The role of nutrition in cardiovascular disease prevention

David Fäh

Berner Fachhochschule
Haute école spécialisée bernoise
Bern University of Applied Sciences

Universität Zürich[UZH]

Learning Objectives

Students will be able to:

- Explain the actual significance of CVD for Switzerland and internationally and describe trends in CVD mortality over time
- Consider the study design when evaluating associations
- Illustrate how dietary risk and protective factors may impact on CVD risk
- Describe the association between consumption of selected foods / nutrients and CVD
- Name at least 2 types of diets that aim at reducing CVD risk including their specific properties

Content

- CVD epidemiology
- Diet and CVD: scientific evidence
- Foods and CVD
- Nutrients and CVD
- Mediterranean diet
- Personalized nutrition

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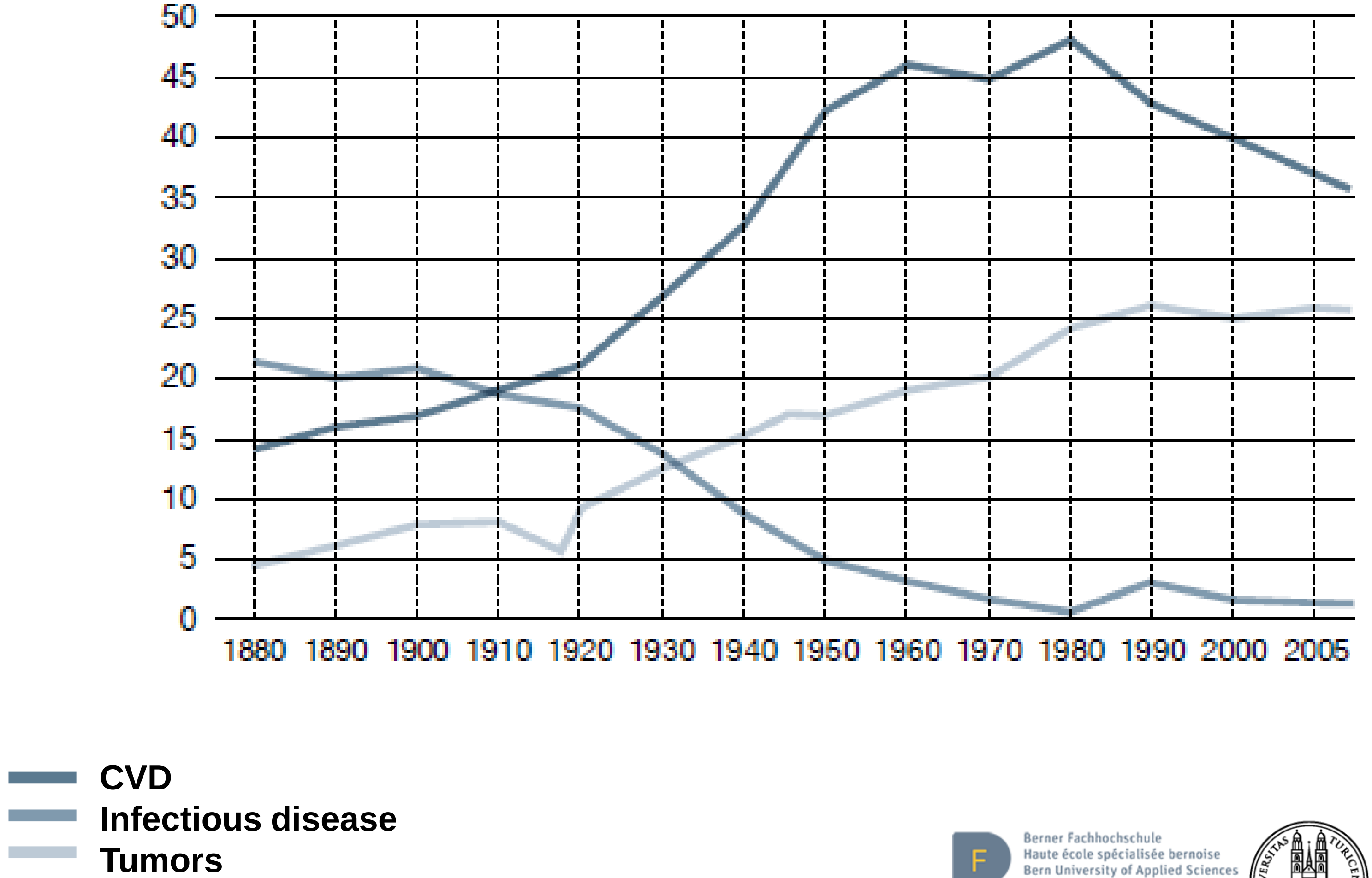
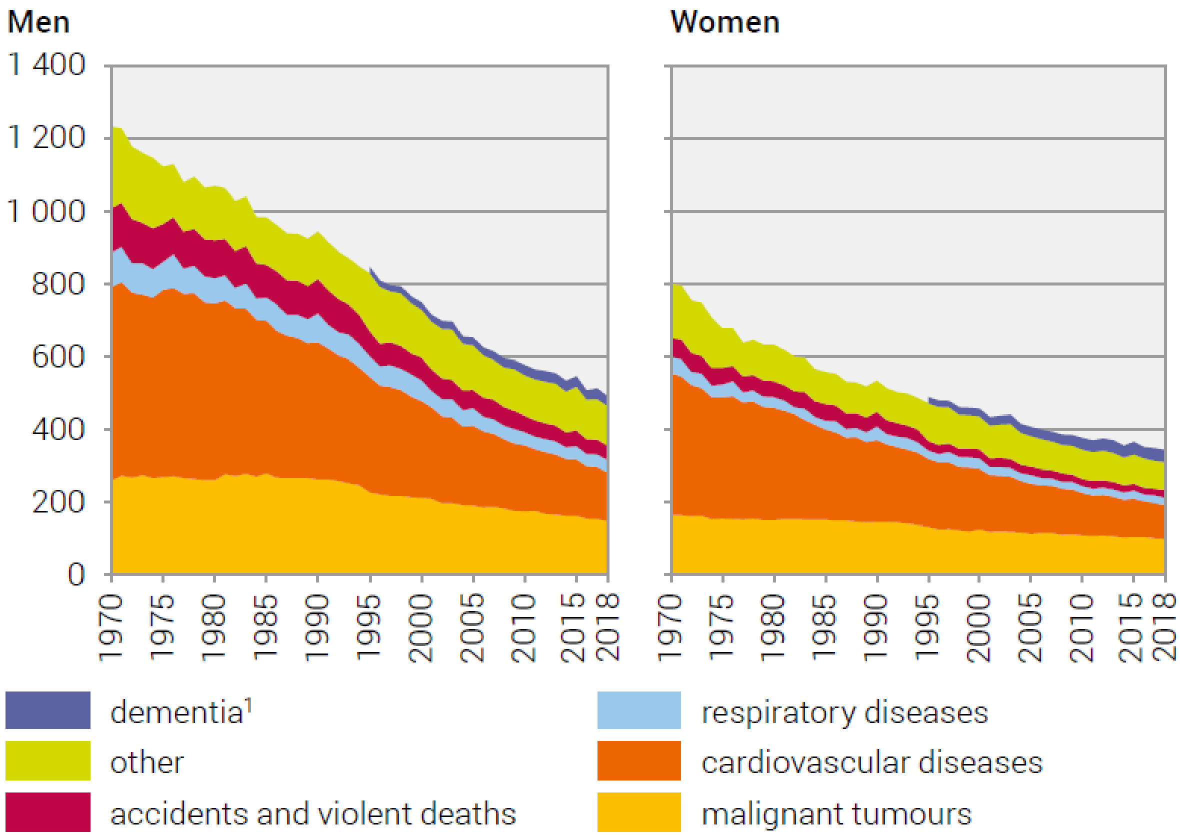
Proportion (%) of death cases attributable to selected disease groups



Standardized mortality rates per 100’000

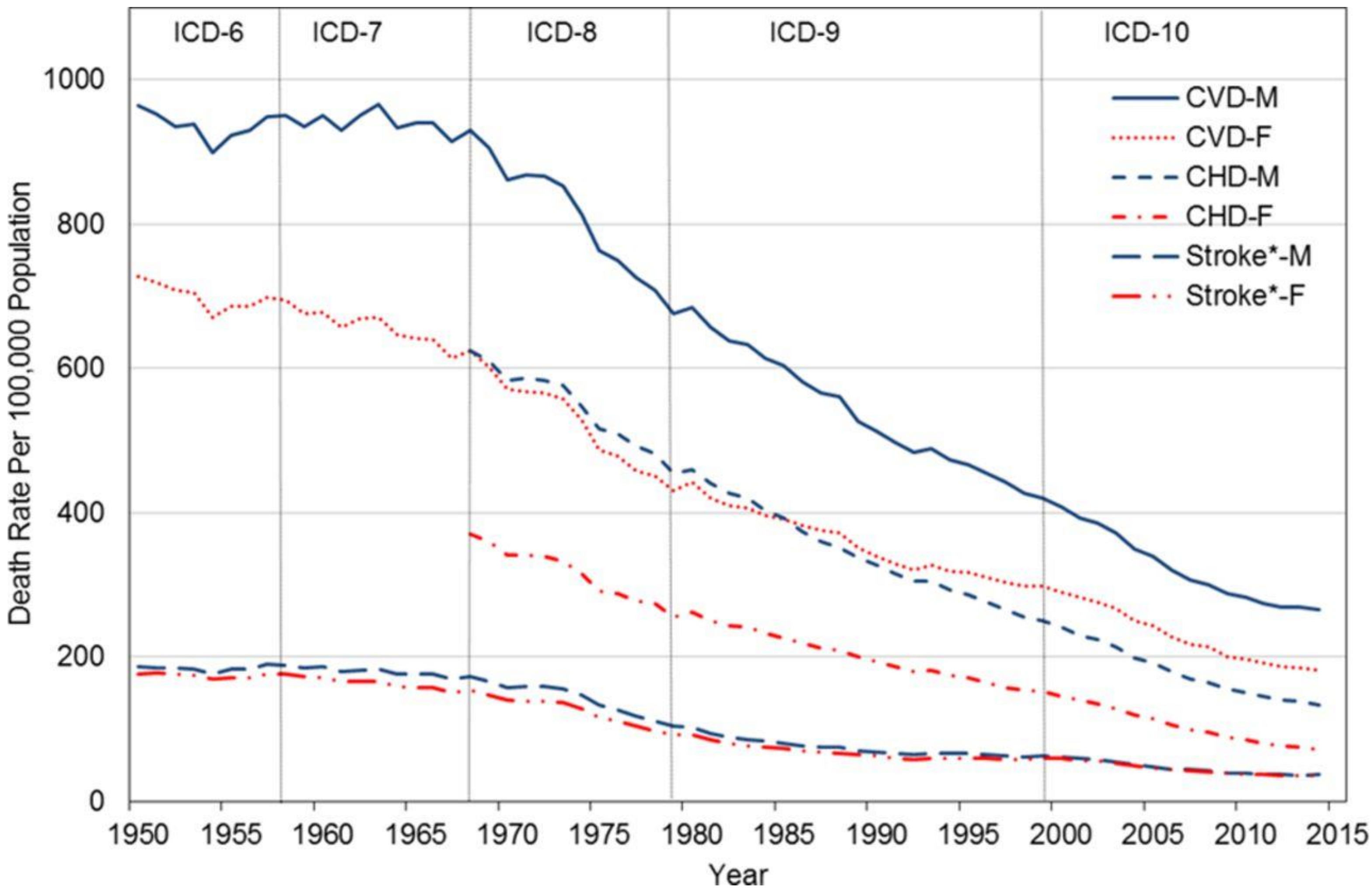


[1] surveyed from 1995 onwards

Source: FSO – Cause of Death Statistics (CoD)

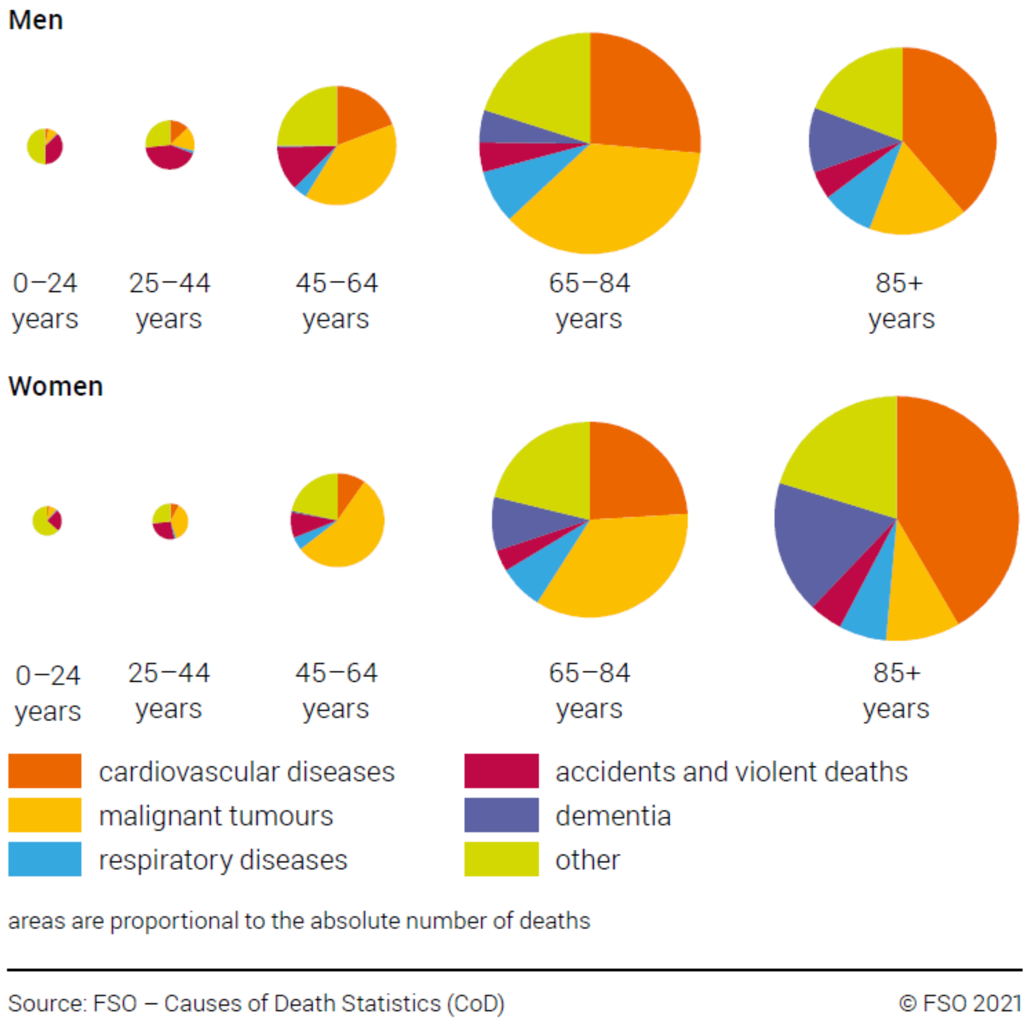
[FSO: Health - Pocket Statistics 2020]

Age-adjusted CVD mortality rates by sex, US



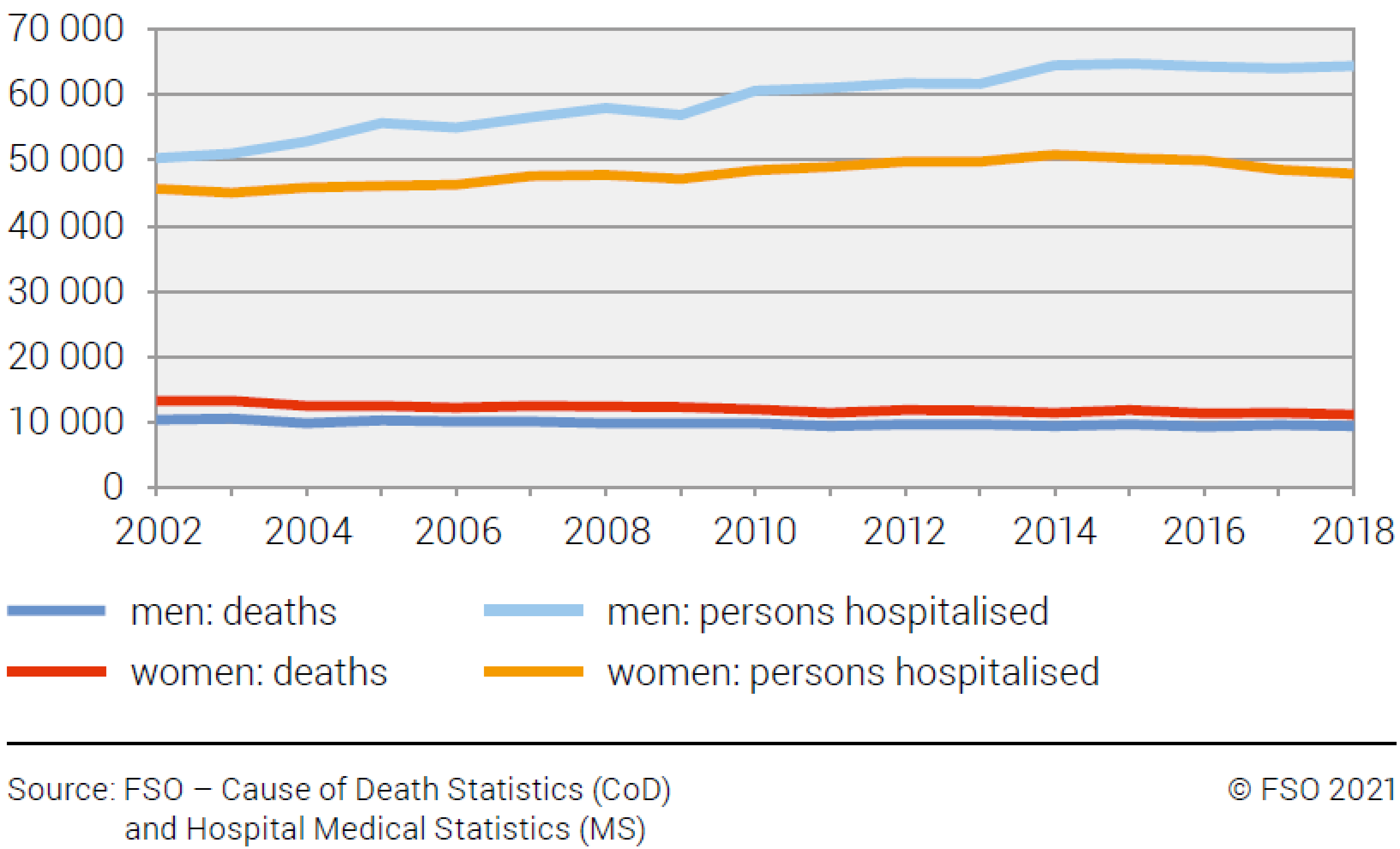
http://circres.ahajournals.org/content/120/2/366

Grouped causes of death, Switzerland, 2018 by age group and sex



FSO: Health - Pocket Statistics 2020

Deaths and persons hospitalised due to cardiovascular diseases, Switzerland



[FSO: Health - Pocket Statistics 2020]

Cardiovascular disease (CVD)

Morbidity

>60 ´000 cases / year of myocardial infarction, stroke and cardiac arrest;
110 ´000 hospital admissions because of CVD (2018)

Schweizerische Herzstiftung, BFS

[FSO: Health - Pocket Statistics 2020]

Cardiovascular disease (CVD)

Mortality

2019: 17’000 † (27% of all †);
IHD: 6´500 †;
Stroke: 3‘400 †
M>F
90% of deaths after age 65

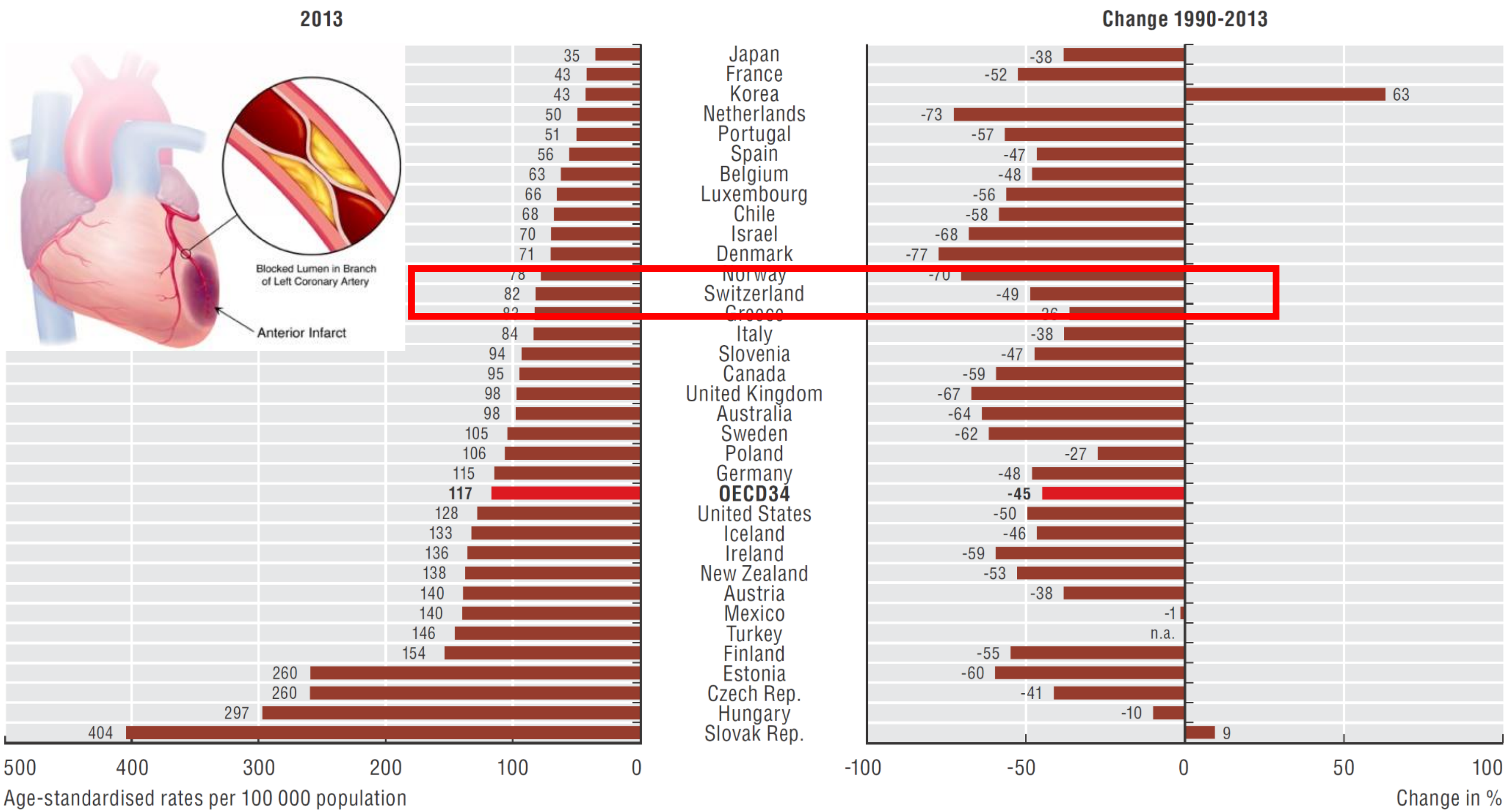
Schweizerische Herzstiftung, BFS

Morbidity

>60´000 cases / year of myocardial infarction, stroke and cardiac arrest;
110´000 hospital admissions because of CVD (2018)

FSO: Health - Pocket Statistics 2020

3.6. Ischemic heart disease mortality, 2013 and change 1990-2013 (or nearest years)



Source: *OECD Health Statistics 2015, http://dx.doi.org/10.1787/health-data-en.*

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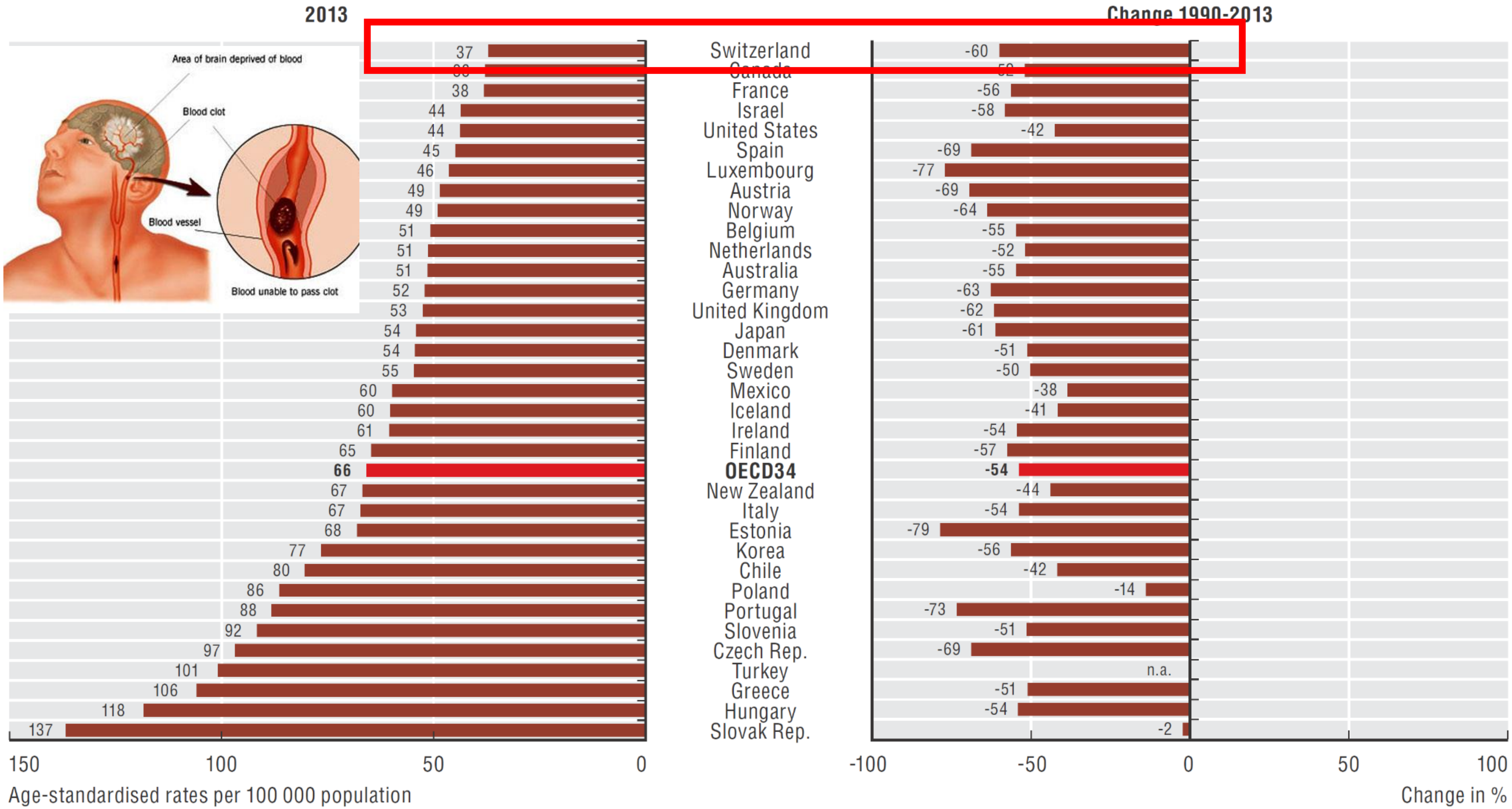
3.7. Cerebrovascular disease mortality, 2013 and change 1990-2013 (or nearest years)



Source: OECD Health Statistics 2015, http://dx.doi.org/10.1787/health-data-en.

