General Principles of Nutrition Support in Cardiac Rehabilitation

2

Helmut Gohlke

The type of nutrition is one of the important factors that contribute to the development of cardiovascular disease. The way we eat is part of our lifestyle and poor dietary habits and physical inactivity together contribute to 15% of the causes of death.[45]

A diet with a low proportion of fruits and vegetables contributes to more than a quarter of the population attributable fraction of coronary artery disease and strokes.[15] Accordingly dietary recommendations are a key element in the management of patients with cardiovascular disease. More and more studies indicate that certain dietary patterns can influence cardiovascular health by modifying risk factors such as obesity, dyslipidemia, and hypertension as well as factors involved in systemic inflammation, insulin sensitivity, oxidative stress, endothelial function, thrombosis, and cardiac rhythm. A diet favorable for cardiovascular prevention can also reduce the risk of cancer substantially. Although the interventional database for dietary endpoint studies in primary as well as in secondary prevention is far from satisfactory, it is highly plausible that the type of nutrition will maintain its importance after a cardiovascular event has occurred. This chapter will review epidemiologic studies, prospective observational cohort studies, metabolic and clinical interventional studies that guide our recommendation for the best possible nutrition in patients at risk for or with established cardiovascular disease. This includes the quantitative and the qualitative aspect of nutrition as well as drinks.

2.1 Overweight and Obesity as Risk Factors for Heart Disease

Overweight and obesity are increasingly common in modern industrialized countries. The relationship between body size and body weight is expressed in the body-mass index (BMI), calculated as weight in kilograms divided by the square of height in meters. A BMI of 25–29.9 is generally referred to as “overweight” and a BMI of≥30 as “obesity.”

H. Gohlke
Chefarzt Abt. Klinische Kardiologie II, Herz-Zentrum, Bad Krozingen, D-79189, Germany
e-mail: helmut.gohlke@herzzentrum.de

J. Niebauer (ed.), *Cardiac Rehabilitation Manual*,
DOI: 10.1007/978-1-84882-794-3_2, © Springer-Verlag London Limited 2011

Only a third of the population in Europe or in the USA has a desirable BMI of less than 25.[36,60]

To evaluate the importance of overweight and obesity for life expectancy more than one million adults in the United States were analyzed in a prospective study, with more than 200,000 deaths occurring during 14 years of follow-up. The relationship between BMI and the risk of death from all causes in four subgroups categorized according to smoking status and history of disease was examined. The prognostically most favorable BMI range in healthy male never smokers was between 23.5 and 24.9, in females between 22.0 and 23.4.

The database was used to assess the relative risk between BMI and mortality. To avoid confounding, Cox models were used for exact age at enrollment, level of education and physical activity, alcohol use, marital status, current use of aspirin, a crude index of fat consumption, vegetable consumption, and (in women) the use of estrogen replacement therapy.

Among subjects with the highest BMI, white men and women had a relative risk of death of 2.58 and 2.00, respectively, compared with those with a BMI of 23.5–24.9. A high BMI was most predictive of death from CVD, especially in men. Heavier men and women in all age groups had an increased risk of death. In black men and women, however, with the highest BMI the risk of death was not significantly increased.[4]

Obesity with a BMI of more than 30.0 favors the early development of atherosclerosis, type 2 diabetes, hypertension, coronary artery disease, acute coronary syndromes and heart failure and shortens life expectancy. A 35-year-old Caucasian man with a BMI of over 35 has a loss of life expectancy of 10 years. Weight gain during adulthood is also a strong and independent risk factor for premature cardiovascular death.

In patients with established coronary disease, obesity was associated with major adverse cardiovascular events (MACE) after adjusting for significant confounders in men but not in women. Further categorization of BMI showed a J-shaped association between BMI and MACE in men, and no association in women.[80]

Obese patients with acute coronary syndrome had a more favorable course after an event; however, the event had occurred 7 years earlier compared to non-obese individuals.[3]

BMI is in itself a strong predictor of overall mortality both above and below the apparent optimum of about 22.5–25 kg/m². The progressive excess mortality above this range is due mainly to vascular disease and is probably largely causal. At 30–35 kg/m², median survival is reduced by 2–4 years; at 40–45 kg/m², it is reduced by 8–10 years.

