Heart Metab. (2014) 63:33–36

Basic food composition: is it just sugar and fat?

Thomas A. B. Sanders
Diabetes and Nutritional Sciences Division, School of Medicine, Kings College London, London, UK

Correspondence: Professor Thomas Sanders, Diabetes and Nutritional Sciences Division, School of Medicine, King's College London, Franklin-Wilkins Building, 150 Stamford Street, London SE1 9NH, United Kingdom.
E-mail: tom.sanders@kcl.ac.uk

Abstract

Dietary guidelines for cardiovascular disease (CVD) prevention apply to the whole population as well as to people with CVD. They are based on the premise that reductions in the mean population serum low-density lipoprotein cholesterol and blood pressure will reduce the population risk of CVD. Guidelines recommend moderating the intake of fat so that it supplies between 25% to 35% of the energy with most of the fat derived from monounsaturated and polyunsaturated fatty acids with a restriction on saturated (<10% energy) and trans fatty acids (1% energy). This can readily be achieved by choosing lean cuts of meat especially chicken, reduced fat dairy foods, using vegetable oils such as olive oil and rapeseed oil for cooking in place of hard fats, and eating oily fish and nuts. It is generally advised that the intake of confectionary sugar, cakes, biscuits, and sugar-sweetened beverages should be restricted, but the intake of fruit, which is rich in sugar, and vegetables should be increased to five portions a day. Restricting salt intake to <6 g/day helps maintain normal blood pressure, but salt intake <3.5 g/day is associated with increased risk of death in patients with heart failure. Obesity contributes to the risk of CVD, therefore, maintaining a body mass index in the range of 20 to 25 kg/m^2 is a desirable, but often an unachievable, goal. Weight maintenance is determined by balancing energy intake with expenditure; however, restricting the intake of foods that provide most of the energy is a key to controlling body weight. ■ Heart Metab. 2014;63:33–36

Keywords: Blood pressure; cardiovascular risk; fat; salt; sugar.

Population-based strategies to prevent cardiovascular disease (CVD) focus on diet and lifestyle modification. Dietary guidelines for the prevention of CVD are broadly similar in Australasia, Western Europe, the United Kingdom,[1] and the United States.[2] Consensus exists regarding the prevention of obesity, which is directly associated with the risk of CVD, and which operates through a variety of pathways including increased blood pressure, hyperlipidemia, and type 2 diabetes. Preventing and managing obesity depends on balancing energy intake with energy expenditure. There is no good evidence to suggest that different ratios of fat to carbohydrates are any better in controlling body weight if intakes are isoenergetic. However, weight maintenance is more likely to be achieved by controlling the portion sizes of drinks and meals as well as avoiding high-energy foods which have low-nutrient density, such as sugar-sweetened beverages, cakes, biscuits, and deep fried snacks. There is controversy concerning other aspects of diet, notably the role of fat vs refined carbohydrates and salt. The nutrient targets aim to reduce fat intake, to partially replace saturated fatty acids (SFA) with unsaturated fatty

Abbreviations

BP: blood pressure; **CHD:** coronary heart disease; **CVD:** cardiovascular disease; **DG:** dietary guidelines; **HDL-C:** high-density lipoprotein cholesterol; **LDL-C:** low-density lipoprotein cholesterol; **MUFA:** monounsaturated fatty acids; **PHVO:** partially hydrogenated vegetable oil; **PUFA:** polyunsaturated fatty acids; **RCT:** randomized controlled trial; **SFA:** saturated fatty acids; **TC:** total serum cholesterol

acids, to avoid foods containing industrial trans fats, and to reduce the intake of added sugars and salt. They have been translated into palatable food-based dietary guidelines, which are illustrated in *Table I*. This article reviews the scientific basis for these guidelines.

The dietary guidelines evidence for CVD prevention

The evidence supporting dietary guidelines is derived from observational data, which is mainly from prospective cohort studies as well as the effects of individual dietary components on surrogate CVD risk markers such as blood pressure (BP) and the serum lipid profile, but rarely from randomized controlled trials (RCTs) with clinical end points. Foods associated with an increased risk of CVD are red meat and sugar-sweetened beverages, and foods associated with a lower risk of CVD are whole grain cereals, nuts, fish, fruit and vegetables, and moderate intakes of alcohol.[2] Milk and yogurt appear to be associated with a lower risk of CVD, but there is some uncertainty regarding cheese. Some of these associations are subject to residual confounding by other health-related behaviors and, therefore, may not be causal.