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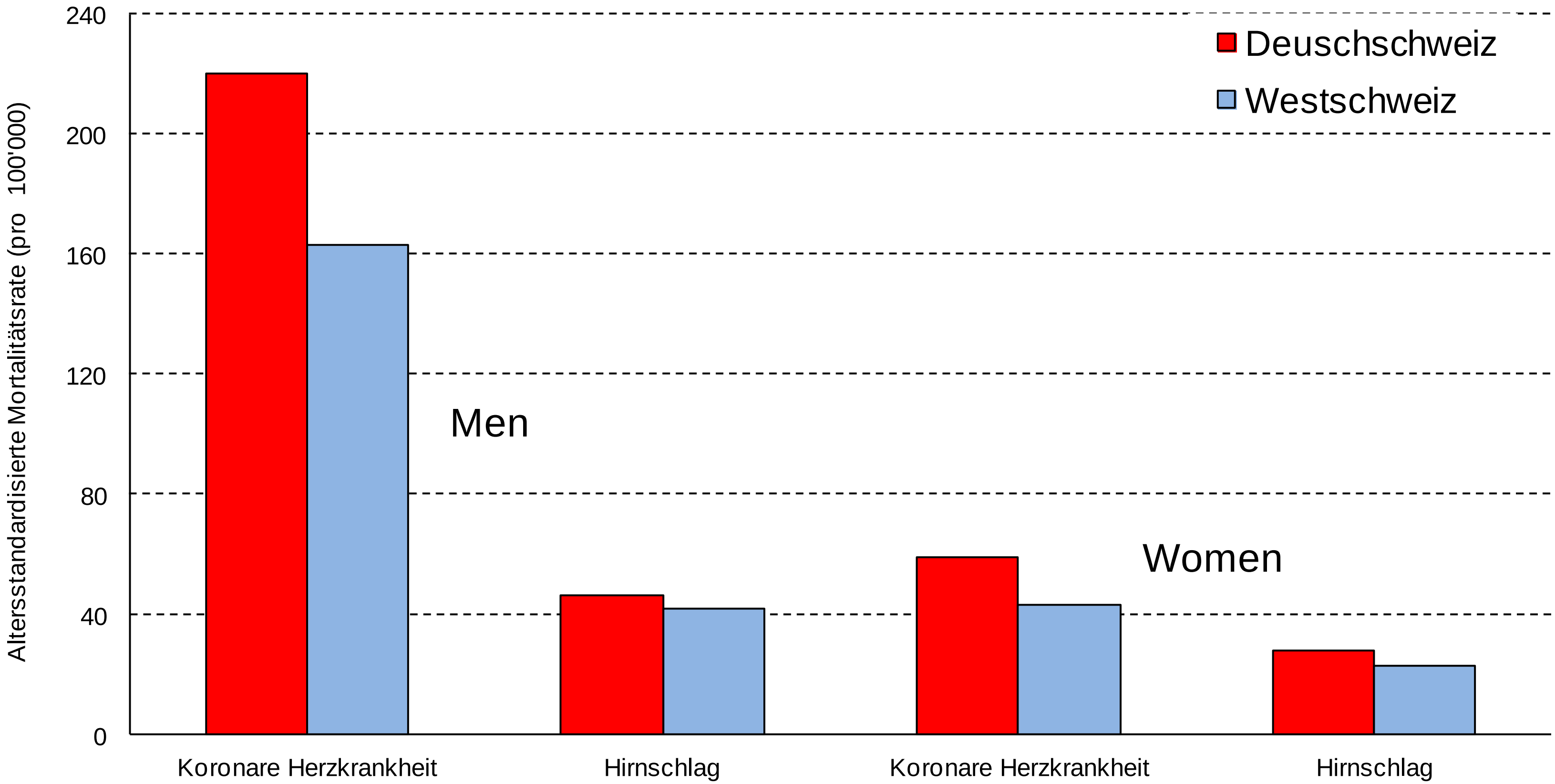
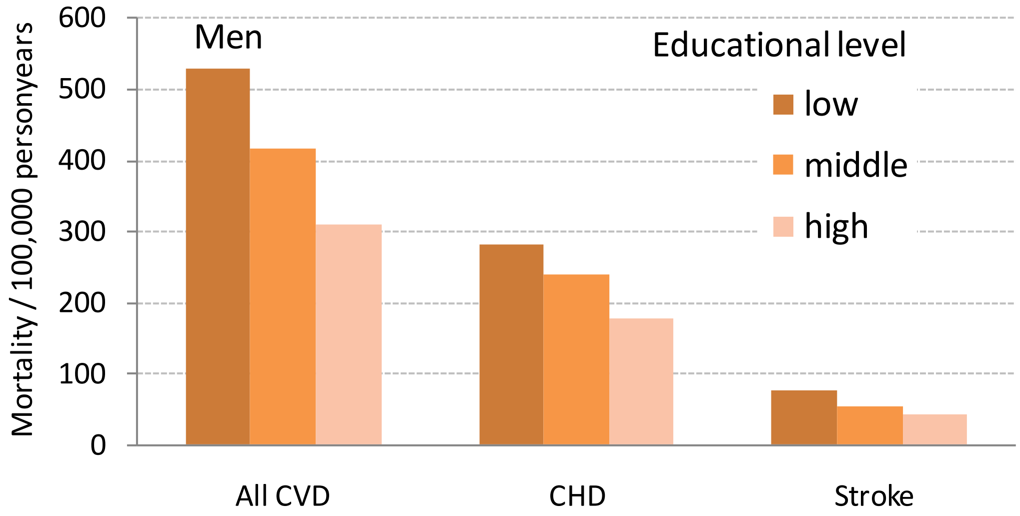
Coronary heart disease & stroke in German and French speaking part of Switzerland

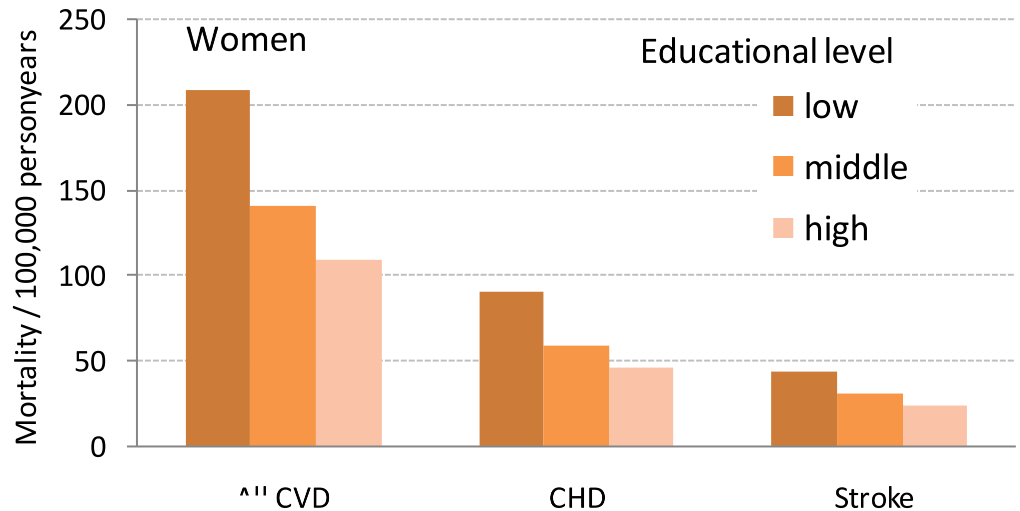


Data: Swiss National Cohort 2000

Faeh et al, JECH 2009 Aug;63(8):639-45

Inequality in CVD-mortality by education





Data: Swiss National Cohort 2000

Faeh et al, BMC Public Health. 2010 Sep 22;10:567

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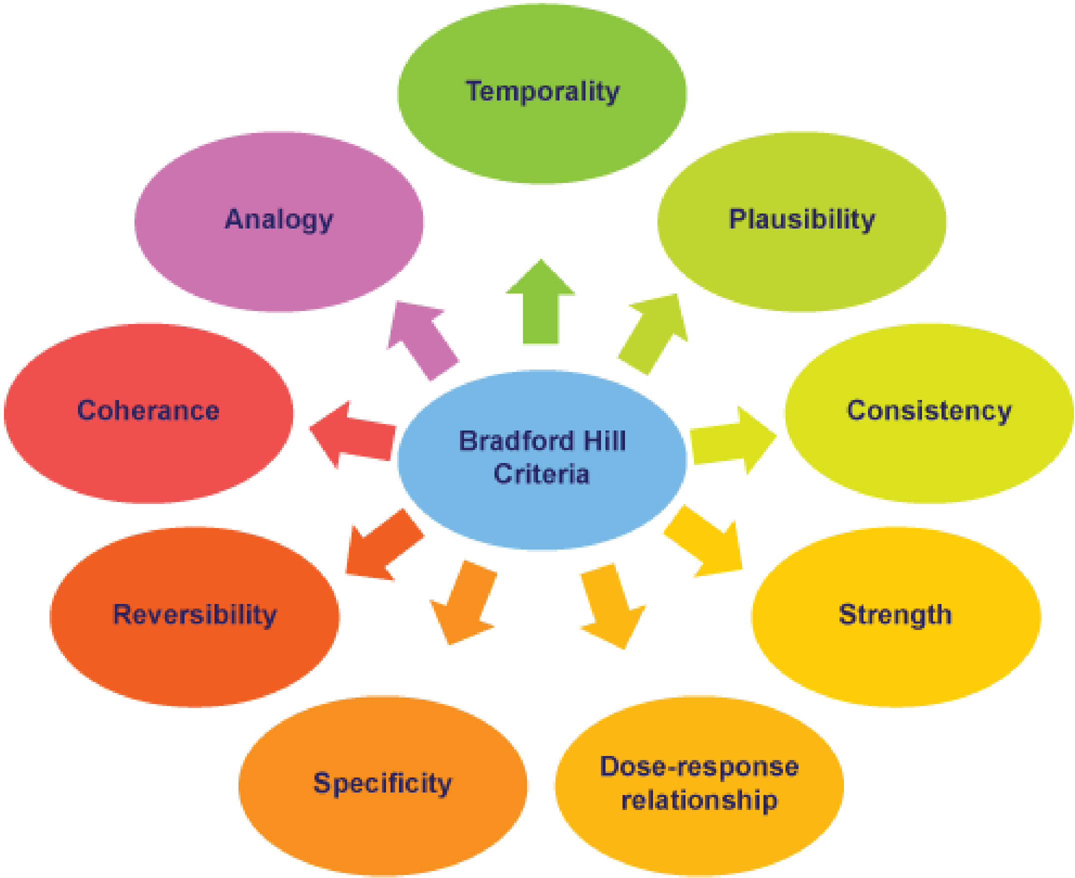
When is an association causal?



https://www.edwardtufte.com/tufte/hill

Bradford-Hill Criteria

1. **Strength** (effect size): A small association does not mean that there is not a causal effect, though the larger the association, the more likely that it is causal.
2. **Consistency** (reproducibility): Consistent findings observed by different persons in different places with different samples strengthens the likelihood of an effect.
3. **Specificity**: Causation is likely if there is a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship.
4. **Temporality**: The effect must occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay).
5. **Biological gradient**: Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect. In other cases, an inverse proportion is observed: greater exposure leads to lower incidence.
6. **Plausibility**: A plausible mechanism between cause and effect is helpful (but Hill noted that knowledge of the mechanism is limited by current knowledge).
7. **Coherence**: Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, Hill noted that "... lack of such [laboratory] evidence cannot nullify the epidemiological effect on associations".
8. **Experiment**: "Occasionally it is possible to appeal to experimental evidence".
9. **Analogy**: The effect of similar factors may be considered.

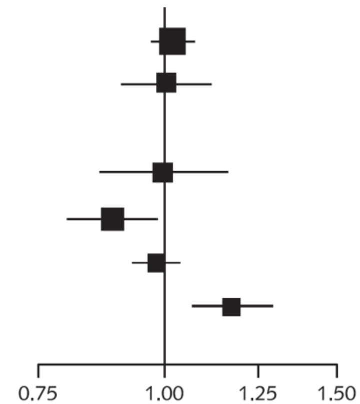
https://www.edwardtufte.com/tufte/hill

Level of Evidence	Type of Study
1a	Systematic reviews of randomized controlled trials (RCTs)
1b	Individual RCTs with narrow confidence interval
2a	Systematic reviews of cohort studies
2b	Individual cohort studies and low-quality RCTs
3a	Systematic reviews of case-control studies
3b	Case-controlled studies
4	Case series and poor-quality cohort and case-control studies
5	Expert opinion

Omega-3 fatty acids and CHD: cohort studies

Figure 1. **RRs for coronary outcomes in prospective cohort studies of dietary fatty acid intake.**

Fatty Acid Intake	Studies, *n*	Participants, *n*	Events, *n*	RR (95% CI)*
Total saturated fatty acids	20	276 763	10 155	1.03 (0.98–1.07)
Total monounsaturated fatty acids	9	144 219	6031	1.00 (0.91–1.10)
Total ω-3 fatty acids				
α-Linolenic	7	157 258	7431	0.99 (0.86–1.14)
Total long-chain ω-3	16	422 786	9089	0.87 (0.78–0.97)
Total ω-6 fatty acids	8	206 376	8155	0.98 (0.90–1.06)
Total trans fatty acids	5	155 270	4662	1.16 (1.06–1.27)



RR (95% CI) Comparing Top vs. Bottom Thirds of Baseline Dietary Fatty Acid Intake

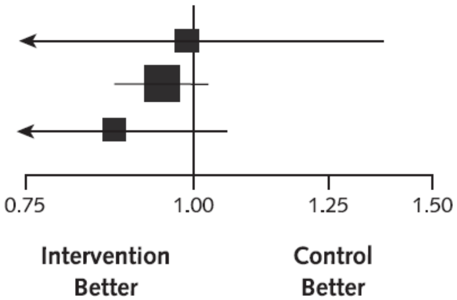
Size of the data marker is proportional to the inverse of the variance of the RR. RR = relative risk.
* Pooled estimate based on random-effects meta-analysis. Corresponding forest plots, I^2 estimates, and pooled RRs based on fixed-effects meta-analysis are provided in **Supplement 1**, available at www.annals.org.

Ann Intern Med. 2014;160:398-406.

Omega-3 fatty acids and CHD: RCTs

Figure 3. **Effect of fatty acid supplementation on risk for coronary event, derived from available randomized, controlled trials.**

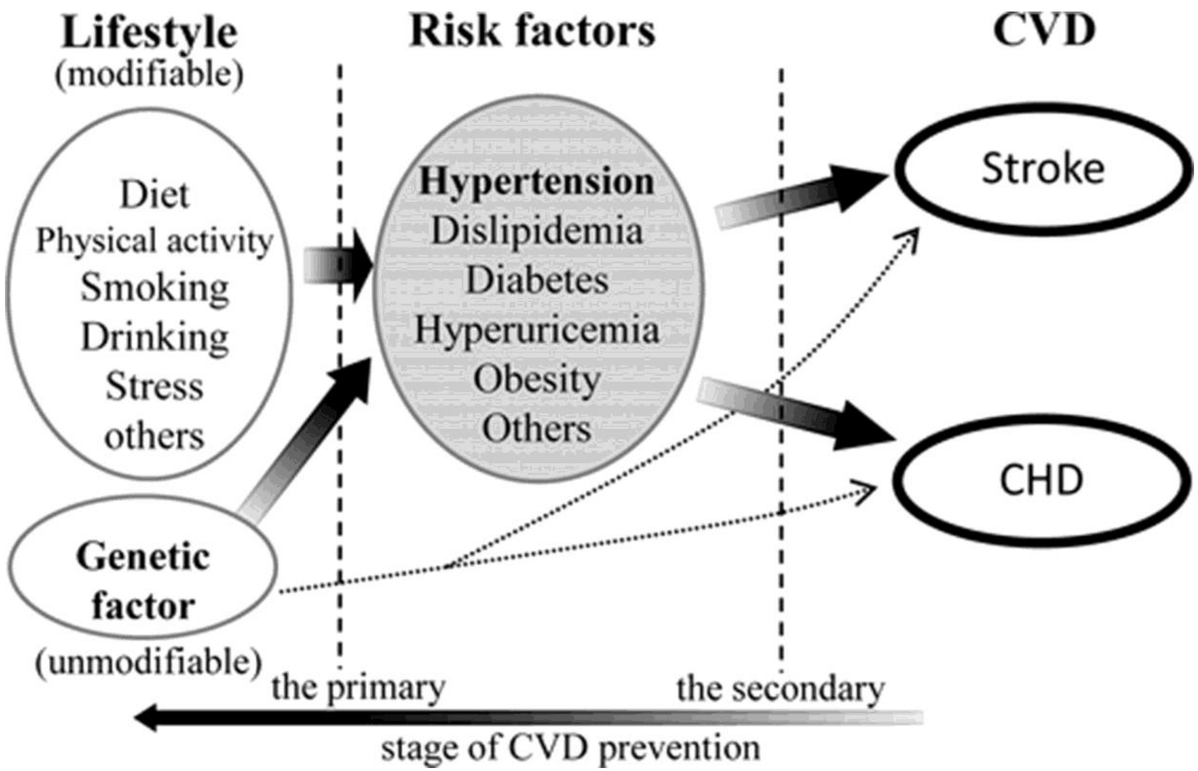
Fatty Acid Supplement	Studies, *n*	Events/Participants, *n/N*		RR (95% CI)*
		Intervention Group	Control Group	
α-Linolenic acid	4	199/9444	220/9422	0.97 (0.69–1.36)
Long-chain ω-3 fatty acid	17	2426/38 303	2548/38 277	0.94 (0.86–1.03)
ω-6 fatty acid†	8	459/7245	515/7231	0.86 (0.69–1.07)



Conclusion: Current evidence does not clearly support cardiovascular guidelines that encourage high consumption of polyunsaturated fatty acids and low consumption of total saturated fats

Ann Intern Med. 2014;160:398-406.

Intermediate risk factors



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CVD: risk factors vs. disease risk

1. Plant sterols (e.g. Becel®): Reduction of blood cholesterol but increase in CVD risk (?)
2. N-3 fatty acids: reduction of triglycerides but virtually no reduction of CVD risk
3. Coffee: Increases LDL-cholesterol and blood pressure; Acrylamide but likely reduces CVD risk & cancer risk
4. Salt: reduction in blood pressure but reduction in CVD risk not accordingly (as could be expected)?
5. Low-Carb diet: improvement in blood lipids and glucose parameters but increase in all-cause mortality
6. Alcohol (moderate consumption): improvement in blood lipids & coagulation factors but no decrease in all-cause mortality (?)

RCT: for diet a lot of limitations

- Impossible to blind complex, behavioral exposures
- Selective dropout/retention in control/intervention group
- Hard to control for multiple exposures (with control diets) by design
- Incomplete adherence in the intervention arm; control group reactivity (awareness of being observed)
- Limited generalizability for the specific exposure and study sample
- Questions about real-world effectiveness (where people choose therapies)

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4863268/;
https://jamanetwork.com/journals/jama/fullarticle/2698337

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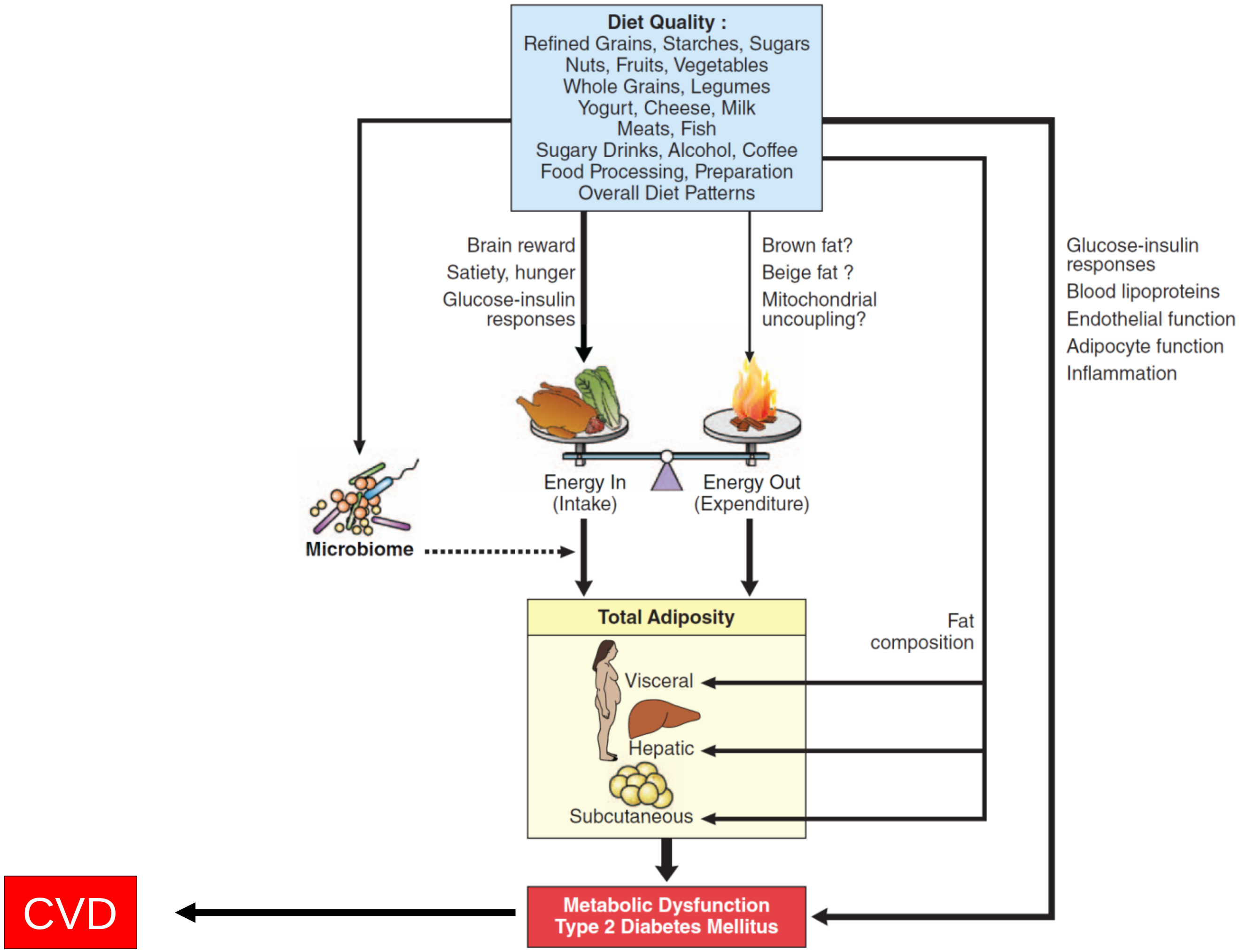
Impact of diet on CVD



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Diet and NCD: risk/protective factors

Risk factors	Protective factors
• Excess calories • Overweight/obesity • Alcohol • Food processing • Red and processed red meat • Salt • Refined sugars (e.g. from sugar sweetened beverages) • Refined Carbohydrates (e.g. from white bread, pasta, white rice) • Unhealthy fats (trans fats) • Food contamination (virus, bacteria, fungus) • Dietary supplements (?) • Milk / dairy (?), Acrylamide	• Low energy density • High micronutrient density • Fruit and vegetables • Nuts / Seeds • Dietary fibres • Legumes • Healthy fats (olive oil) • Fish (?) • Healthy Eating behaviour

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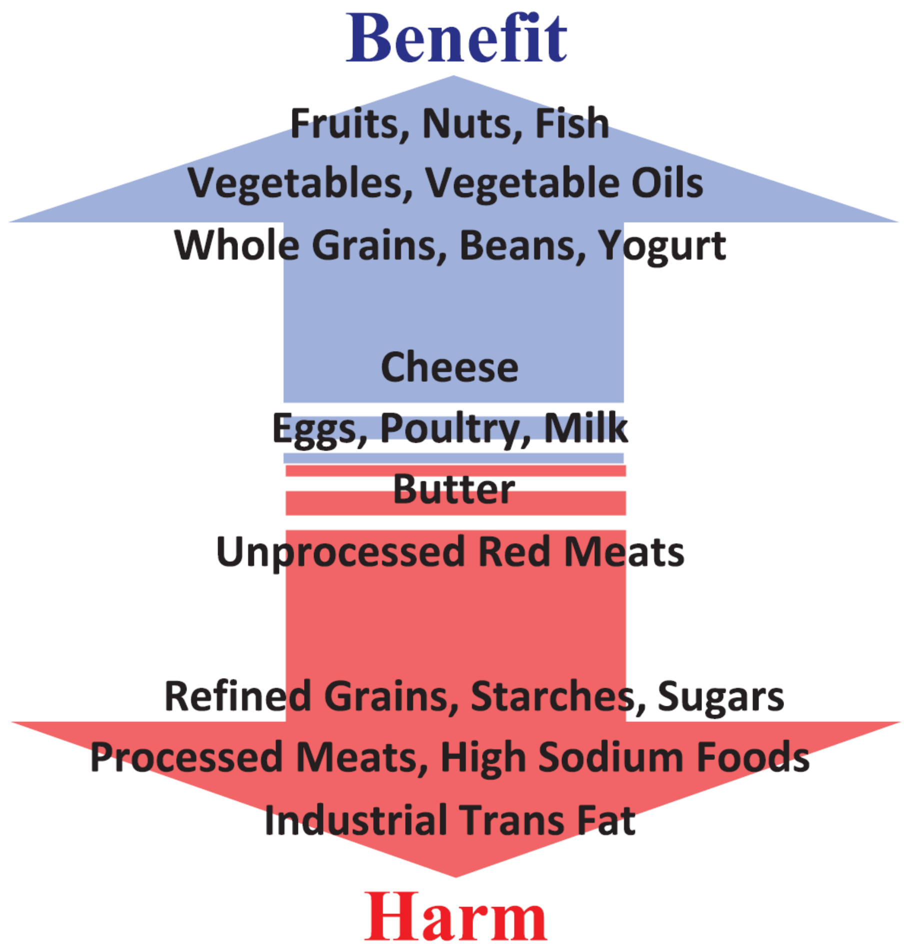
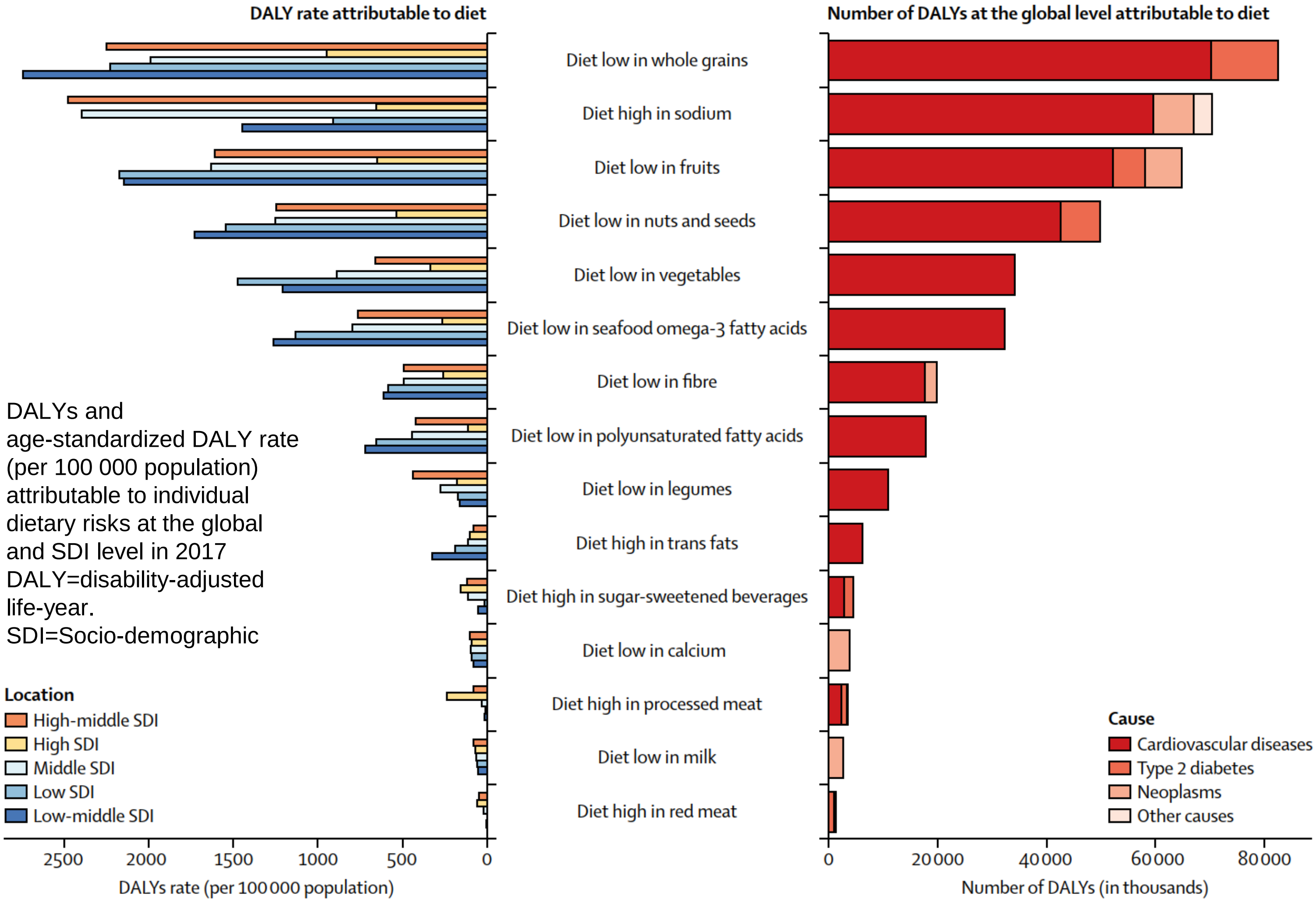