The increased risk for CHD through excess body weight in most population-based studies may be mediated partly through its impact on individual risk factors such as hypertension, diabetes, and dyslipidemia.[80] Obesity however also results in reduced nitric oxide bioavailability, increased vascular tone, arterial stiffening, increased systolic and pulse pressures, and an overall atherogenic vascular phenotype.

Additional independent mechanisms may include chronic oxidative stress, local activation of the renin–angiotensin system, and a low-grade inflammatory state; the latter two may have their origin in the abdominal visceral fatty tissue.[22]

2.1.1 Abdominal Obesity

An increased waist circumference has been recognized as an additional – possibly independent – risk factor for myocardial infarction and may be present despite a normal BMI.[60]

In the EPIC-Study[60] waist circumference was measured either at the narrowest circumference of the torso or at the midpoint between the lower ribs and the iliac crest.[81] Hip circumference was measured horizontally at the level of the largest lateral extension of the hips or over the buttocks.

The association of BMI, waist circumference, and waist-to-hip ratio with the risk of death was examined among more than 350,000 European subjects who had no major chronic diseases. General as well as abdominal adiposity were associated with the risk of death. The data support the use of waist circumference or waist-to-hip ratio in addition to BMI for assessment of the risk of death, particularly among persons with a lower BMI.[60]

The risk for metabolic diseases is increased with a waist circumference of greater than 80 cm in women and 94 cm in men. Persons with abdominal obesity (the android pattern) are in a proinflammatory, prodiabetic and prothrombogenic state. Visceral fatty tissue has been recognized as an active endocrine organ playing a central role in lipid and glucose metabolism. It produces a large number of hormones and cytokines involved in the development of metabolic syndrome, diabetes mellitus, and vascular diseases,[22] whereas weight reduction and increasing physical activity improve the adipose tissue function.

2.1.2 Caloric Restriction

Increasing evidence from laboratory animals indicates that caloric restriction profoundly affects the physiological and pathophysiological alterations associated with aging and markedly increases lifespan in several species, including mammals. Although the ability of caloric restriction to prolong the lifespan in humans has not been demonstrated conclusively, it now seems plausible that caloric restriction may attenuate visceral fat accumulation and counteract the deleterious aspects of obesity. The cardio protective effects of short-term caloric restriction are probably mediated by increased production of adiponectin and the associated activation of AMP-activated protein kinase.[68]

Caloric restriction has also cardiac-specific effects that ameliorate aging-associated changes in diastolic function. These beneficial effects on cardiac function might be mediated by the effect of caloric restriction on blood pressure, systemic inflammation, and myocardial fibrosis.

Recent studies show that prolonged caloric restriction in obese type 2 diabetes patients decreases BMI and improves glucoregulation associated with decreased myocardial triglyceride content and improved diastolic heart function.[23]

2.1.3 Weight Loss

If weight loss is intended, the daily caloric intake should be reduced by 500–800 kcal and physical activity should be increased, which will lead to a reduction of 1 kg of weight per 14 days. Mediterranean and low-carbohydrate diets may be effective alternatives to low-fat diets. In a recent trial in moderately obese subjects, the low-carbohydrate diet had more favorable effects on lipids and the Mediterranean diet lead to a better glycemic control which suggest that personal preferences and metabolic considerations might allow

individualized tailoring of dietary interventions. The mean weight loss over 2 years was between 3.3 and 5.5 kg with a plateau reached after 1 year suggesting that the lifestyle modification was difficult to maintain.[66]

Diet induced weight reduction over 2 years may reverse to some degree the atherosclerotic process; weight reduction was associated with a significant regression of measurable carotid vascular wall volume in a 3-dimensional echo-study. The effect was similar in low-fat, Mediterranean, or low-carbohydrate strategies and correlated with the weight loss-induced decline in blood pressure.[67]

How the caloric restriction can be implemented in the individual overweight or obese patient remains an unresolved problem the discussion of which exceeds the scope of this chapter. A recent trial compared five ways of providing support for lifestyle modification. High-frequency telephone contact with a dietitian led to the same weight loss as a frequent personal contact and more weight loss than with low-frequency contact or e-mail contact or no contact at all.[11]

The findings illustrate that a frequent contact is necessary to keep up the motivation for healthy lifestyle changes in patients trying to lose weight.