It would be an immense challenge to conduct a long-term trial of dietary modification on CVD because incidence rates are relatively low below the age of 60 years, thus requiring the recruitment of tens of thousands of people to change their diet for many years. It is impossible to conduct placebo-controlled, RCTs with diet, however, this has been done with some components such as vitamins, minerals, and long-chain n-3 polyunsaturated fatty acids (PUFA). RCTs of dietary antioxidant vitamins and selenium supplements (vitamin A, C, E, β-carotene, and selenium),[3] homocysteine-lowering vitamins (folic acid, vitamin B6, and vitamin B12)[4] show no effect on CVD outcomes. RCTs show that the risk of CVD may be increased by vitamin D and calcium supplementation,[5] and that long-chain n-3 PUFA supplements may reduce the risk of cardiac death,[6] but not CVD incidence or all-cause mortality in secondary prevention. Consequently, dietary supplements are not recommended for CVD prevention.

Nutrient	Target	Food-based guidelines
Saturated fatty acids	<10% energy	Choose lean cuts of meat, poultry, and chicken—avoid fatty meat and meat products. Choose reduced fat dairy produce, use monounsaturated oils for food preparation (ie, olive oil, rapeseed oil)
Trans fatty acids	<1%	Avoid fats containing partially hydrogenated vegetable oils
Total fat	25% to 35% energy	Avoid deep-fried foods, use oils and fat spreads sparingly
Oily fish	0.2 g of n-3 poly unsaturated fatty acids	At least 1 portion of oily fish/week: fresh tuna, mackerel, herring, pilchard, sardine, salmon
Whole grains	Encouraged, but amount not specified	Choose whole grain breakfast cereals, whole grain bread, brown rice, brown pasta
Added sugars	<60 g added sugar	Restrict the intake of confectionery sugar, cakes, biscuits, and sugar-sweetened beverages
Fruit and vegetables	Potassium >3.5 g/day	5 portions/day. One portion is 80 g, but fruit juice only counts as one portion
Salt	<6 g/day	Do not add salt to food, choose foods with a reduced salt content
Energy intake	body mass index: 20 to 25 kg/m^2	Balance energy intake with energy expenditure

Table I *Dietary guidelines for cardiovascular disease prevention and their translation into food-based dietary guidelines.*

Level and type of fat

High fat intake contributes to hyperlipidemia, which is the major underlying process in atherosclerosis. SFA (12 to 16 carbons long) and trans fatty acids increase total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) compared with carbohydrates, monounsaturated fatty acids (MUFA), or PUFA.[7] Replacing fat with carbohydrates lowers high-density lipoprotein cholesterol (HDL-C) regardless of the glycemic index of the carbohydrate intake,[8] but this may not be of any clinical significance.[9] A 1 mmol/L decrease in LDL-C lowers CVD death and major events by 10% and 20%, respectively.[10] Therefore, a 5% reduction in energy from SFA that would lower LDL-C by up to 0.3 mmol/L would be predicted to decrease the risk of fatal and non-fatal CVD by 3% and 6%, respectively. SFA intake has markedly decreased over the past 20 to 30 years in many countries including the UK, which is mainly due to the replacement of animal and hydrogenated fats with vegetable oils,[7] and as a result, the average TC has decreased. SFA intake remains above the target of 10% in many Western countries, typically in the range of 11% to 14%, but further reductions in SFA intake may have only a modest impact on CVD.

Prospective cohort studies found that trans fatty acids, which result from industrial partially hydrogenated vegetable oils (PHVO), are associated with a 24% increased risk of CVD.[11] PHVO were used to make margarine and shortenings for biscuits, cakes, and pastries. However, PHVO is now rarely used in Europe and the level of exposure in the United States has declined. In November 2013, the Food and Drug Administration issued a preliminary determination that PHVO are no longer recognized as safe. Nowadays, margarines contain fewer trans fatty acids and SFAs than butter.