Figure 3. Evidence-based dietary priorities for cardiometabolic health. The placement of each food/factor is based on its net effects on cardiometabolic health, across all risk pathways and clinical end points, and the strength of the evidence, as well. For dietary factors not listed (eg, coffee, tea, cocoa), the current evidence remains insufficient to identify these as dietary priorities for either increased or decreased consumption (see Table 3).

http://circ.ahajournals.org/content/133/2/187.long

DALYs attributable to dietary risks, 2017



DALYs and age-standardized DALY rate (per 100 000 population) attributable to individual dietary risks at the global and SDI level in 2017
DALY=disability-adjusted life-year.
SDI=Socio-demographic

http://circ.ahajournals.org/content/133/2/187.long

Published online April 3, 2019 http://dx.doi.org/10.1016/S0140-6736(19)30041-8

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	Endpoint	No. of studies	No. of subjects	No. of events	Unit	RR	Reference
Fruits	CHD	16 PCs	817,155	13,786	Each 1 serving/day (100 g)	0.94 (0.91, 0.98)	Gan Y 2015
	Stroke	8 PCs	377,159	9,706	Each 1 serving/day (100 g)	0.82 (0.75, 0.91)	Hu D 2014
	Diabetes	7 PCs	368,232	21,063	Each 1 serving/day (100 g)	0.94 (0.89, 1.00)	Li M 2014
Vegetables	CHD	14 PCs	705,316	13,135	Each 1 serving/day (100 g)	0.95 (0.92, 0.98)	Gan Y 2015
	Stroke	6 PCs	342,118	8,854	Each 1 serving/day (100 g)	0.94 (0.90, 0.99)	Hu D 2014
	Diabetes	5 PCs	173,995	18,758	Each 1 serving/day (100 g)	0.98 (0.89, 1.08)	Li M 2014
Green leafy vegetables	Diabetes	3 PCs	127,148	13,331	Each 1 serving/day (100 g)	0.76 (0.62, 0.94)	Li M 2014
Legumes	Stroke	6 PCs	254,628	6,690	Each 4 servings/wk (400 g)	0.98 (0.84, 1.14)	Afshin A 2014
	CHD	4 PCs	198,904	6,514	Each 4 servings/wk (400 g)	0.86 (0.78, 0.94)	Afshin A 2014
	Diabetes	2 PCs	100,179	2,746	Each 4 servings/wk (400 g)	0.78 (0.50, 1.14)	Afshin A 2014
Whole grains	CHD	6 PCs	--	5,383	high vs low	0.78 (0.71, 0.86)	Tang G 2015
	Stroke	4 PCs	207,984	877	2.5 vs. 0.2 servings/day	0.83 (0.68, 1.14)	Mellen P 2008
	Diabetes	10 PCs	385, 868	19,829	Each 1 serving/day (50 g)	0.81 (0.74, 0.89)	Aune D 2013
Nuts and seeds	CHD death	5 PCs, 1 RCT	206,114	6,749	Each 4 servings/week (4 oz [113 g])	0.76 (0.69, 0.84)	Afshin A 2014
	Nonfatal CHD	3 PCs, 1 RCT	141, 390	4,280	Each 4 servings/week (4 oz [113 g])	0.78 (0.67, 0.92)	Afshin A 2014
	Diabetes	5 PC, 1 RCT	230,216	13,308	Each 4 servings/week (4 oz [113 g])	0.87 (0.81, 0.94)	Afshin A 2014
Fish	CHD Death	12 PCs	282,075	4,195	2-4 servings/week vs. ≤3 servings/month	0.79 (0.67, 0.92)	Zheng J 2012
	Stroke	8 PCs	394,958	16,890	> 5 vs. 1 serving/week	0.88 (0.81, 0.96)	Chowdhury R 2012
	Diabetes	13 PCs	481,489	20,830	Each 1 serving/day (100 g)	1.12 (0.94, 1.34)	Wu J 2012
Unprocessed red meats	CVD death	13 PCs	1,070,215	24,241	high vs low	1.12 (0.95, 1.33)	Abete I 2014
	Stroke	5 PCs	239,251	9,593	Each 1 serving/day (100 g)	1.13 (1.03, 1.23)	Chen G 2013
	Diabetes	9 PCs	447,333	28,206	Each 1 serving/day (100 g)	1.19 (1.04, 1.37)	Pan A 2011
Processed red meats	CVD death	6 PCs	1,186,761	35,537	Each 1 serving/day (50 g)	1.24 (1.09, 1.40)	Abete I 2014
	Stroke	5 PCs	239,251	9,593	Each 1 serving/day (50 g)	1.11 (1.02, 1.20)	Chen G 2013
	Diabetes	8 PCs	372,391	26,234	Each 1 serving/day (50 g)	1.51 (1.25, 1.83)	Pan A 2011
White meat (poultry, rabbit)	CVD death	5 PCs	1,197,805	31,535	Each 1 serving/day (100 g)	1.00 (0.87, 1.15)	Abete I 2014
Total dairy	CHD	10 PCs	253,260	8,792	high vs low	0.94 (0.82, 1.07)	Qin L 2015
	Stroke	16 PCs	764,635	28,138	high vs low	0.88 (0.82, 0.94)	Hu D 2014
	Diabetes	14 PCs	459,790	35,863	Each 1 serving/day	0.98 (0.96, 1.01)	Chen M 2014
Milk	CHD	6 PCs	259,162	4,391	Each 1 serving/day (200 ml)	1.00 (0.96, 1.04)	Soedamah-Muthu S 2011
	Stroke	9 PCs	525,609	22,382	high vs low	0.91 (0.82, 1.01	Hu D 2014
	Diabetes	7 PCs	167,982	15,149	Each 1 serving/day (200 g)	0.87 (0.72, 1.04)	Aune D 2013
Cheese	CHD	7 PCs	--	--	high vs low	0.84 (0.71, 1.00)	Qin L 2015
	Stroke	5 PCs	282,439	9,919	high vs low	0.94 (0.89, 0.995)	Hu D 2014
	Diabetes	8 PCs	242,960	17,620	Each 1 serving/day (50 g)	0.92 (0.86, 0.99)	Aune D 2013
Butter	CHD	5 PCs	--	--	high vs low	1.02 (0.88, 1.20)	Qin L 2015
	Stroke	3 PCs	173,853	5,299	high vs low	0.95 (0.85, 1.07)	Hu D 2014
Yogurt	CHD	5 PCs	--	--	high vs low	1.06 (0.90, 1.34)	Qin L 2015
	Diabetes	9 PCs	408,096	32,995	Each 1 serving/day (½ cup)	0.82 (0.70, 0.96)	Chen M 2014
Eggs	CHD	7 PCs	263,938	5,847	Each 1 serving/day (1 egg)	0.99 (0.85, 1.15)	Rong Y 2013
	Stroke	6 PCs	210,404	7,579	Each 1 serving/day (1 egg)	0.91 (0.81, 1.02)	Rong Y 2013
	Diabetes	5 PCs	69,297	4,889	≥1 egg/day vs. never or <1 egg/week	1.42 (1.09, 1.86)	Shin J 2013
100% fruit juice	Diabetes	11 PCs	407,288	34,549	Each 1 serving/day (8 oz.)	1.06 (0.98, 1.14)	Imamura F 2015
Sugar-sweetend beverages	Diabetes, non-BMI adjusted	13 PCs	421,973	36,492	Each 1 serving/day (8 oz.)	1.42 (1.19, 1.69)	Imamura F 2015
	Diabetes, BMI adjusted	17 PCs	464,937	38,253	Each 1 serving/day (8 oz.)	1.27 (1.10, 1.46)	Imamura F 2015
	CHD	4 PCs	194,664	7,396	Each 1 serving/day (8 oz.)	1.17 (1.10, 1.24)	Xi B 2015
Coffee—	CVD	29 PCs	--	--	3 vs. 0 cups/day, nonlinear	0.89 (0.85, 0.93)	Ding M 2014
Caffeneited	Diabetes	11 PCs	--	--	Each 1 serving/day (1 cup)	0.91 (0.89, 0.94)	Ding M 2014
Decaffeneited	Diabetes	11 PCs	--	--	Each 1 serving/day (1 cup)	0.94 (0.91, 0.98)	Ding M 2014
Tea	CHD	7 PCs	235,368	8,328	Each 1 serving/day (1 cup)	0.90 (0.81, 0.996)	Zhang C 2015
	Diabetes	14 PCs	503,165	35,574	Each 1 serving/day (1 cup)	0.98 (0.96, 0.995)	Yang W 2014
	Stroke	8 PCs	307,968	11,329	Each 1 serving/day (1 cup)	0.94 (0.90, 0.973)	Zhang C 2015

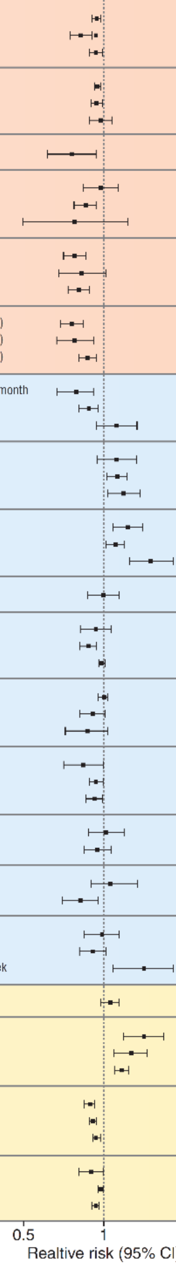
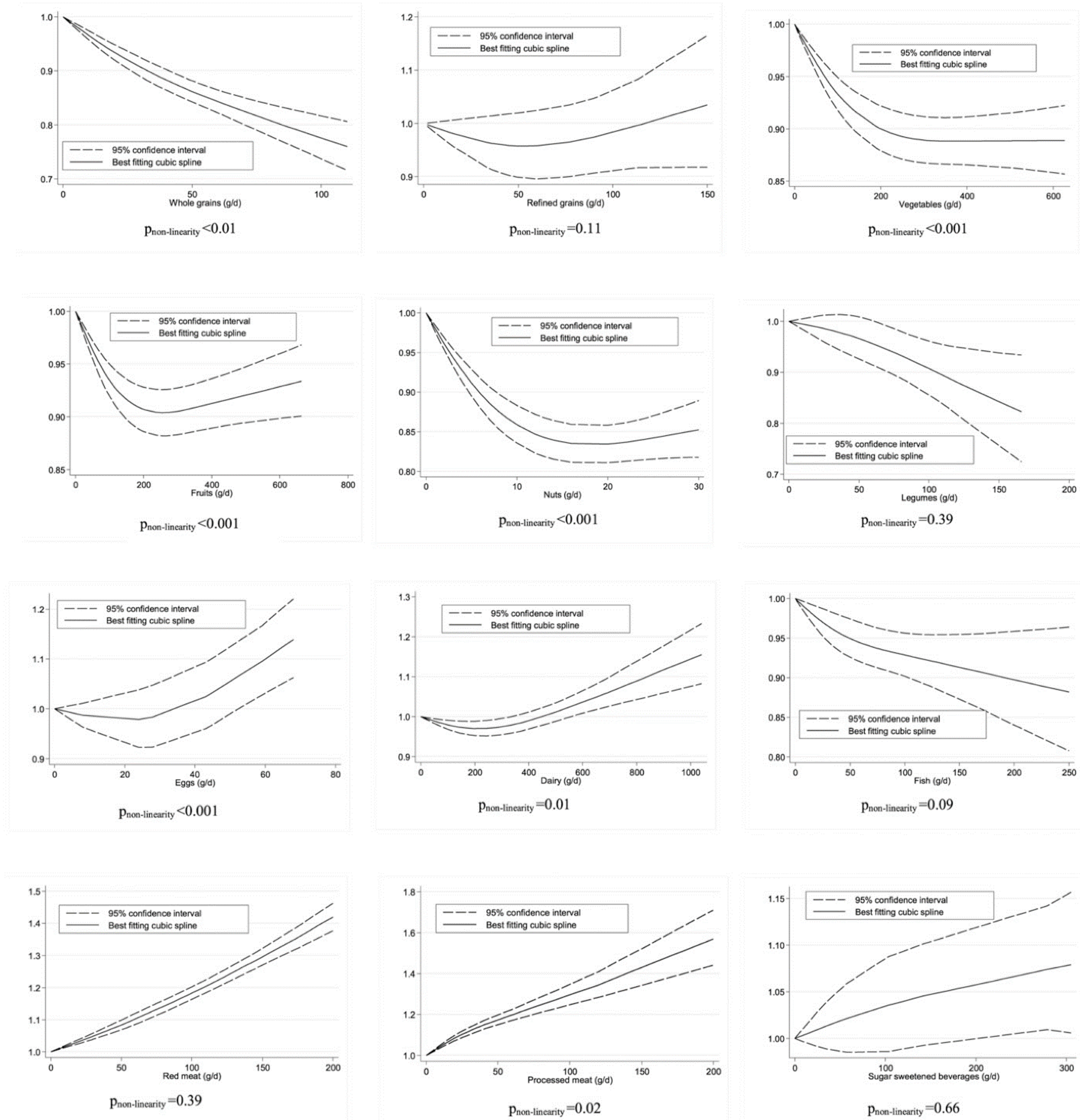


Figure 6. Meta-analyses of foods and coronary heart disease, stroke, and diabetes mellitus. BMI indicates body mass index; CHD, coronary heart disease; CI, confidence interval; CVD, cardiovascular disease; PC, prospective cohort; RCT, randomized clinical trial; and RR,

https://www.ncbi.nlm.nih.gov/pubmed/26746178

Food (group) and all-cause mortality



http://ajcn.nutrition.org/content/105/6/1462.full.pdf

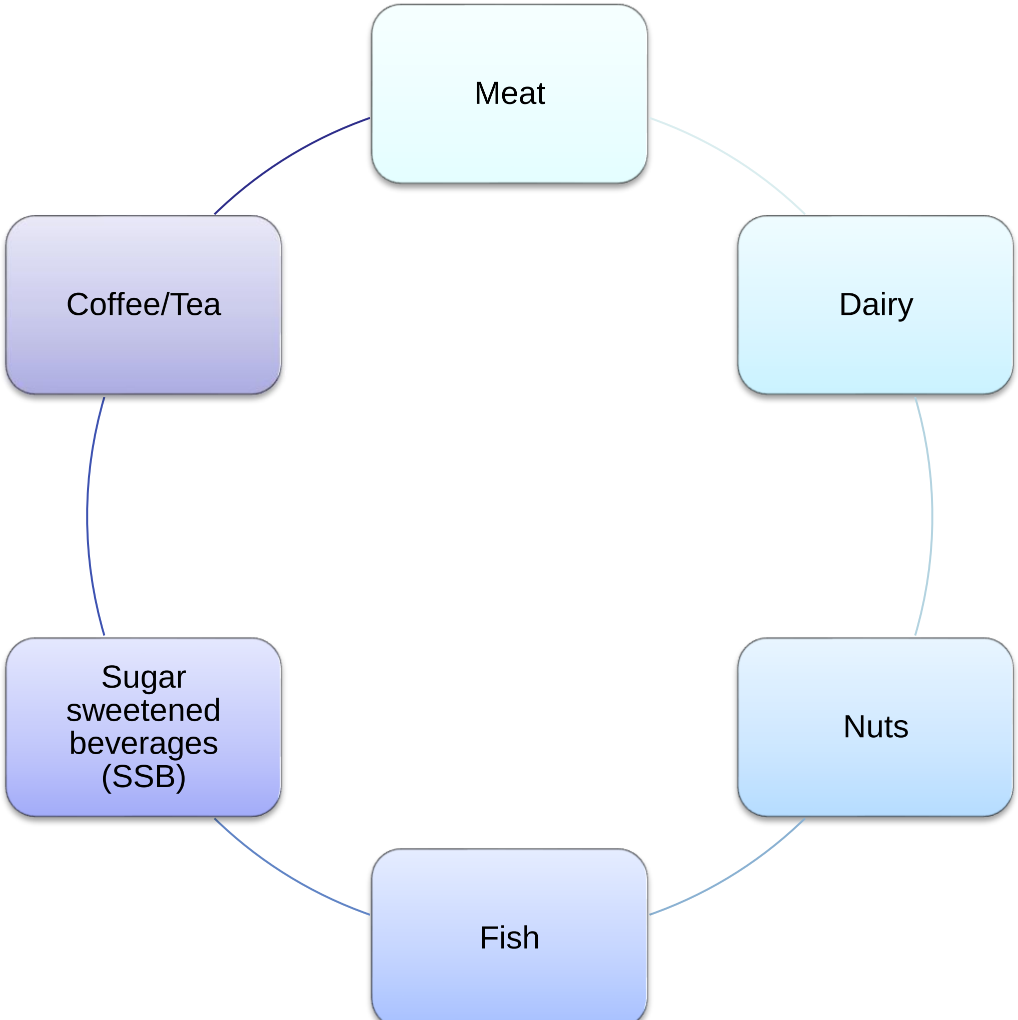
Concordance and Controversy Related to Diet and Health

	Broad Concordance and Less Controversy or Uncertainty†	General Concordance but Some Remaining Controversy and Uncertainty	Substantial Controversy and Uncertainty	Insufficient Evidence for Meaningful Conclusions
Benefits of:	Fruits, nonstarchy vegetables, nuts/seeds, legumes, yogurt Dietary fiber, potassium Moderate alcohol use Mediterranean-style or higher fat DASH-style diet patterns	Seafood, whole grains Certain vegetable oils (eg, soybean, canola, extra virgin olive) n-3 and n-6 polyunsaturated fats, plant-derived monounsaturated fats Phenolic compounds	Cheese, low-fat milk Certain vegetable oils (eg, corn, sunflower, safflower) Total or animal-derived monounsaturated fats Coffee, tea, cocoa Vitamin D, magnesium, fish oil	Whole-fat milk Starchy vegetables other than potatoes Coconut oil
Harms of:	Partially hydrogenated vegetable oils, processed meats High sodium Sugar-sweetened beverages, foods rich in refined grains, starches, added sugars Greater than moderate alcohol use	Moderate sodium White/russet potatoes High glycemic index/load	Saturated fats, dietary cholesterol Unprocessed red meats, eggs Butter	Whole-fat milk Palm oil
Little effect of:	Total fat	Total carbohydrate Isolated antioxidant vitamins, calcium	Poultry 100% fruit juice Total protein, specific amino acids Noncaloric sweeteners	Concepts of local, organic, farmed/wild, grass fed, genetic modification

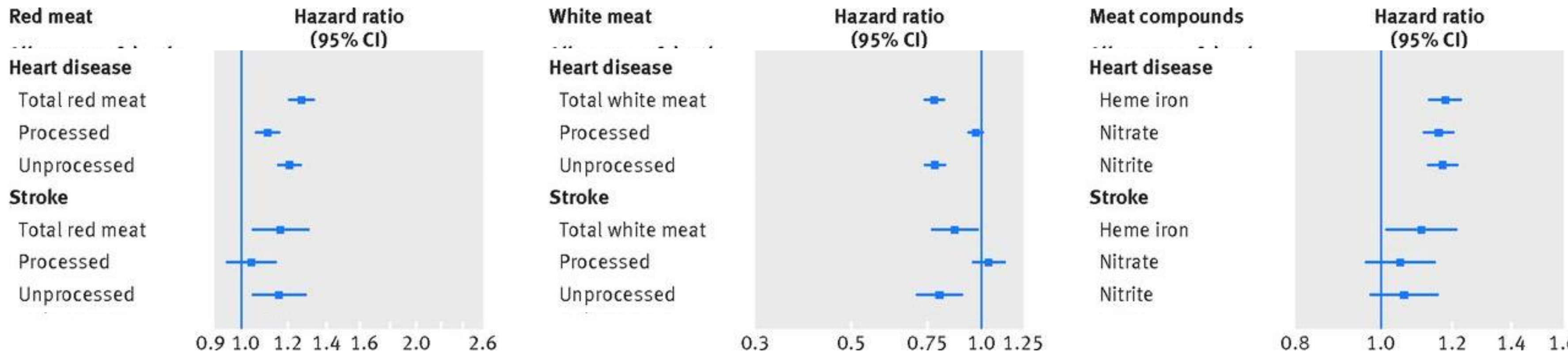
DASH indicates Dietary Approaches to Stop Hypertension.
*See article text for details on these topics and on other foods and nutrients.
†Some amount of controversy can be identified for almost any topic in science.

http://circ.ahajournals.org/content/133/2/187.long

Foods


Meat
Dairy
Nuts
Fish
Sugar sweetened beverages (SSB)
Coffee/Tea

Meat (ingrediences) and CVD mortality



BMJ 2017;357:j1957

Milk/dairy and health

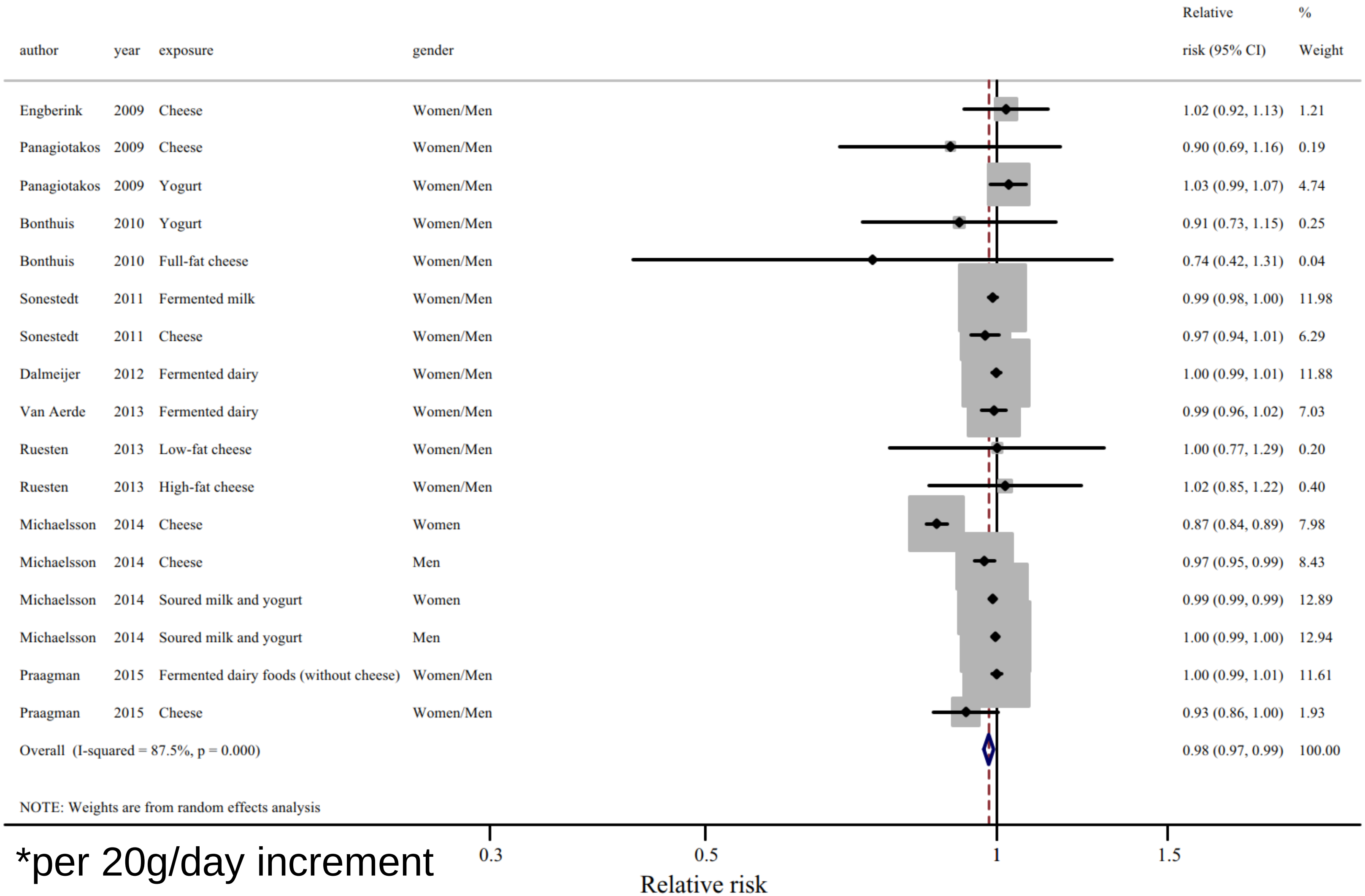
- Overall: weak scientific evidence, conflicting results

- For most NCDs: either slightly beneficial or neutral impact with the exception of prostate cancer for which there is limited evidence for an increased risk

- The evidence is strongest for a protective effect against T2DM and against colorectal cancer

Krieger, JP. (2018). Les effets sur la santé des produits laitiers et carnés: que disent les données épidémiologiques? (NOVANIMAL Working Papers No.4). Zurich: Université de Zurich. doi: 10.21256/zhaw-1404