A recent comparison of weight loss diets with different compositions of fat, protein, and carbohydrates showed that the average weight loss over 2 years was similar (about 4 kg) in the low fat average protein group, in low fat high protein group, in high fat average protein, and in the high fat high protein group – and was altogether somewhat disappointing. Thus the composition of the diet was of less importance than the attendance of the participants in the weight counseling sessions. Behavioral factors and motivation for change appear to be of greater importance for weight loss than macronutrient composition of the diet.[63]

It has also been suggested that an individual approach to the societal problem of obesity is bound to fail, because obesity is favored by societal conditions.[30]

Network phenomena appear to be relevant to the biologic and behavioral trait of obesity, and obesity appears to spread through social ties. The distribution of overweight in the community bears similarities to the spread of an infectious disease. These findings have implications for clinical and public health intervention.

The increasing prevalence of overweight in our communities is a threat particularly to the health of the children in our society. Communities have to get engaged to achieve a beneficial impact and early results of such endeavors appear promising.[62]

A general lack of exercise is certainly one important component of this problem. The average American is spending in his free time 150 h/month in front of the TV set, i.e., 5 h/day.

2.2 Individual Components of the Diet

Several individual components of the diet are of particular metabolic importance, although for the patient a dietary pattern rather than picking individual components should guide the eating habits.

2.2.1 Intake of Total Fats vs. Unsaturated Fats

The consumption of fat as a risk modifying factor has been debated since the early results of the Seven Countries Study. Probably the largest and most detailed analysis of the effects of fat consumption was performed over 14 years among more than 80,000 women in the Nurses' Health Study cohort.[56]

Higher intakes of trans-fat and, to a smaller extent, saturated fat were associated with increased risk, whereas higher intakes of non hydrogenated polyunsaturated and monounsaturated fats and olive oil were associated with decreased risk. Because of opposing effects of different types of fat, total fat as percentage of energy is not appreciably associated with CHD risk.

Fat consumption has therefore to be looked at in a more differentiated manner.

From the preventive aspect saturated fatty acids, trans-fatty acids and cholesterol consumption should be reduced and increased fish consumption recommended. Trans-fatty acids are of greater importance in the USA and are associated with a markedly increased risk for CHD.[74]

In the Nurses' Health study the quartile of women with the highest erythrocyte trans-fat content – as a validated indicator of trans-fat consumption – had a relative risk of 3.3 for CHD after adjustment for the usual risk factors.[56] As compared with the consumption of an equivalent amount of calories from saturated or cis-unsaturated fats, the consumption of trans-fatty acids raises levels of low-density lipoprotein (LDL) cholesterol, reduces levels of high-density lipoprotein (HDL) cholesterol, and increases the ratio of total cholesterol to HDL cholesterol, a powerful predictor of the risk of CHD. Trans-fats also increase the blood levels of triglycerides as compared with the intake of other fats, increase levels of Lp(a) lipoprotein, and reduce the particle size of LDL cholesterol all of which are considered unfavorable for the CHD risk.[47]

Trans-fatty acids are considered so important in the USA that the FDA has required that nutrition labels for all conventional foods and supplements must indicate the content of trans-fatty acids, and their use was prohibited in the state of New York in 2007.[57] Also in Denmark the content of trans-fats in foods has to be below 2%.

The consumption of saturated fatty acids decreases the anti-inflammatory activity of HDL-lipoprotein and inhibits endothelial function, whereas the consumption of polyunsaturated fatty acids improves the anti-inflammatory activity of HDL-lipoprotein and endothelial function.[53]

A breakfast rich in saturated fats increases the CV reaction in response to psychological stress in healthy young adults, whereas the addition of walnuts to a fat rich meal improves acutely the flow-dependent endothelial dilatation.

The predominant consumption of a western diet characterized by frequent use of red and processed meat, fried foods, soft drinks, refined cereal products and a low consumption of fruits, vegetables, fish and whole grain products is associated with an increased rate of the metabolic syndrome whereas dairy consumption provides some protection. Also the consumption of a Mediterranean diet enriched with 30 g mixed nuts per day decreased the prevalence of a metabolic syndrome compared with a low fat control diet.[64]

2.2.2 N-3-Fatty Acids

Large long term observational studies in women in the Nurses' Health Study[25] and in men in the Physicians' Health Study and the Zutphen Study,[73] in randomized clinical trials after myocardial infarction,[21] and experimental studies have evaluated the effects of fish and n–3 fatty acid consumption on fatal CHD and sudden cardiac death (SCD).

