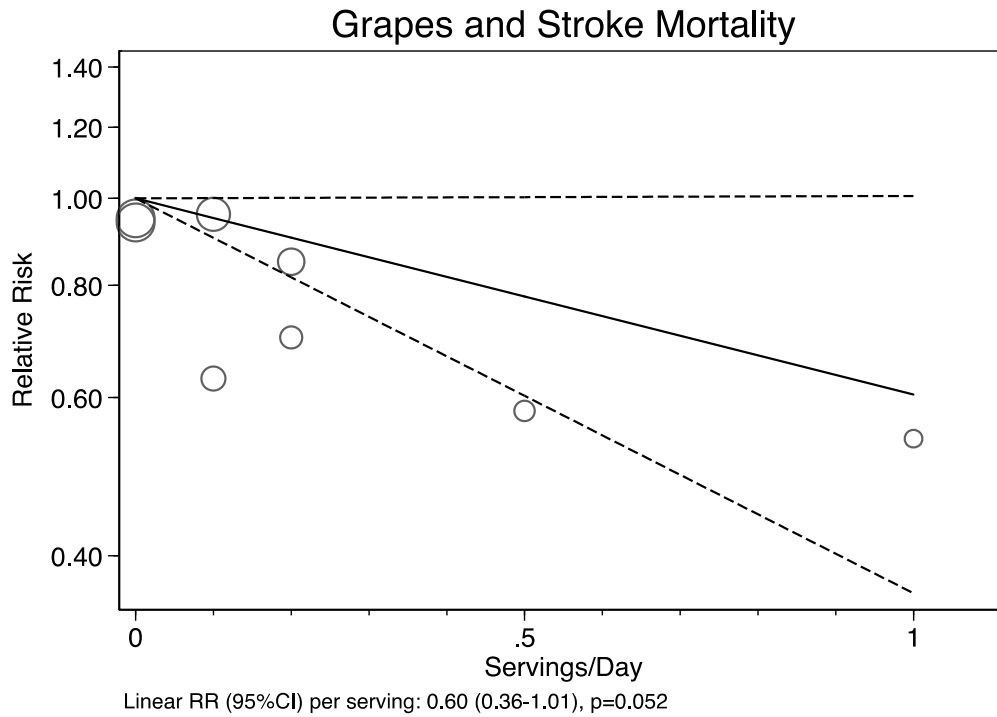


Figure S165. Linear dose-response relation between increasing grapes intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

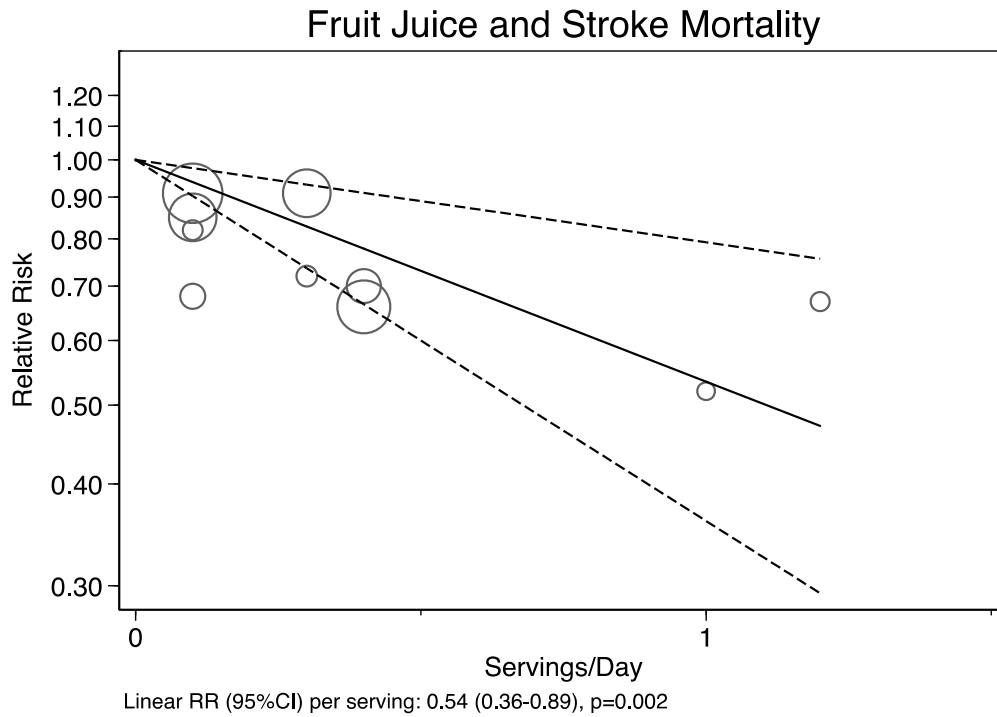


Figure S166. Linear dose-response relation between increasing fruit juice intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

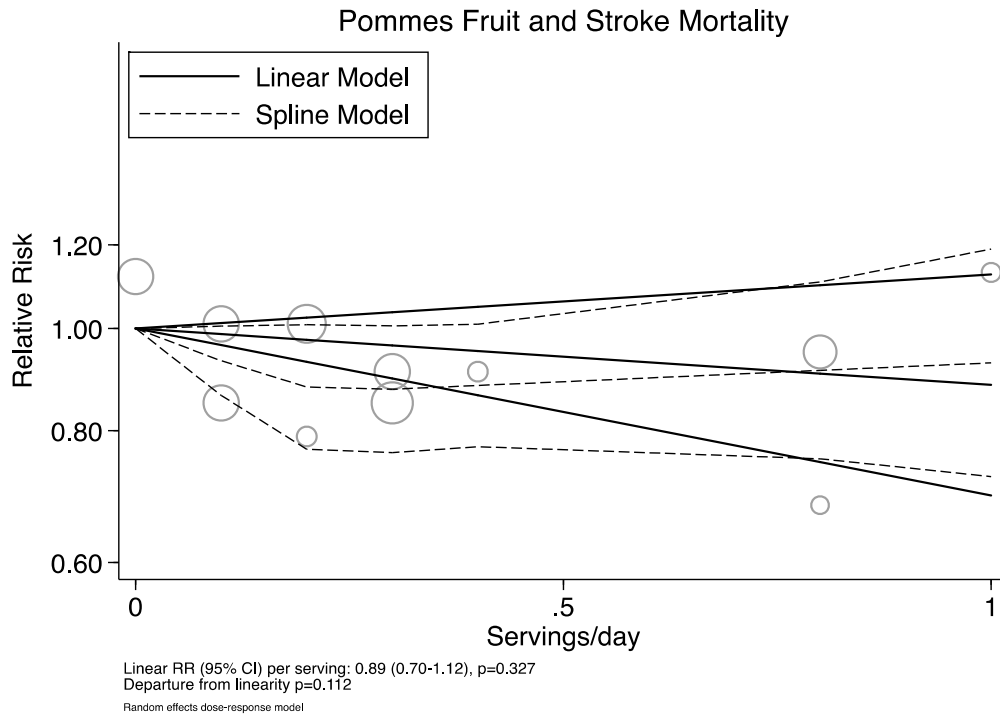


Figure S167. Linear and cubic-spline dose-response relation between increasing pomme fruit intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

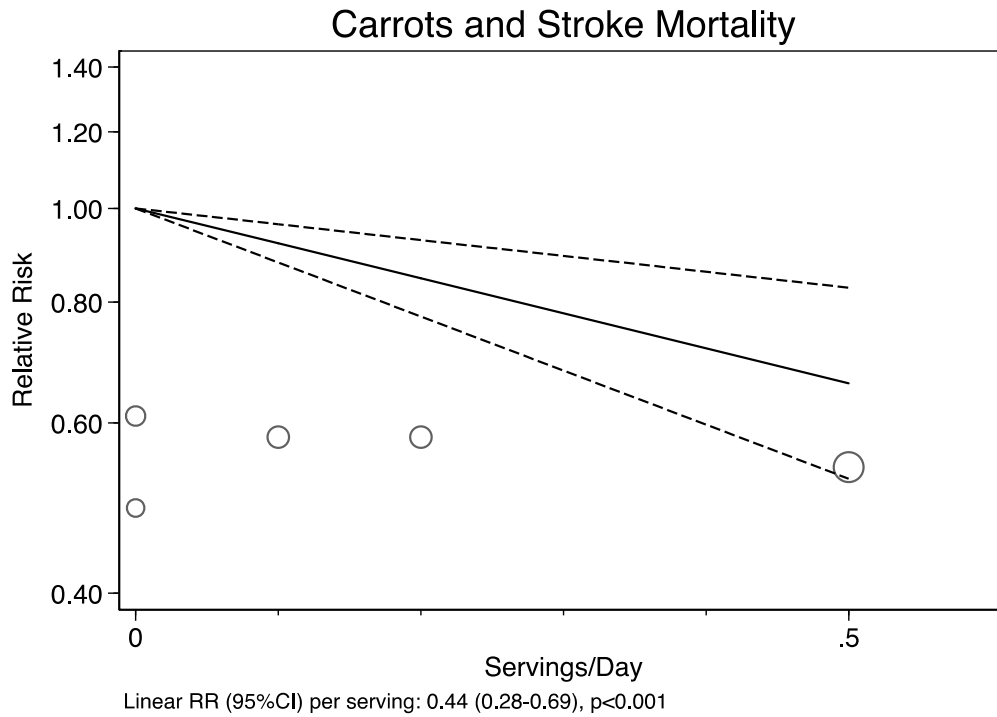


Figure S168. Linear dose-response relation between increasing intake of carrots and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

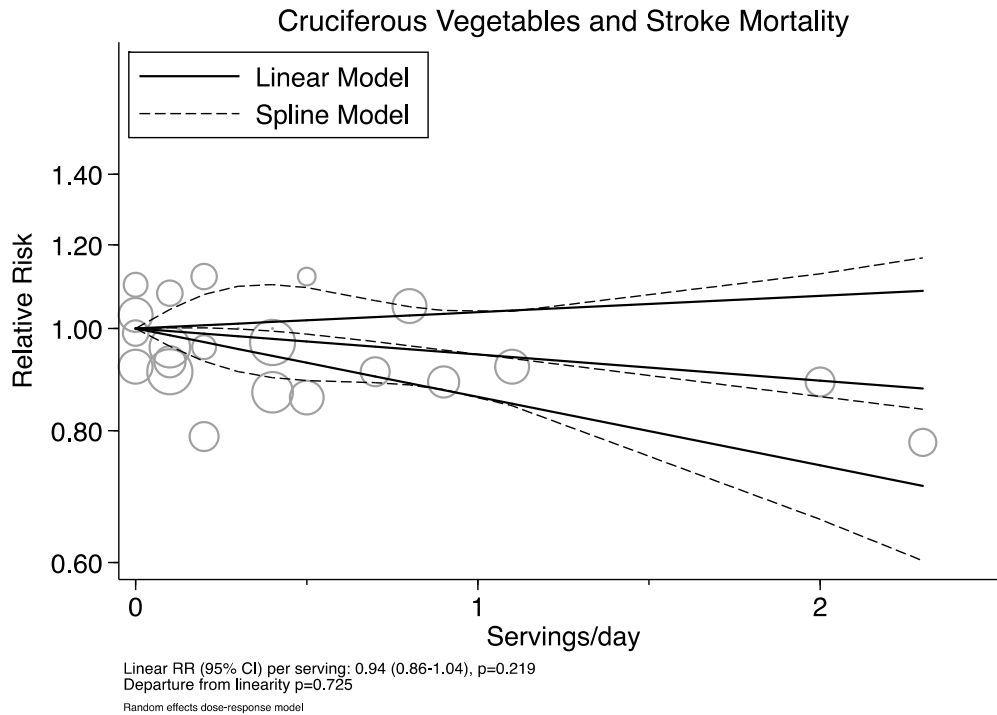


Figure S169. Linear and cubic-spline dose-response relation between increasing cruciferous vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

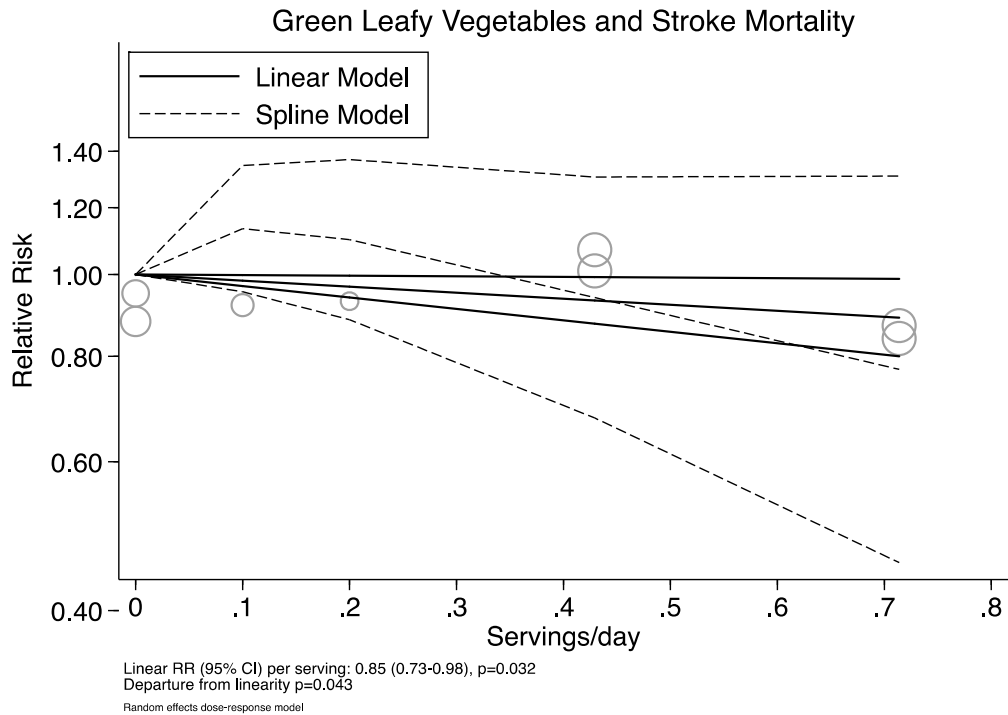


Figure S170. Linear and cubic-spline dose-response relation between increasing green leafy vegetable intake and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. Cubic spline data were modeled with fixed-effects restricted cubic spline with 3 knots and using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk. All data was kept on the original dose scale. The fitted trend for each model is represented by a central line (solid lines for linear model; dashed lines for cubic spline model) with 95% confidence intervals represented by the outer lines. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

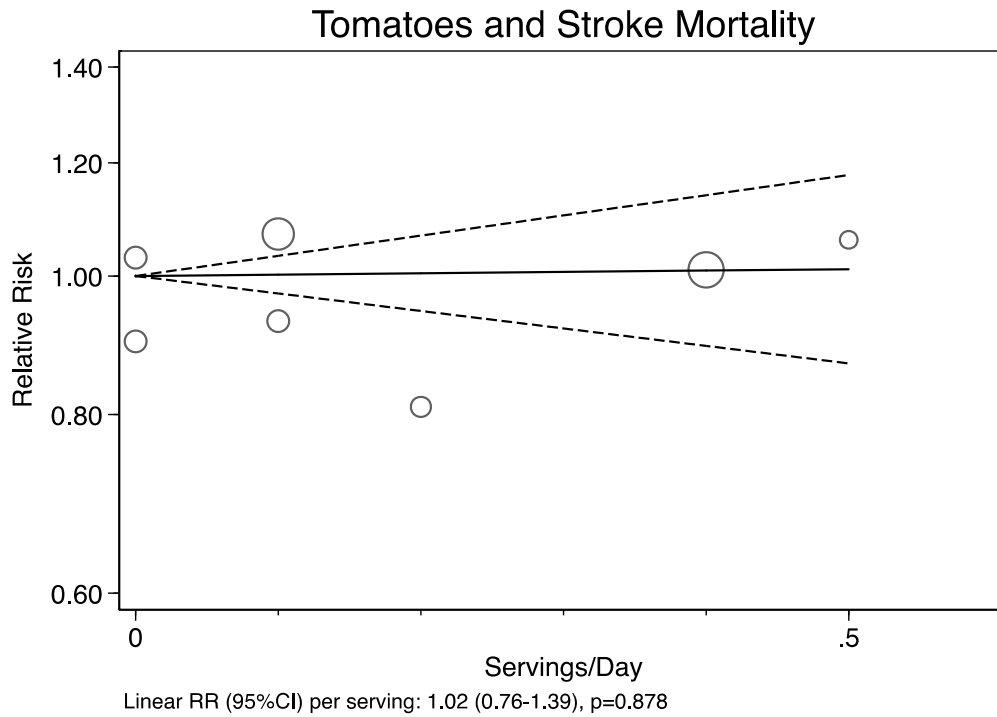


Figure S171. Linear dose-response relation between increasing intake of tomatoes and stroke mortality. Linear dose-response data was modeled using the Greenland and Longnecker²³ method to estimate the covariances of multivariable-adjusted relative risk, with kept on the original dose scale. Dashed lines represent the pointwise 95% confidence intervals for the fitted linear trend represented by a solid line. Individual observations are represented by the circles, with the weight of the study in the overall analysis represented by the size of the circles.

TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

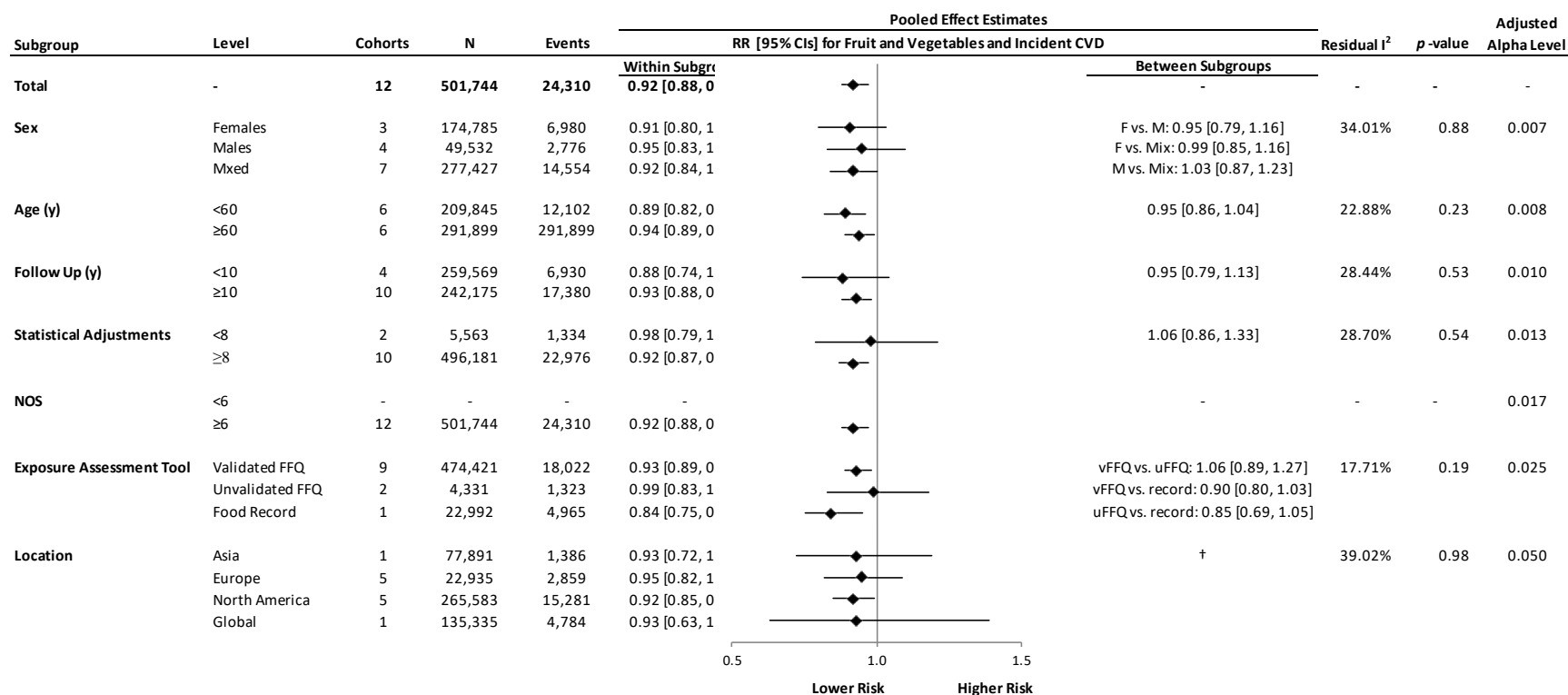


Figure S172. Categorical subgroup analyses of total fruit and vegetable intake and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.98 [0.74, 1.31]; Europe vs. Global 0.99 [0.64, 1.51]; Europe vs. North America 0.97 [0.83, 1.14]; Asia vs. Global 0.99 [0.62, 1.60]; Asia vs. North America 1.00 [0.78, 1.32]; Global vs. North America 1.02 [0.68, 1.53];

FRUIT AND CARDIOVASCULAR DISEASE INCIDENCE

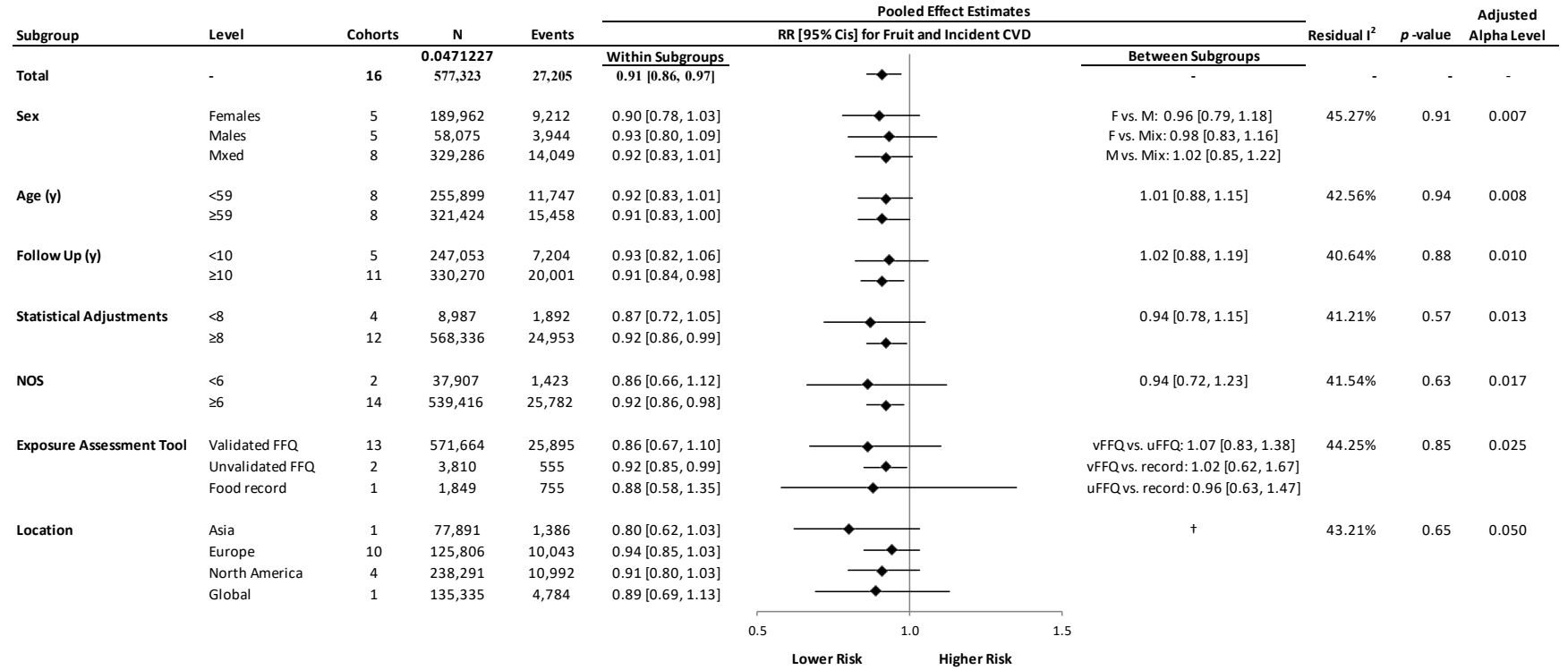


Figure S173. Categorical subgroup analyses of fruit intake and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
 † Europe vs. Asia 0.85 [0.65, 1.12]; Europe vs. Global 0.94 [0.73, 1.23]; Europe vs. North America 0.96 [0.82, 1.13]; Asia vs. Global 0.90 [0.63, 1.29]; Asia vs. North America 0.88 [0.67, 1.18]; Global vs. North America 0.98 [0.74, 1.29]

VEGETABLES AND CARDIOVASCULAR DISEASE INCIDENCE

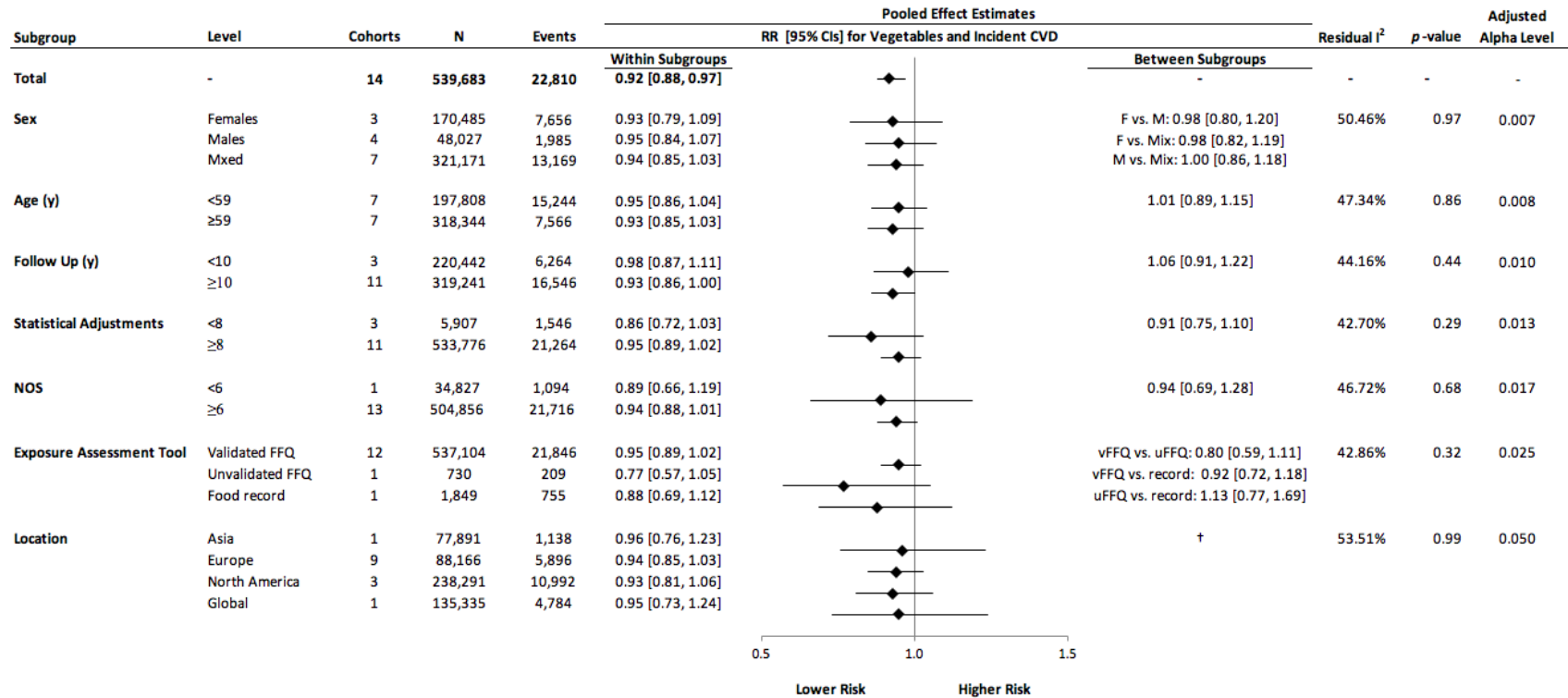


Figure S174. Categorical subgroup analyses of intake of vegetables and cardiovascular disease incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
 † Europe vs. Asia 1.03 [0.79, 1.33]; Europe vs. Global 1.01 [0.76, 1.34]; Europe vs. NA 0.99 [0.84, 1.16]; Asia vs. Global 1.01 [0.71, 1.45]; Asia vs. NA 1.04 [0.79, 1.37]; Global vs. NA 1.03 [0.76, 1.38]

TOTAL FRUIT AND VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

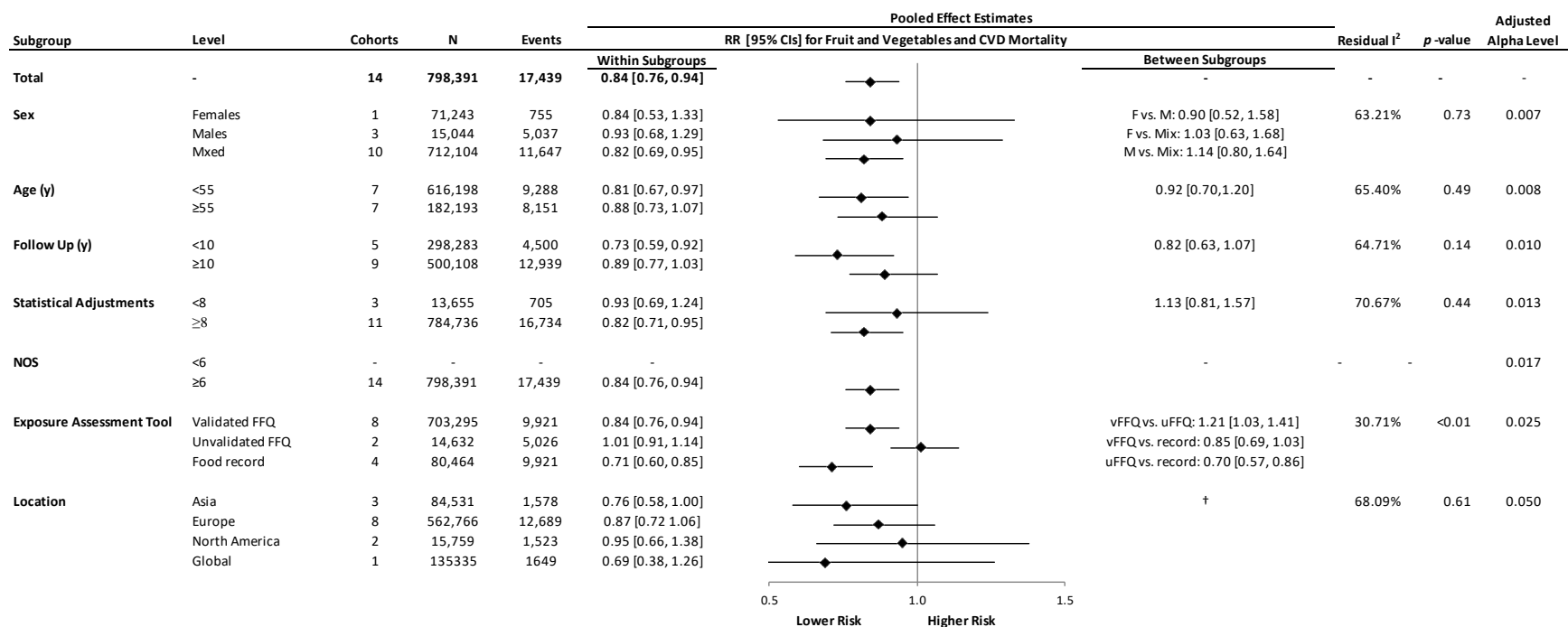


Figure S175. Categorical subgroup analyses of total fruit and vegetable intake and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.87 [0.63, 1.22]; Europe vs. Global 0.79 [0.42, 1.49]; Europe vs. North America 1.09 [0.72, 1.66]; Asia vs. Global 1.10 [0.57, 2.13]; Asia vs. North America 0.80 [0.50, 1.27]; Global vs. North America 0.73 [0.36, 1.47]

FRUIT AND CARDIOVASCULAR DISEASE MORTALITY

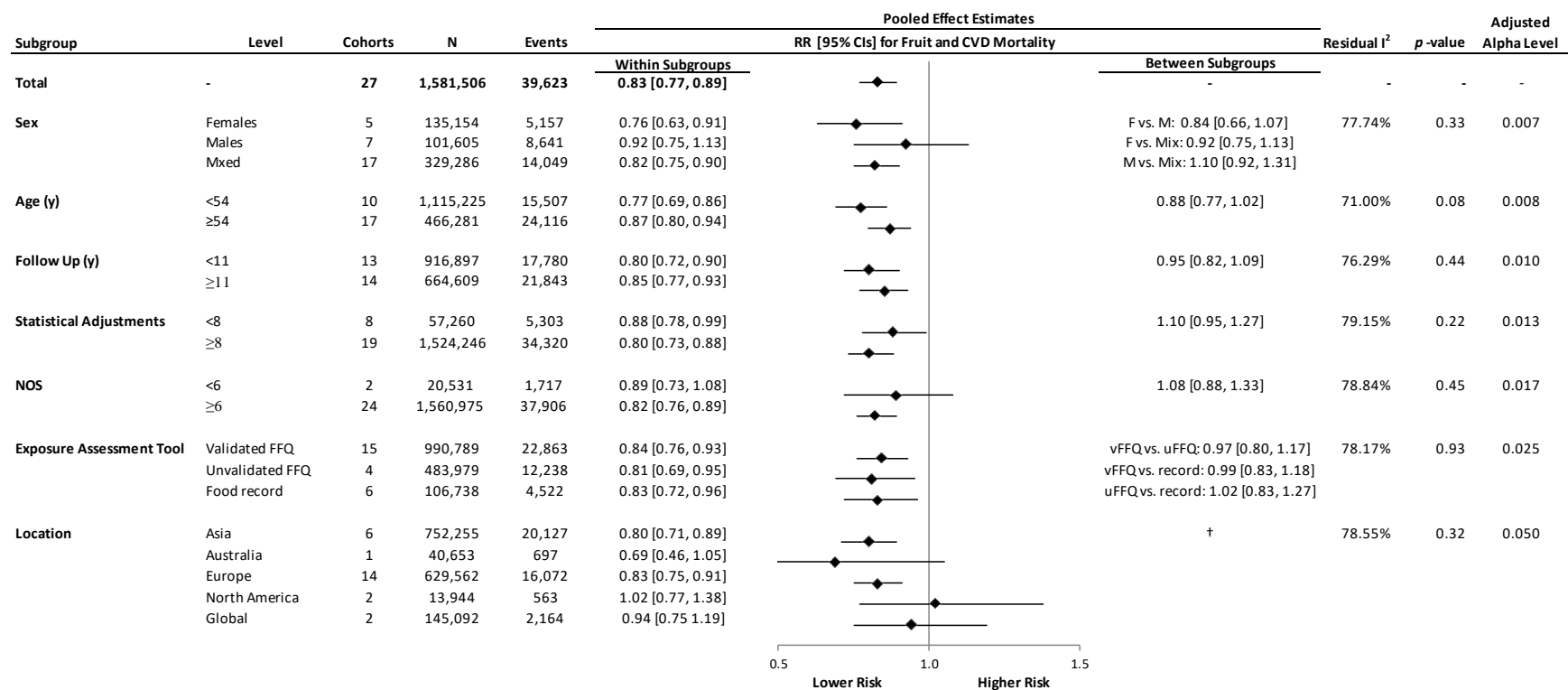


Figure S176. Categorical subgroup analyses of fruit intake and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
 † Europe vs. Asia 0.96 [0.83, 1.13]; Europe vs. Australia 0.84 [0.55, 1.28]; Europe vs. Global 1.14 [0.89, 1.47]; Europe vs. North America 1.25 [0.92, 1.70]; Asia vs. Australia 1.15 [0.75, 1.77]; Asia vs. Global 0.85 [0.65, 1.10]; Asia vs. North America 0.77 [0.56, 1.06]; Australia vs. Global 0.73 [0.46, 1.18]; Australia vs. North America 0.67 [0.41, 1.12]; Global vs. North America 0.92 [0.63, 1.33]