Fermented dairy and CVD mortality*



*per 20g/day increment

Eur J Epidemiol. 2017 Apr;32(4):269-287

All-cause Mortality and protein source

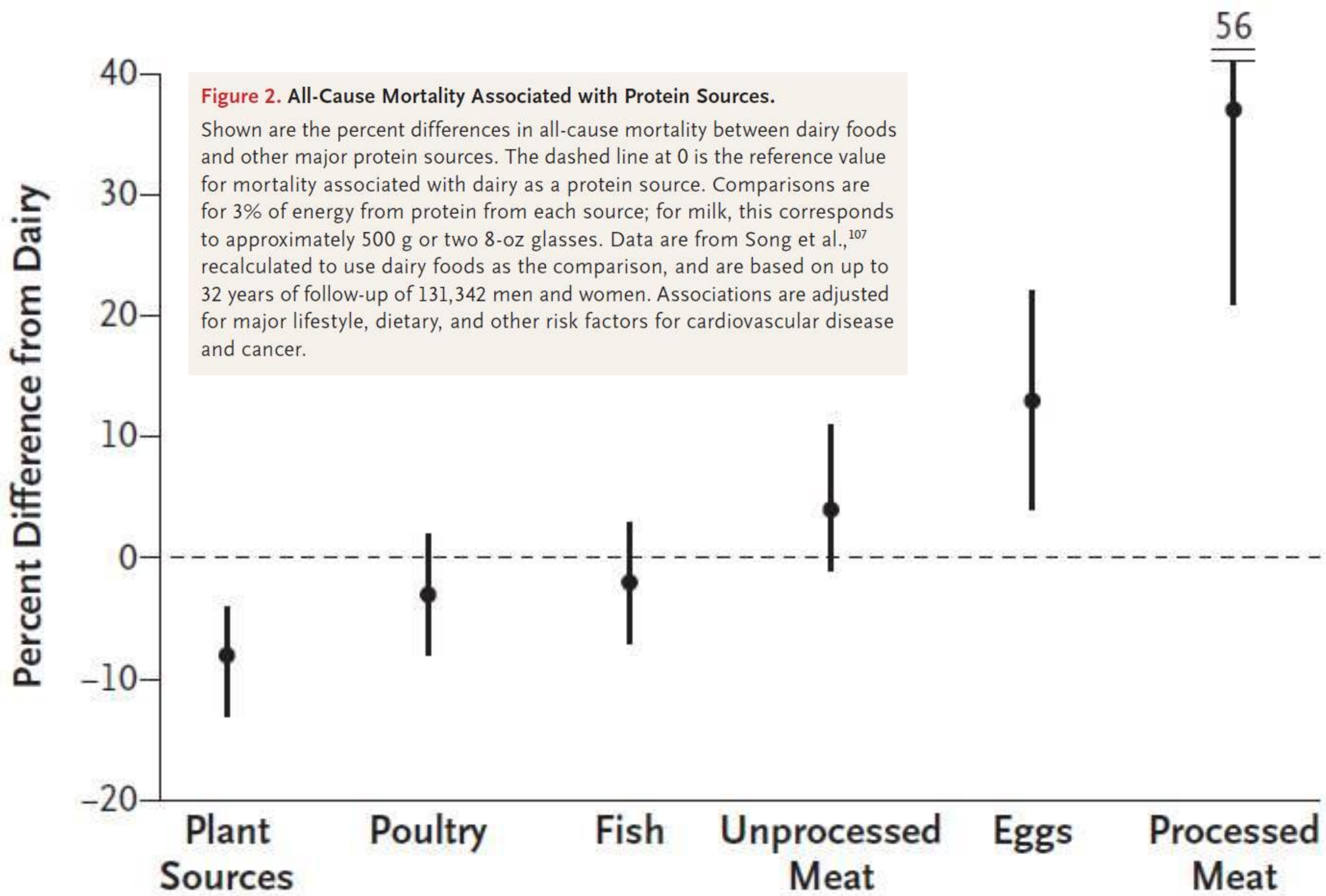
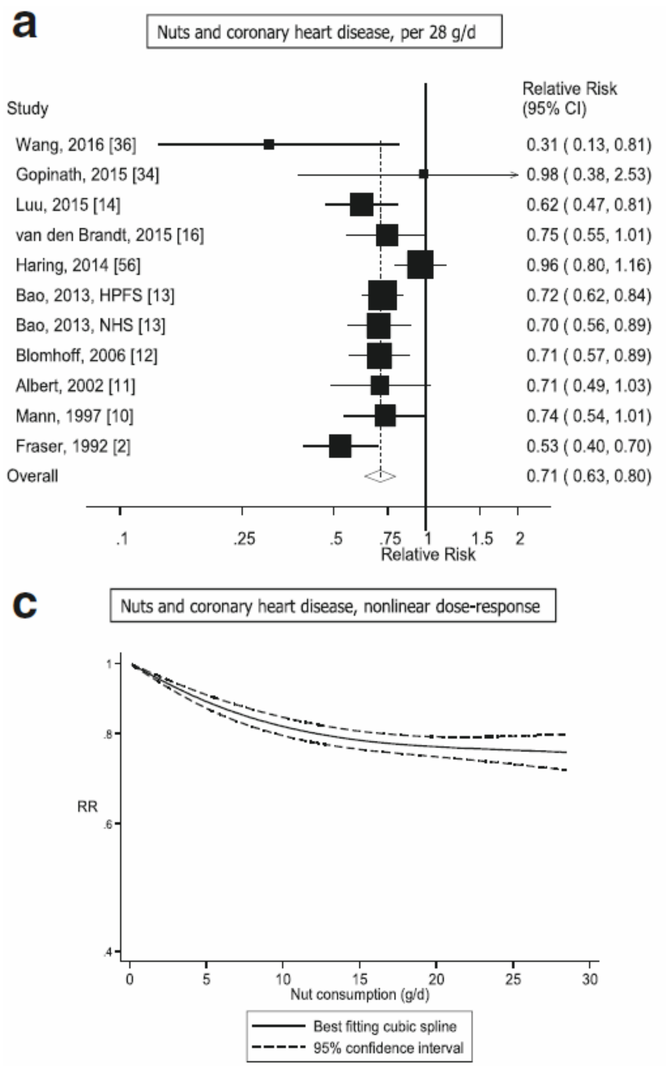


Figure 2. All-Cause Mortality Associated with Protein Sources.

Shown are the percent differences in all-cause mortality between dairy foods and other major protein sources. The dashed line at 0 is the reference value for mortality associated with dairy as a protein source. Comparisons are for 3% of energy from protein from each source; for milk, this corresponds to approximately 500 g or two 8-oz glasses. Data are from Song et al.,[107] recalculated to use dairy foods as the comparison, and are based on up to 32 years of follow-up of 131,342 men and women. Associations are adjusted for major lifestyle, dietary, and other risk factors for cardiovascular disease and cancer.

N Engl J Med 2020; 382:644-654; https://www.aerzteblatt.de/blog/109428/Wie-gesund-ist-Milch

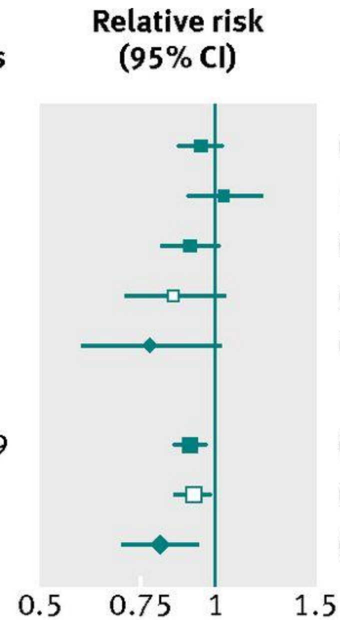
Nuts and CHD mortality



https://bmcmedicine.biomedcentral.com/articles/10.1186/s12916-016-0730-3

Fish / n-3 PUFA intake and cerebrovascular disease

	No of studies	No of participants	No of events	Relative risk (95% CI)
Long chain omega 3 fatty acids				
All cohort studies	14	305 119	5374	0.94 (0.86 to 1.02)
Circulating biomarker studies	4	4096	1177	1.04 (0.90 to 1.20)
Dietary studies	10	301 023	4197	0.90 (0.80 to 1.01)
	4	201 551	1141	0.85 (0.70 to 1.03)
	4	201 551	710	0.77 (0.59 to 1.01)
Fish consumption				
	11	366 787	11 349	0.91 (0.86 to 0.97)
	7	284 178	5002	0.93 (0.87 to 0.99)
	7	284 178	1783	0.81 (0.70 to 0.94)



■ Any cerebrovascular disease
□ Ischaemic stroke
◆ Haemorrhagic stroke

https://www.bmj.com/content/345/bmj.e6698

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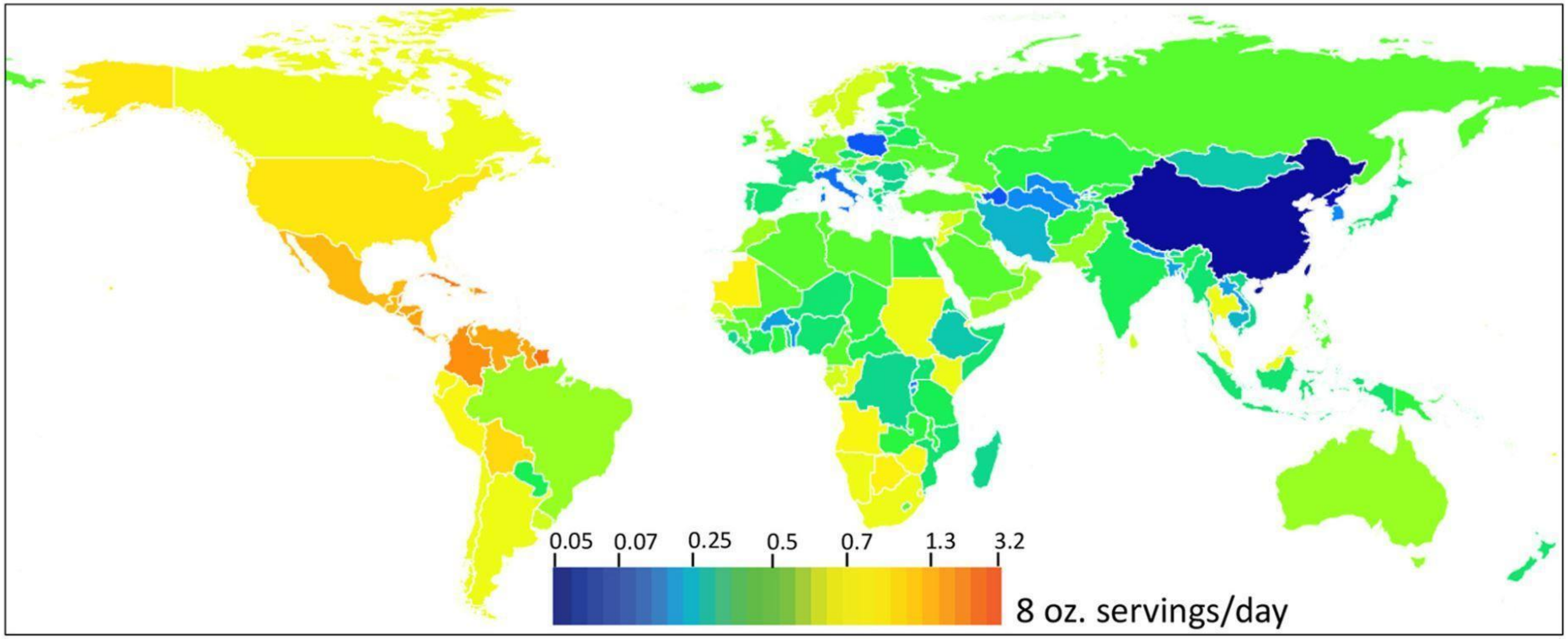
Fish and CVD risk

- Protective effect on CVD probably overestimated, partially conflicting results
- Reduced consumption of red/processed meat: real advantage of fish consumption?
- Impact depending on pre-existing CVD?
- Possibly large regional differences regarding the fish-mortality association (depending e.g. on preparation)
- Mercury, dioxins and others as potential contaminant

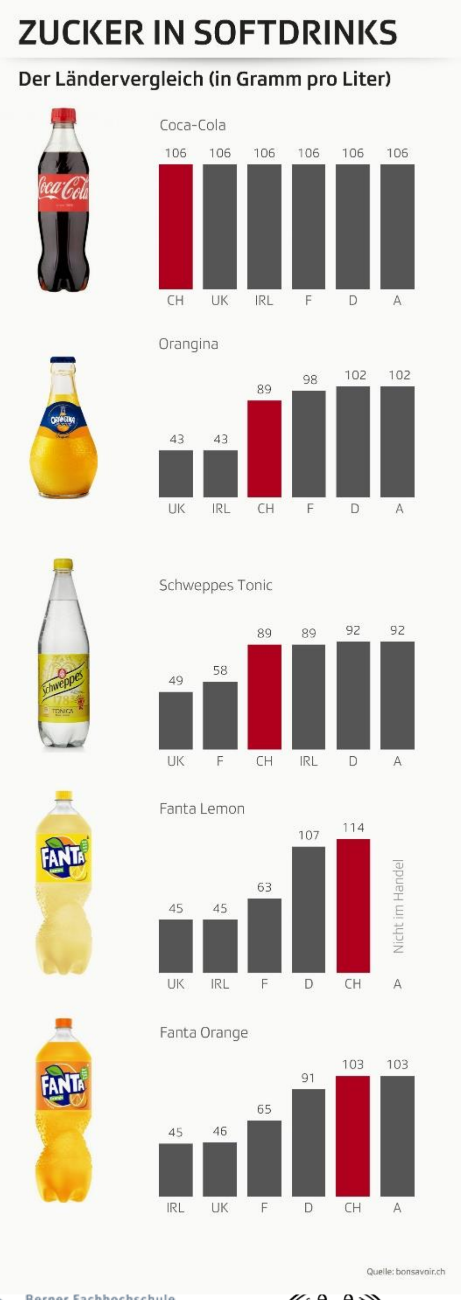
https://www.elsevier.com/about/press-releases/research-and-journals/investigators-find-something-fishy-with-the-classical-evidence-for-dietary-fish-recommendations
https://www.cambridge.org/core/journals/public-health-nutrition/article/fish-consumption-and-risk-of-allcause-and-cardiovascular-mortality-a-doseresponse-metaanalysis-of-prospective-observational-studies/210580CDB6D398F204F6C5D80282C9EE

SSB consumption globally (8oz=2.5 dl)



[Cochrane Database Syst Rev. 2019 Jun 12;6:CD012292.]

Sugar in SSB, Switzerland



https://www.srf.ch/news/schweiz/fanta-co-das-suessere-leben-in-der-schweiz

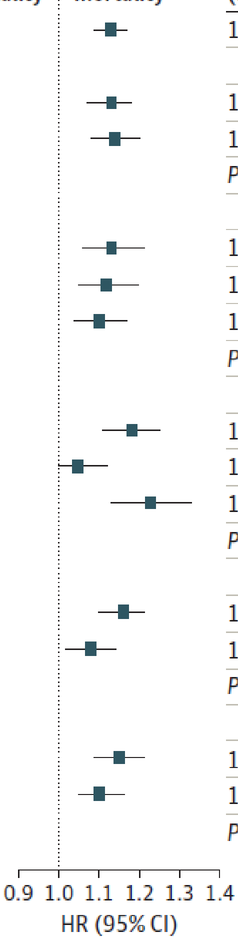
SSBs and CVD risk

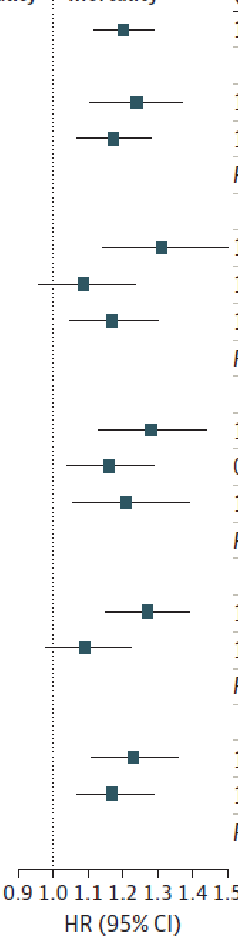
- Generally weak evidence, bias and low quality
- Moderate increase of CVD risk
- Effect mediated by blood pressure, obesity, insulin resistance, hyperuricemia?
- Substantial (residual) confounding
- Similar impact of Zero-beverages and sugar sweetened juices?

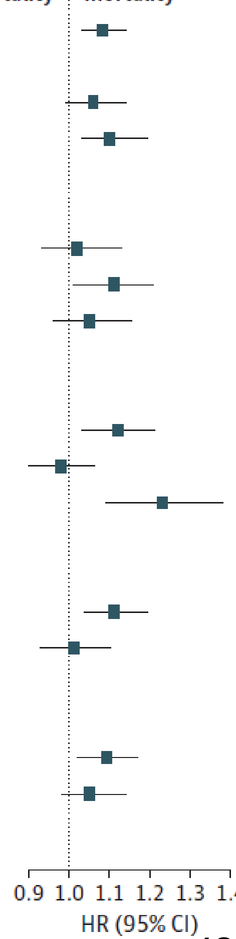
http://care.diabetesjournals.org/content/33/11/2477.short
https://www.cambridge.org/core/journals/british-journal-of-nutrition/article/sugarsweetened-beverages-and-risk-of-hypertension-and-cvd-a-doseresponse-metaanalysis/51C7F6378F3A4B04D2C23FAAFCC06D88
https://onlinelibrary.wiley.com/doi/abs/10.1111/ijcp.12841

SSB and mortality (all causes)

Variable	Total Soft Drinks HR (95% CI)	Artificially-Sweetened Soft Drinks HR (95% CI)	Sugar-Sweetened Soft Drinks HR (95% CI)
Overall	1.13 (1.09-1.17)	1.20 (1.12-1.29)	1.08 (1.03-1.14)
Sex			
Men	1.13 (1.07-1.18)	1.23 (1.11-1.37)	1.06 (0.99-1.14)
Women	1.14 (1.08-1.20)	1.17 (1.07-1.28)	1.10 (1.02-1.19)
	P for interaction = .91	*P* for interaction = .17	*P* for interaction = .73
Smoking status			
Never	1.13 (1.06-1.21)	1.31 (1.14-1.49)	1.02 (0.93-1.13)
Former	1.12 (1.05-1.20)	1.09 (0.96-1.24)	1.11 (1.01-1.21)
Current	1.10 (1.04-1.17)	1.17 (1.05-1.30)	1.05 (0.97-1.15)
	P for interaction = .05	*P* for interaction = .003	*P* for interaction = .41
BMI			
<25	1.18 (1.11-1.25)	1.27 (1.12-1.43)	1.11 (1.03-1.21)
25 to <30	1.05 (1.00-1.12)	1.16 (1.04-1.29)	0.98 (0.90-1.06)
≥30	1.23 (1.13-1.33)	1.22 (1.07-1.39)	1.23 (1.10-1.39)
	P for interaction = .003	*P* for interaction = .54	*P* for interaction = .002
Physical activity index			
Inactive	1.16 (1.10-1.21)	1.26 (1.15-1.39)	1.11 (1.04-1.19)
Active	1.08 (1.02-1.14)	1.09 (0.98-1.22)	1.01 (0.93-1.10)
	P for interaction = .42	*P* for interaction = .04	*P* for interaction = .45
Alcohol consumption			
Below median	1.15 (1.09-1.21)	1.23 (1.11-1.36)	1.09 (1.02-1.17)
Above median	1.10 (1.05-1.16)	1.17 (1.07-1.29)	1.05 (0.98-1.14)
	P for interaction = .02	*P* for interaction = .20	*P* for interaction = .67







JAMA Intern Med. 2019 Sep 3. doi: 10.1001/jamainternmed.2019.2478

Coffee, tea and CVD risk

- Coffee consumption associated with lower CVD risk with maximum effect found with 4 cups/day
- Probably no increased risk with heavy coffee consumption (>10 cups/day)
- Tea: similar impact compared to Coffee
- Tea and coffee: also associated with lower type 2 diabetes risk
- Type of coffee (filter, espresso, instant) not so important

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3945962/pdf/nihms-543017.pdf

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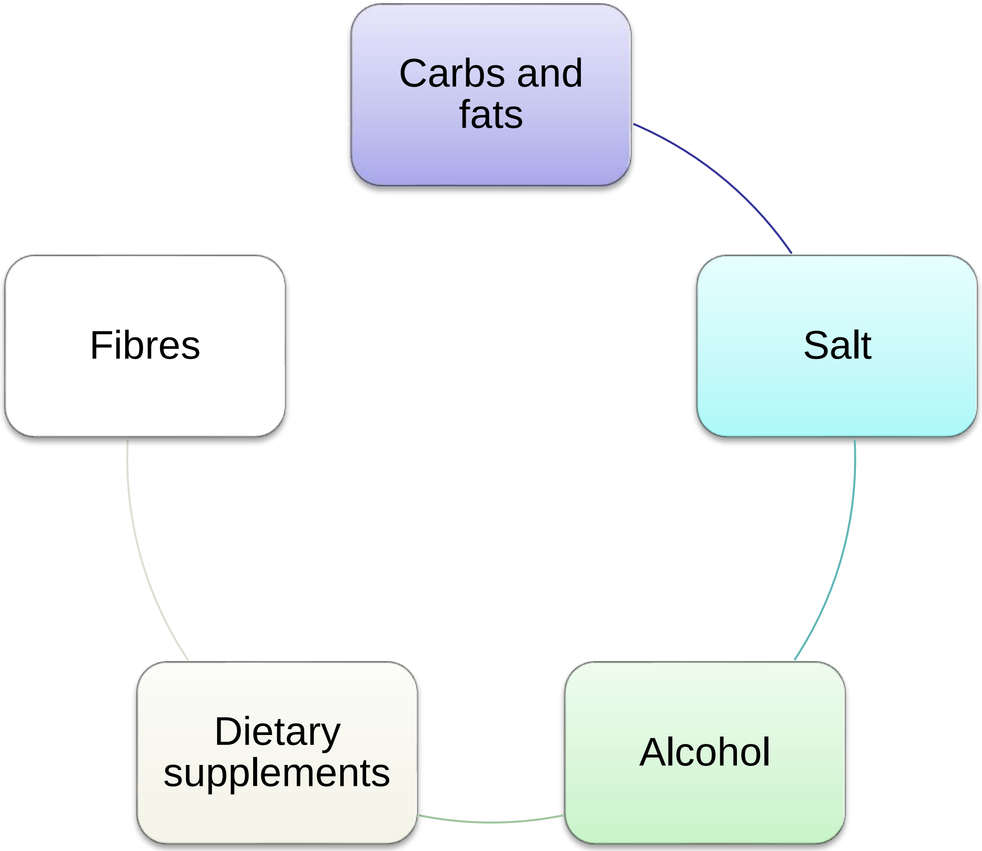
Content

- CVD epidemiology
- Diet and CVD: scientific evidence
- Foods and CVD
- Nutrients and CVD
- Mediterranean diet
- Personalized nutrition

Nutrients



Trans-fats

Outcome	No of studies /comparisons	No of events /participants	Risk ratio (95% CI)	Relative risk (95% CI)	P	P_{het}	I^2 (%)
Total trans fats							
All cause mortality	2/2	2141/20 346		1.34 (1.16 to 1.56)	<0.001	0.07	70
CHD mortality	5/6	1234/70 864		1.28 (1.09 to 1.50)	0.003	0.66	0
CHD total	6/7	4579/145 922		1.21 (1.10 to 1.33)	<0.001	0.43	0
Ischemic stroke	3/4	1905/190 284		1.07 (0.88 to 1.28)	0.50	0.03	67
Type 2 diabetes	6/6	8690/230 135		1.10 (0.95 to 1.27)	0.21	0.01	66
Industrial trans fats							
All cause mortality	1/2	11 890/71 464		0.98 (0.92 to 1.04)	0.52	0.52	0
CHD mortality	2/2	3018/93 394		1.18 (1.04 to 1.33)	0.009	0.68	0
CHD total	2/2	454/69 848		1.42 (1.05 to 1.92)	0.02	0.22	34
Ischemic stroke	0	0/0		-	-	-	-
Type 2 diabetes	0	0/0		-	-	-	-
Ruminant trans fats							
All cause mortality	1/2	11 890/71 464		1.04 (0.92 to 1.18)	0.51	0.31	4
CHD mortality	2/3	3018/93 394		1.01 (0.71 to 1.43)	0.95	0.01	79
CHD total	3/4	828/73 546		0.93 (0.73 to 1.18)	0.55	0.13	46
Ischemic stroke	0	0/0		-	-	-	-
Type 2 diabetes	5/5	1153/12 942		0.58 (0.46 to 0.74)	<0.001	0.22	30

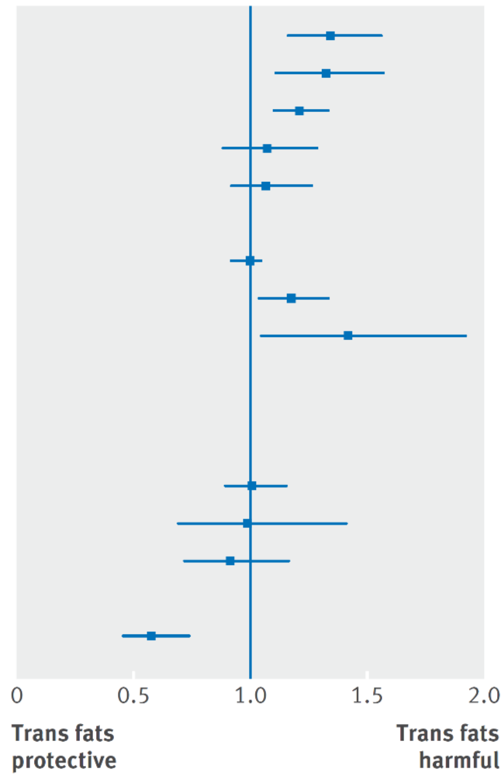


Fig 4 | Summary most adjusted relative risks of total trans fat, industrial trans fat, and ruminant trans fat and all cause mortality, CHD mortality, total CHD, ischemic stroke, and type 2 diabetes. For total trans fats effect estimate for is fixed effect analysis; all others random effects analyses. P value is for Z test of no overall association between exposure and outcome; P_{het} is for test of no differences in association measure among studies; I^2 is proportion of total variation in study estimates from heterogeneity rather than sampling error

Saturated fats

Outcome	No of studies /comparisons	No of events /participants	Relative risk (95% CI)	P	P_{het}	I^2 (%)
All cause mortality	5/7	14 090/99 906	0.99 (0.91 to 1.09)	0.91	0.17	33
CHD mortality	11/15	2970/101 712	1.15 (0.97 to 1.36)	0.10	<0.001	70
CVD mortality	3/5	3792/90 501	0.97 (0.84 to 1.12)	0.69	0.29	19
CHD total	12/17	6383/267 416	1.06 (0.95 to 1.17)	0.29	0.02	47
Ischemic stroke	12/15	6226/339 090	1.02 (0.90 to 1.15)	0.79	0.002	59
Type 2 diabetes	8/8	8739/237 454	0.95 (0.88 to 1.03)	0.20	0.61	0