These different studies provide strong concordant evidence that modest consumption of fish or fish oil, reduces risk of coronary death and total mortality significantly and may favorably affect other clinical outcomes. Intake of 250 mg/day of EPA and DHA appears sufficient for primary prevention with little additional benefit with higher intakes. An omega-3-index has been proposed which describes the percentage of EPA + DHA of total fatty acids measured in red blood cells. An omega-3 index of >8% is associated with 90% less risk for sudden cardiac death, as compared to an omega-3 index of<4%; the index could be used as a benchmark for supplementation of omega-3-fatty acids.[79] However, interventional studies are necessary to confirm this percentage as a treatment goal.

The concordance of findings from different studies also suggests that effects of fish or fish oil on CHD death and SCD do not vary depending on presence or absence of established CHD. The evidence is strong and consistent, and the magnitude of this effect is considerable. Because more than one-half of all CHD deaths and two-thirds of SCD occur among individuals without recognized heart disease, modest consumption of fish or fish oil, together with smoking cessation and regular moderate physical activity, should be among the first-line lifestyle modifications for prevention of CHD death and SCD.

In the Health Professionals Follow-up Study, the associations between different patterns of intake of seafood and plant PUFAs and incident CHD among 45,722 men were investigated over the course of 14 years. N-3 PUFAs from both seafood and plant sources may reduce CHD risk, with little apparent influence from background n-6 PUFA intake. The alpha-linolenic acid content in selected plant oils, nuts and seeds is shown in Table. 2.1.

Plant-based n-3 PUFAs may particularly reduce CHD risk when seafood-based n-3 PUFA intake is low, which has implications for populations with low consumption or availability of fatty fish.[46]

Table 2.1 Alpha-linolenic acid content in selected plant oils, nuts and seeds estimated daily requirements 1.3–2.7 g

	Alpha-linolenic acid content; g/tablespoon
Flaxseed oil	8.5
Flaxseed	2.2
Walnut oil	1.4
Canola oil	1.3
Soja oil	0.9
Walnuts	0.7
Olive oil	0.1

The mechanisms underlying the protective effects of marine omega-3 fatty acids are poorly under-stood. Telomere length is an emerging marker of biological age. Telomeres are tandem repeat DNA sequences (TTAGGG)n that form a protective cap at the ends of eukaryotic chromosomes. Among patients with coronary artery disease, there was an inverse relationship between baseline blood levels of marine omega-3 fatty acids and the rate of telomere shortening over 5 years, suggesting that this might be one mechanism by which n-3 PUFAs might have a protective effect.[16]

2.2.3 Omega-6[n-6]-Fatty Acids

Dietary recommendations for omega-6 polyunsaturated fatty acids (PUFAs) traditionally focused on the prevention of essential fatty acid deficiency. But n-6-PUFAs are also increasingly seen as competitors for n-3 PUFAs whose benefit in reducing CV-disease is well established. Therefore it appears important to define "optimal" intakes of n-6-PUFAs to reduce risk for chronic disease, particularly CHD. The American Heart Association has recently summarized the current evidence on the consumption of omega-6 PUFAs, particularly linolenic acid (LA), and CHD risk. LA an 18-carbon fatty acid with two double bonds (18:2 omega-6), is the primary dietary omega-6 PUFA.[24]

The Third Adult Treatment Panel of the National Cholesterol Education Program recommends PUFA consumption up to 10% of daily calories, noting that there are no large populations that have consumed large quantities of polyunsaturated fatty acids for long periods; the European Commission recommends 4% to 8%.

Randomized trials in humans have shown reduced CHD risk with omega-6 PUFA intakes of 11–21% of energy for up to 11 years with no evidence of harm.

Advice to reduce omega-6 PUFA intakes is usually given in the intention to lower the ratio of dietary omega-6 to omega-3 PUFAs. Although increasing omega-3 PUFA consumption and tissue levels reduce the risk for CHD, it is unlikely that decreasing omega-6 levels will do the same.