Cohort studies show a lower risk of CVD when SFAs are replaced by PUFAs, but not by refined carbohydrates or MUFA.[12] They also show that replacing trans fatty acids with SFAs, MUFAs, or PUFAs decreases the risk of CVD.[11]

Diets high in fat have the potential to increase energy intake because fats contain 9 kcal/g compared with 3.75 kcal/g for carbohydrates. Furthermore, meals high in fat (both saturated and unsaturated) promote postprandial lipemia that can result in the formation of atherogenic remnant particles, cause endothelial dysfunction and activate factor VII coagulant activity.[7] Consequently, it is desirable to avoid meals high in fat. Furthermore, longer-term studies show that a lower fat intake does lead to a small 0.51 kg/m^2 reduction in body mass index.[13]

Type of carbohydrate

Carbohydrates constitute the major source of food energy in most human diets. In the rural area of developing countries where CVD is still uncommon and serum TC concentrations are low, carbohydrates provide over 60% of the food energy mainly as starch from cereal crops such as rice, wheat, maize, or tubers. Prospective cohort studies in the United States found that high intake of sugar-sweetened beverages was associated with a greater risk of CVD.[14] There has also been a substantial debate regarding the effects of fructose versus glucose, but a meta-analysis of metabolic feeding studies[15] showed minimal differences between fructose and glucose: fructose slightly improved plasma glucose and body weight, but it slightly increased fasting and postprandial triglycerides. High intake of sugar-sweetened beverages may contribute to obesity rather than having a direct effect on CVD risk.

Prospective studies generally show fruit consumption to be associated with a lower risk of CVD. While fruit typically contains 10% to 20% sugar by weight, it also has vitamin C and potassium. Consequently, dietary guidelines encourage fruit consumption, but advocate restricting the intake of added sugars.

Salt

High salt intake is linked with BP and restricting salt intake lowers BP. For each 100 mmol reduction in sodium intake per day, a reduction in systolic/diastolic BP in hypertensive and normotensive subjects of 5.4/2.8 and 2.4/1.1 mm Hg, respectively can be expected.[16] The impact of salt restriction on BP is greater in people over the age of 50, when renin concentrations are low, and in black Africans. To put these effects in context, a 20 mm Hg difference in systolic BP doubles the risk of CVD.

Salt is often added to food at the table or during food preparation, however, about 70% comes from processed food. The foods that are particularly high in salt are pickled foods such as ham, bacon, sausages, cheese, olives, some soups, and soy sauce (≈20% salt). Bread, which contains 1% to 2% salt, is also an important contributor to salt intake. However, it is

hard to measure salt intake from dietary records and it is more reliably estimated from measuring sodium output in urine: a urinary sodium excretion of 100 mmol/day is equivalent to a salt intake of 6 g/day. A recent meta-analysis reported a 2.59 relative risk of death[17] in heart failure patients with a salt intake <3.5 g/day. Thus, the relationship between salt intake and blood pressure may be J-shaped with both low and high intakes having adverse effects on CVD.

Many people find it very difficult to follow low salt diets, in part, because the salt content of food is difficult to understand, and because people prefer salty food. The UK Food Standards Agency persuaded the food industry to gradually reduce the salt content of processed foods especially bread, cereals, sauces, and ready meals and then introduced a traffic light–labeling system for the salt levels in processed foods. Average salt intakes in the UK have decreased to 8 g/day and the average BP has fallen, which has contributed to the 55% decrease in CVD mortality that has occurred in the UK since 1997.[18] Yet, salt intake remains high (>15 g/day) in some other European countries.

Conclusion

Dietary advice to prevent CVD has focused on reductions in SFA, sugar, and salt intake, but controlled studies where compliance to treatment is good only show modest changes in LDL-C and BP with a minimal impact on obesity. A more promising approach to CVD prevention is to modify the overall dietary pattern as in the DASH (Dietary Approaches to Stop Hypertension)[19] and OMNIHEART (Optimal Macronutrient Intake Trial to Prevent Heart Disease)[20] trials, which were designed to mimic dietary patterns associated with a lower risk of CVD.[21] ■