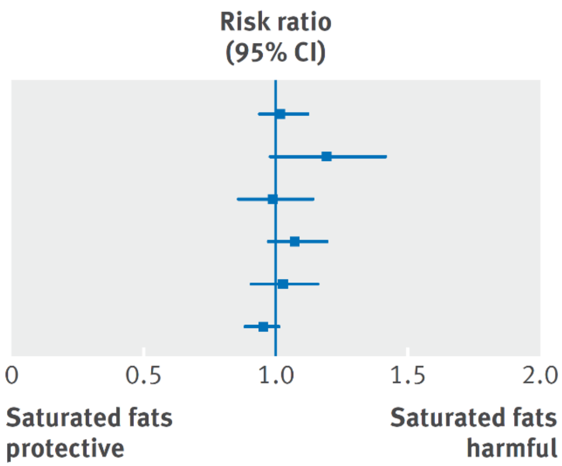


Fig 2 | Summary most adjusted relative risks for saturated fat intake and all cause mortality, CHD mortality, CVD mortality, total CHD, ischemic stroke, and type 2 diabetes. All effect estimates are from random effects analyses. P value is for Z test of no overall association between exposure and outcome; P_{het} is for test of no differences in association measure among studies; I^2 is proportion of total variation in study estimates from heterogeneity rather than sampling error

http://www.bmj.com/content/351/bmj.h3978

Saturated fat, replaced by…

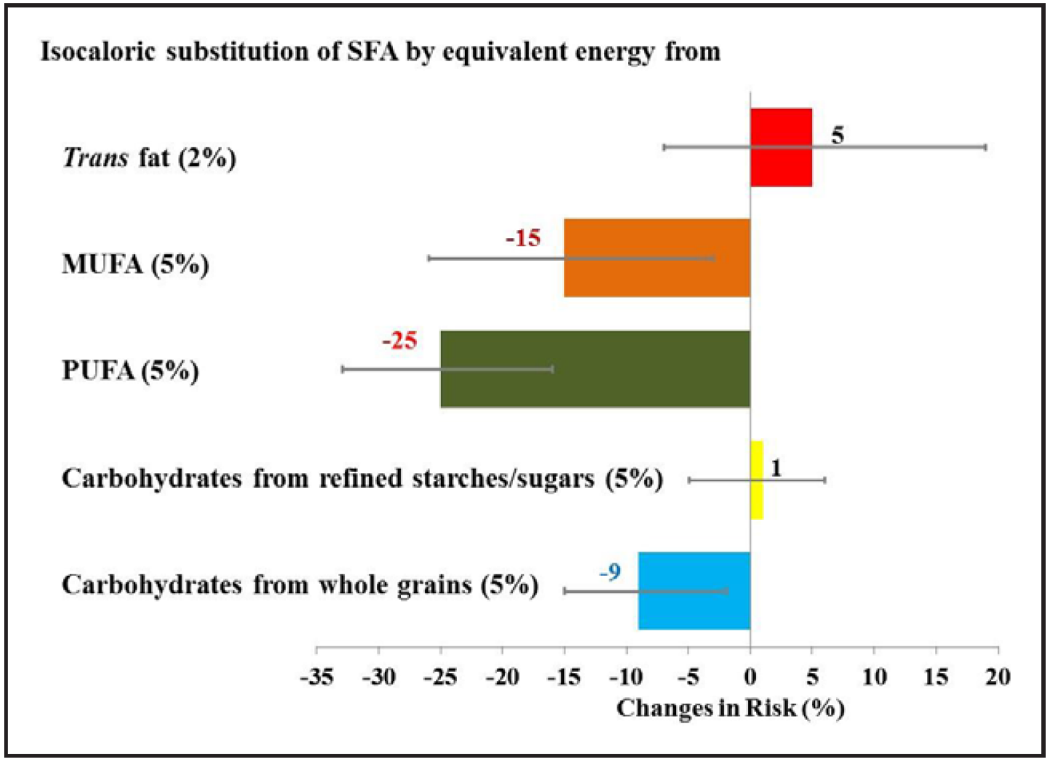


Figure 3. Replacement of saturated fat with other types of fat or carbohydrates.
Association with risk of cardiovascular disease in the Nurses' Health Study and Health Professionals Follow-Up Study. Multivariable adjustment. MUFA indicates monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; and SFA, saturated

http://circ.ahajournals.org/content/early/2017/06/15/CIR.0000000000000510

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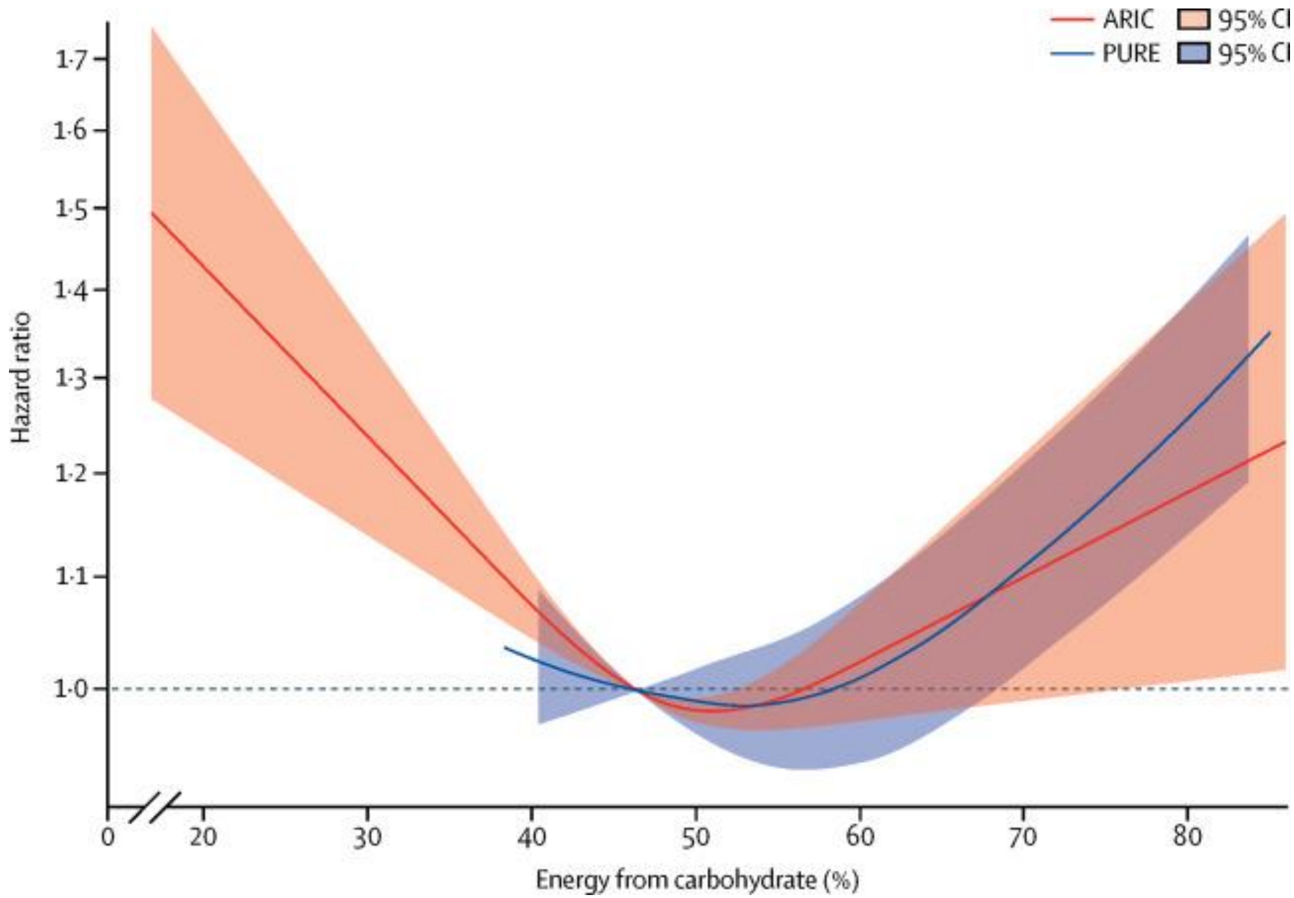
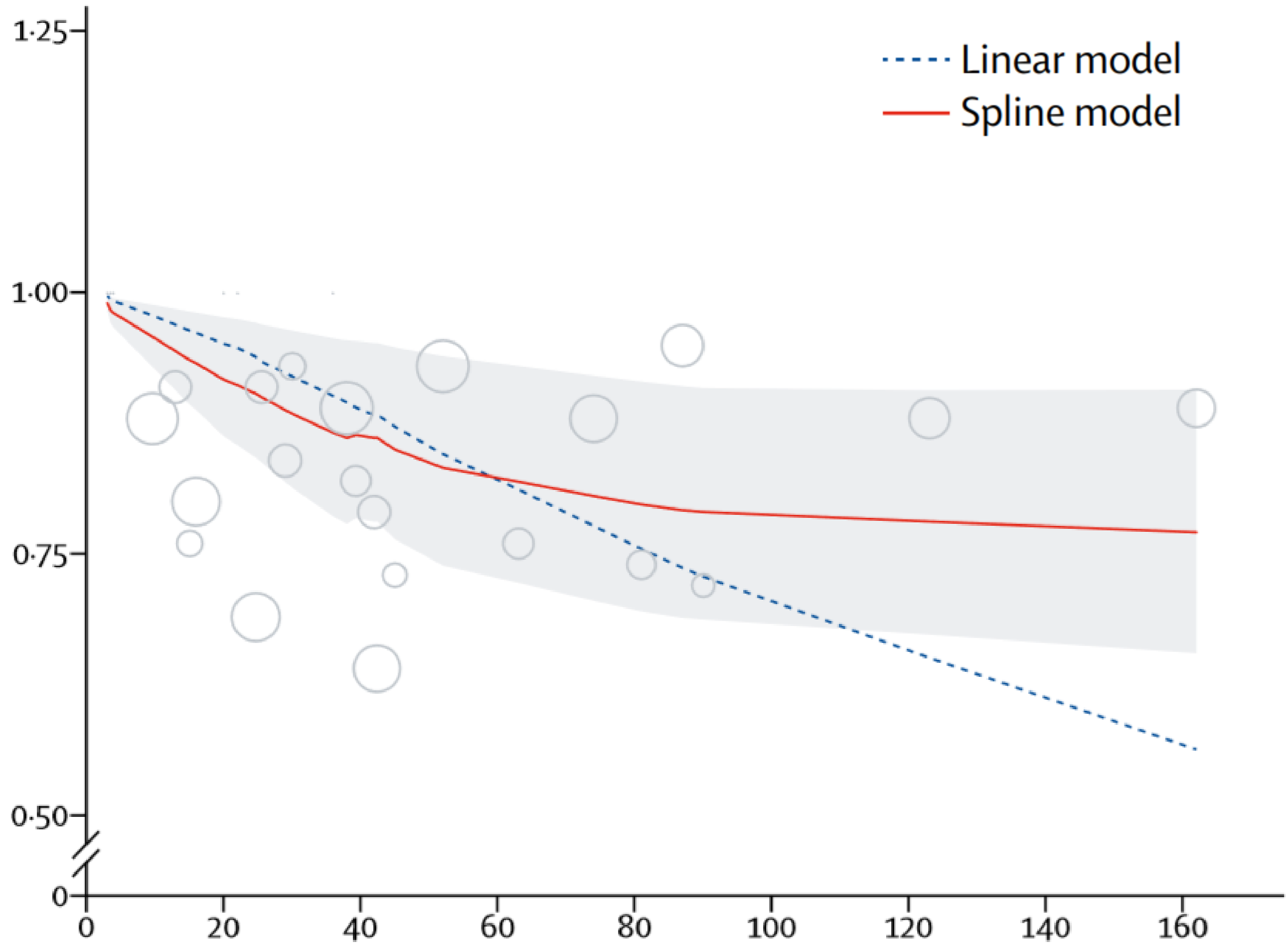
Energy% Carbohydrate intake and all-cause mortality



Lancet Public Health 2018; 3: e419–28

Whole Grain Intake (g per day) and Coronary Heart Disease risk



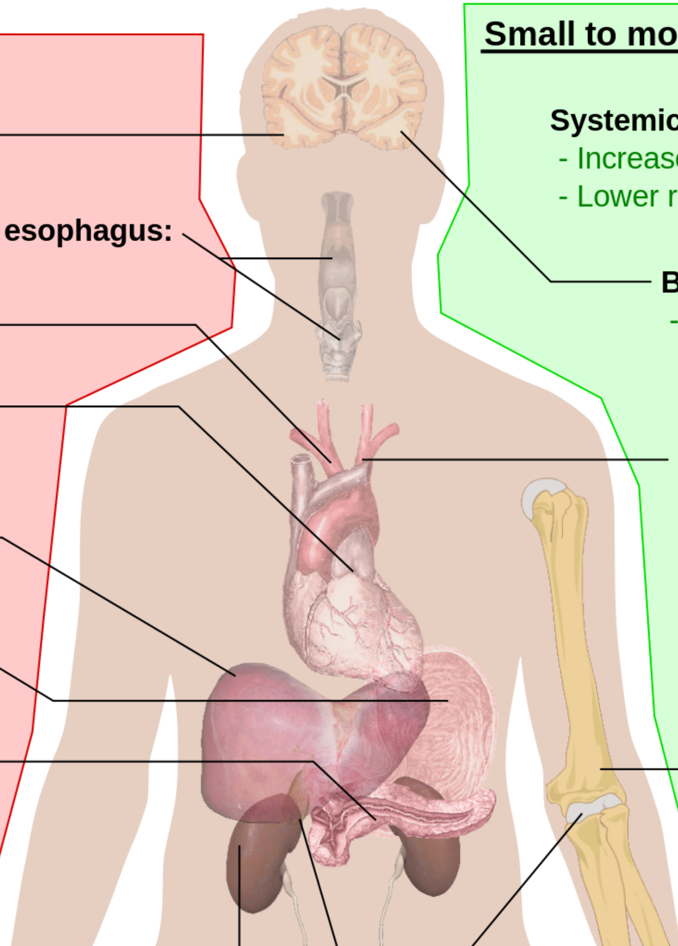
Lancet 2019; 393: 434–45

Alcohol

Potential long-term effects of

Ethanol

Red - generally "bad"
Green - generally "good"



Large consumption

Brain:
- Impaired development
- Wernicke-Korsakoff syndrome
  * Vision changes
  * Ataxia
  * Impaired memory
- Psychological
  * Cravings
  * Irritability
  * Antisociality
  * Depression
  * Anxiety
  * Panic
  * Psychosis
  * Hallucinations
  * Delusions
  * Sleep disorders

Mouth, trachea and esophagus:
- Cancer

Blood:
- Anemia

Heart:
- Alcoholic cardio-myopathy

Liver:
- Cirrhosis
- Hepatitis

Stomach:
- Chronic gastritis

Pancreas:
- Pancreatitis

Peripheral tissues:
- Increased risk of diabetes type 2

Small to moderate consumption

Systemic:
- Increases insulin sensitivity
- Lower risk of diabetes

Brain:
- Reduce the number of silent infarcts

Blood:
- Increases HDL
- Decreases thrombosis
- Reduces fibrinogen
- Increases fibrinolysis
- Reduces artery spasm from stress
- Increases coronary blood flow

Skeletal:
- Higher bone mineral density

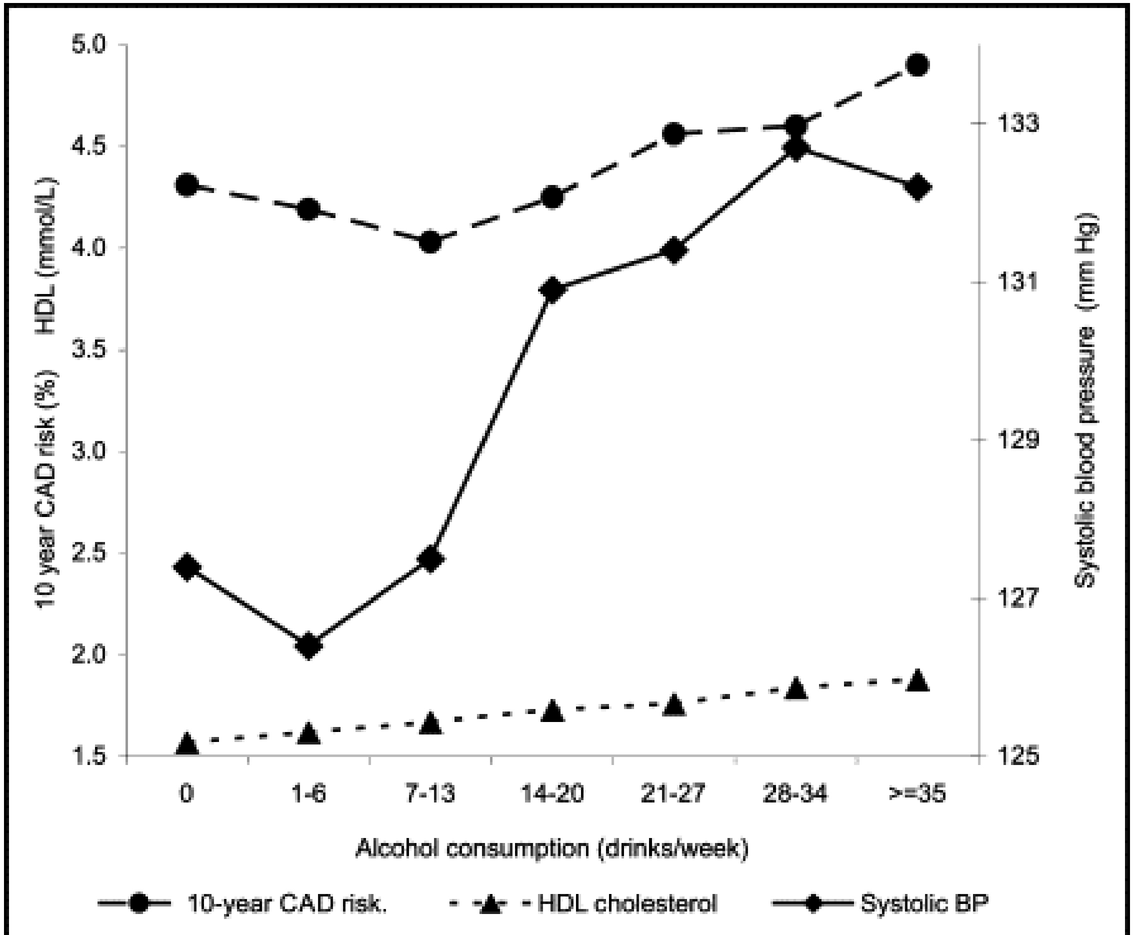
Effects linked with both small and large consumption

Joints:
- Reduced risk of rheumatoid arthritis

Gallbladder:
- Reduced the risk of developing gallstones

Kidney:
- Reduced risk of developing kidney stones

Association between alcohol consumption and cardiovascular risk, Lausanne



HDL cholesterol, systolic blood pressure (BP), and 10-year CAD risk according to last week alcohol consumption.

Am J Cardiol. 2009 Feb 1;103(3):361-8

Alcohol and risk / protecive factors

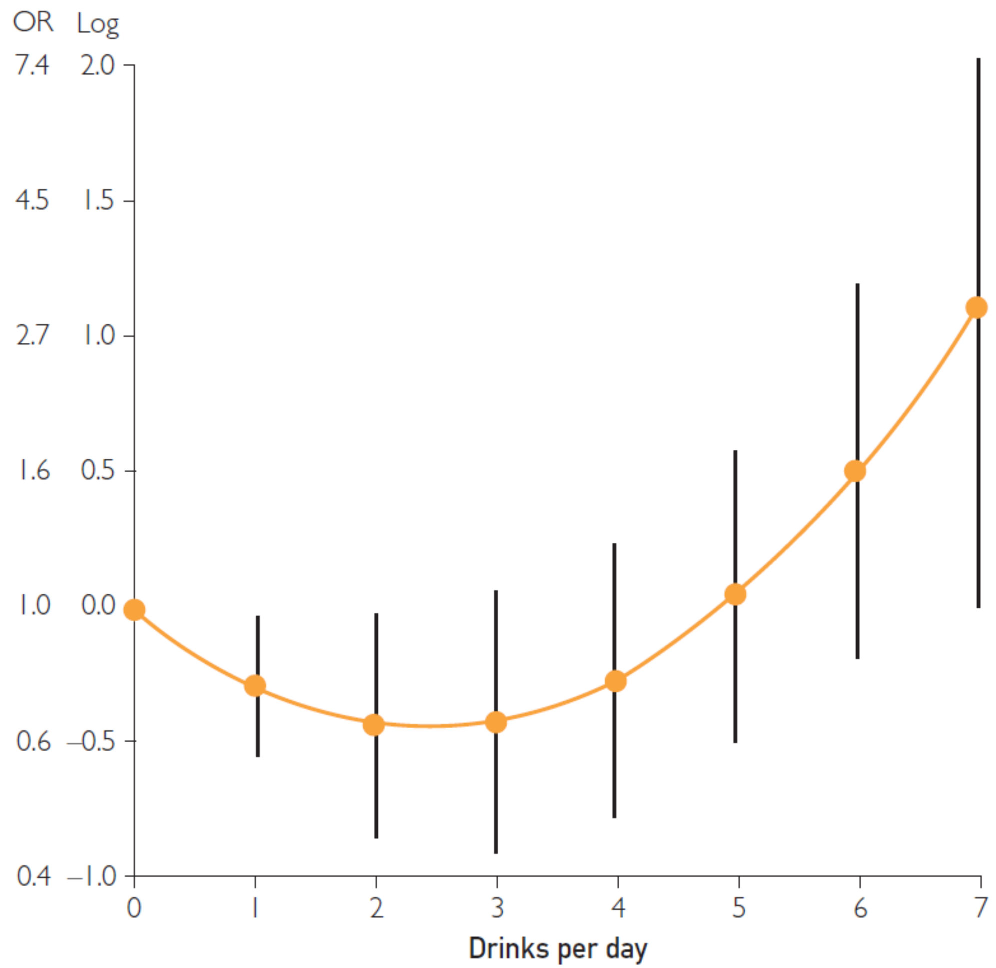
Table 2 | Summary of pooled mean difference in biomarker level after alcohol use

Biomarker	No of pooled studies	No of pooled participants	Type of model	Pooled mean difference in biomarker level (95% CI)
High density lipoprotein cholesterol (mmol/L)	33	796	Random	0.094 (0.064 to 0.123)*†
Low density lipoprotein cholesterol (mmol/L)	24	513	Random	−0.11 (−0.22 to 0.006)†
Total cholesterol (mmol/L)	26	596	Fixed	0.00 (−0.066 to 0.067)
Triglycerides (mmol/L)	31	752	Fixed	0.016 (−0.018 to 0.051)
Apolipoprotein A1 (g/L)	16	374	Random	0.101 (0.073 to 0.129)*†
Lp(a) lipoprotein (mg/dL)	3	114	Fixed	0.80 (−4.17 to 5.76)
C reactive protein (mg/L)	5	186	Fixed	−0.11 (−0.31 to 0.10)
Interleukin 6 (pg/mL)	2	144	Fixed	0.502 (−3.482 to 4.486)
Tumour necrosis factor α (pg/mL)	3	121	Fixed	−0.469 (−32.02 to 31.08)
Plasminogen activator inhibitor 1 (ng/mL)	3	67	Fixed	3.285 (−0.898 to 7.469)
Tissue plasminogen activator (ng/mL)	3	67	Fixed	0.754 (−0.132 to 1.641)
Fibrinogen (g/L)	7	387	Fixed	−0.20 (−0.29 to −0.11)*
Adiponectin (mg/L)	4	108	Fixed	0.56 (0.39 to 0.72)*

*Indicates significant ($P<0.01$) change in biomarker level after alcohol use compared with a period of no alcohol use.
†Heterogeneity detected across pooled studies, where Q statistic $P<0.10$.

http://www.bmj.com/content/342/bmj.d636.long

Alcohol consumption and ischemic stroke risk



Mayo Clin Proc. 2014 Mar;89(3):382-93

Alcohol/mortality: J-shape of association

- Protective effect (mainly CVD): a consequence of poor study design rather than «real» effect of alcohol?
- Misclassification of individuals
- Change of (and reason for) alcohol consumption status not considered
- Relative risks (compared with abstainers) may be misleading in the sense that they overestimate the benefit of moderate alcohol consumption

http://onlinelibrary.wiley.com/doi/10.1111/add.13451/epdf

Alcohol & mortality: data from 600’000 individuals

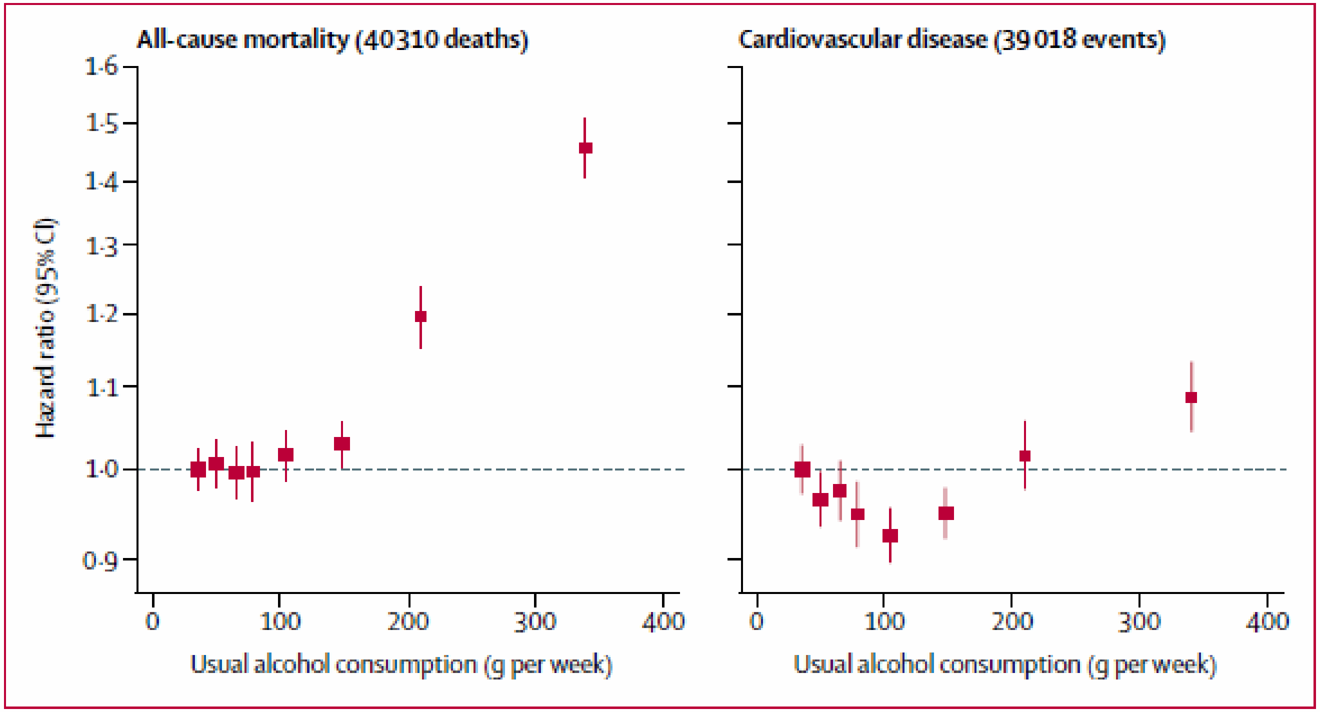


Figure 1: Associations of usual alcohol consumption with all-cause mortality and the aggregate of cardiovascular disease in current drinkers
Cardiovascular disease was defined as an aggregate of myocardial infarction, coronary heart disease, and stroke. Hazard ratios are adjusted for age, smoking, and history of diabetes, and stratified by sex and EPIC centre. The reference category is the lowest baseline alcohol consumption category (between 0 and 25 g/week). HRs are plotted against the mean usual alcohol consumption in each category. Sizes of the boxes are proportional to the inverse of the variance of the log-transformed hazard ratios. Vertical lines represent 95% CIs.

http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(18)30134-X.pdf

Alcohol & CVD mortality (n=600’000)

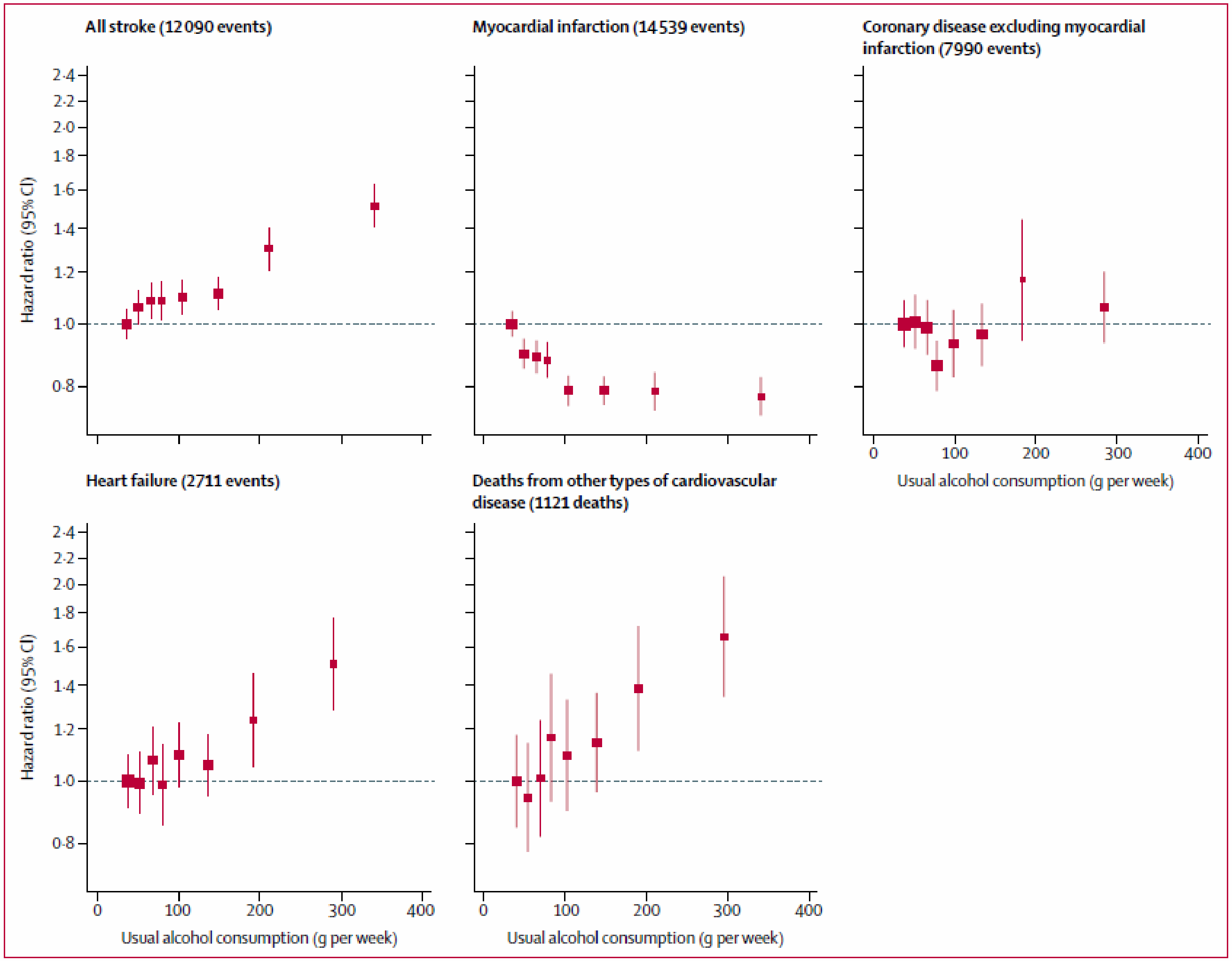


Figure 2: **Associations of usual alcohol consumption with cardiovascular subtypes in alcohol drinkers**
Hazard ratios are adjusted for age, smoking, and history of diabetes, and stratified by sex and EPIC centre. The reference category is the lowest baseline alcohol consumption category (between 0 and 25g/week). Hazard ratios are plotted against the mean usual alcohol consumption in each category. Studies with fewer than five events of any outcome were excluded from the analysis of that outcome. Sizes of the boxes are proportional to the inverse of the variance of the log-transformed hazard ratios. Vertical lines represent 95% CIs. Deaths from other cardiovascular disease include the following outcomes: cardiac dysrhythmia, hypertensive disease, sudden death, and aortic aneurysm.