The data rather suggest that higher intakes appear to be safe and may be even more beneficial (as part of a low–saturated-fat, low-cholesterol diet) but seen in the context of other prudent lifestyle and dietary recommendations.

2.2.4 Olive Oil

Olive oil has been associated with longevity and good cardiovascular health since the seven-countries study and is an essential part of the Mediterranean diet. Olive oil contains aside from monounsaturated oleic acid also micronutrients like phenolic components, which have antioxidative, anti-inflammatory and antithrombotic properties. The long term consumption of olive oil improves endothelial function in persons with hypercholesterolemia decreases oxidability of LDL-cholesterol in vitro and increases the antioxidative capacity of human plasma.[17] The latter can be shown after only short term intake of olive

oil; the higher the polyphenol content of the olive oil the stronger the increase in HDL, the decrease of the total-cholesterol/HDL-cholesterol-ratio and the decrease of the oxidative stress-indicators.[8] The ratio of monounsaturated to saturated fatty acids in the diet is of prognostic importance. The German, the European and the American cardiac societies consider olive oil as a favorable component of the diet.

2.2.5 Fruits and Vegetables

The role of fruits and vegetables for the prevention of ischemic events has been examined in the Nurses' Health Study and the Health Professionals study. 84,251 women 34–59 years of age who were followed for 14 years in the Nurses' Health Study and 42,148 men 40–75 years of age who were followed for 8 years in the Health Professionals' Follow-Up Study were free of diagnosed cardiovascular disease, cancer, and diabetes at baseline. Participants in both studies completed mailed questionnaires about medical history, health behaviors, and occurrence of cardiovascular and other outcomes every 2 years.

After adjustment for standard cardiovascular risk factors, persons in the highest quintile of fruit and vegetable intake had a 20% lower relative risk for coronary heart disease and a 31% lower risk for ischemic stroke[29] compared with those in the lowest quintile of intake. Each 1-serving/day increase in intake of fruits or vegetables was associated with a 4–6% lower risk for coronary heart disease or ischemic stroke. Green leafy vegetables and vitamin C–rich fruits and vegetables contributed most to the apparent protective effect of total fruit and vegetable intake. The optimal effect was reached with five servings per day, which is the current recommendation.

In the European Prospective Investigation into Cancer and Nutrition on more than 19,000 men and women aged 45–79 each 50 g consumption of fruit or vegetable decreased total mortality by 20%.[31]

A recent multicentre study from Europe[54] extends these findings to a diabetic population, where intake of vegetables, legumes, and fruits was associated with reduced risks of all-cause and CVD mortality. The findings support the current state of evidence from general population studies suggesting that the protective potential of vegetable and fruit intake is also seen in diabetes patients.

2.2.6 Whole Grain Products

Although whole grain products are metabolically favorable their prognostic implications have not been adequately examined. Whole grain products decrease total cholesterol and LDL-cholesterol by about 18%, decrease postprandial glucose levels, decrease the risk for type 2 diabetes mellitus and improve insulin sensitivity in overweight and obese adults. Their influence on bodyweight however remains unresolved. There are no prospective studies evaluating the effect of whole grain products or diets on coronary death or on the occurrence of coronary artery disease. A retrospective analysis of ten American and

European studies found consumption of dietary fiber from cereals and fruits inversely associated with risk of coronary heart disease: for each 10 g cereal or fruit fiber intake, risk reductions of 10% and 16% respectively for all coronary events were observed and 25% and 30% risk reductions respectively for deaths; there was however no risk reductions for vegetable fiber intake. The results were similar for men and women.[58]

Whole grain products for breakfast could prevent the occurrence of heart failure, as a recent observational sub-study on 21,000 physicians over more than 20 years from the British Physician's Health Study suggested. Whether this was achieved via prevention of hypertension and/or myocardial infarction is unclear at present.[12]

2.2.7 Snacks and Sweets

2.2.7.1 Nuts and Almonds

The diet of the coronary patient is characterized by quite a few restrictions and some degree of fat and cholesterol reduction. Therefore the addition of snacks to the diet is often more than welcome, particularly if these snacks have a beneficial effect on the course of the disease or at least modify risk factors in a favorable way or improve endothelial function.