VEGETABLES AND CARDIOVASCULAR DISEASE MORTALITY

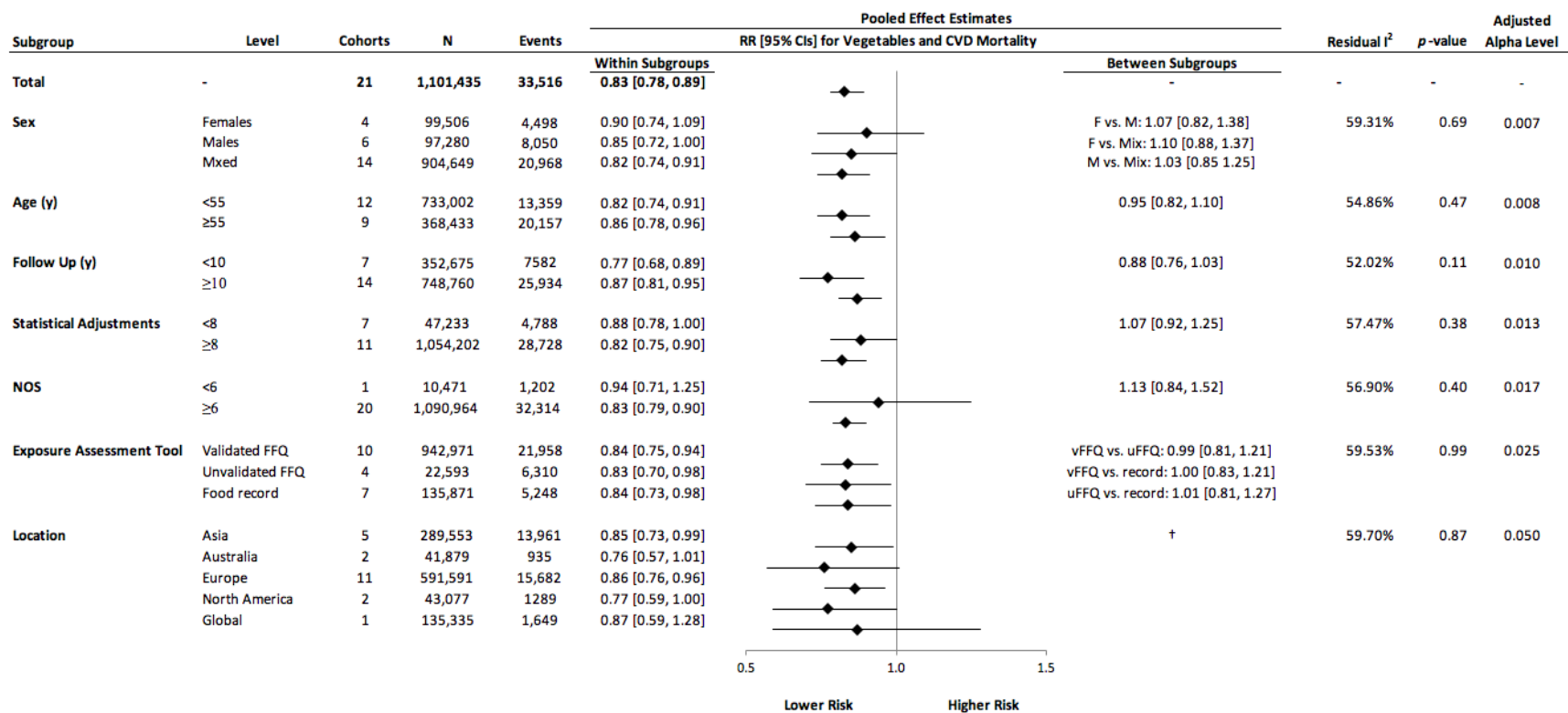


Figure S177. Categorical subgroup analyses of intake of vegetables and cardiovascular disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CVD – cardiovascular disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
 † Europe vs. Asia 0.99 [0.82, 1.19]; Europe vs. Australia 0.88 [0.65, 1.20]; Europe vs. Global 1.01 [0.68, 1.52]; Europe vs. North America 0.90 [0.67, 1.20]; Asia vs. Australia 1.12 [0.81, 1.56]; Asia vs. Global 0.98 [0.64, 1.48]; Asia vs. North America 1.11 [0.81, 1.50]; Australia vs. Global 0.87 [0.54, 1.41]; Australia vs. North America 0.98 [0.66, 1.46]; Global vs. North America 1.13 [0.71, 1.81]

TOTAL FRUIT AND VEGETABLES AND CORONARY HEART DISEASE INCIDENCE

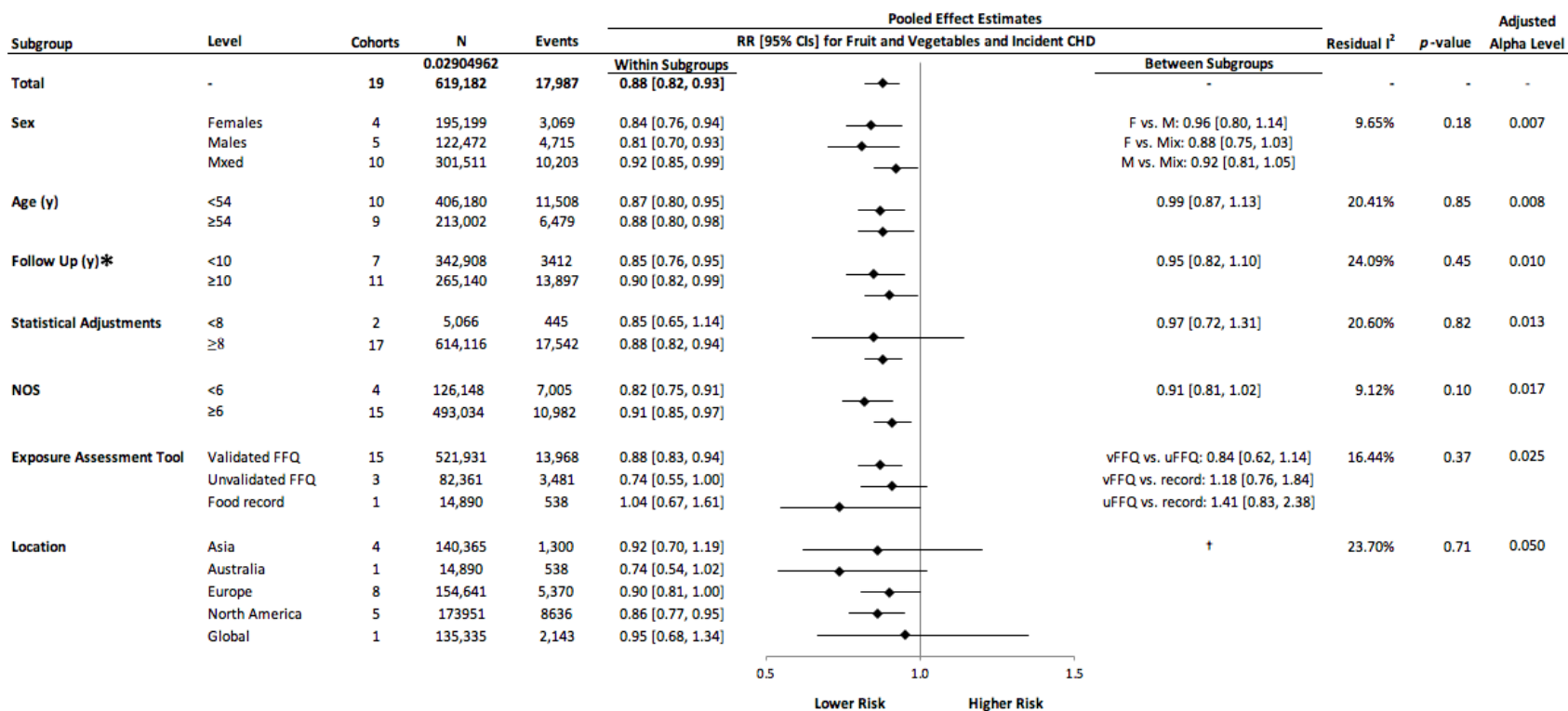


Figure S178. Categorical subgroup analyses of total fruit and vegetable intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. * Follow-up years includes 17 cohorts as Bingham et al. 2008 (EPIC Norfolk) did not report follow-up time. † Europe vs. Asia 1.02 [0.77, 1.35]; Europe vs. Australia 0.82 [0.59, 1.14]; Europe vs. Global 1.06 [0.74, 1.51]; Europe vs. North America 0.95 [0.83, 1.10]; Asia vs. Australia 1.24 [0.82, 1.87]; Asia vs. Global 0.96 [0.63, 1.48]; Asia vs. North America 1.07 [0.81, 1.42]; Australia vs. Global 0.78 [0.49, 1.24]; Australia vs. North America 0.86 [0.62, 1.21]; Global vs. North America 1.11 [0.78, 1.58]

FRUIT AND CORONARY HEART DISEASE INCIDENCE

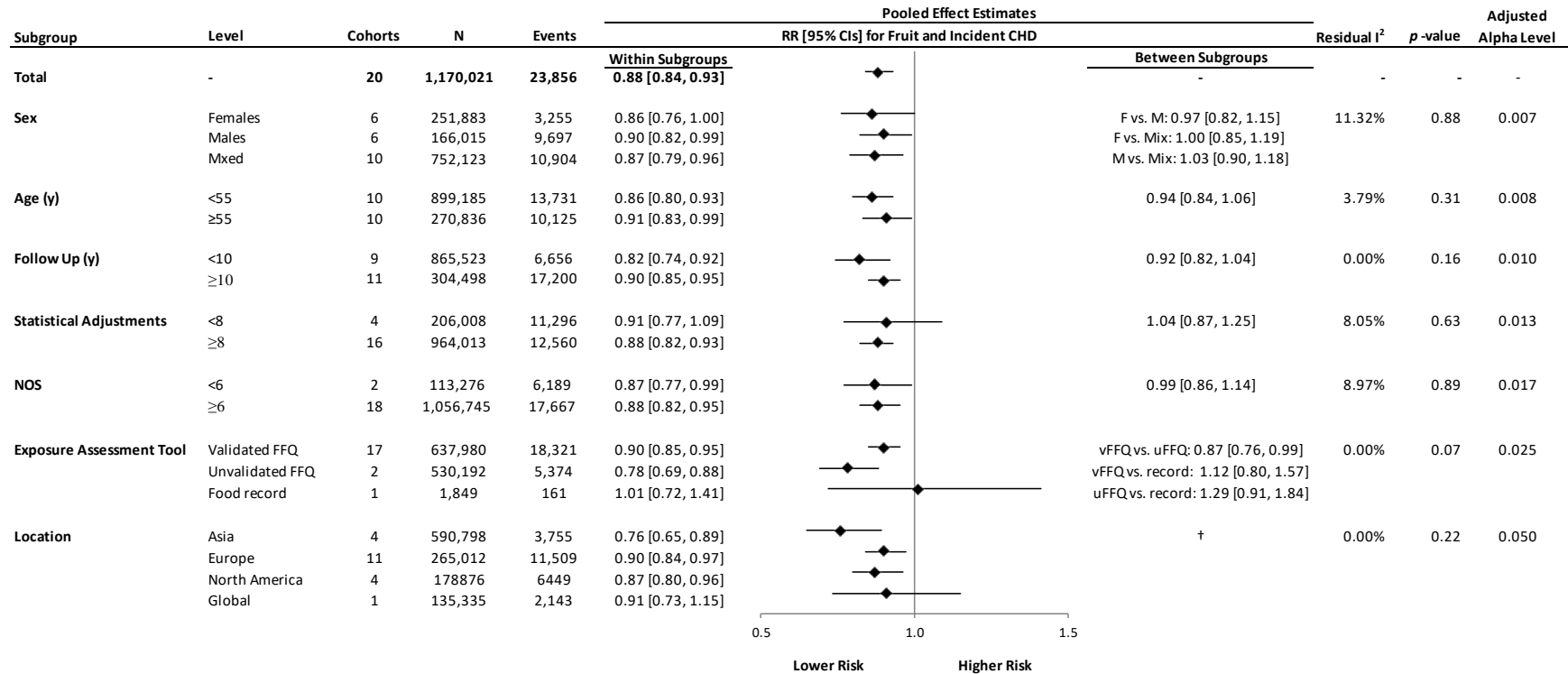


Figure S179. Categorical subgroup analyses of fruit intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.84 [0.71, 0.99]; Europe vs. Global 1.01 [0.79, 1.29]; Europe vs. North America 0.96 [0.85, 1.08]; Asia vs. Global 0.83 [0.63, 1.10]; Asia vs. North America 0.87 [0.73, 1.04]; Global vs. North America 1.05 [0.82, 1.34]

VEGETABLE AND CORONARY HEART DISEASE INCIDENCE

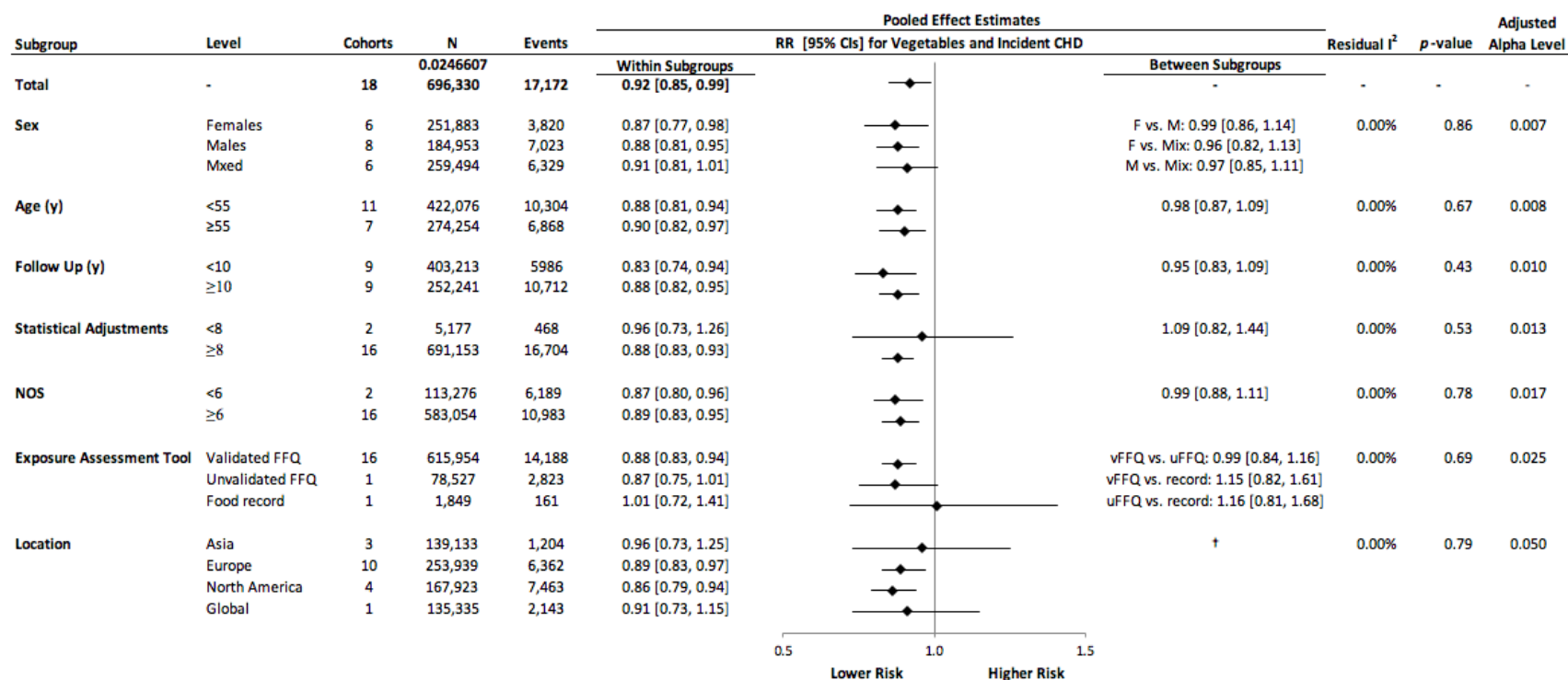


Figure S180. Categorical subgroup analyses of intake of vegetables and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs. Asia 1.07 [0.81, 1.41]; Europe vs. Global 1.02 [0.80, 1.30]; Europe vs. NA 0.96 [0.85, 1.08]; Asia vs. Global 1.05 [0.74, 1.49]; Asia vs. NA 1.11 [0.84, 1.48]; Global vs. NA 1.07 [0.83, 1.37]