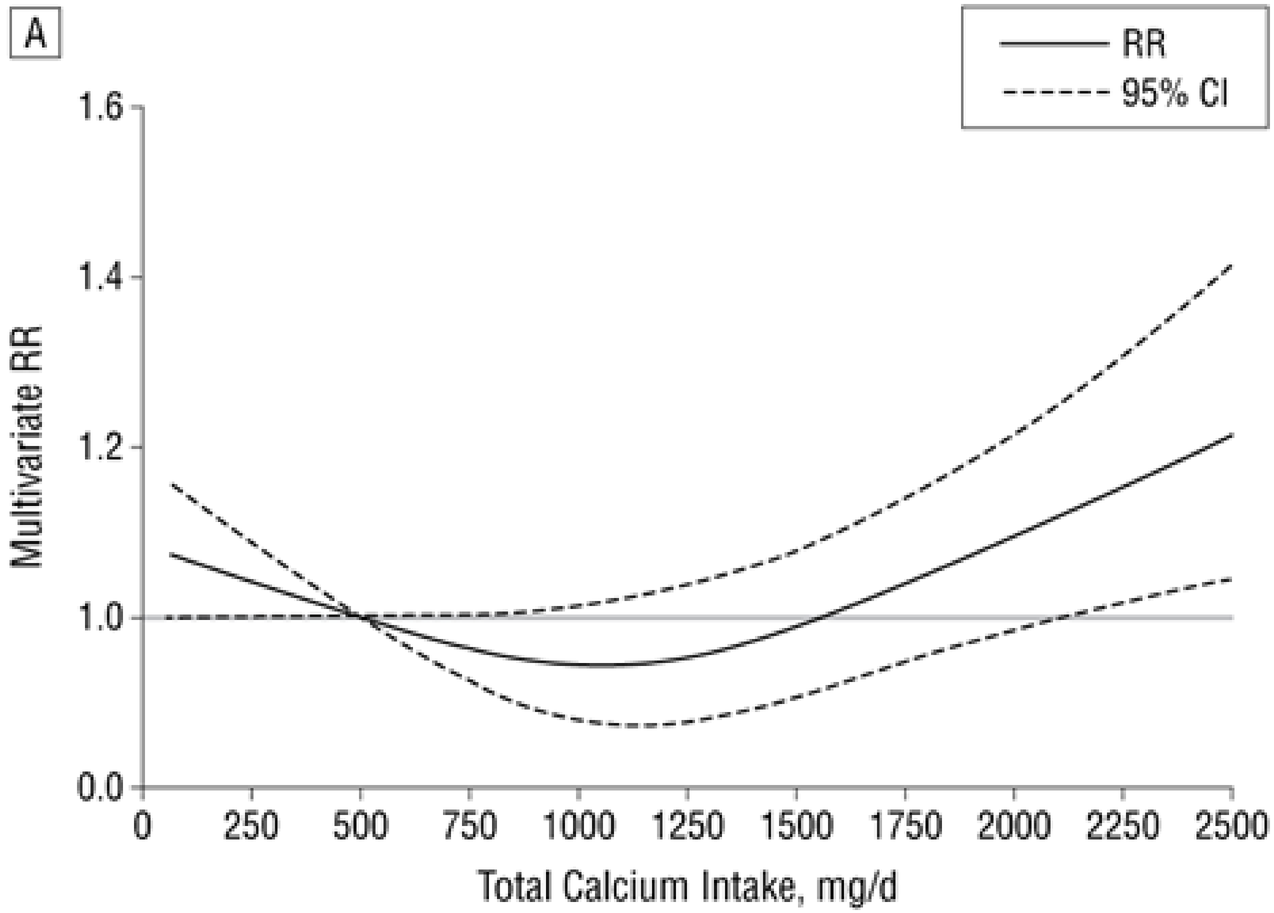
http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(18)30134-X.pdf

Meta analysis: Supplements and mortality in general populations

- "We found **no evidence** to support antioxidant supplements for primary or secondary prevention. Beta-carotene and vitamin E seem to **increase mortality**, and so may higher doses of vitamin A. Antioxidant supplements need to be considered as medicinal products and should undergo sufficient evaluation before marketing."

http://www.cochrane.org/CD007176/LIVER_antioxidant-supplements-for-prevention-of-mortality-in-healthy-participants-and-patients-with-various-diseases

Total calcium intake and CVD mortality



http://archinte.jamanetwork.com/article.aspx?articleid=1568523

Dietary supplements and CVD risk

- No evidence to support antioxidant supplements for primary or secondary prevention. Beta-carotene and vitamin E seem to increase mortality, and so may higher doses of vitamin A
- Among patients with diabetic nephropathy, high doses of B vitamins compared with placebo resulted in a greater decrease in GFR and an increase in vascular events
- Vitamin D3 could decrease mortality in elderly people living independently or in institutional care but could also be harmful in higher doses

http://www.ncbi.nlm.nih.gov/pubmed/22419320; http://www.ncbi.nlm.nih.gov/pubmed/20424250
http://www.ncbi.nlm.nih.gov/pubmed/24414552; http://www.bmj.com/content/355/bmj.i6201
http://jamanetwork.com/journals/jamainternalmedicine/article-abstract/2478897

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Supplements and Cardiovascular Health

β-Carotene	Some cohort studies have linked low serum levels or low dietary intake of β-carotene with higher CVD risk. Trials of β-carotene supplements document no benefit in the general population and increased risk of lung cancer in patients who were at high risk of lung cancer.
Calcium	Meta-analysis of trials suggests that calcium supplementation could increase the risk of myocardial infarction. No evidence for cardiometabolic benefits.
Vitamin D	Evidence from observational studies indicates that low serum vitamin D levels, which are largely determined by sun exposure, are associated with higher risk of CVD. Trials of vitamin D supplementation have not shown reductions in risk of CVD. Additional trials using higher doses of vitamin D supplementation are ongoing.
Vitamin E	Several prospective cohort studies have linked vitamin E consumption or supplementation with lower risk of CHD. Trials have failed to show reductions in CVD events with supplemental vitamin E, and 2 meta-analyses suggest that high-dose vitamin E supplements may increase total mortality.
Folic acid, Vitamins B_6, B_{12}	Observational studies have associated low folate intake, low serum folate levels, and high homocysteine levels with higher risk of CVD outcomes. Trials have confirmed that folic acid supplementation lowers blood homocysteine levels. Long-term trials have not documented benefits of folic acid with or without vitamin B_6 and vitamin B_{12} on CVD outcomes. In some trials, supplemental folic acid was associated with increased risk of CVD.
Fish oil	Multiple cohort studies have documented an inverse relationship between fish intake and subsequent CHD, in particular, CHD death. A meta-analysis of trials, largely in higher-risk populations, demonstrated a reduction in cardiac death with fish oil supplementation, largely because of the benefits in patients with prevalent CHD.
Multivitamins	Although some cohort studies have seen lower CVD risk with multivitamin supplements, several trials, rated to be of fair to poor quality, have not documented any clear CVD benefit of multivitamin use in mixed populations.

CHD indicates coronary heart disease; and CVD, cardiovascular disease.
Table updated from Mozaffarian et al.[19]

http://circ.ahajournals.org/content/133/2/187.long

Supplements and Cardiovascular Health

β-Carotene	Some cohort studies have linked low serum levels or low dietary intake of β-carotene with higher CVD risk. Trials of β-carotene supplements document no benefit in the general population and increased risk of lung cancer in patients who were at high risk of lung cancer.
Calcium	Meta-analysis of trials suggests that calcium supplementation could increase the risk of myocardial infarction. No evidence for cardiometabolic benefits.
Vitamin D	Evidence from observational studies indicates that low serum vitamin D levels, which are largely determined by sun exposure, are associated with higher risk of CVD. Trials of vitamin D supplementation have not shown reductions in risk of CVD. Additional trials using higher doses of vitamin D supplementation are ongoing.
Vitamin E	Several prospective cohort studies have linked vitamin E consumption or supplementation with lower risk of CHD. Trials have failed to show reductions in CVD events with supplemental vitamin E, and some meta-analyses suggest that high-dose vitamin E supplements may increase total mortality.
Folic acid, Vitamins B_6, B_{12}	Observational studies have associated low folate intake, low serum folate levels, and high homocysteine levels with higher risk of CVD outcomes. Trials have confirmed that folic acid supplementation lowers blood homocysteine levels. Long-term trials have not documented benefits of folic acid with or without vitamin B_6 and vitamin B_{12} on CVD outcomes. In some trials, supplemental folic acid was associated with increased risk of CVD.
Fish oil	Multiple cohort studies have documented an inverse relationship between fish intake and subsequent CHD, in particular, CHD death. A meta-analysis of trials, largely in higher-risk populations, demonstrated a reduction in cardiac death with fish oil supplementation, largely because of the benefits in patients with prevalent CHD.
Multivitamins	Although some cohort studies have seen lower CVD risk with multivitamin supplements, several trials, rated to be of fair to poor quality, have not documented any clear CVD benefit of multivitamin use in mixed populations.

Forget dietary supplements

CHD indicates coronary heart disease; and CVD, cardiovascular disease.
Table updated from Mozaffarian et al.[19]

http://circ.ahajournals.org/content/133/2/187.long

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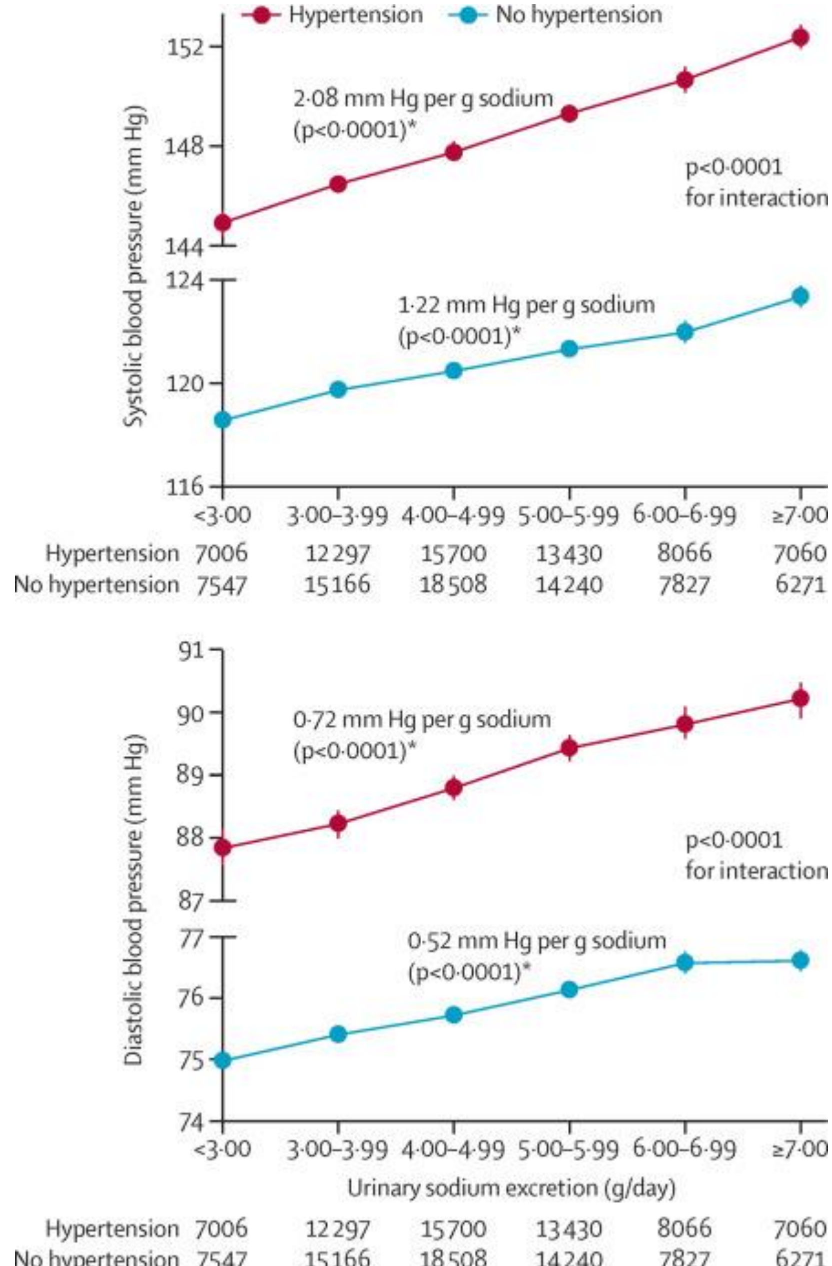
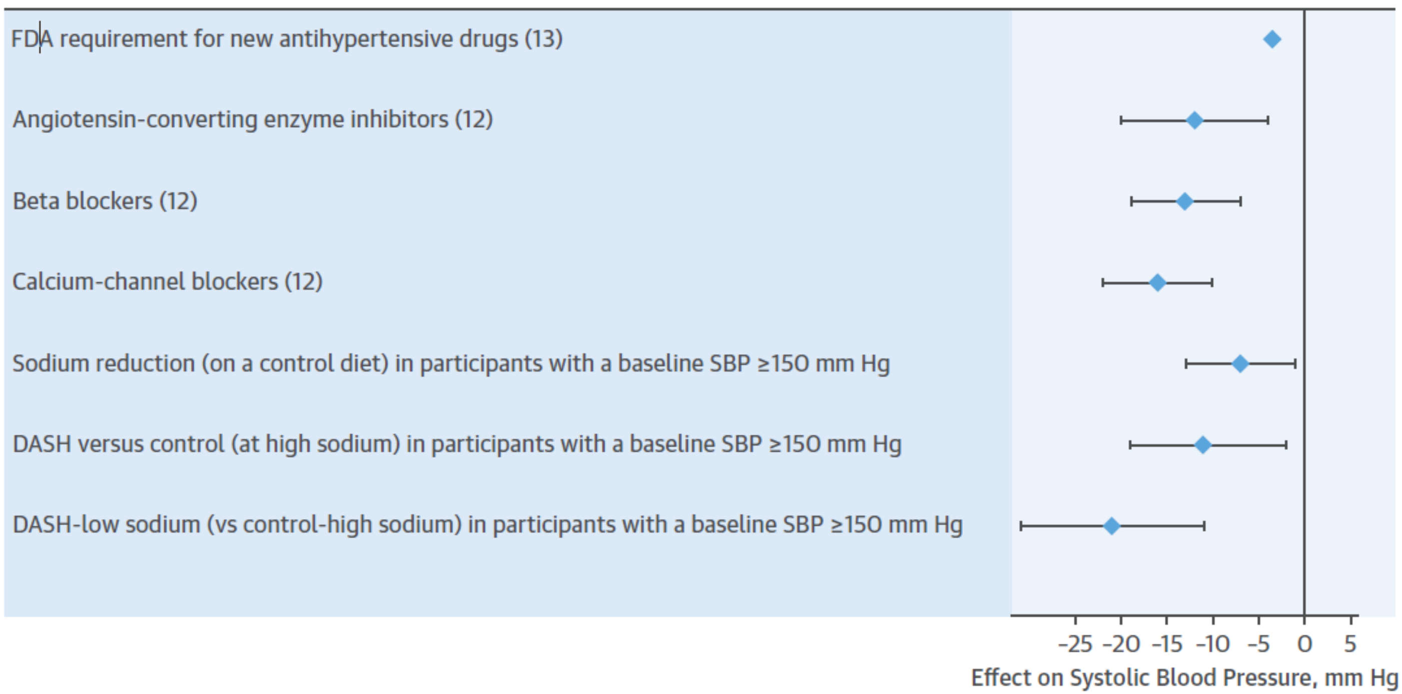
Sodium Excretion (Urine) and Blood Pressure



http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(16)30467-6/abstract

DASH diet: SBP-reduction in comparison

CENTRAL ILLUSTRATION **The BP Effects of the DASH Diet**

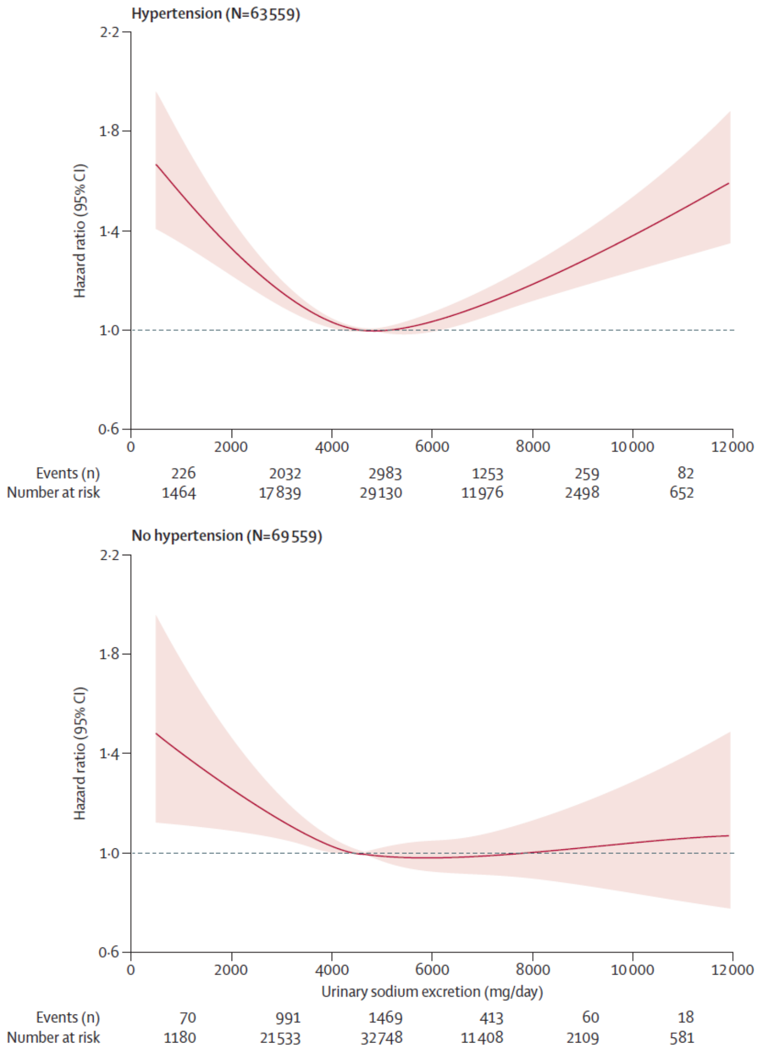


Juraschek, S.P. et al. J Am Coll Cardiol. 2017;70(23):2841-8.

Sodium reduction, alone or combined, compared with average BP effects of antihypertensive drug therapies and the FDA requirement for new antihypertensive drugs. Estimates for antihypertensive drug classes are taken from Manisty et al. (12). The FDA requirement for new antihypertensive drugs is taken from a committee meeting of the Center for Drug Evaluation and Research (2014) (13). BP = blood pressure; DASH = Dietary Approaches to Stopping Hypertension; FDA = Food and Drug Administration; SBP = systolic blood pressure.

https://www.sciencedirect.com/science/article/pii/S0735109717410989?via%3Dihub

Sodium excretion (urinary) and CVD



http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(16)30467-6/abstract

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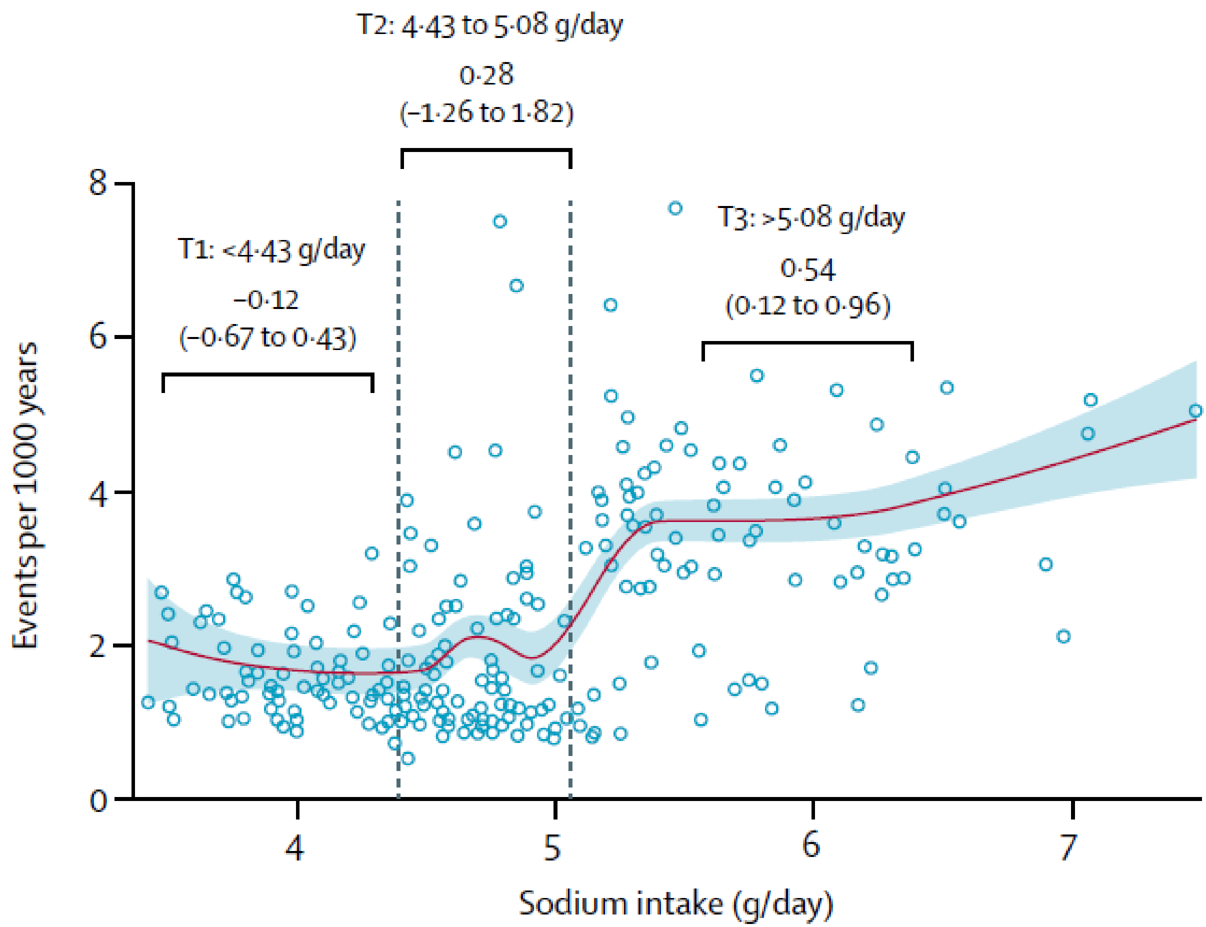
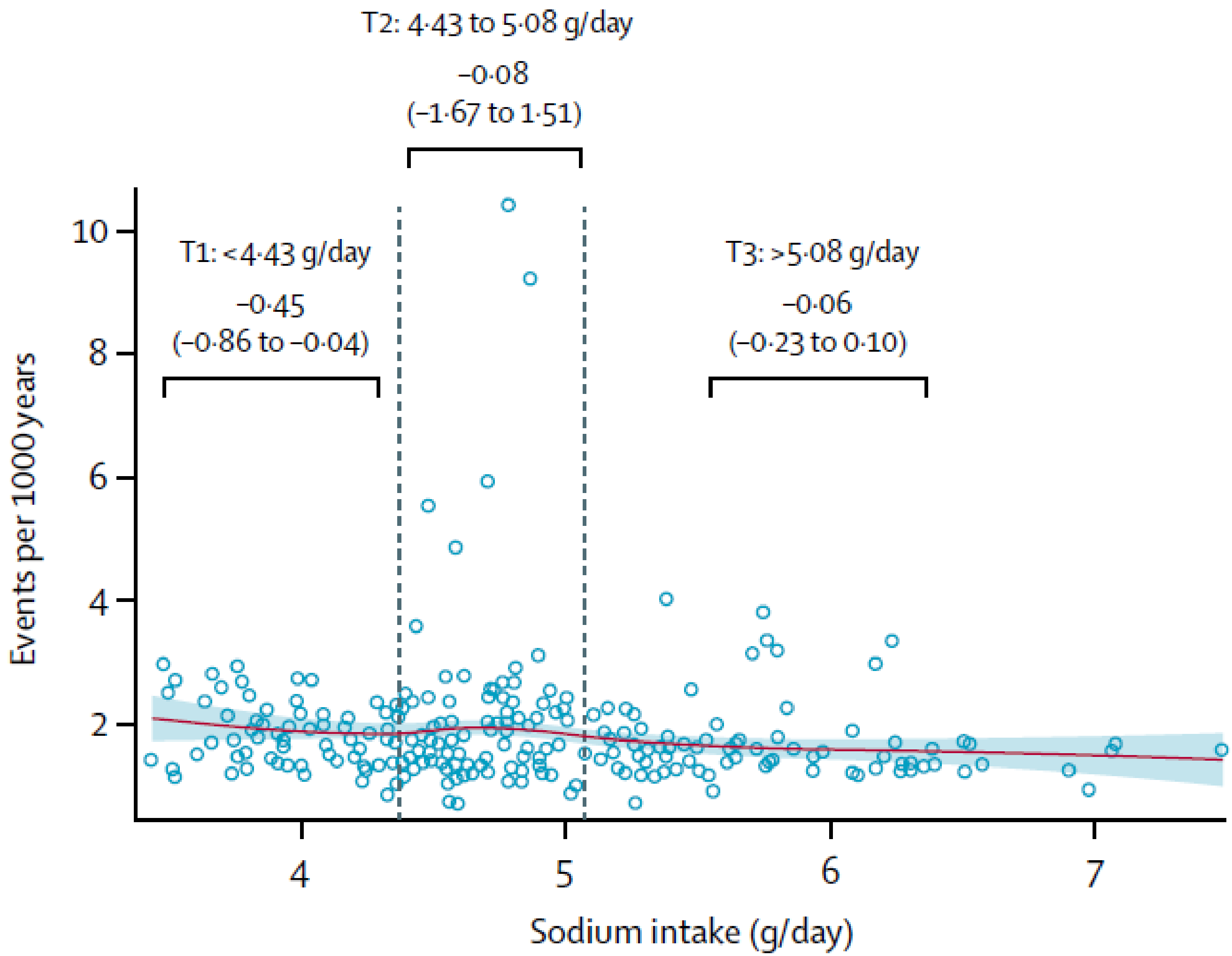
Sodium (Estimated **Intake)** and Stroke (Incidence)