Tree-Nuts, pea nuts and almonds have been thoroughly analyzed with respect to their effect on prognosis and lipid profile of persons with hypercholesterolemia.

Observations from the Adventist Health Study have shown that frequent consumption of nuts is associated with a substantial, independent reduction in the risk of myocardial infarction and death from ischemic heart disease. Similarly, in the Nurses' Health Study nut consumption was associated with reduced CHD risk. This inverse association between nut consumption and CHD risk has been consistently found in several studies. In the prospective Physicians' Health Study, an inverse association between nut consumption and total coronary heart disease death was primarily due to a reduction in the risk of sudden cardiac death.

The average nut consumption in the Adventists' and the Nurses' Health Study was about 20 g/day – a handful. In a randomized nutritional study, moderate quantities of walnuts (84 g/day) within a cholesterol-lowering diet favorably modified the lipoprotein profile in normal men and decreased serum levels of LDL-cholesterol by 16% if the intake of total dietary fat and calories was maintained. The ratio of LDL cholesterol to HDL cholesterol was also lowered by the walnut diet.

The beneficial effect of nuts on prognosis is plausible. Nuts are rich in monounsaturated and polyunsaturated fatty acids, which makes them a palatable choice of healthy fats. Monounsaturated fats may contribute to decreased CHD risk by amelioration of lipid profile, by reducing postprandial triglyceride concentrations, and by decreasing soluble inflammatory adhesion molecules in patients with hypercholesterolemia. Moreover, the relatively high arginine content of nuts has been suggested as one of the potential biologic mechanisms for their cardioprotective effect, because consumption of arginine-rich foods is associated with lower CRP levels.[35]

Also, almonds used as supplements in the diet of hyperlipidemic subjects significantly reduce coronary heart disease risk factors: 73 g of almonds produced a significant 9.4% reduction of LDL-Cholesterol, a 12% reduction of the LDL: HDL-ratio, a 7.8% reduction of Lipoprotein (a) and a reduction of oxidized LDL concentrations by 14.0% – all significant and most likely beneficial for the course of the disease.

Also a macadamia nut-based diet high in monounsaturated fat has potentially beneficial effects on cholesterol and low density lipoprotein cholesterol levels when compared with a typical American diet. These changes are probably a result of the nonfat (protein and fiber) as well as the monounsaturated fatty acid components of the nut but other additive effects of the numerous bioactive constituents found in almonds may contribute to this effect.

A traditional Mediterranean diet enriched with nuts in a weight reduction program enhanced – as mentioned above – the reversion of Metabolic Syndrome by 30% compared with the control diet group.[64]

In addition, the consumption of peanuts and other nuts is significantly associated with a lower risk of gallstone disease – a welcomed side effect in persons with increased cholesterol levels. A recent review found the prognostic effects of nut and peanut studies somewhat greater than expected on the basis of the magnitude of the blood cholesterol-lowering seen from the diet. Thus, in addition to a favorable fatty acid profile, nuts and peanuts may contain other bioactive compounds that could contribute to their multiple cardiovascular benefits. Other macronutrients include plant protein and fiber; micronutrients including potassium, calcium, magnesium, and tocopherols; and phytochemicals such as phytosterols, phenolic compounds, resveratrol, and arginine. Nuts and peanuts are food sources that are a composite of numerous cardioprotective nutrients and if routinely incorporated in a healthy diet, the population risk of CHD would therefore be expected to decrease markedly.[35,43]

2.2.7.2 Chocolate

In the sixteenth century, Aztec Emperor Montezuma was a keen admirer of cocoa, calling it a “divine drink, which builds up resistance and fights fatigue. A cup of this precious drink permits a man to walk for a whole day without food” (Hernán Cortés, 1519). In the language of the Aztecs, this drink was called chocolatl. With the discovery of the New World, cocoa came to Europe in the sixteenth century. Also today the consumption of chocolate is often associated with or followed by an intense feeling of pleasure and gratification, where the desire of repetitive consumption is often difficult to resist (personal experience and unpublished observation). The total chocolate consumption in Germany is approximately 11.4 kg per person and year and only second to the Swiss who consume 11.7 kg/year however including the sales to tourists[82] (Fig. 2.1).