CITRUS FRUIT AND CORONARY HEART DISEASE INCIDENCE

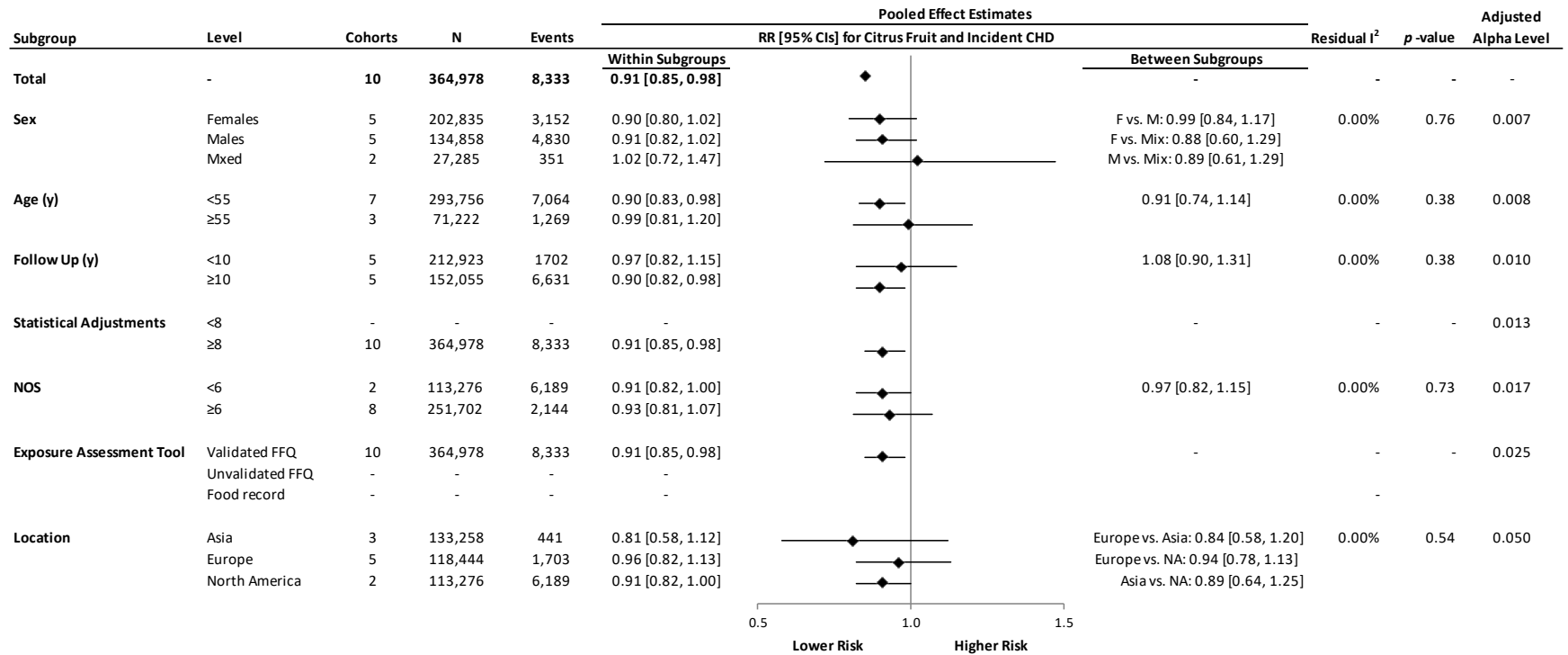


Figure S181. Categorical subgroup analyses of citrus fruit intake and incident coronary heart disease. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

FRUIT AND CORONARY HEART DISEASE MORTALITY

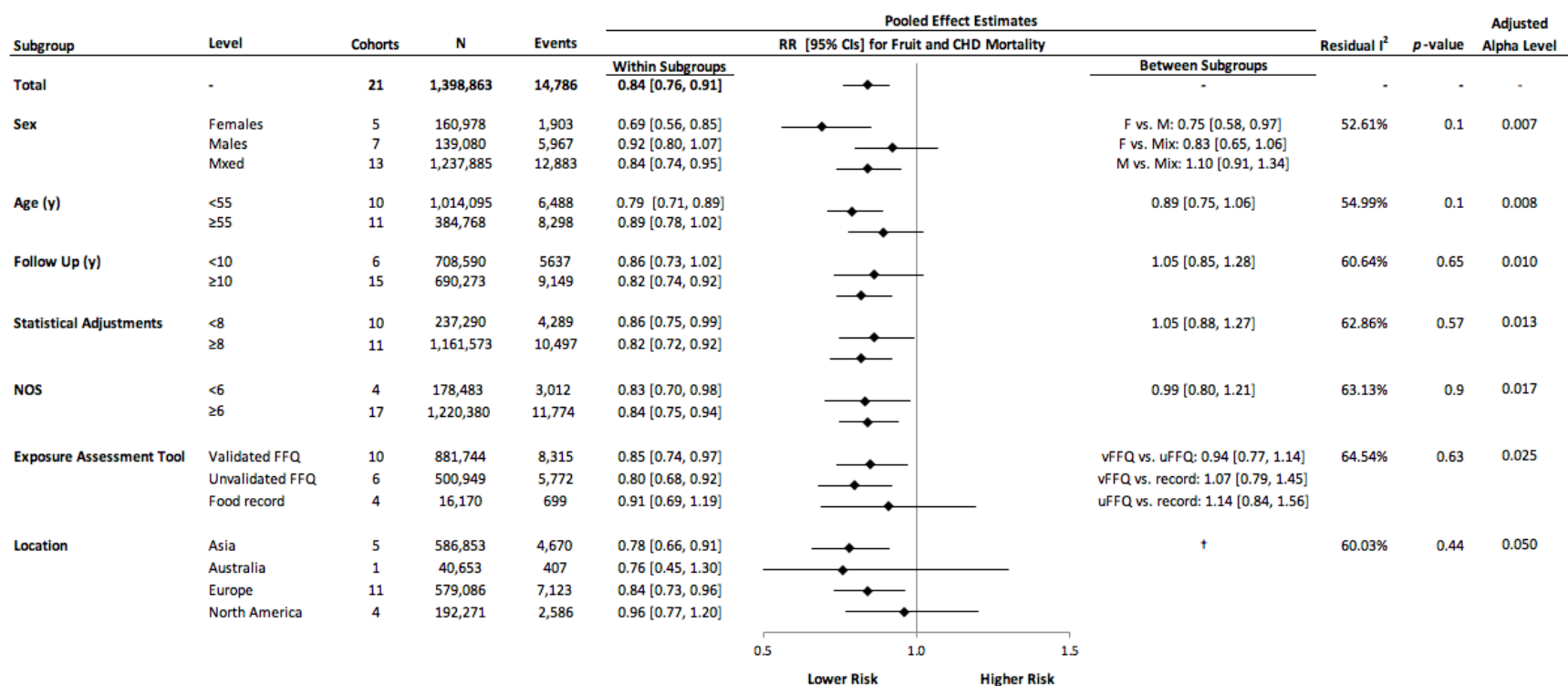


Figure S182. Categorical subgroup analyses of fruit intake and coronary heart disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs. Asia 0.93 [0.76, 1.14]; Europe vs. Australia 0.91 [0.53, 1.57]; Europe vs. North America 1.15 [0.89, 1.47]; Asia vs. Australia 1.01 [0.59, 1.77]; Asia vs. North America 0.81 [0.62, 1.06]; Australia vs. North America 0.80 [0.45, 1.41]

VEGETABLES AND CORONARY HEART DISEASE MORTALITY

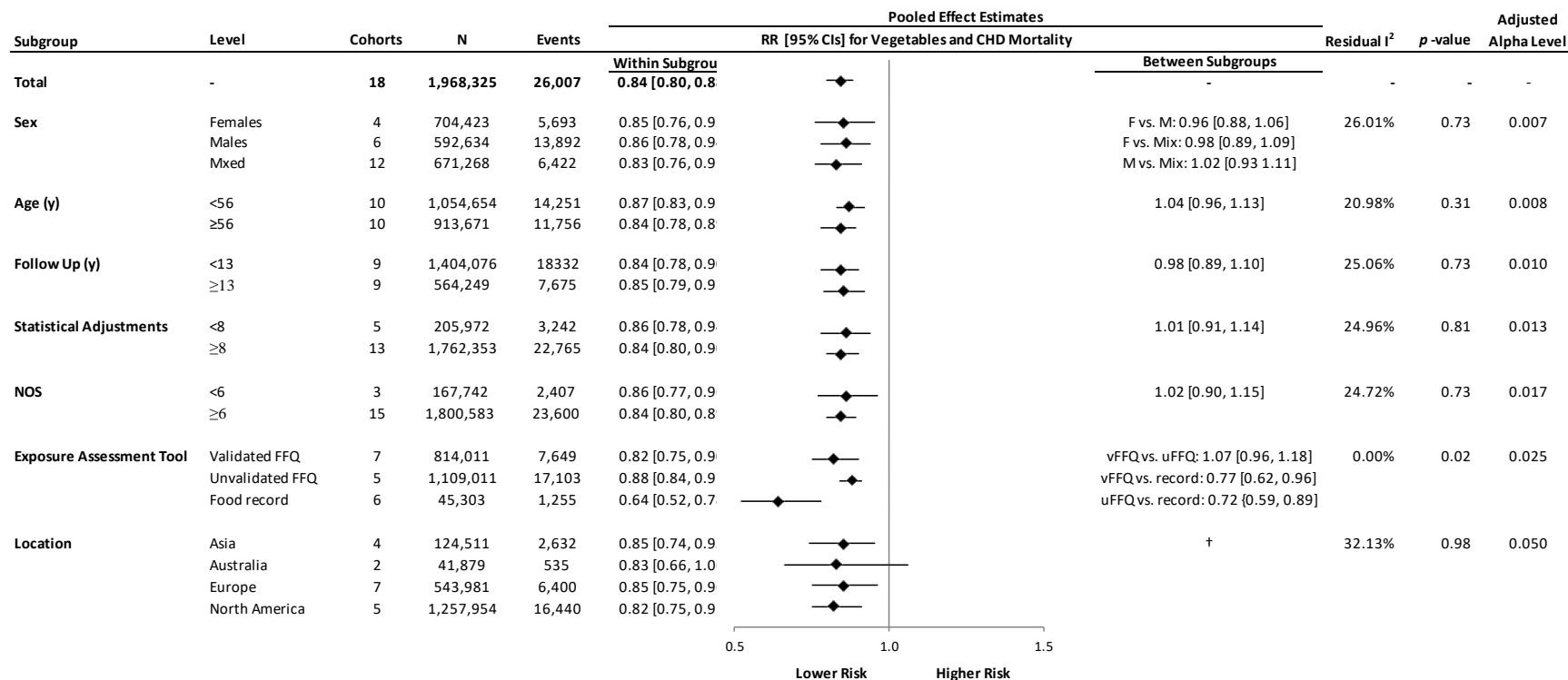


Figure S183. Categorical subgroup analyses of intake of vegetables and coronary heart disease mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs. Asia 0.98 [0.83, 1.17]; Europe vs. Australia 0.98 [0.75, 1.28]; Europe vs. North America 0.97 [0.83, 1.14]; Asia vs. Australia 1.01 [0.77, 1.32]; Asia vs. North America 1.02 [0.87, 1.19]; Australia vs. North America 1.01 [0.78, 1.30]

TOTAL FRUIT AND VEGETABLES AND STROKE INCIDENCE

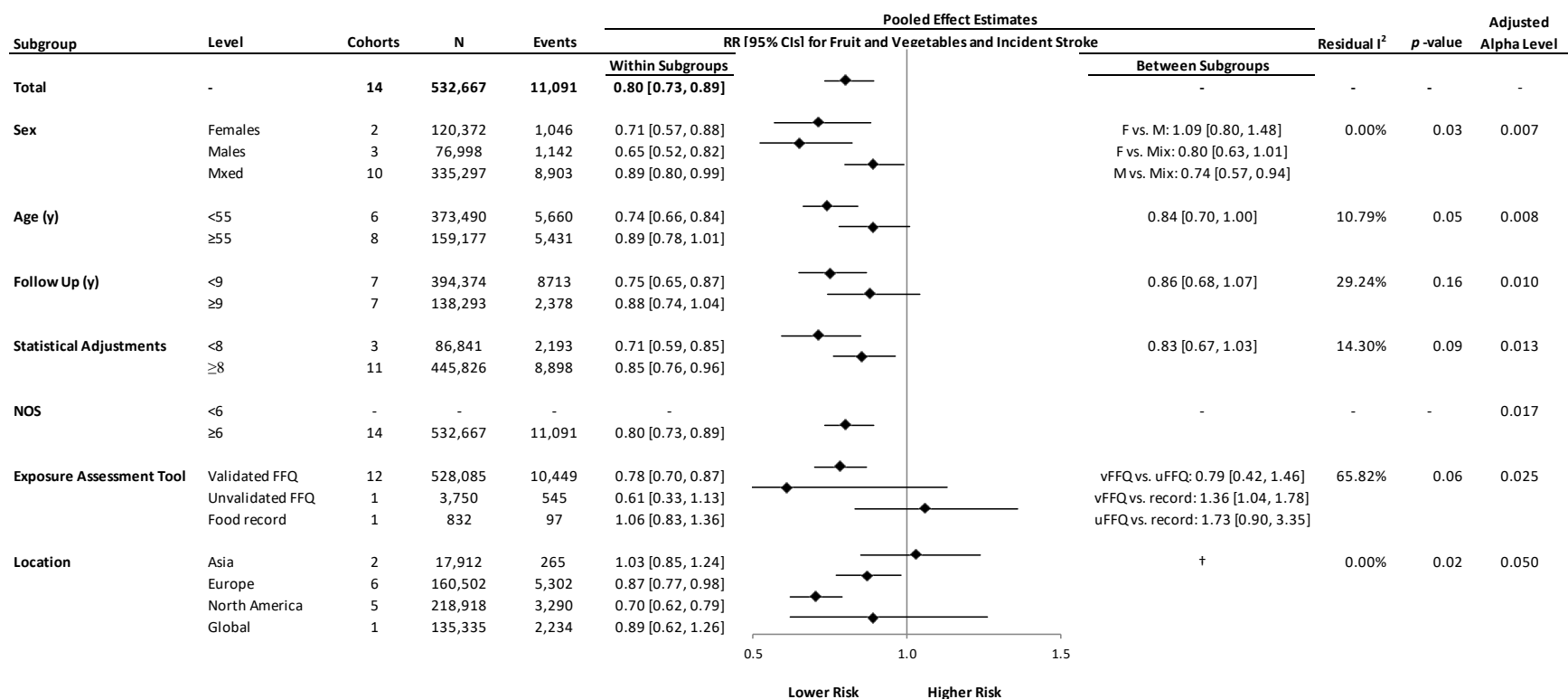


Figure S184. Categorical subgroup analyses of total fruit and vegetable intake and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs Asia 1.17 [0.94, 1.47]; Europe vs Global 1.02 [0.70, 1.48]; Europe vs NA 0.81 [0.68, 0.96]; Asia vs Global 1.16 [0.77, 1.72]; Asia vs NA 1.46 [1.16, 1.84]; Global vs NA 1.27 [0.87, 1.84]

FRUIT AND STROKE INCIDENCE

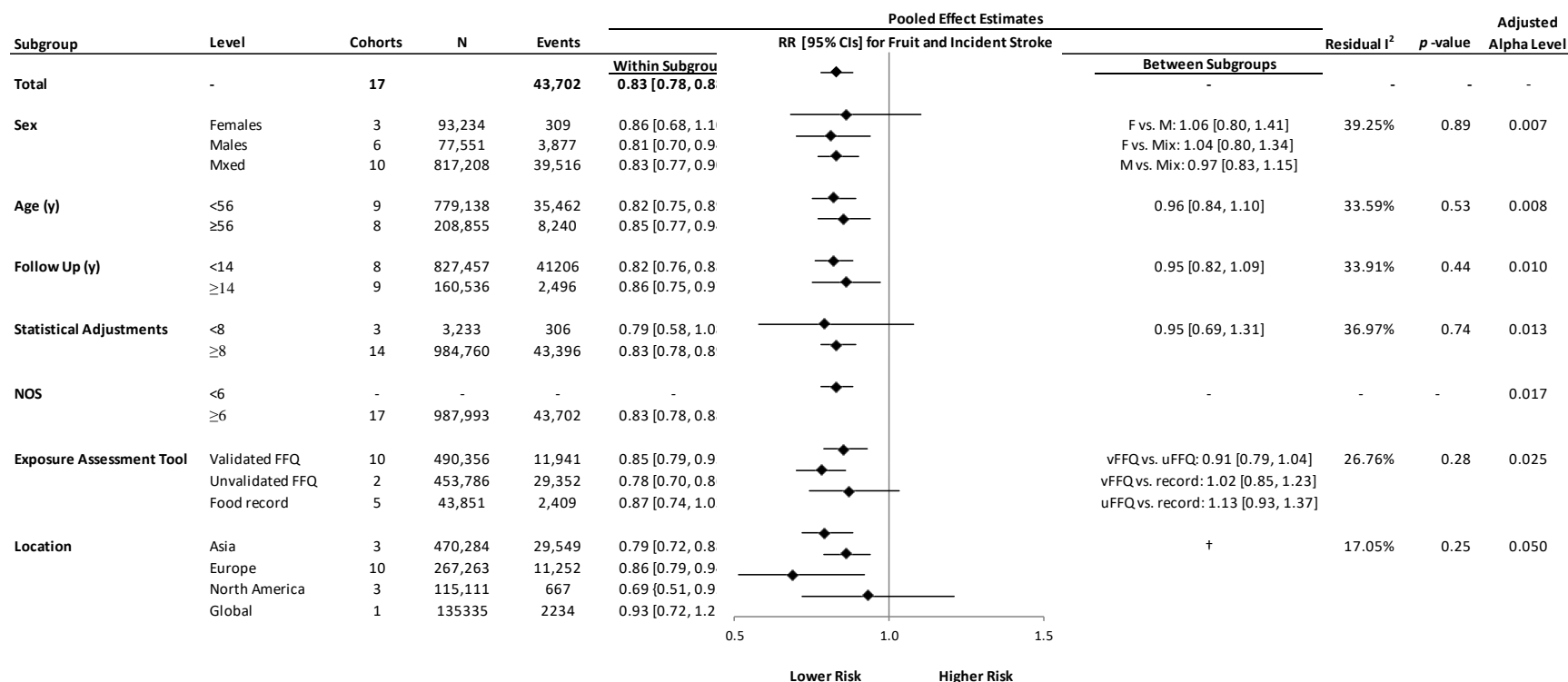


Figure S185. Categorical subgroup analyses of fruit intake and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals. † Europe vs. Asia 0.92 [0.81, 1.05]; Europe vs. Global 1.09 [0.82, 1.42]; Europe vs. North America 0.80 [0.59, 1.09]; Asia vs. Global 0.85 [0.65, 1.12]; Asia vs. North America 1.15 [0.85, 1.57]; Global vs. North America 1.36 [0.92, 2.01]