REFERENCES

1. Department of Health. Nutritional aspects of cardiovascular disease. Report on Health and Social Subjects. 1994. No 46. HMSO. London.
2. Mozaffarian D, Appel LJ, Van Horn L. Components of a cardioprotective diet: new insights. Circulation. 2011;123:2870-2891.
3. Bjelakovic G, Nikolova D, Gluud LL, Simonetti RG, Gluud C. Antioxidant supplements for prevention of mortality in healthy participants and patients with various diseases. Cochrane Database Syst Rev. 2012;3:CD007176.
4. Armitage JM, Bowman L, Clarke RJ, et al; Study of the Effectiveness of Additional Reductions in Cholesterol and Homocysteine (SEARCH) Collaborative Group. Effects of homocysteine-lowering with folic acid plus vitamin B12 vs placebo on mortality and major morbidity in myocardial infarction survivors: a randomized trial. JAMA. 2010;303:2486-2494.
5. Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the women's health initiative limited access dataset and meta-analysis. BMJ. 2011;342:d2040.
6. Rizos EC, Ntzani EE, Bika E, Kostapanos MS, Elisaf MS. Association between omega-3 fatty acid supplementation and risk of major cardiovascular disease events: a systematic review and meta-analysis. JAMA. 2012;308:1024-1033.
7. Sanders TA. Reappraisal of SFA and cardiovascular risk. Proc Nutr Soc. 2013;72:390-398.
8. Jebb SA, Lovegrove JA, Griffin BA, et al; RISCK Study Group. Effect of changing the amount and type of fat and carbohydrate on insulin sensitivity and cardiovascular risk: the RISCK (Reading, Imperial, Surrey, Cambridge, and Kings) trial. Am J Clin Nutr. 2010;92:748-758.
9. Voight BF, Peloso GM, Orho-Melander M, et al. Plasma HDL cholesterol and risk of myocardial infarction: a mendelian randomisation study. Lancet. 2012;380:572-580.
10. Baigent C, Blackwell L, Emberson J, et al; Cholesterol Treatment Trialists' (CTT) Collaboration. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. Lancet. 2010;376:1670-1681.
11. Mozaffarian D, Clarke R. Quantitative effects on cardiovascular risk factors and coronary heart disease risk of replacing partially hydrogenated vegetable oils with other fats and oils. Eur J Clin Nutr. 2009;63:S22-S33.
12. Sanders TA. Protective effects of dietary PUFA against chronic disease: evidence from epidemiological studies and intervention trials. Proc Nutr Soc. 2014;73:73-79.
13. Hooper L, Abdelhamid A, Moore HJ, Douthwaite W, Skeaff CM, Summerbell CD. Effect of reducing total fat intake on body weight: systematic review and meta-analysis of randomised controlled trials and cohort studies. BMJ. 2012;345:e7666.
14. de Koning L, Malik VS, Kellogg MD, Rimm EB, Willett WC, Hu FB. Sweetened beverage consumption, incident coronary heart disease, and biomarkers of risk in men. Circulation. 2012;125:1735-1741.
15. Sievenpiper JL, de Souza RJ, Cozma AI, Chiavaroli L, Ha V, Mirrahimi A. Fructose vs. glucose and metabolism: do the metabolic differences matter? Curr Opin Lipidol. 2014;25:8-19.
16. He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. Cochrane Database Syst Rev. 2013;4:CD004937.
17. Taylor RS, Ashton KE, Moxham T, Hooper L, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease: a meta-analysis of randomized controlled trials (Cochrane review). Am J Hypertens. 2011;24:843-853.
18. Bajekal M, Scholes S, Love H, et al. Analysing recent socioeconomic trends in coronary heart disease mortality in England, 2000-2007: a population modelling study. PLoS Med. 2012;9:e1001237.
19. Sacks FM, Svetkey LP, Vollmer WM, et al; DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) Diet. N Engl J Med. 2001;344:3-10.
20. Appel LJ, Sacks FM, Carey VJ, et al; OmniHeart Collaborative Research Group. Effects of protein, monounsaturated fat and carbohydrate intake on blood pressure and serum lipids: results of the OMNIHEART randomized trial. JAMA. 2005;294:2455-2464.
21. European Commission. Health: nutritional and physical activity section. http://ec.europa.eu/health/nutrition_physical_activity/policy/index_en.htm. Accessed March 19, 2014.