Because of its high caloric content (500–600 kcal/100 g) chocolate consumption may be an important aspect of overall energy balance in men as well as in women. It could contribute to 7–8 kg overweight/year in Germany if chocolate was used as an add-on to a

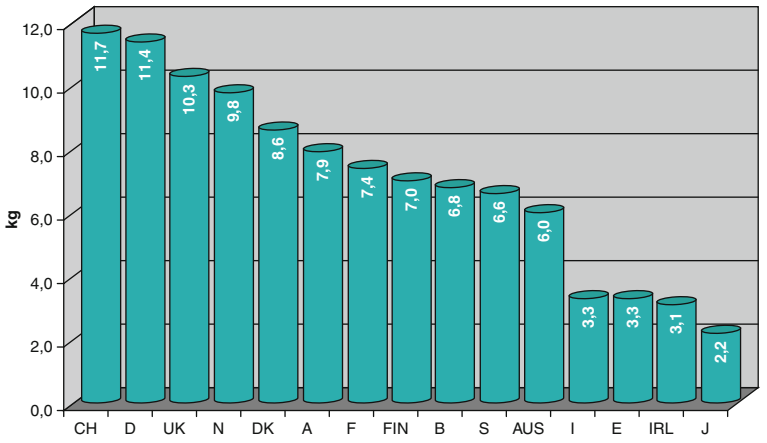

Fig. 2.1 Per capita annual chocolate consumption in kg in different countries (Swiss 2009, other countries 2008)[82]

normocaloric diet. The high fat and sugar content limit its use in a diet with the aim to minimize risk factors. Yet regular cocoa consumption – an essential ingredient for chocolate production – prevented high blood pressure in Kuna Indians of Panama. Recent investigations have shown, that flavanol-rich dark chocolate induces coronary vasodilation, improves coronary vascular function, and decreases platelet adhesion even in a short term experiment 2 h after consumption. These immediate beneficial effects were paralleled by a significant reduction of serum oxidative stress and were positively correlated with changes in serum epicatechin concentration.[18] The possible beneficial effects of cocoa on cardiovascular health by activation of nitric oxide (NO) and influencing antioxidant, anti-inflammatory, and antiplatelet effects, which in turn might improve endothelial function, lipid levels, blood pressure, insulin resistance, and eventually clinical outcome have been reviewed recently.[7] Cocoa is contained in dark chocolate rather than in milk chocolate. The content of the bitter tasting flavanols is responsible for the vasodilating and antioxidative effects of the chocolate, whereby epicatechin is probably the dominant, if not the sole, mediator. Interestingly the procyanidins, which are polymerized chains of epicatechin and catechin, and which represent the vast majority of the polyphenol content of cocoa are also present in red wine and presumably responsible for the beneficial vascular effects. Unfortunately – or rather on purpose – in the regular production process of the chocolate, the bitter tasting flavanols are largely eliminated by a process called "dutching." Accordingly, the liberal consumption of chocolate as a preventive measure is probably limited by the bitter taste and in the regular chocolate these prognostic beneficial effects have been eliminated to better please the taste of the majority of the consumers (Fig. 2.2).

Thus, chocolate with high flavanol content has beneficial effects on endothelial function and can probably be enjoyed without untoward effects as a snack by persons who are fond of bitter chocolate. The bitter flavor will probably prevent any excessive caloric intake.

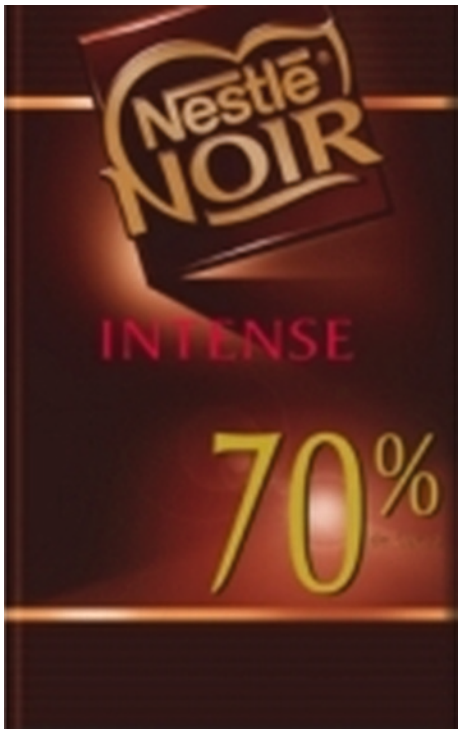

Fig. 2.2 A 70% Chocolate which improves endothelial function[18]