VEGETABLES AND STROKE INCIDENCE

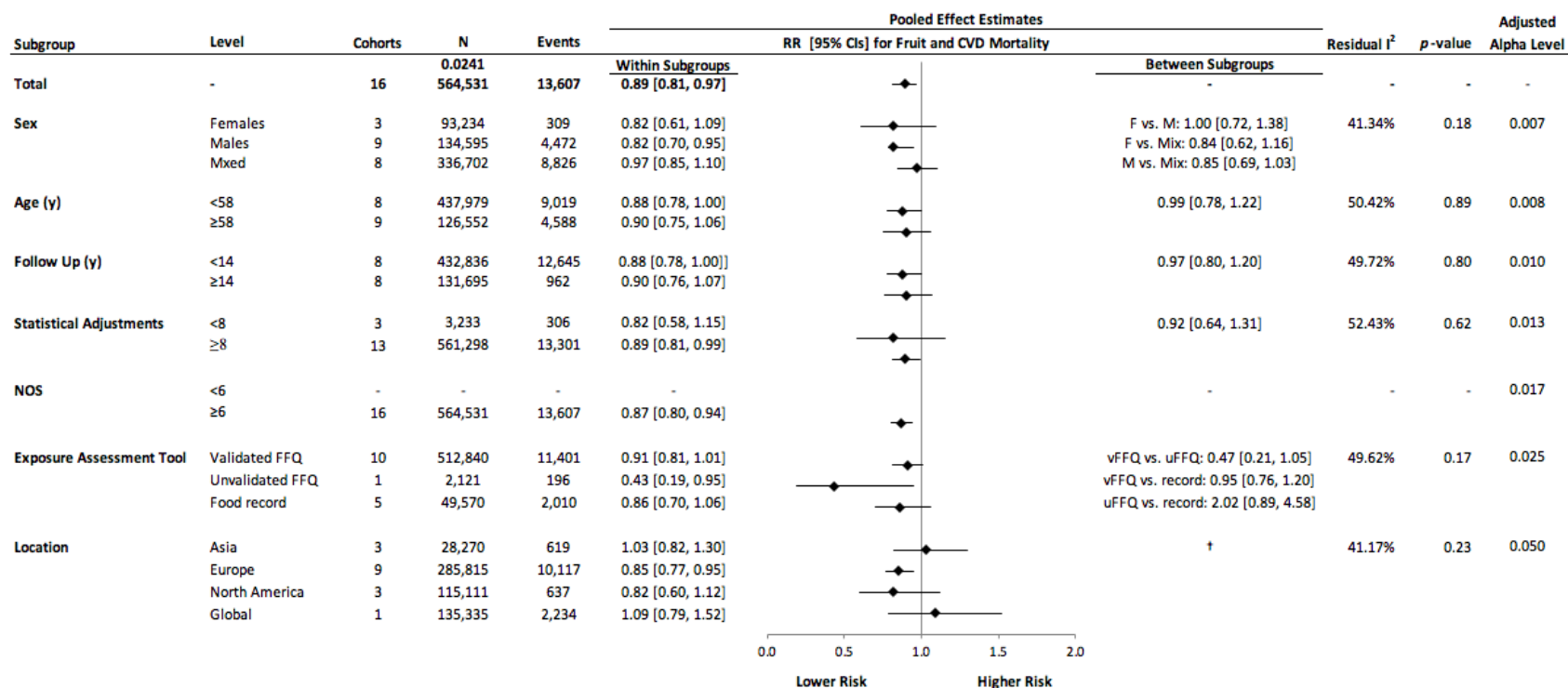


Figure S186. Categorical subgroup analyses of intake of vegetables and stroke incidence. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. CHD – coronary heart disease; FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.
 † Europe vs. Asia 1.21 [0.94, 1.56]; Europe vs. Global 1.28 [0.91, 1.81]; Europe vs. NA 0.96 [0.69, 1.33]; Asia vs. Global 0.94 [0.63, 1.40]; Asia vs. NA 1.26 [0.86, 1.86]; Global vs. NA 1.34 [0.85, 2.10]

FRUIT AND STROKE MORTALITY

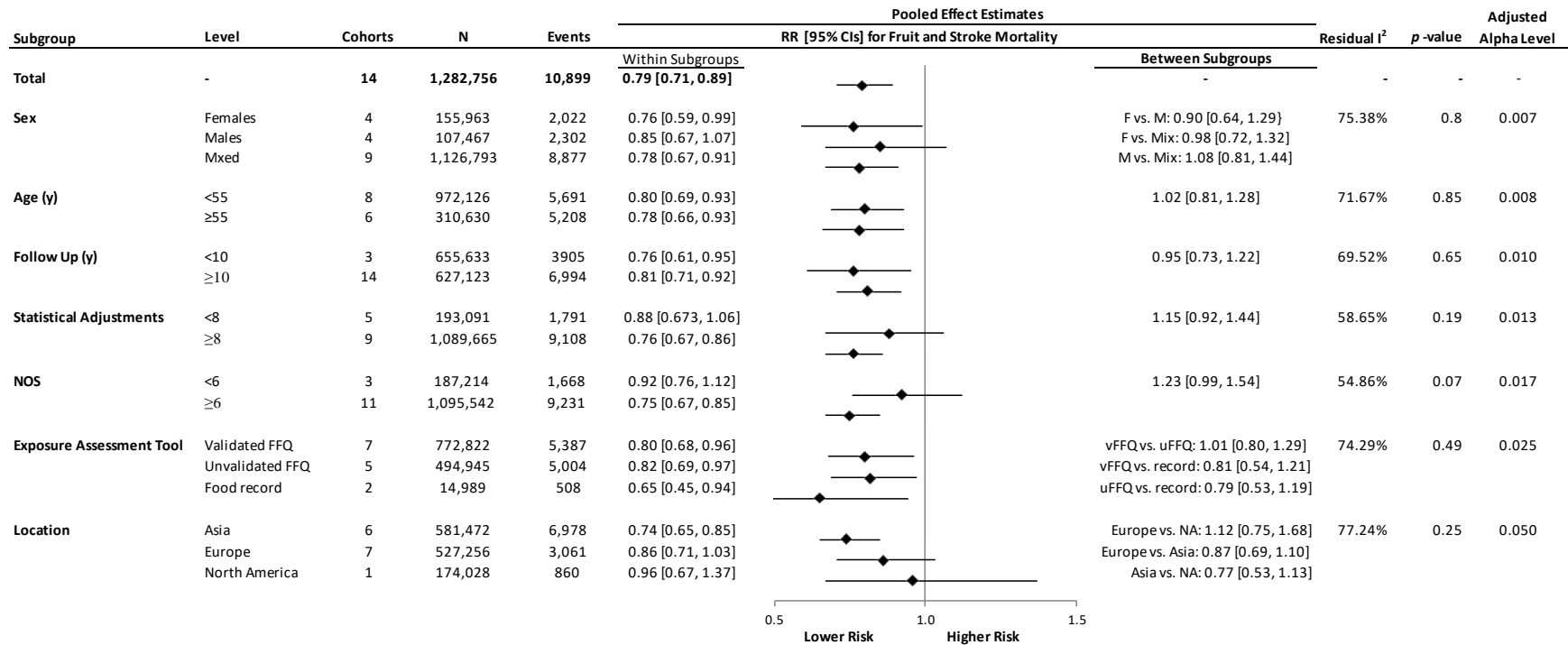


Figure S187. Categorical subgroup analyses of fruit intake and stroke mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

VEGETABLES AND STROKE MORTALITY

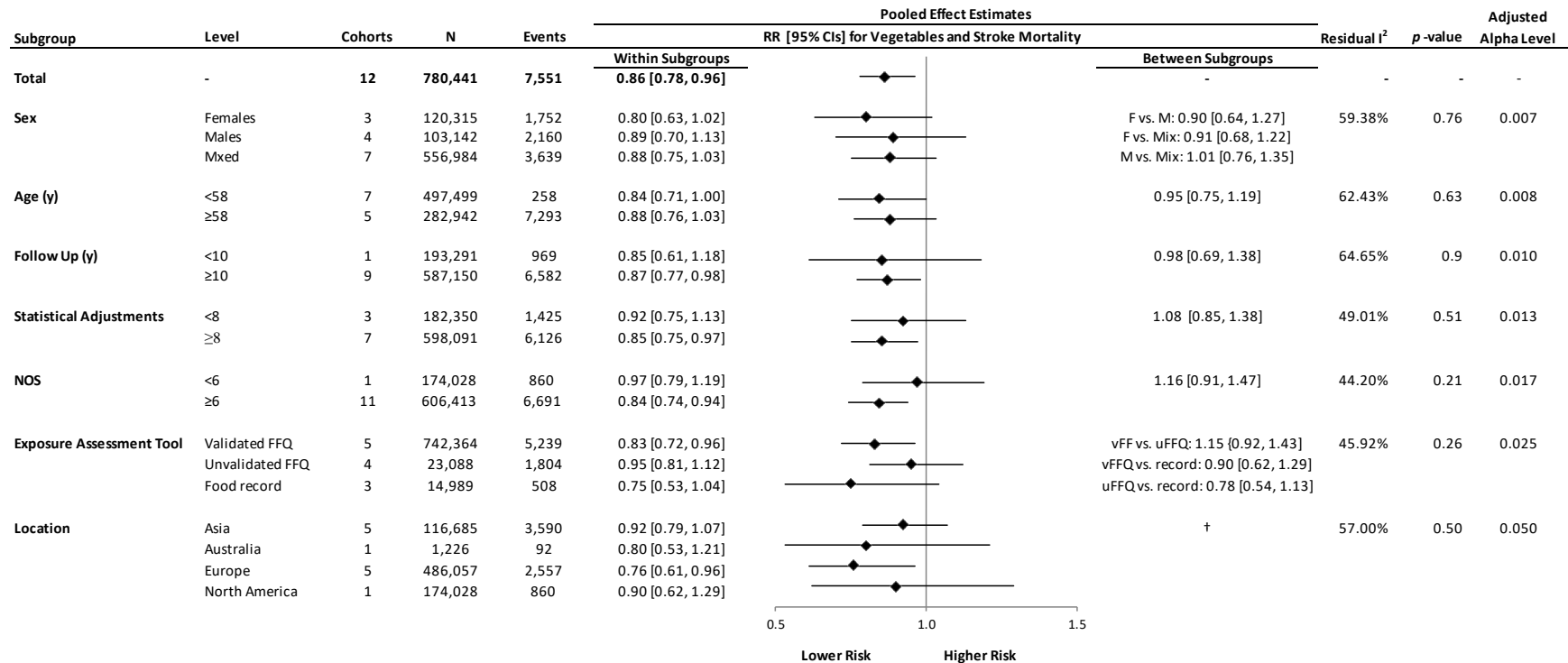


Figure S188. Categorical subgroup analyses of intake of vegetables and stroke mortality. Point estimates for within subgroup level are the pooled effect estimates and are represented by a black diamond. The residual I² value indicates the inter-study heterogeneity unexplained by the subgroup. FFQ – food frequency questionnaire; NOS – Newcastle-Ottawa Scale; RR – relative risk; 95% CIs – 95% confidence intervals.

† Europe vs. Asia 1.20 [0.92, 1.57]; Europe vs. Australia 1.05 [0.66, 1.67]; Europe vs. North America 1.17 [0.76, 1.80]; Asia vs. Australia 1.44 [0.74, 1.77]; Asia vs. North America 1.03 [0.69, 1.53]; Australia vs. North America 0.90 [0.52, 1.55]