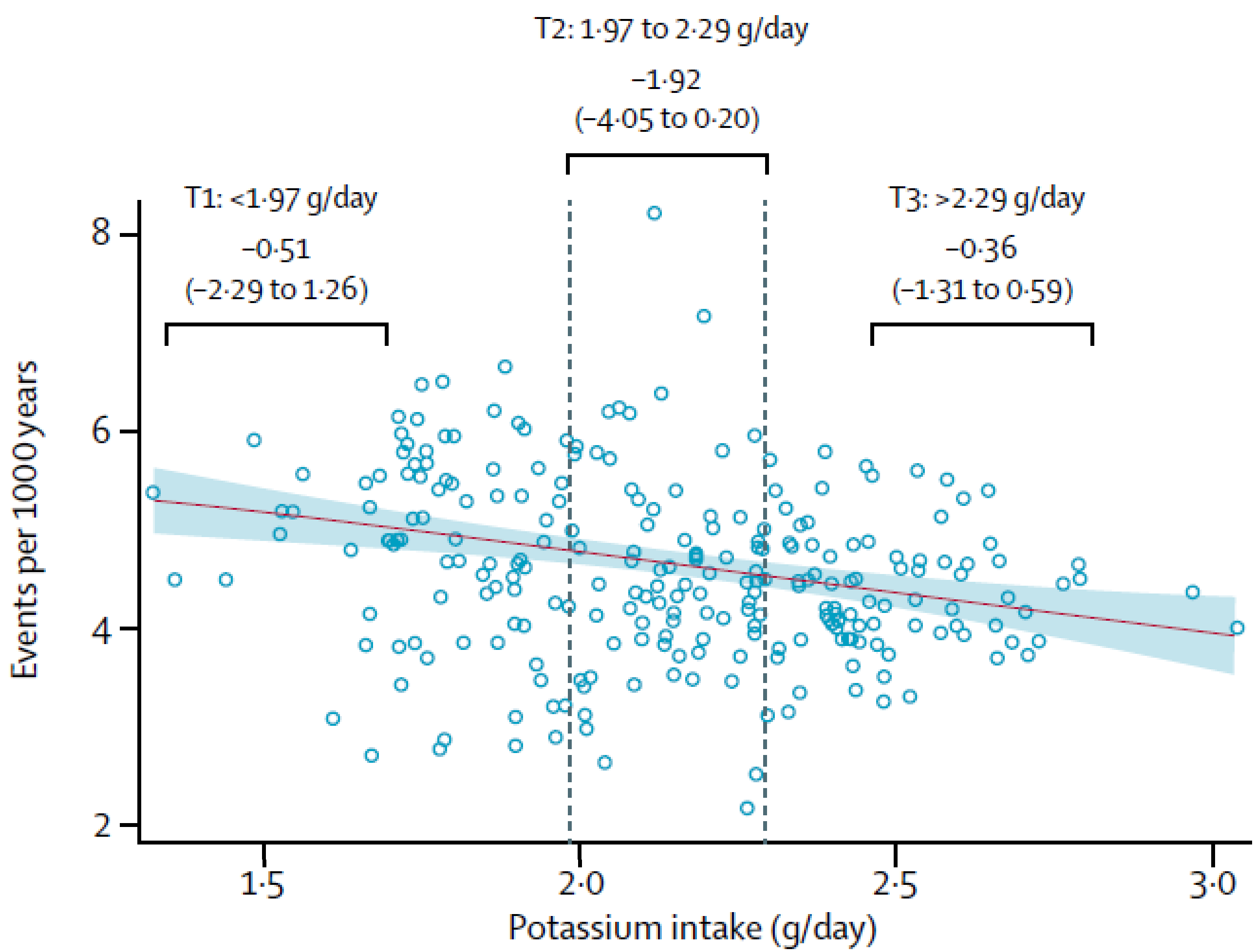
[Lancet, 09 Aug 2018, 392(10146):496-506](#)

Sodium (Estimated **Intake**) and Myocardial Infarction (Incidence)



[Lancet, 09 Aug 2018, 392(10146):496-506](#)

Potassium (Estimated Intake) and Cardiovascular Diseases (Incidence)



[Lancet, 09 Aug 2018, 392(10146):496-506](#)

Potassium / Sodium excretion: CVD-Risk

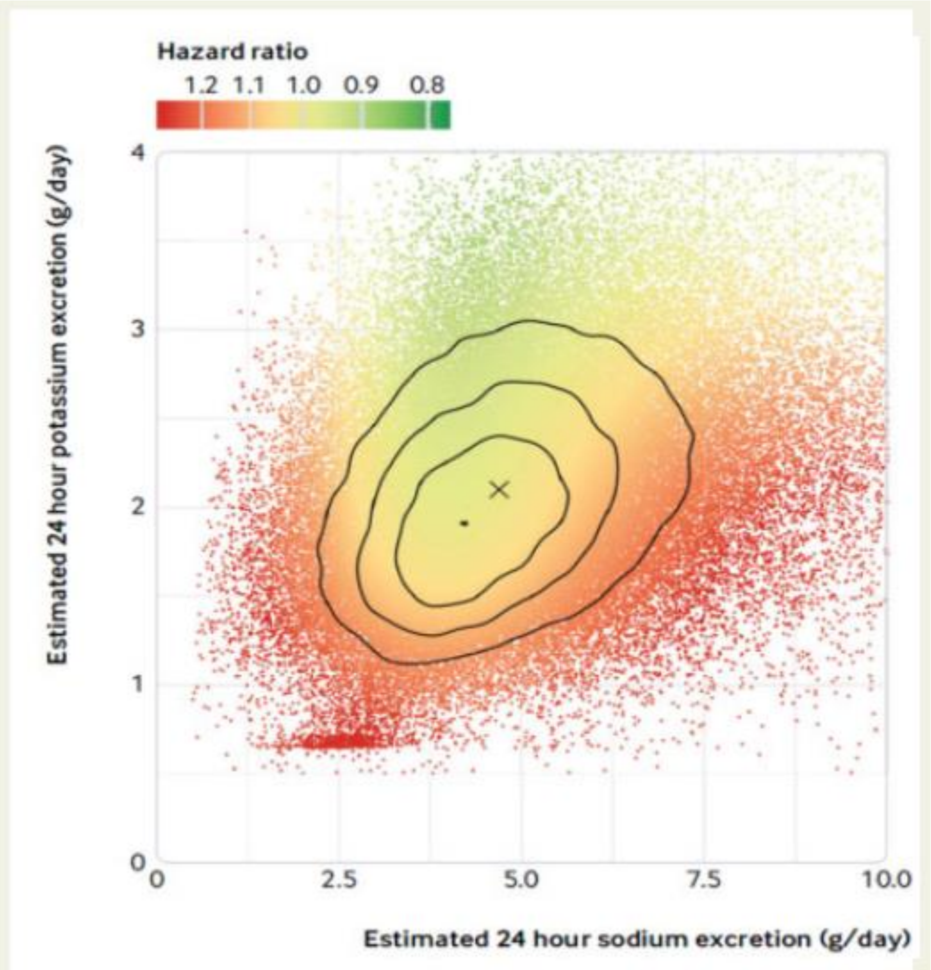
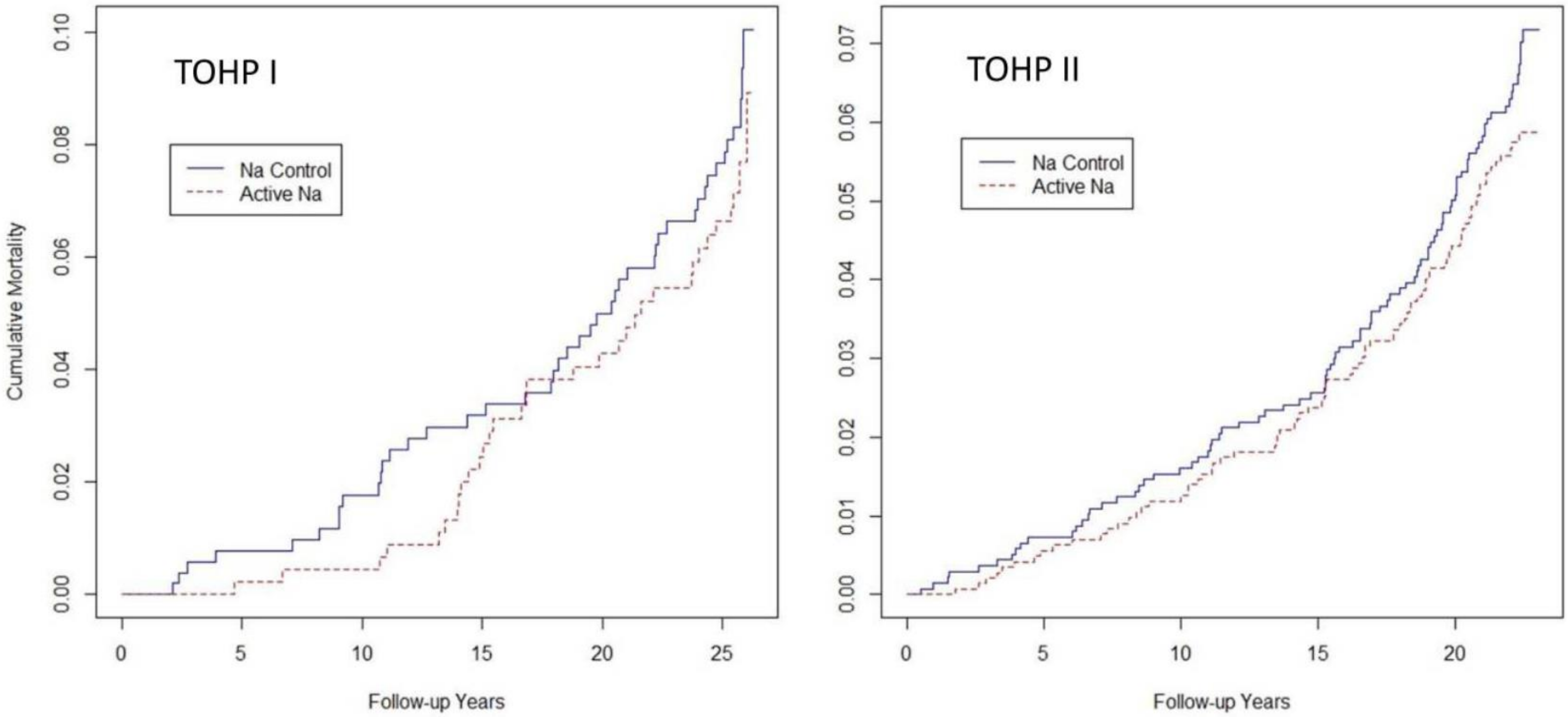


Figure 3 Association of combined sodium and potassium intake with mortality/cardiovascular risk (PURE study) (O'Donnell *et al.*[80]). Heat map of risk for composite of cardiovascular events or death showing lowest risk in region of moderate sodium intake 3–5 g/day and higher potassium intake and highest risk in region of extremes of sodium excretion and low potassium excretion. The reference hazard for these hazard ratios was set at a value of sodium daily excretion/ intake of 5.00 g and potassium daily excretion/intake of 2.25 g (median excretion of sodium and potassium), marked as X. The overlaid lines represent joint distribution quartiles; each region contains a quarter of the analysed participants. $r = 0.34$.

European Heart Journal, Volume 41, Issue 35, 14 Sep 2020, P 3363-3373, https://doi.org/10.1093/eurheartj/ehaa586

Total mortality by randomized sodium intervention group, TOHP*



*Trials of Hypertension Prevention

J Am Coll Cardiol. 2016 Oct 11; 68(15): 1609–1617.

Sodium excretion and Renin activity

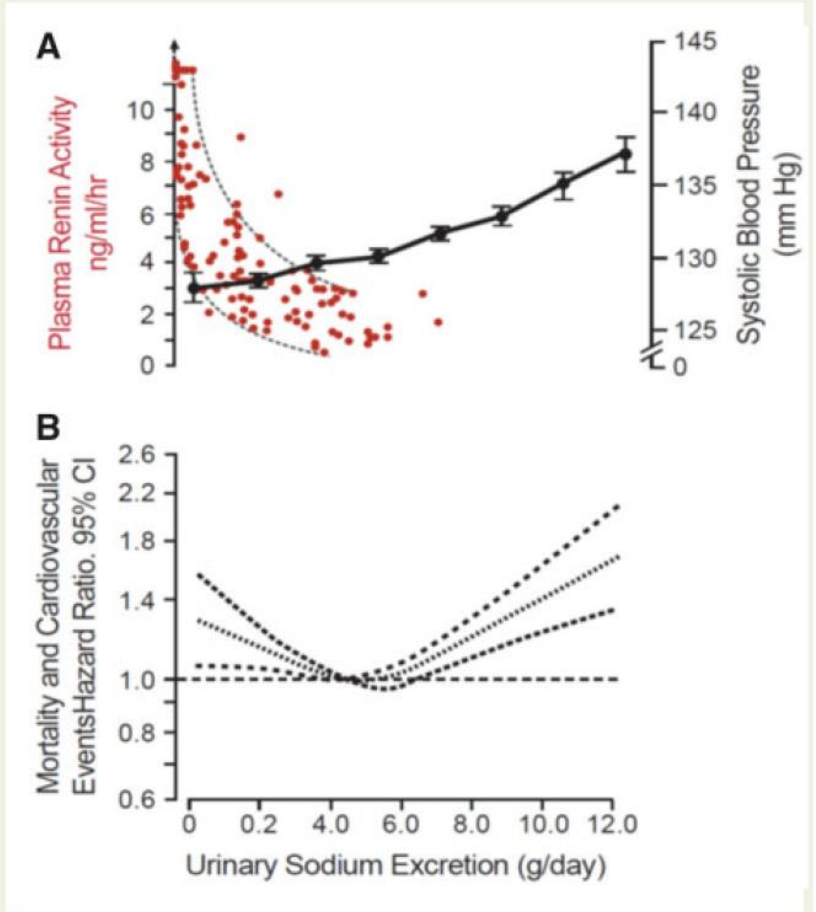


Figure 2 (*A* and *B*) Association of sodium intake with (*A*) plasma renin activity and systolic blood pressure; and (*B*) mortality and cardiovascular events (adapted from and O'Donnell *et al.*[51] and Brunner and Gavras[52]).

European Heart Journal, Volume 41, Issue 35, 14 Sep 2020, P 3363-3373, https://doi.org/10.1093/eurheartj/ehaa586

Disadvantages of sodium reduction

- A salt reduction from approx. 12g to approx. 5g...
  - ...reduced blood pressure in hypertensive patients by 5.5/2.9 mmHg (SBD/DBD)
  - ...increased the renin concentration by 55%.
  - ...increased the aldosterone concentration by 127%.
  - ...increased the concentration of adrenaline (14%) and norepinephrine (27%)
  - ...increased the concentration of cholesterol (3%) and triglycerides (6%)

Cochrane Database Syst Rev. 2017 Apr 9;4:CD004022

Sodium intake: populations at risk

- Focus on populations with **high intake**…

Sodium intake categories	Sodium (salt) g/day	Sodium (mmol/day)	~Teaspoons of salt
Low sodium intake	Sodium <2.3 g/day (salt <5.75 g/day)	Sodium <100 mmol	<1 teaspoon of salt
Moderate sodium intake	Sodium 2.3–4.6 g/day (salt 5.75–11.5 g/day)	Sodium 100–200 mmol/day	1–2 teaspoons of salt
High sodium intake	Sodium >4.6 g/day (Salt 11.5 g/day)	Sodium >200 mmol/day	>2 teaspoons of salt

- and with higher **age**…
- and **increased** blood pressure

European Heart Journal, Volume 41, Issue 35, 14 Sep 2020, P 3363-3373,
https://doi.org/10.1093/eurheartj/ehaa586

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Content

- CVD epidemiology
- Diet and CVD: scientific evidence
- Foods and CVD
- Nutrients and CVD
- Mediterranean diet
- Personalized nutrition

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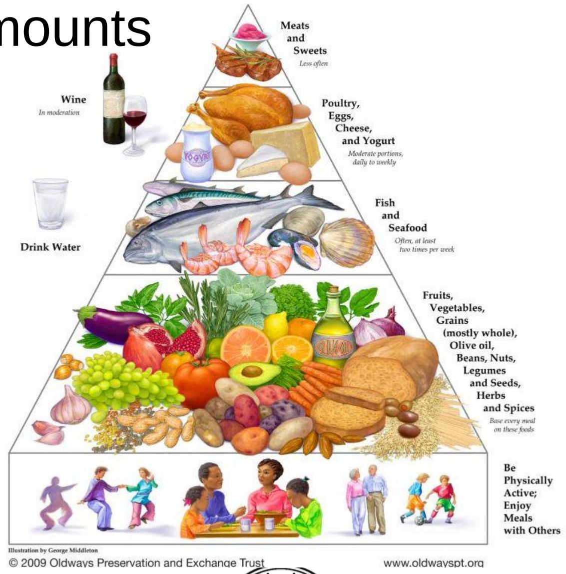
Mediterranean Diet

1. Mainly plant foods: fruits, vegetables, grains, nuts, seeds
2. Starchy food should contain fibres: Whole grains, legumes, quinoa
3. Minimally processed and seasonally fresh foods
4. Fresh fruits as the typical daily dessert
5. Extra Vergine olive oil as the principal source of dietary fat
6. Dairy, poultry, and fish in low to moderate amounts
7. Less than five eggs per week
8. Red meat in low frequency and amounts
9. Wine in low to moderate amounts

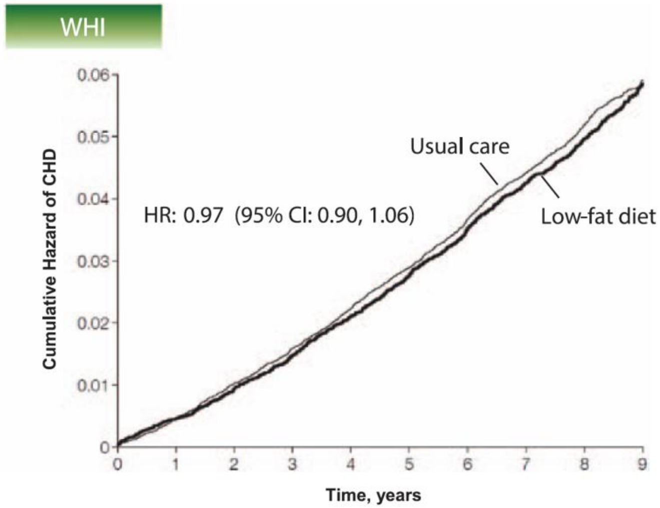
   (one to two glasses per day for men

   and one glass per day for women)

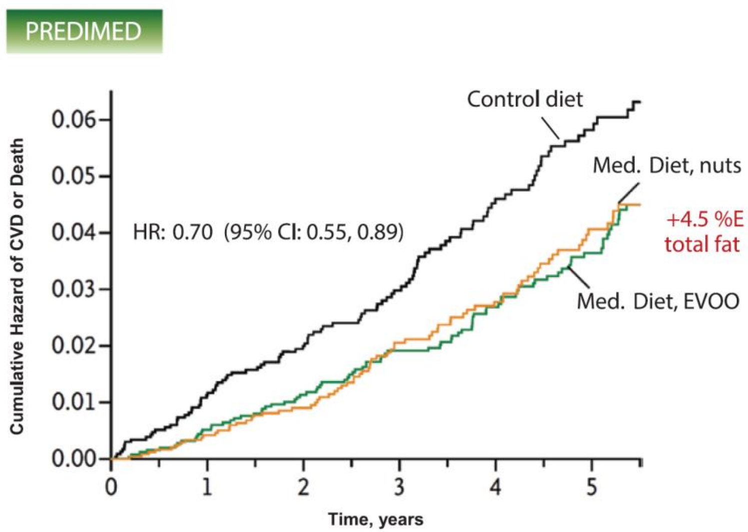


https://www.sciencedirect.com/science/article/pii/S0889852908000856?via%3Dihub

Diets and CVD: Evidence from RCTs



WHI
Women’s Health Initiative

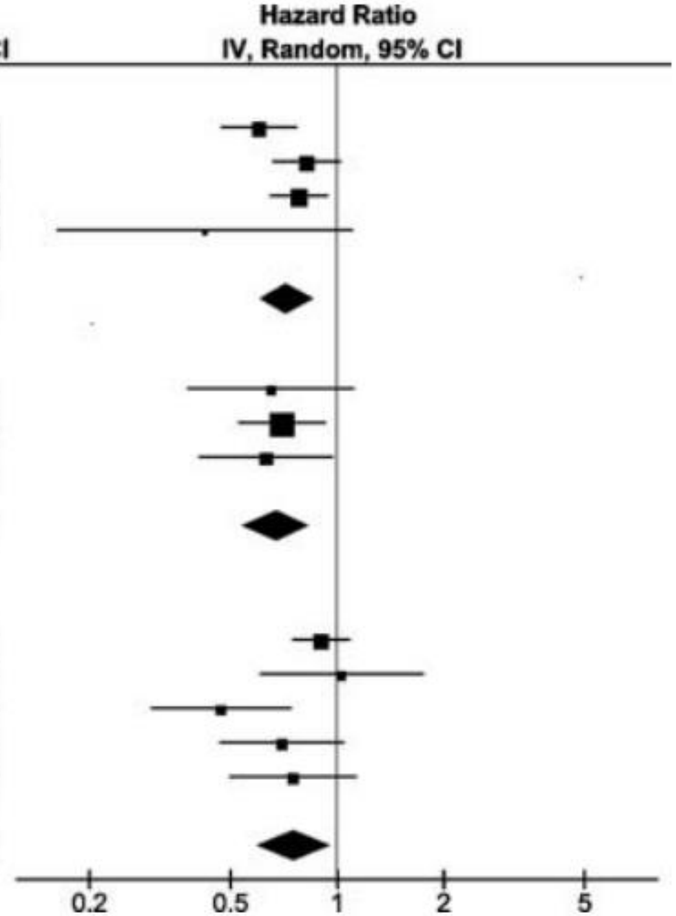


PREDIMED
Prevención con Dieta Mediterránea

https://jamanetwork.com/journals/jama/fullarticle/202339
http://www.nejm.org/doi/full/10.1056/NEJMoa1200303

Mediterranean diet and CVD, **prospective studies**, highest vs. lowest adherence

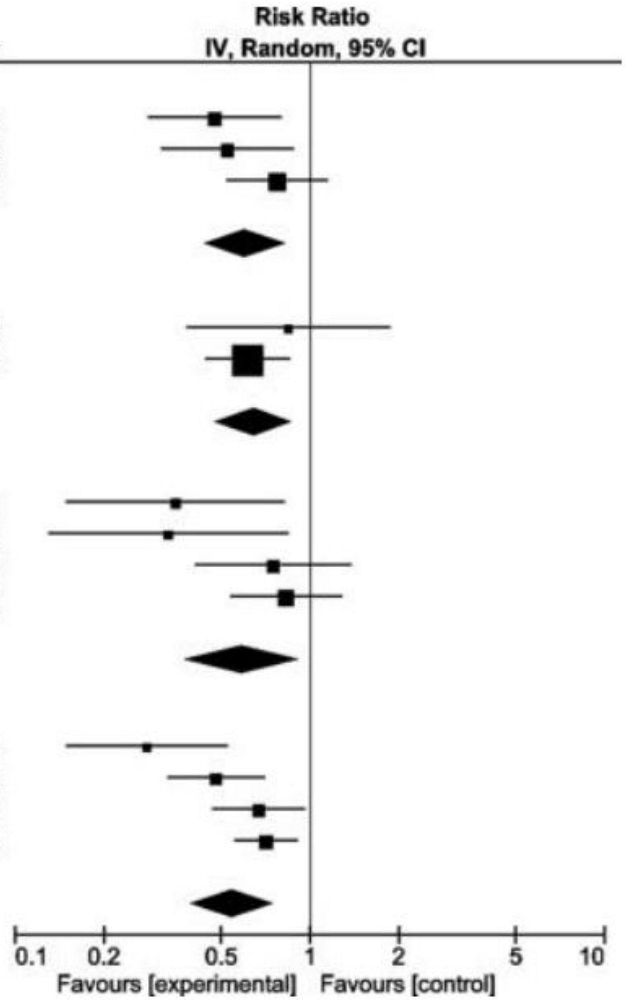
Study or Subgroup	log[Hazard Ratio]	SE	Weight	Hazard Ratio IV, Random, 95% CI
CHD incidence				
Buckland et al, 2009	-0.5108	0.1246	28.1%	0.60 [0.47, 0.77]
Dilis et al, 2012	-0.1985	0.1107	31.7%	0.82 [0.66, 1.02]
Fung et al, 2009	-0.2485	0.093	36.9%	0.78 [0.65, 0.94]
Martinez-Gonzales et al, 2011	-0.8675	0.4924	3.2%	0.42 [0.16, 1.10]
Total (95% CI)			**100.0%**	**0.72 [0.60, 0.86]**
MI incidence				
Gardener et al, 2011	-0.4308	0.2739	15.9%	0.65 [0.38, 1.11]
Hoevenaar-Blomet al, 2012	-0.3567	0.1419	59.3%	0.70 [0.53, 0.92]
Tognon et al, 2013	-0.462	0.2192	24.8%	0.63 [0.41, 0.97]
Total (95% CI)			**100.0%**	**0.67 [0.54, 0.83]**
Stroke incidence				
Fung et al, 2009	-0.1054	0.093	32.5%	0.90 [0.75, 1.08]
Gardener et al, 2011	0.0296	0.2673	13.6%	1.03 [0.61, 1.74]
Agnoli et al, 2011	-0.755	0.2291	16.5%	0.47 [0.30, 0.74]
Hoevenaar-Blomet al, 2012	-0.3567	0.2032	18.9%	0.70 [0.47, 1.04]
Tognon et al, 2013	-0.2877	0.2069	18.5%	0.75 [0.50, 1.13]
Total (95% CI)			**100.0%**	**0.76 [0.60, 0.96]**