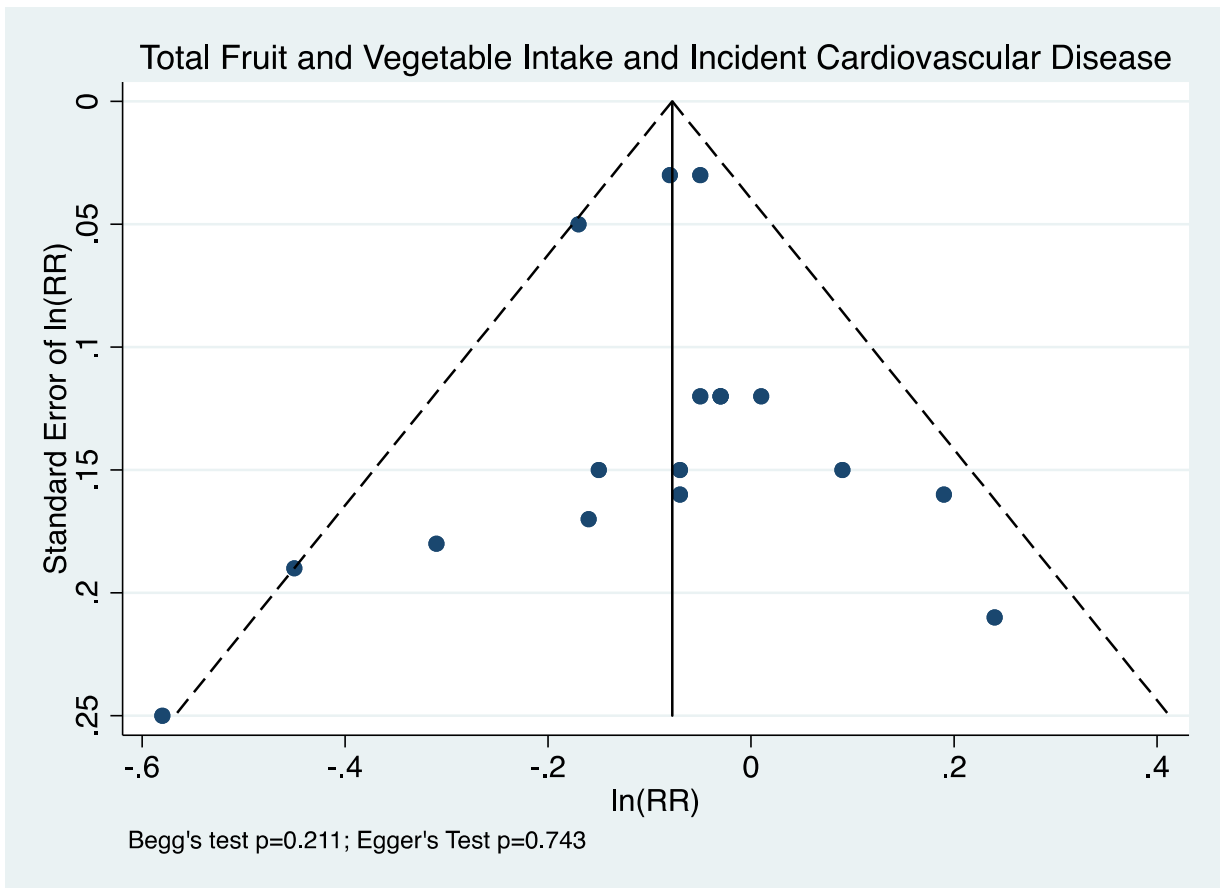


Figure S189. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

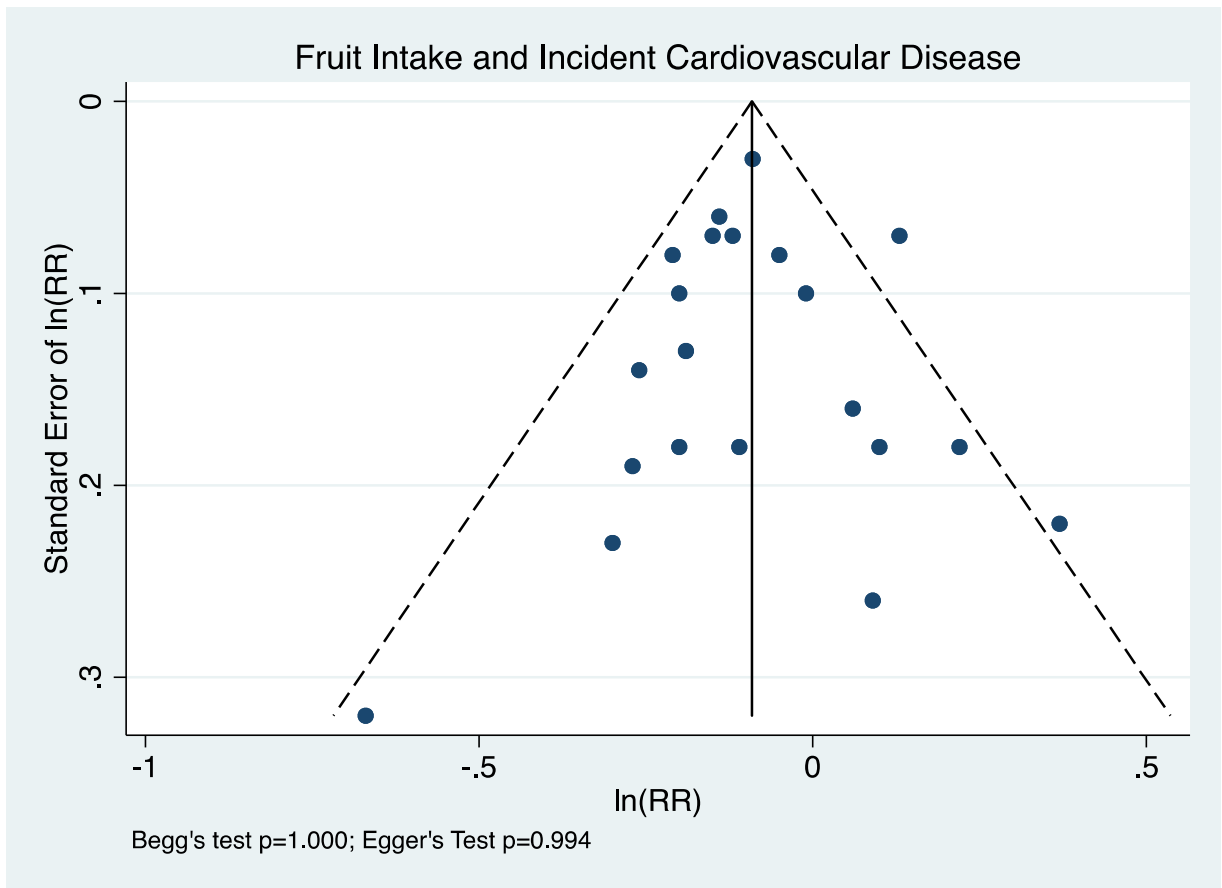


Figure S190. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

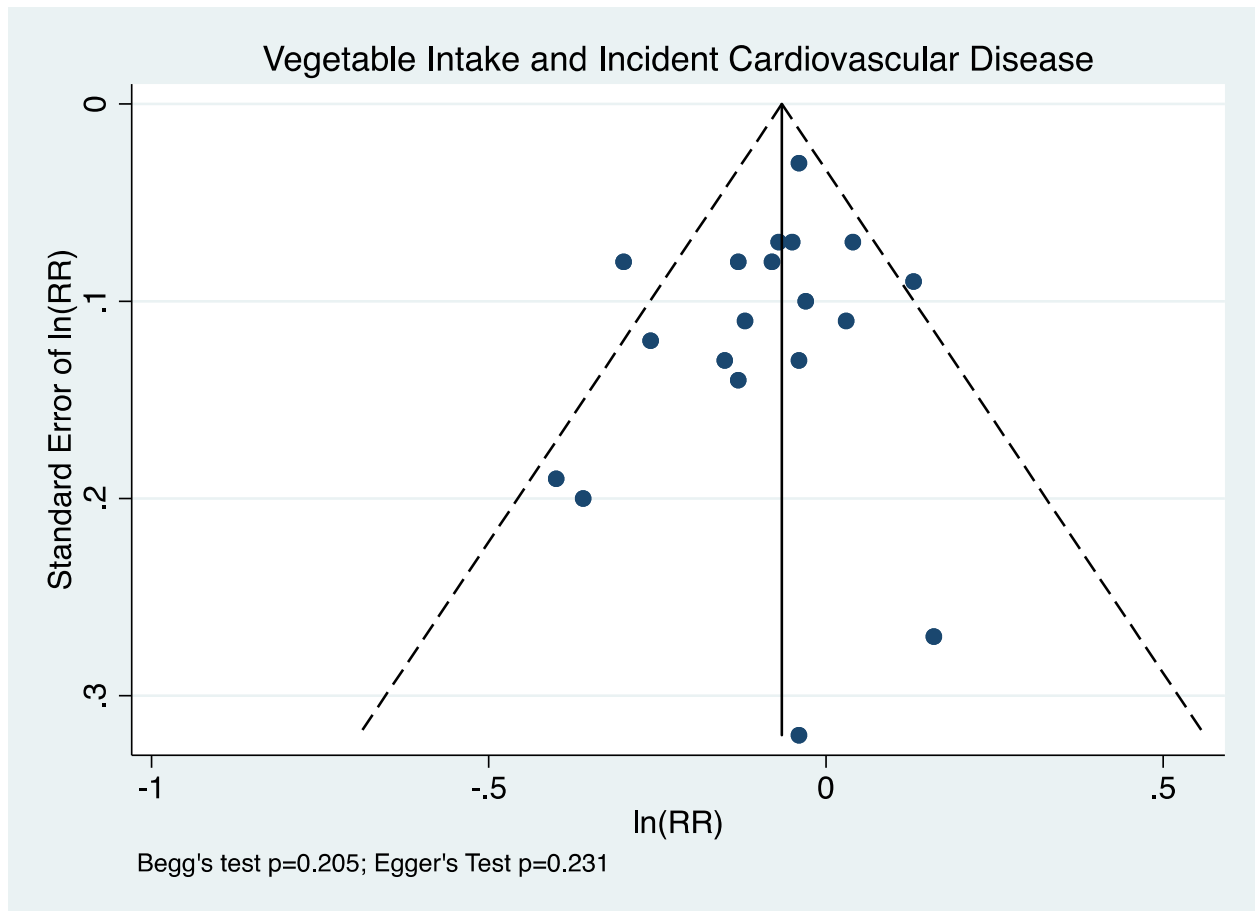


Figure S191. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease incidence comparing the highest and lowest quantiles of vegetable intake. The vertical line represents the pooled effect estimated expressed as Ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the Ln(RR).

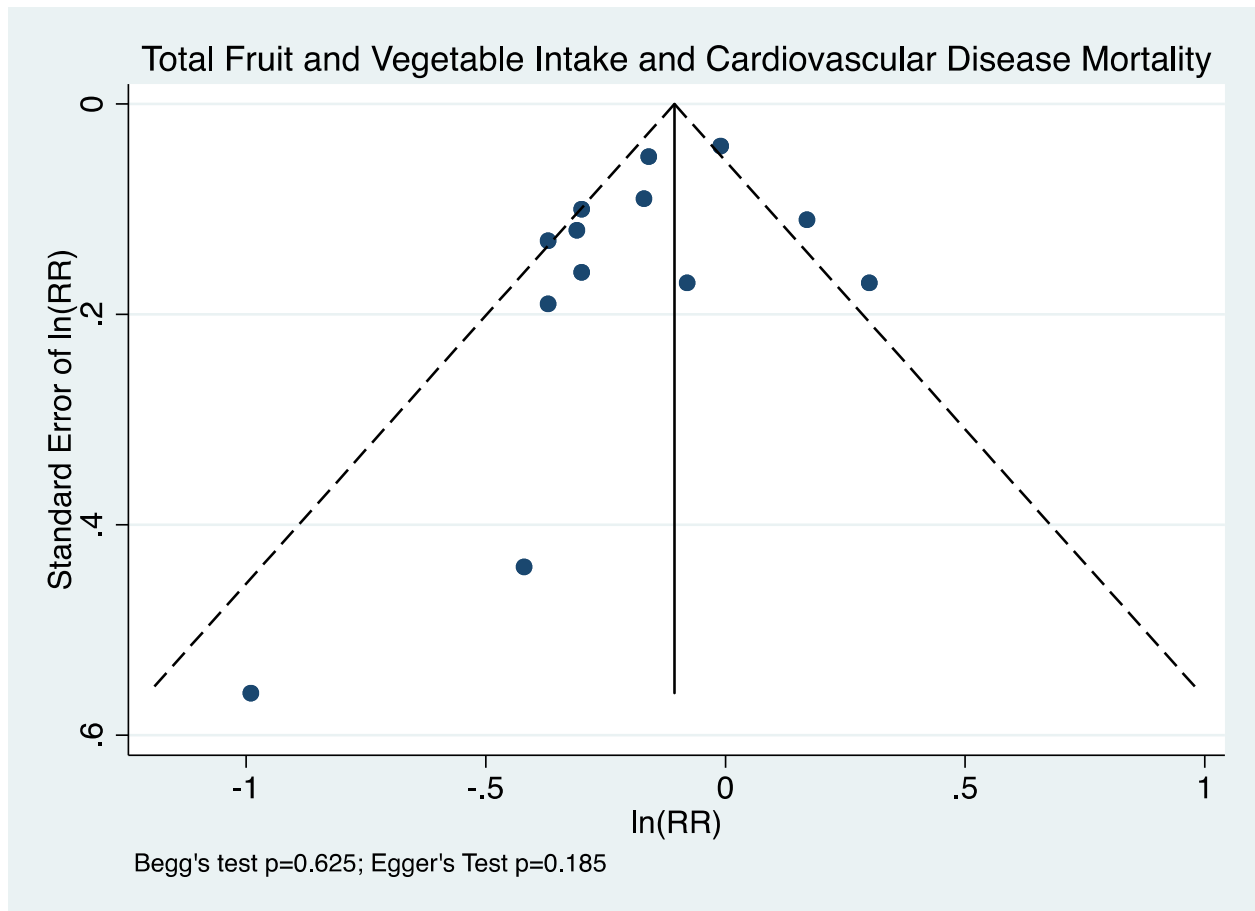


Figure S192. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease mortality comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

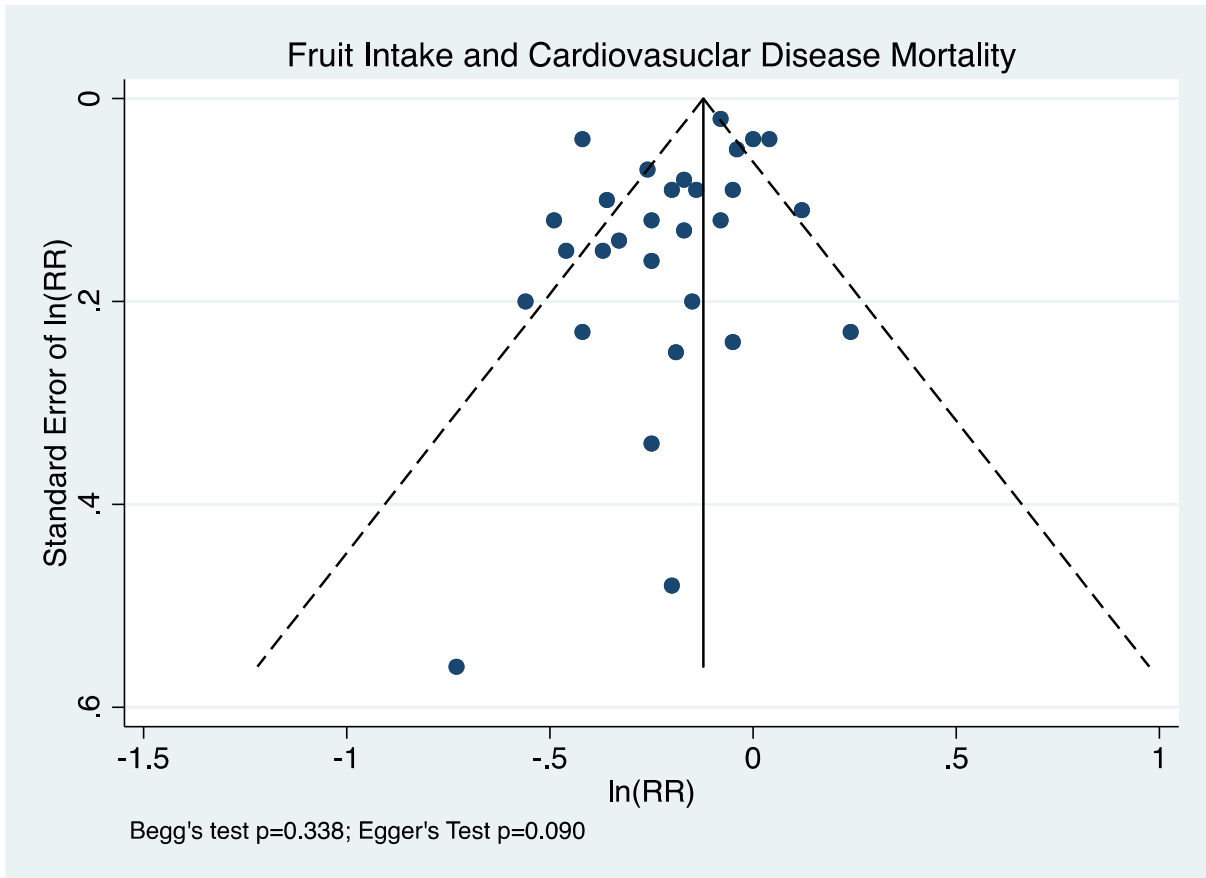
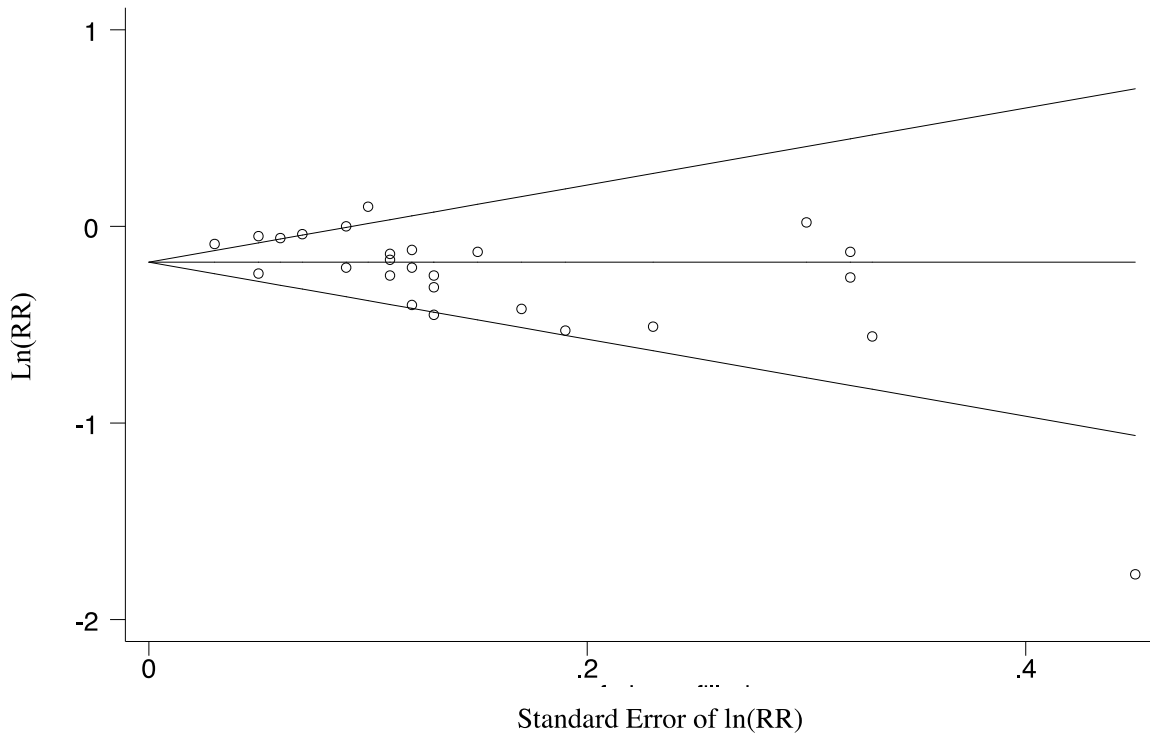


Figure S193. Funnel plot of natural logarithm relative risk [Ln(RR)] for cardiovascular disease mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

Vegetable Intake and Cardiovascular Disease Mortality



Imputed RR accounting for publication bias: N/A

P-value: N/A

Figure S194. Funnel plot for trim-and-fill analysis for coronary heart disease mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [Ln(RR)]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.

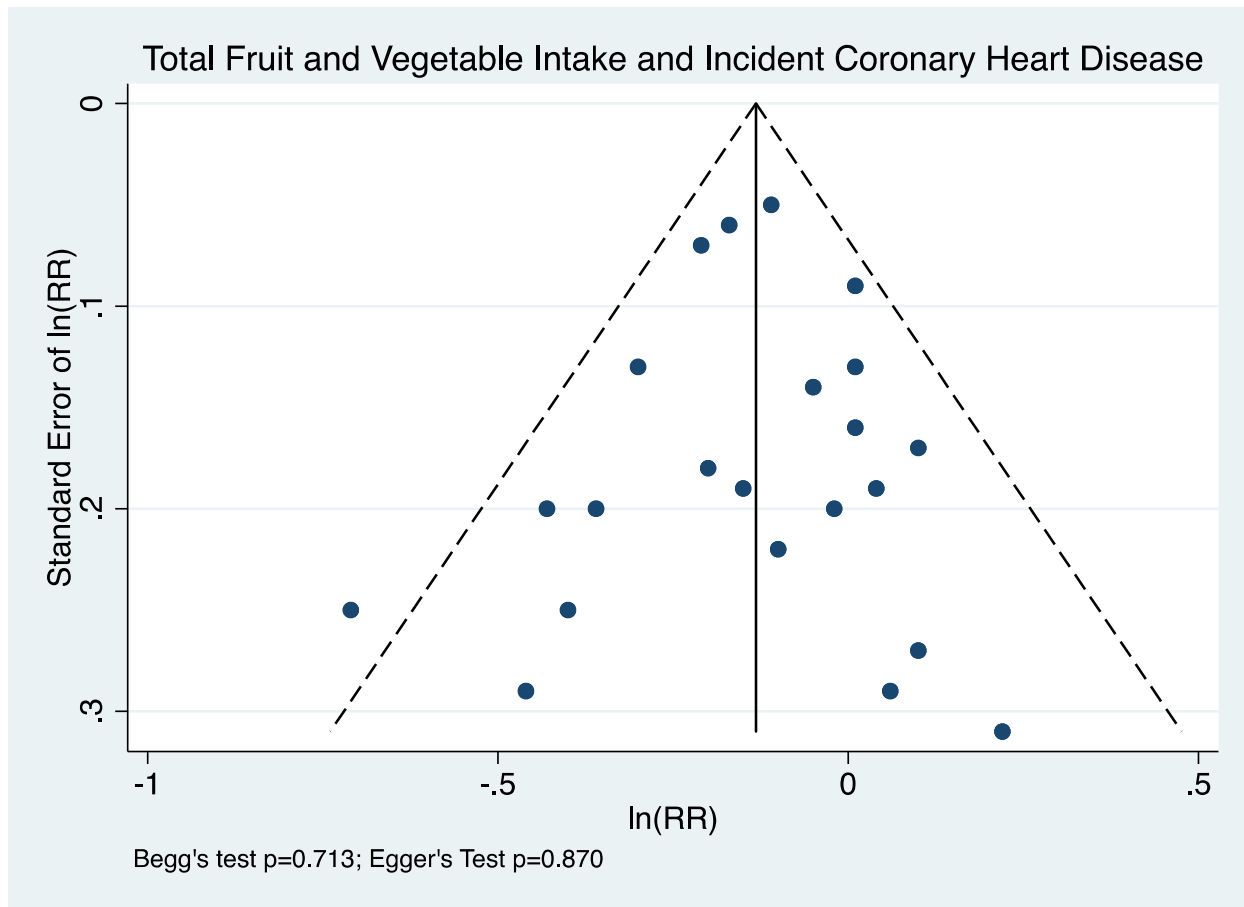


Figure S195. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

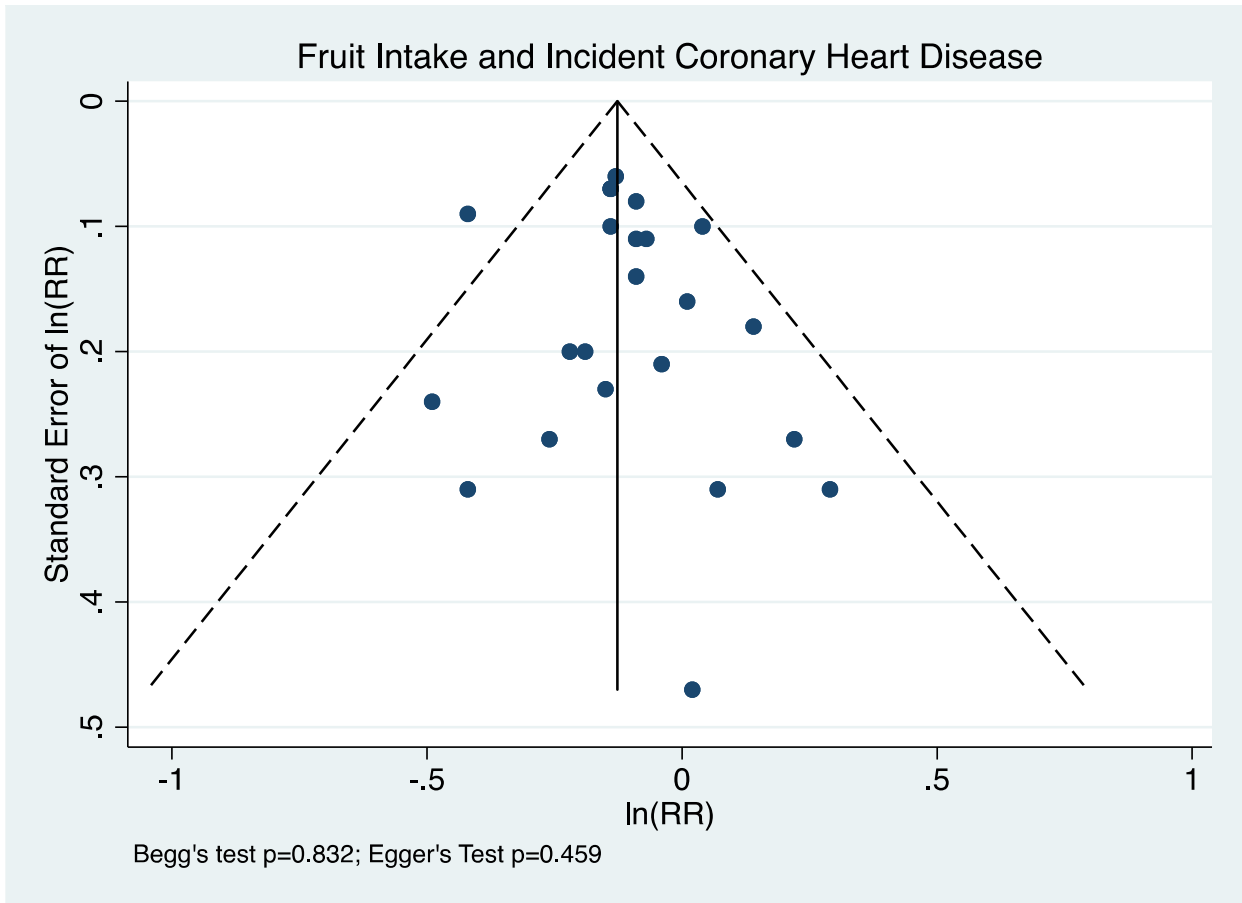


Figure S196. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

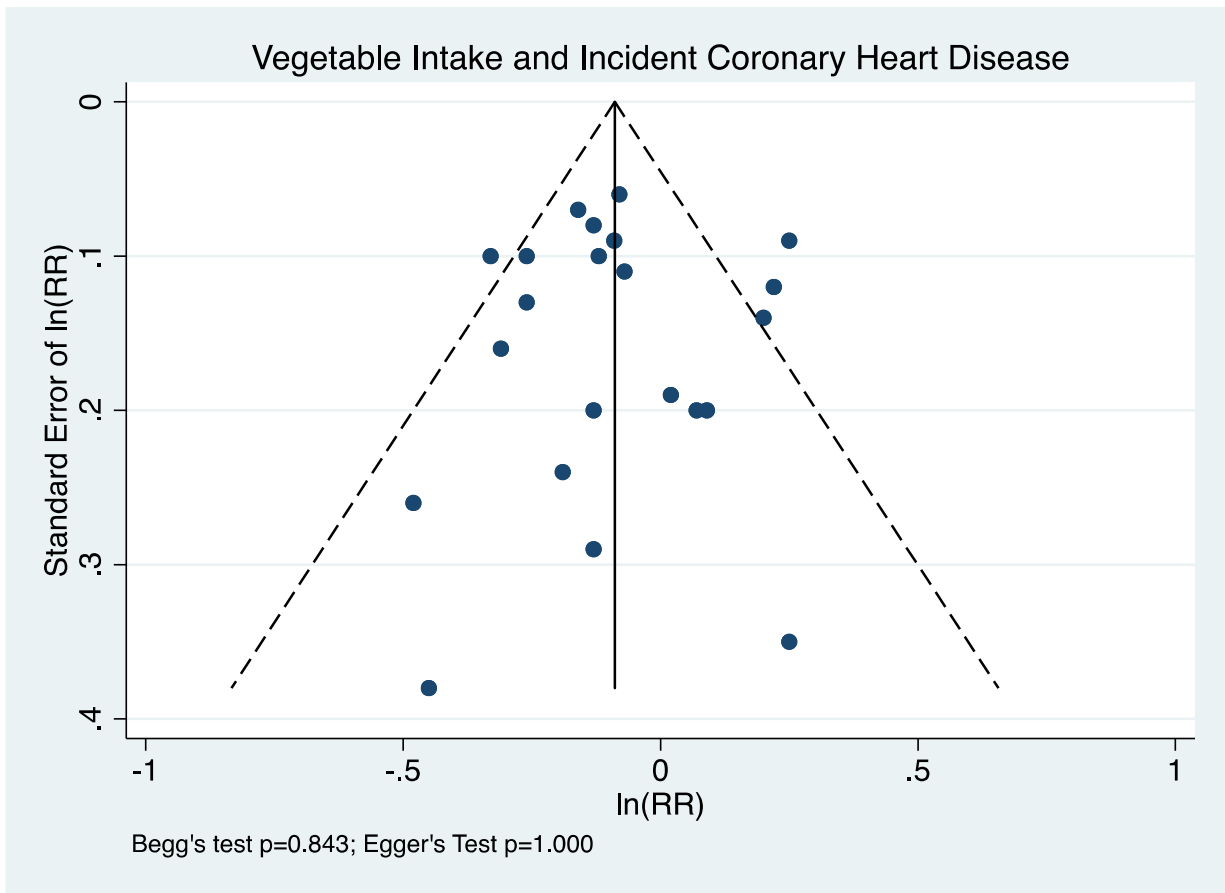


Figure S197. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease incidence comparing the highest and lowest quantiles of vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

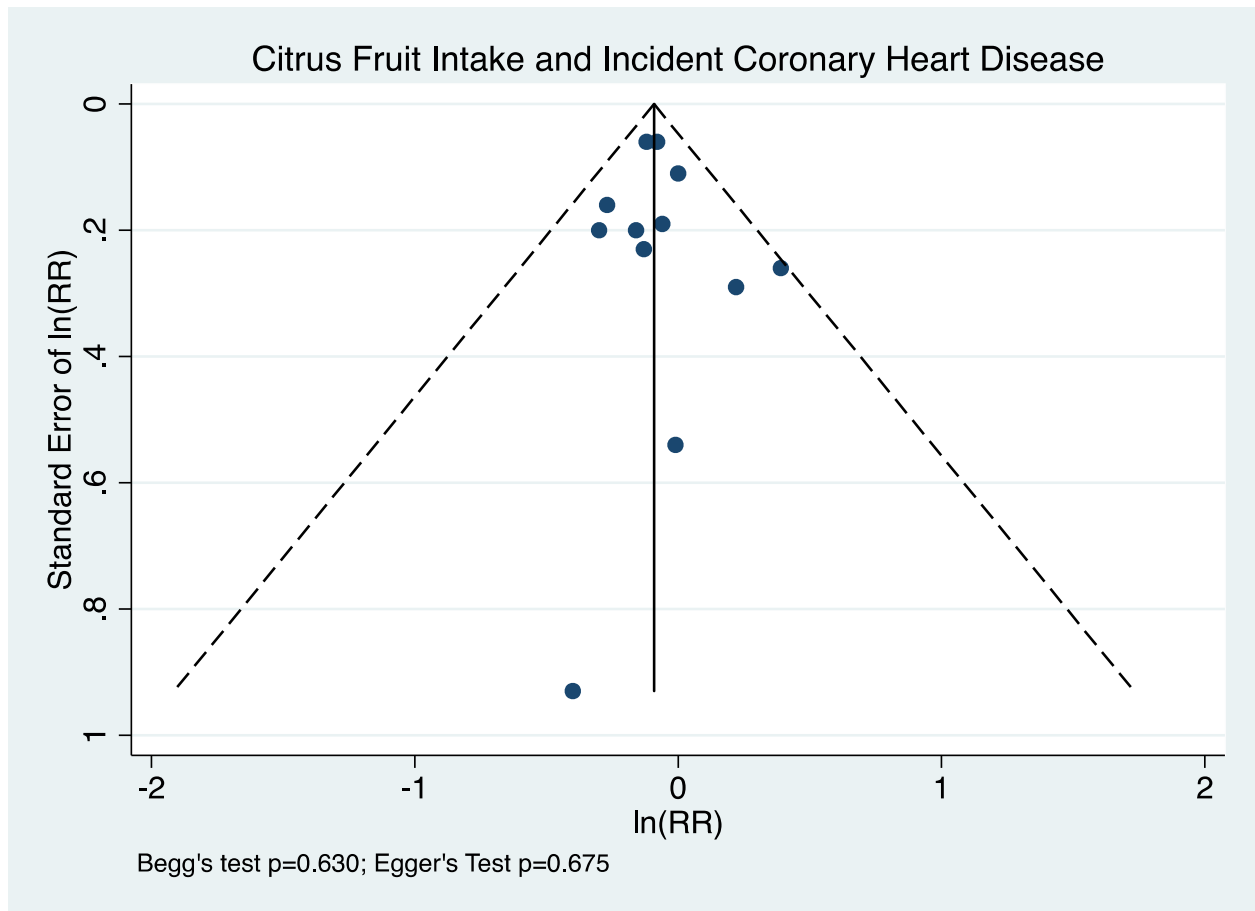


Figure S198. Funnel plot of natural logarithm relative risk [$\ln(\text{RR})$] for coronary heart disease incidence comparing the highest and lowest quantiles of citrus fruit intake. The vertical line represents the pooled effect estimated expressed as $\ln(\text{RR})$. Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the $\ln(\text{RR})$.

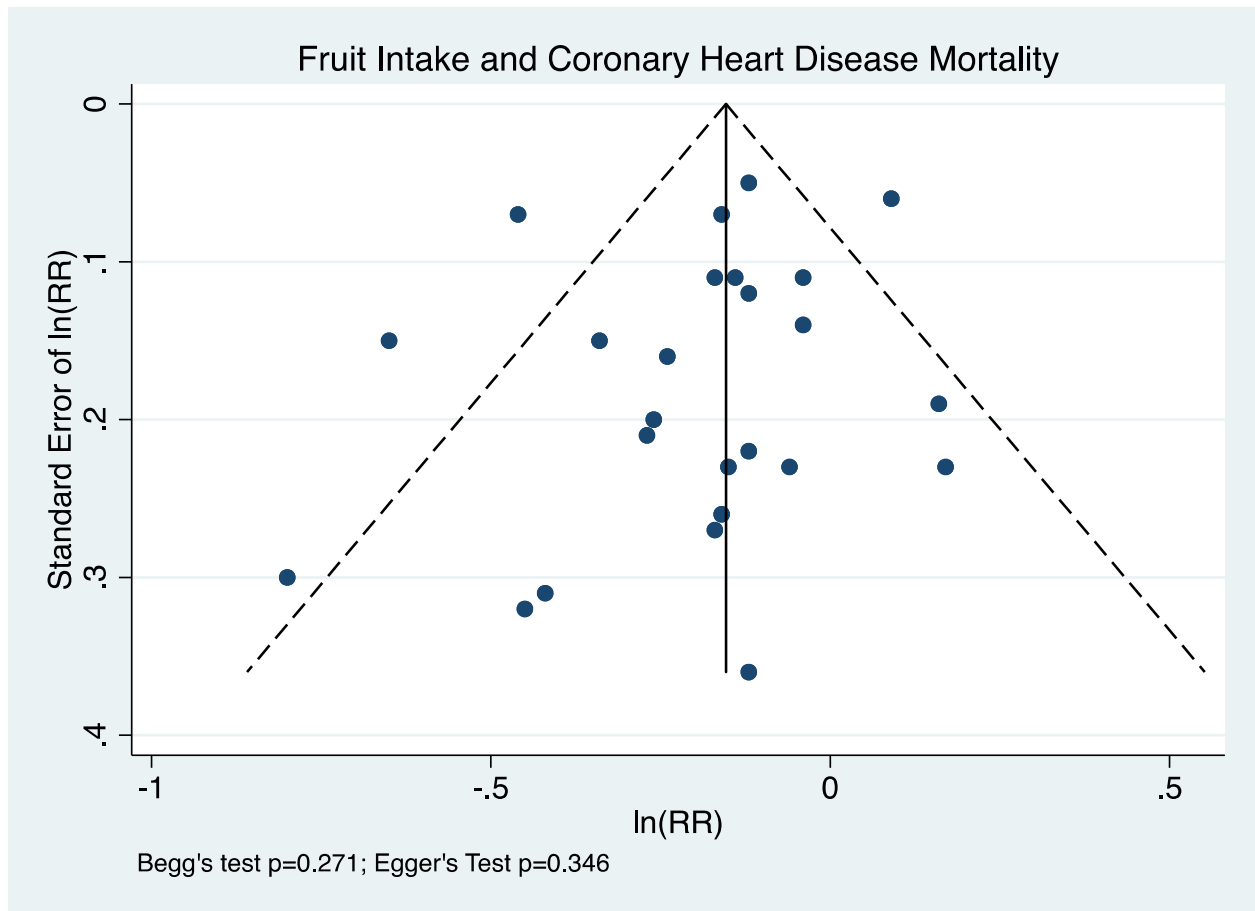
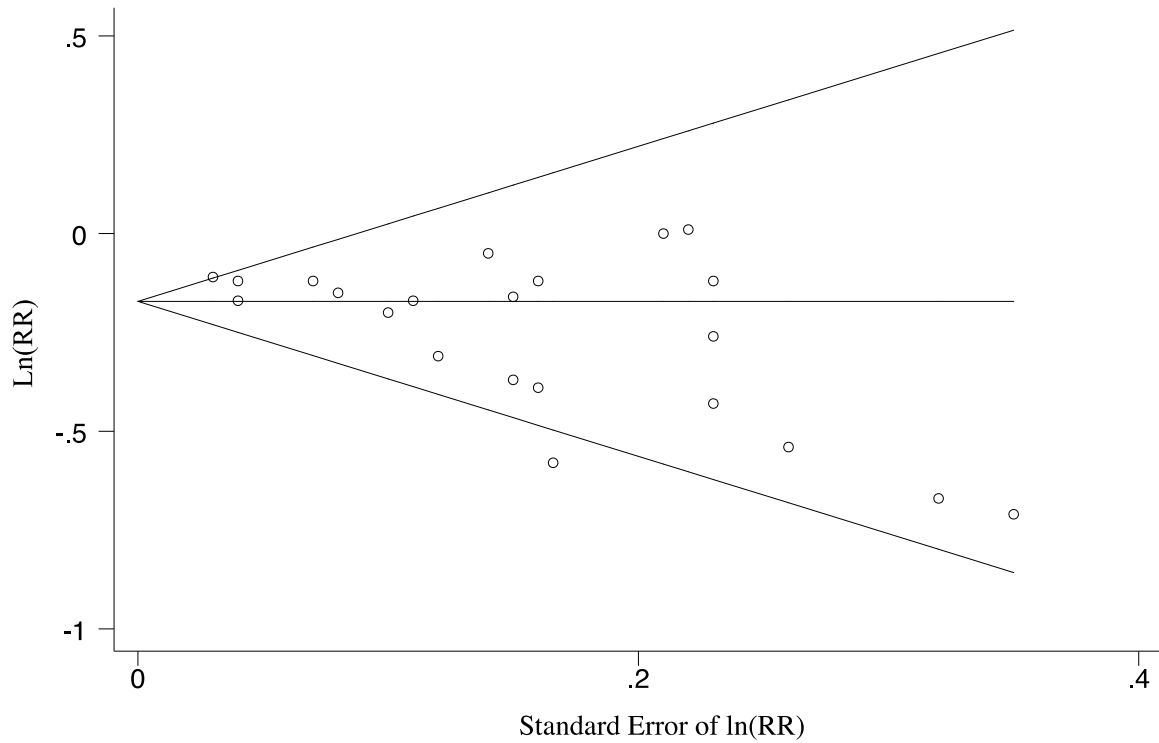


Figure S199. Funnel plot of natural logarithm relative risk [Ln(RR)] for coronary heart disease mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

Vegetable Intake and Coronary Heart Disease Mortality



Imputed RR accounting for publication bias: N/A
P-value: N/A

Figure S200. Funnel plot for trim-and-fill analysis for coronary heart disease mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [Ln(RR)]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.