CRITICAL REVIEWS IN FOOD SCIENCE AND NUTRITION, 2017, VOL. 57, NO. 15, 3218–3232, https://doi.org/10.1080/10408398.2015.1107021

Mediterranean diet and CVD, **RCTs**, highest vs. lowest adherence

Study or Subgroup	log[Risk Ratio]	SE	Weight	Risk Ratio IV, Random, 95% CI	Year
MI incidence					
Singh et al, 2002	-0.755	0.2643	28.6%	0.47 [0.28, 0.79]	2002
Giannuzzi et al, 2008	-0.6539	0.2639	28.7%	0.52 [0.31, 0.87]	2008
Estruch et al, 2013	-0.2614	0.2003	42.7%	0.77 [0.52, 1.14]	2013
Total (95% CI)			**100.0%**	**0.60 [0.44, 0.82]**	
Stroke incidence					
Giannuzzi et al, 2008	-0.1744	0.4047	14.5%	0.84 [0.38, 1.86]	2008
Estruch et al, 2013	-0.4943	0.1667	85.5%	0.61 [0.44, 0.85]	2013
Total (95% CI)			**100.0%**	**0.64 [0.47, 0.86]**	
CVD mortality					
de Lorgeril et al, 1999	-1.0498	0.4323	18.8%	0.35 [0.15, 0.82]	1999
Singh et al, 2002	-1.1087	0.4753	16.5%	0.33 [0.13, 0.84]	2002
Giannuzzi et al, 2008	-0.2877	0.3081	27.8%	0.75 [0.41, 1.37]	2008
Estruch et al, 2013	-0.1863	0.2193	36.9%	0.83 [0.54, 1.28]	2013
Total (95% CI)			**100.0%**	**0.59 [0.38, 0.93]**	
Composite					
de Lorgeril et al, 1999	-1.273	0.3185	16.0%	0.28 [0.15, 0.52]	1999
Singh et al, 2002	-0.734	0.1912	25.5%	0.48 [0.33, 0.70]	2002
Giannuzzi et al, 2008	-0.4005	0.1809	26.5%	0.67 [0.47, 0.96]	2008
Estruch et al, 2013	-0.3425	0.1211	32.0%	0.71 [0.56, 0.90]	2013
Total (95% CI)			**100.0%**	**0.55 [0.39, 0.76]**	



CRITICAL REVIEWS IN FOOD SCIENCE AND NUTRITION, 2017, VOL. 57, NO. 15, 3218–3232,
https://doi.org/10.1080/10408398.2015.1107021

CVD-preventive diets

- Mediterranean diet
- DASH: **D**ietary **A**pproaches to **S**top **H**ypertension
- TLC: **T**herapeutic **L**ifestyle **C**hanges
- Healthy Eating Index (HEI)
- Prudent diet
- Harvard Concept («Willet pyramid»)
- Recommendations of the AHA (American Heart Association)

- The are all quite similar

Table 1. Healthy Dietary Patterns in the 2015-2020 Dietary Guidelines Advisory Committee Report

Component	Healthy US Pattern	Healthy Mediterranean-style Pattern	DASH	Healthy Vegetarian Pattern
Total fruit (cups)[a]	**2**	**2.5**	**4**	**2**
Whole fruit (not juice)	—	—	—	—
Total vegetables (cups)[a,b]	**2.5**	**2.5**	**4**	**2.5**
Dark greens	1.5/wk	1.5/wk	—	1.5/wk
Red/orange	5.5/wk	5.5/wk	—	5.5/wk
Starchy	5/wk	5/wk	—	5/wk
Legumes	1.5/wk	1.5/wk	4-5/wk[c]	3/wk
Total grains (oz equivalent)[a]	**6**	**6**	**6**	**6.5**
Whole grains	3	3	3	3.5
Refined grains	3	3	3	3
Dairy (cups)[a]	**3**	**2**	**3**	**3**
Proteins (oz equivalent)[a]	**5.5**	**6.5**	—	**3.5**
Nuts/seeds	4/wk	4/wk	4-5/wk	7/wk
Red and processed meats	12.5/wk	12.5/wk	≤ 6/wk	—
Poultry	10.5/wk	10.5/wk	—	—
Seafood	8/wk	15/wk	—	—
Eggs	3/wk	3/wk	—	3/wk
Processed soy (tofu)	0.5/wk	0.5/wk	—	8/wk
Fats[a]				
Solid fats, g (tsp)	18 (2)	17 (0.9)	2-3	21 (2.3)
Oils, g (tsp)	27 (3)	27 (3)	—	27 (3)
Sweets, added sugars, g (tsp)[a]	30 (7.5)	29 (7.25)	—	36 (9)
Sugar-sweetened beverages/fruit juice	—		≤ 5/wk	—

DASH = Dietary Approach to Stop Hypertension; g = grams.

[a] Values are expressed as amount per day and are boldfaced. Scoring standards are based on cup and ounce equivalents, where 1 oz =28.3 g and 1 cup = 225 mL.

[b] Other vegetables and starchy vegetables are not shown here but contribute to total vegetables.

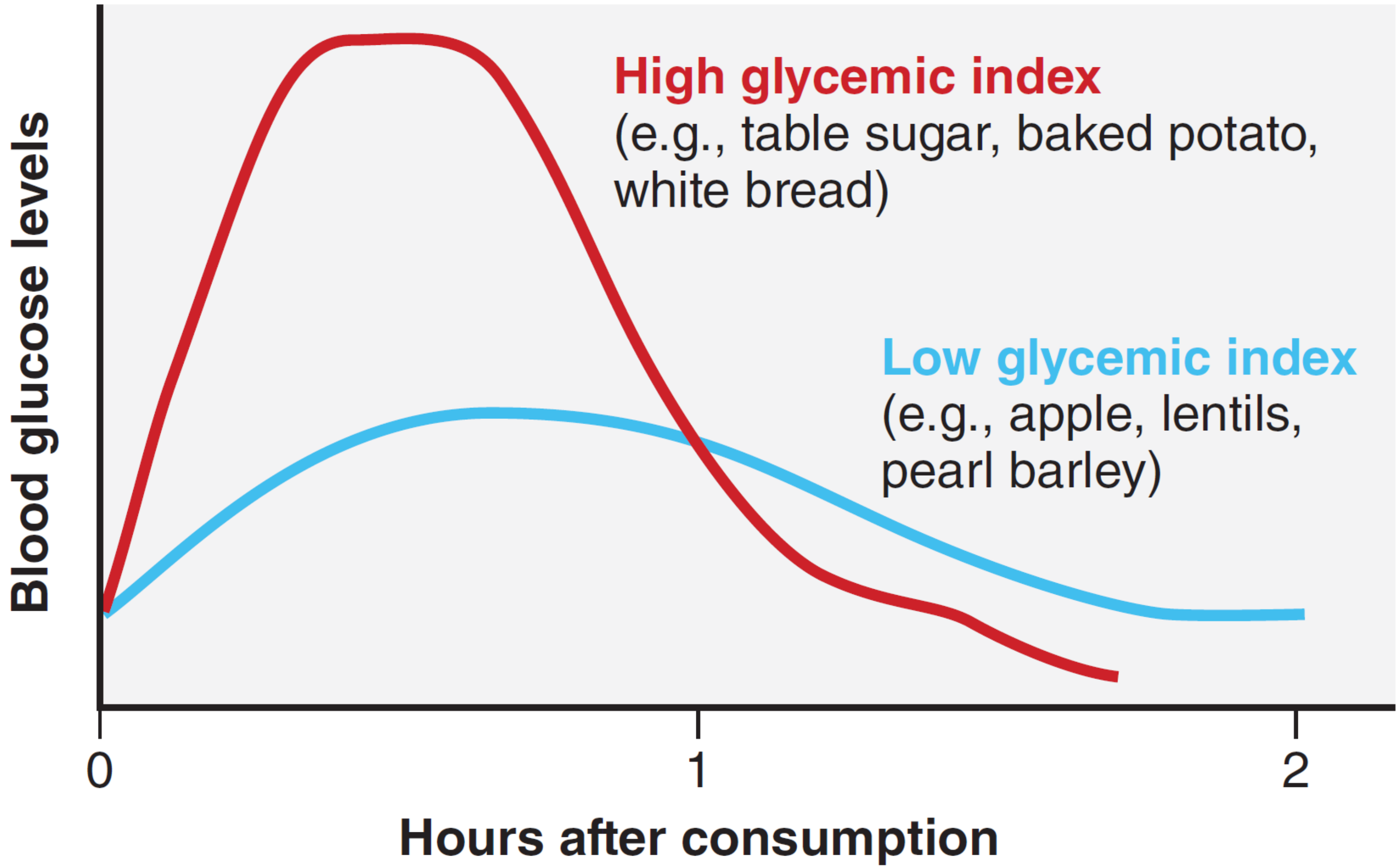
[c] The total amount, includes the amount counted toward protein foods.

http://www.npjournal.org/article/S1555-4155(16)30490-1/abstract

Content

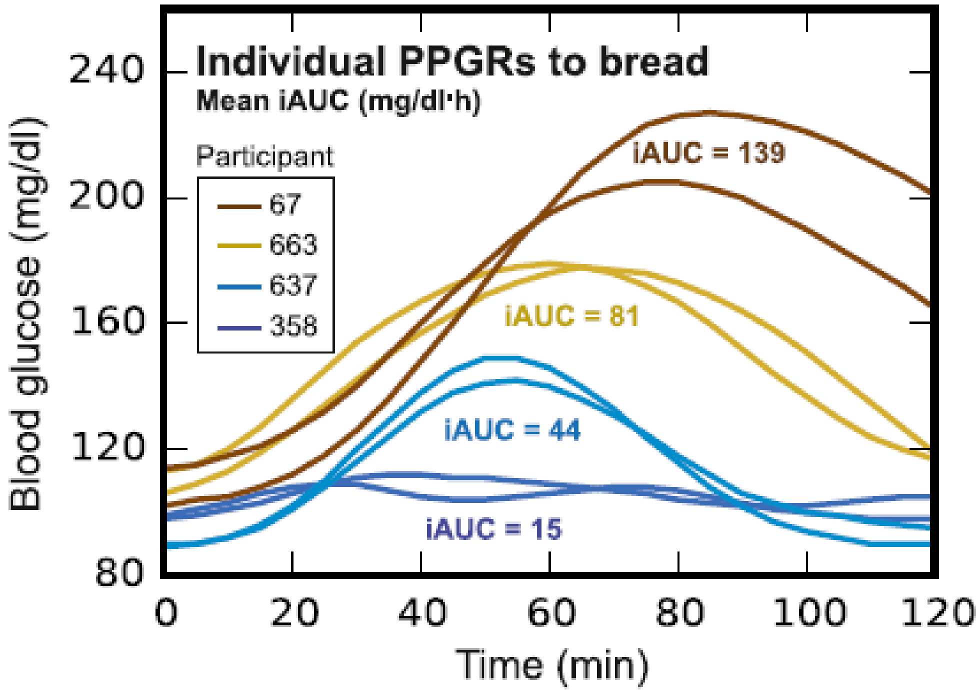
- CVD epidemiology
- Diet and CVD: scientific evidence
- Foods and CVD
- Nutrients and CVD
- Mediterranean diet
- **Personalized nutrition**

Blood glucose and glycemic index (GI)



http://circ.ahajournals.org/content/133/2/187.long

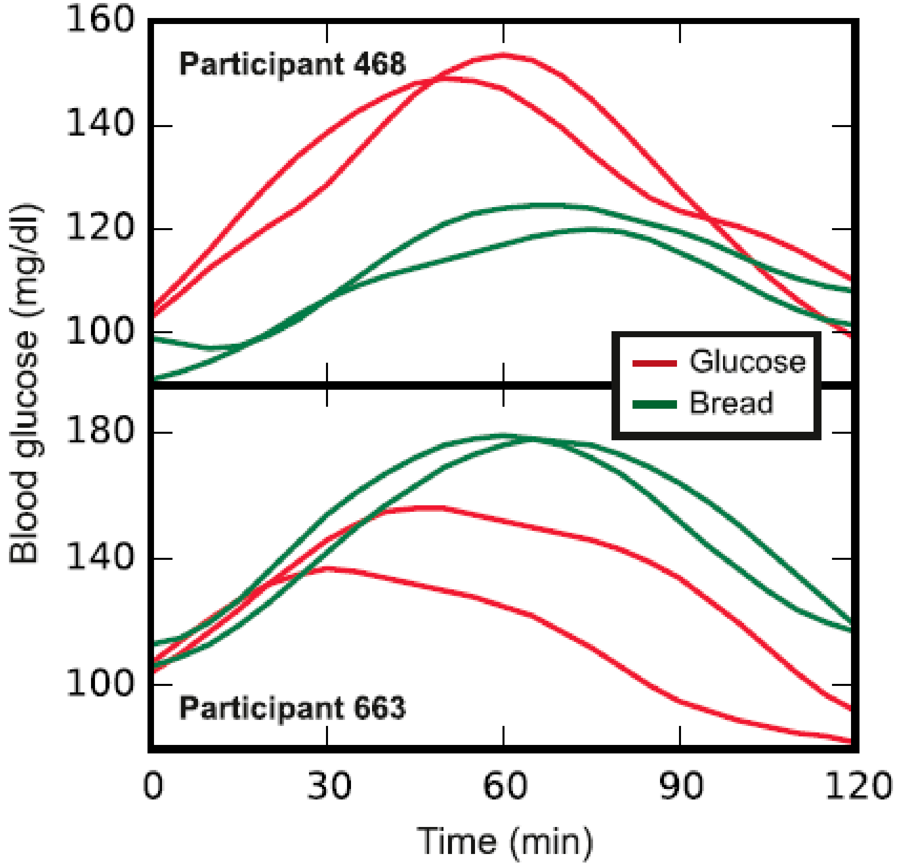
Individual glucose response after bread intake



PPGR: postprandial glycemic responses

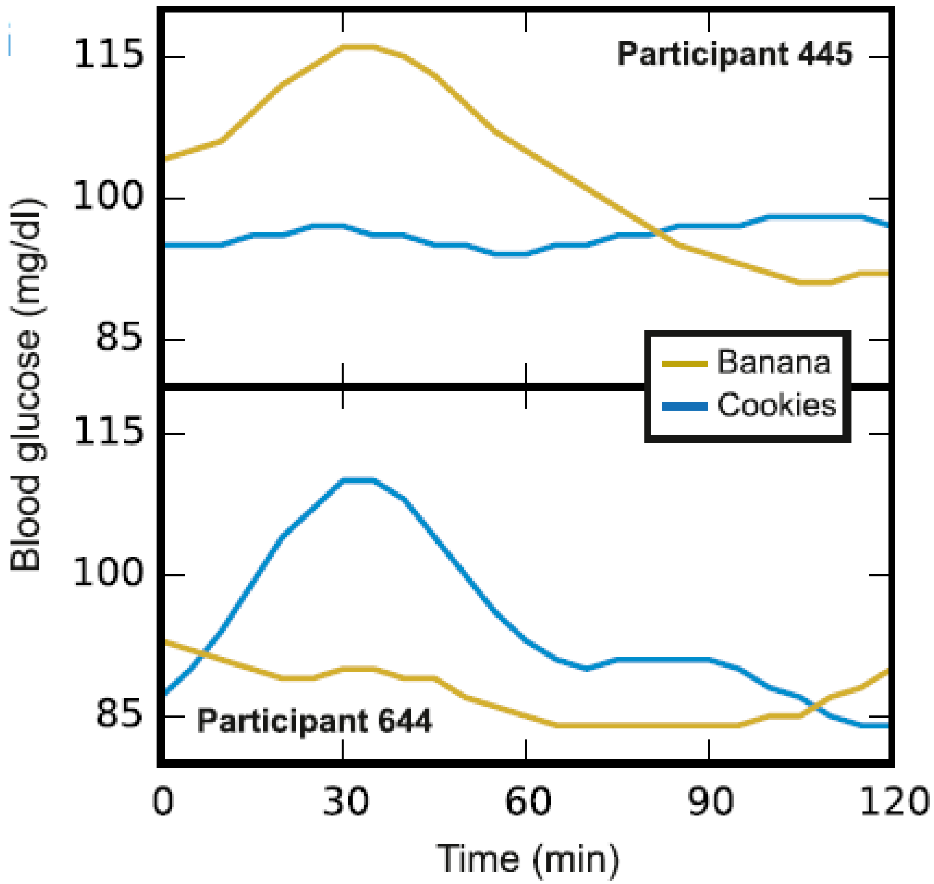
https://www.sciencedirect.com/science/article/pii/S0092867415014816?via%3Dihub
https://www.sciencedirect.com/science/article/pii/S1550413117302887?via%3Dihub

Individual glucose response after bread and glucose intake



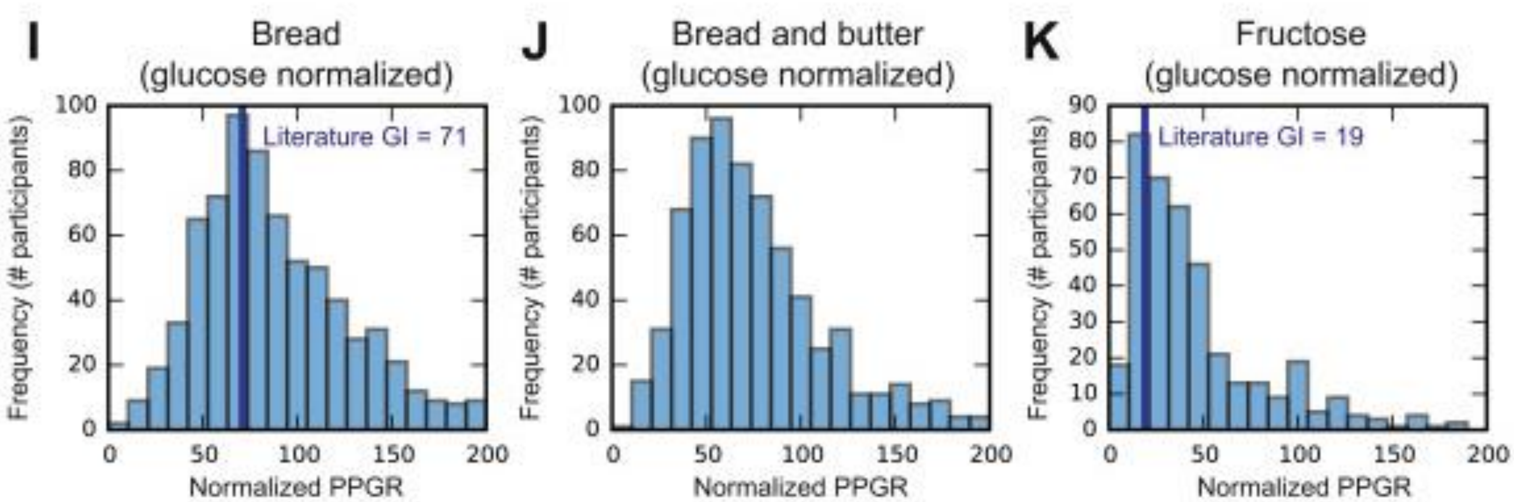
https://www.sciencedirect.com/science/article/pii/S0092867415014816?via%3Dihub
https://www.sciencedirect.com/science/article/pii/S1550413117302887?via%3Dihub

Individual glucose response after intake of banana and cookies



https://www.sciencedirect.com/science/article/pii/S0092867415014816?via%3Dihub
https://www.sciencedirect.com/science/article/pii/S1550413117302887?via%3Dihub

Inter-individual variability of the glycemic index (GI)



Literature GI: GI-value found in tables

PPGR: postprandial glycemic responses

https://www.sciencedirect.com/science/article/pii/S0092867415014816?via%3Dihub
https://www.sciencedirect.com/science/article/pii/S1550413117302887?via%3Dihub

Meat(-products): potential mechanisms

- Salt in processed meat: blood pressure↑
- Curing (nitrate/nitrite): impairs vascular function and insulin sensitivity, induces atherosclerosis and cancer
- Fats that negatively influence blood lipids and blood coagulation
- Iron overload: free radicals-> can affect important molecules (including DNA) and cells
- Substances that are metabolized by enteral bacteria to potentially harmful substances which influence cholesterol metabolism and vascular function

Krieger, JP. (2018). Les effets sur la santé des produits laitiers et carnés: que disent les données épidémiologiques? (NOVANIMAL Working Papers No.4). Zurich: Université de Zurich. doi: 10.21256/zhaw-1404

Curr Atheroscler Rep. 2012 Dec;14(6):515-24; BMJ 2017;357:j1957
Public Health Nutr. 2012 Dec;15(12):2287-94; Circulation. 2010 Jun 1;121(21):2271-83

Lost life years from alcohol (n=600'000)

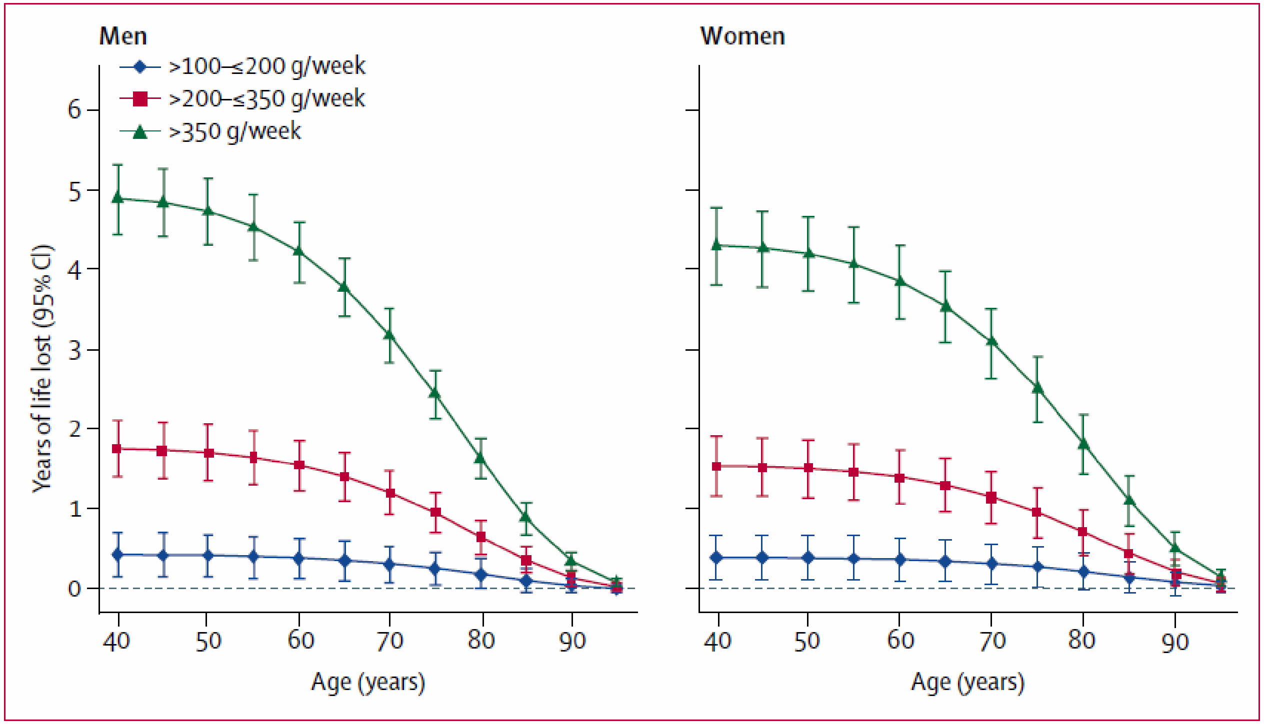


Figure 4: **Estimated future years of life lost by extent of reported baseline alcohol consumption compared with those who reported consuming >0–≤100 g per week**

The estimates of cumulative survival from 40 years of age onwards in the alcohol-drinking groups were calculated by applying hazard ratios (specific to age at risk) for all-cause mortality associated with categorised baseline alcohol consumption to US death rates at the age of 40 years or older. Mean usual levels of alcohol consumption within each baseline alcohol consumption category were 56, 123, 208 and 367 g per week, respectively, for the groups >0–≤100 g per week, >100–≤200 g per week, >200–≤350 g per week, and >350 g per week.

http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(18)30134-X.pdf

Dietary fat intake an blood cholesterol

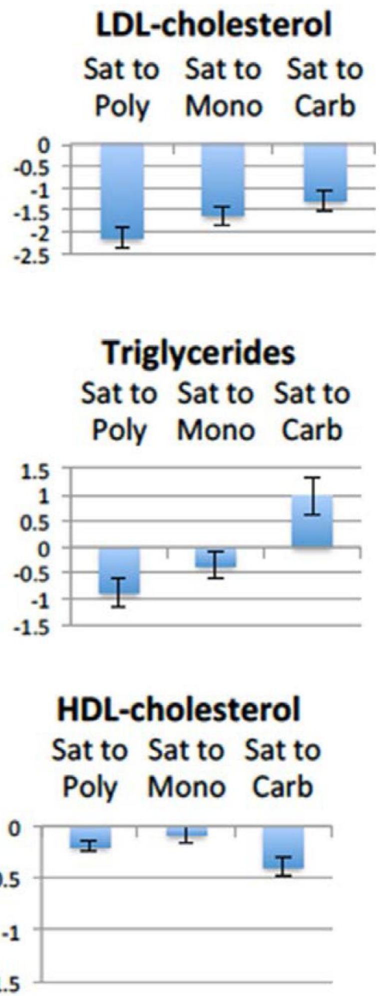


Figure 5. Effects of dietary fat and carbohydrates on blood low-density lipoprotein (LDL) cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol (mg/dL) in meta-regression analysis.
Left, Replacing saturated fat (Sat) with polyunsaturated fat (Poly) (n-6), monounsaturated fat (Mono), or carbohydrates (Carb). **Right**, Replacing carbohydrates with individual saturated fatty acids, lauric, myristic, palmitic, or stearic acid. Error bars show 95% confidence intervals. Data from Mensink.[4]

http://circ.ahajournals.org/content/early/2017/06/15/CIR.0000000000000510

Health effects of the Mediterranean diet	Magnitude
Risk reduction for coronary heart disease mortality	20-40%
Risk reduction for cancer	20-30%
Risk reduction for all cause mortality	17-25%
Risk reduction for cardiovascular disease	25-45%
Risk reduction for type 2 diabetes mellitus (incidence)	25-30%
Reduction body weight, BMI, waist circumference	In average 5 kilograms sustainable weight reduction
Improvement of components of the metabolic syndrome	about 30-40%

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4192546/pdf/12170_2014_Article_416.pdf