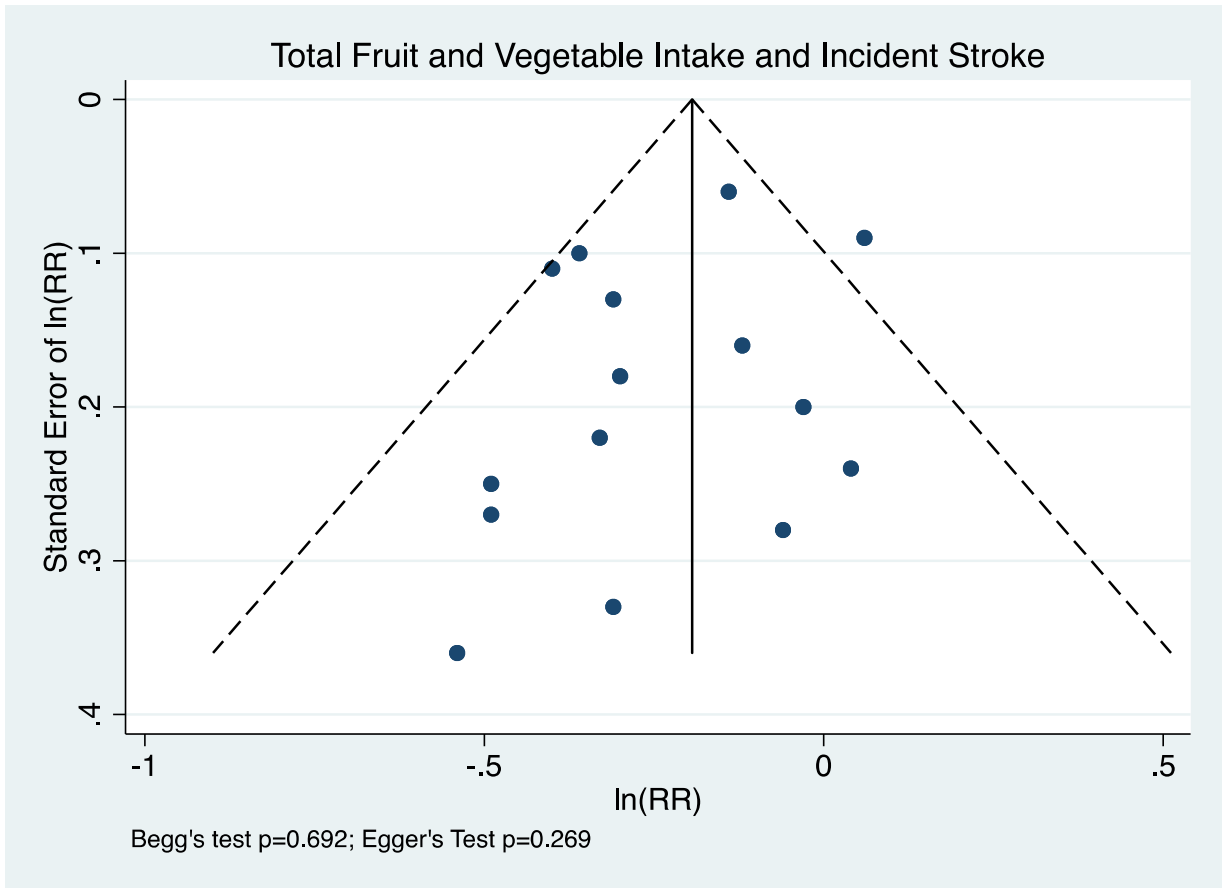


Figure S201. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke incidence comparing the highest and lowest quantiles of total fruit and vegetable intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

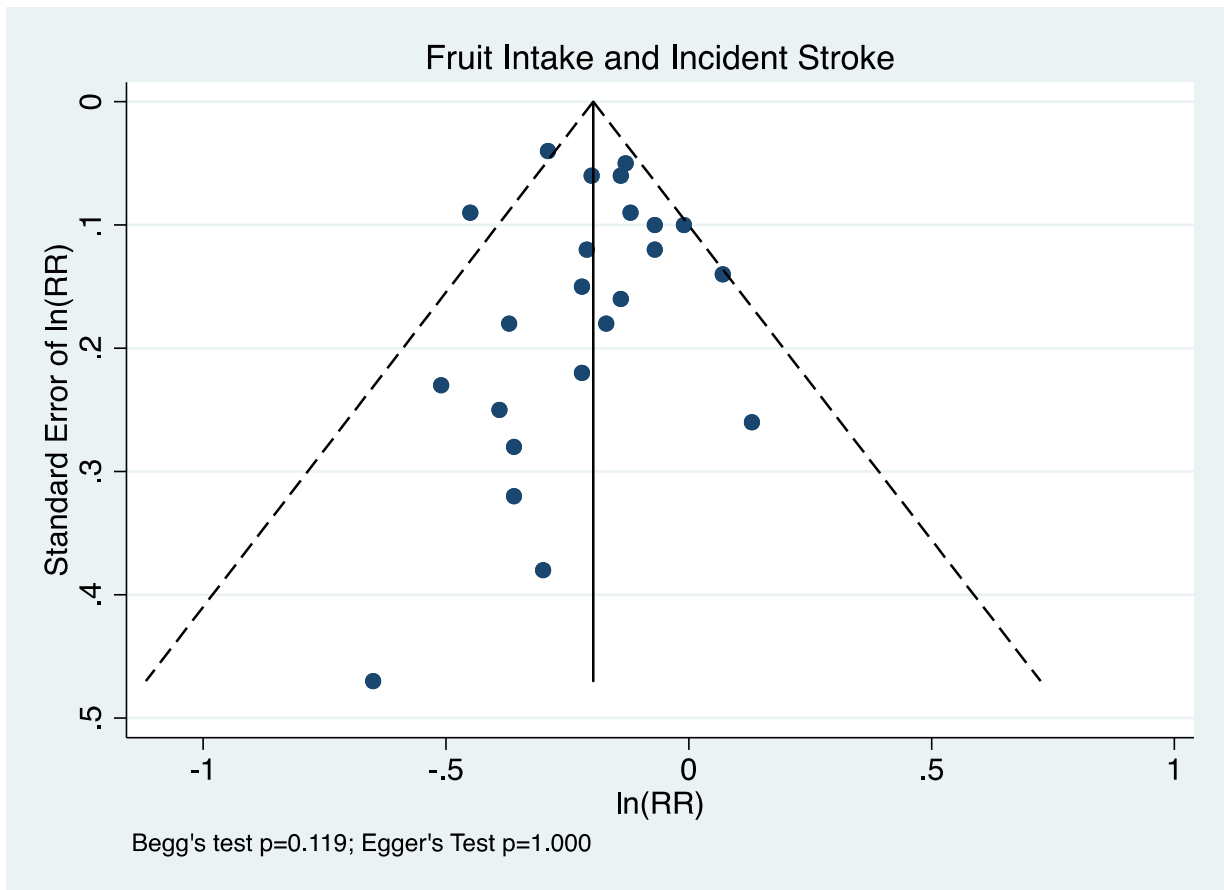
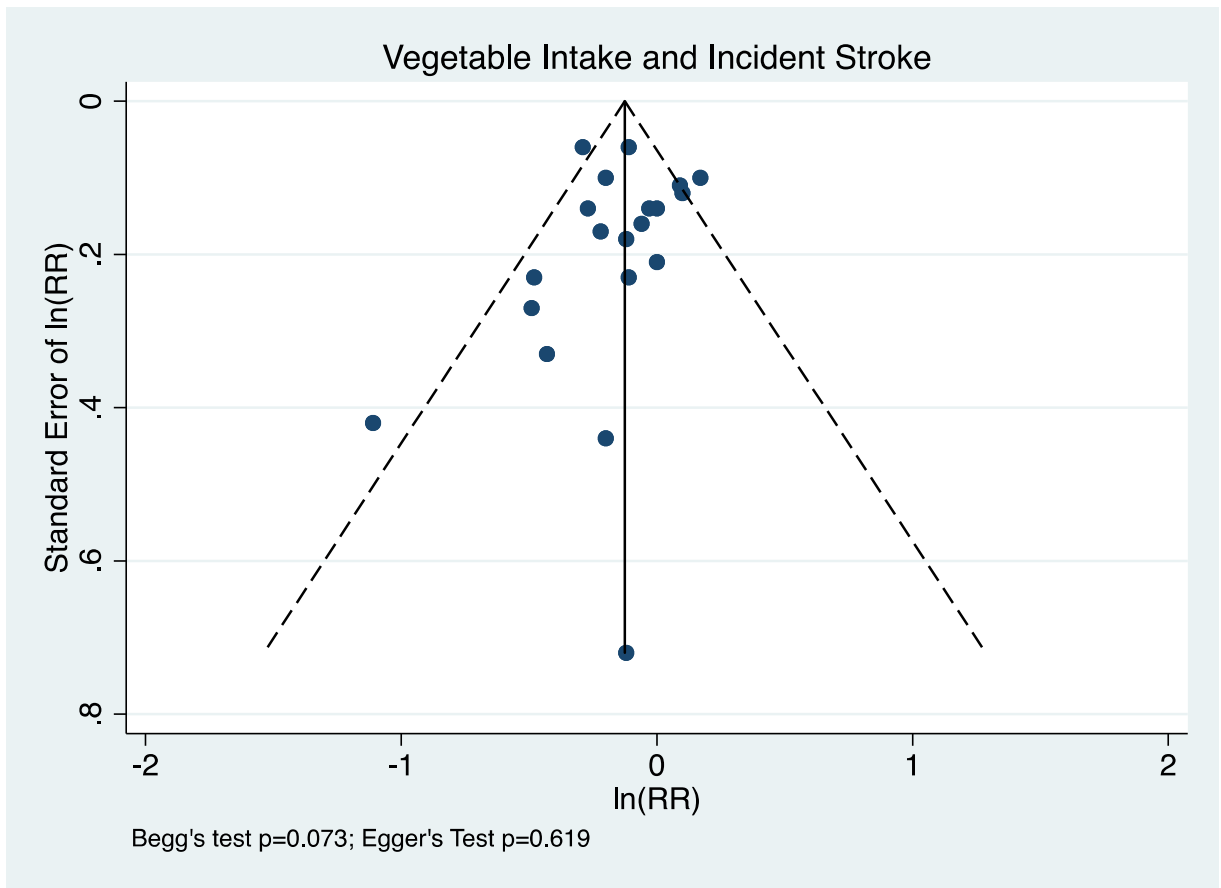
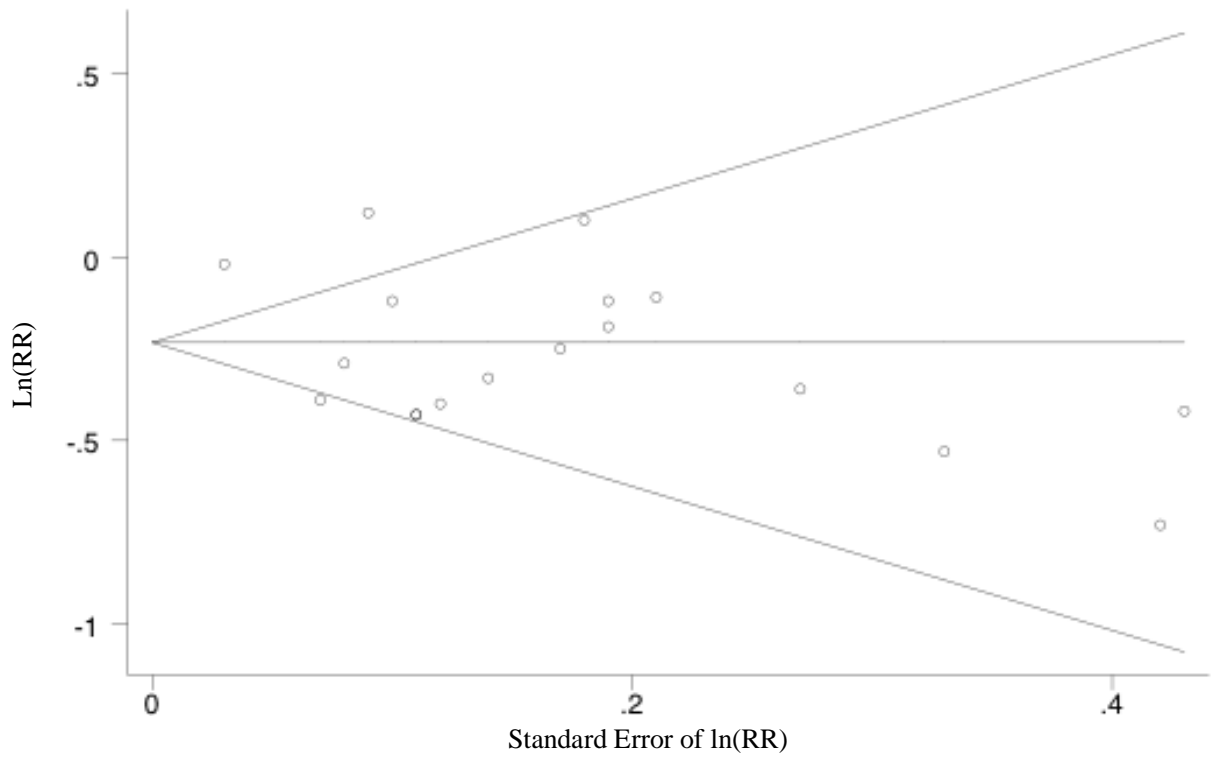


Figure S202. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke incidence comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).



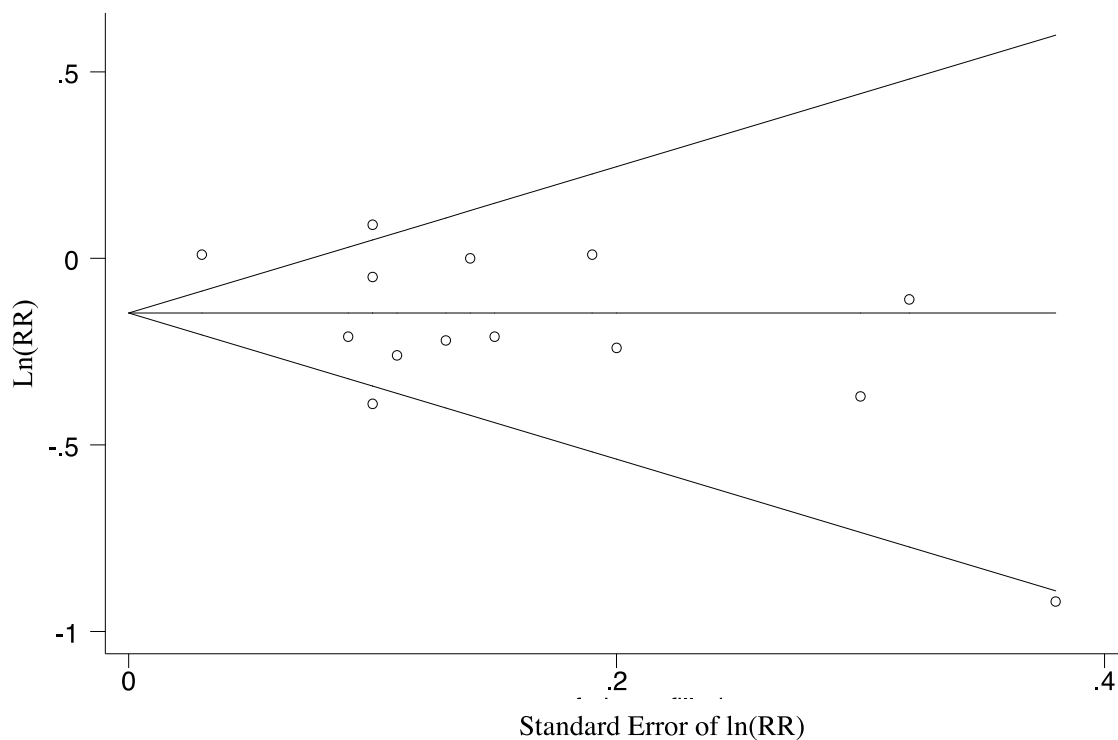
Fruit Intake and Stroke Mortality



Imputed RR accounting for publication bias: N/A
P-value: N/A

Figure S204. Funnel plot of natural logarithm relative risk [Ln(RR)] for stroke mortality comparing the highest and lowest quantiles of fruit intake. The vertical line represents the pooled effect estimated expressed as ln(RR). Dashed lines represent pseudo-95% confidence intervals. The circles represent risk estimates for each comparison, and the horizontal lines represent standard errors of the ln(RR).

Vegetable Intake and Stroke Mortality



Imputed RR accounting for publication bias: N/A

P-value: N/A

Figure S205. Funnel plot for trim-and-fill analysis for stroke mortality comparing the highest and lowest quantiles of vegetable intake. The horizontal line represents the pooled effect estimate expressed as the natural logarithm of relative risk [$\ln(RR)$]. The diagonal lines represent the pseudo-95% confidence intervals of the RR. The clear circles represent the effect estimates for each included study.