2.2.8 Non Pharmacological Decrease of the Postprandial Rise in Glucose

The postprandial rise in glucose appears to be of some importance for the development of diabetes and cardiovascular events; it also correlates with indicators of oxidative stress. A moderate (20 g of alcohol) "aperitif" results in a decrease of the postprandial rise in glucose, two table spoons of vinegar e.g., with salad before a meal with a high glycemic index have a similar effect (Fig. 2.3) as well as almonds, walnuts or peanuts (Fig. 2.4).

These components – which are part of the Mediterranean diet – can result in a noticeable decrease of postprandial lipid and glucose levels.[55]

2.2.9 Glycemic Index

The glycemic index (GI) is an empiric measure describing the influence of carbohydrates on glucose-insuline-homeostasis based on the extent to which they raise blood glucose levels 2 h after their consumption. Less refined carbohydrates with a high fiber content have a lower GI.

Food consumption with a low GI decreases postprandial glucose, insulin levels, and triglycerides, improves the total cholesterol/HDL cholesterol ratio, may support the

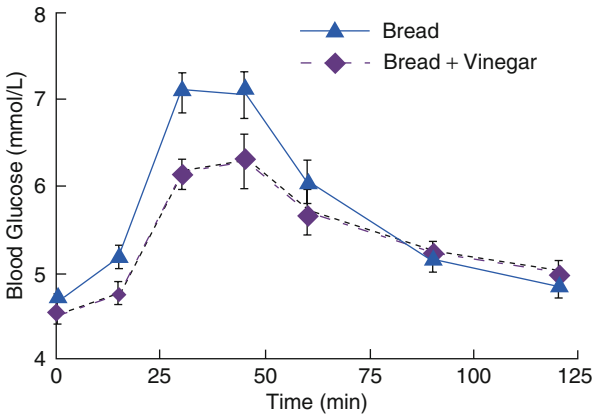

Fig. 2.3 Vinegar Reduces Post-Prandial Glucose The addition of 2 tablespoons of vinegar to 2 slices of white bread significantly reduced the post-prandial glucose increase; modified from *O'Keefe et al. 2008*[55]

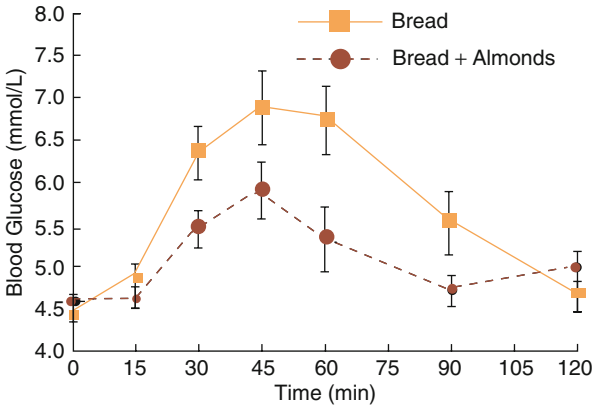

Fig. 2.4 Almonds Reduce Post-Prandial Glucose The post-prandial increase in the area under the curve forglucose was reduced by 58% when 90 g of almonds were added to a high glycemic index meal ($p < 0.01$) modified from *O'Keefe et al. 2008*[55]

decrease of body weight and possibly has – via this pathway – a favorable effect on the development of diabetes and CHD. This concept may be particularly useful in type III hyperlipoproteinemia.[61] However, whether these improvements translate into improved clinical outcomes is not known.

In randomized trials, reduced-glycemic-index diets have not resulted in increased weight loss beyond that explained by caloric restriction. In some aspects low-glycemic-index diets have features resembling the Mediterranean diet.[43]

2.2.10 Mediterranean Diet

The concept of cardiovascular prevention by nutrition has moved from focusing on individual components of a diet to emphasizing a food pattern. The Mediterranean diet has been favored since the Seven Countries Study as promoting longevity and good cardiovascular health.

In general, the type of diet is part of the lifestyle and there may be some residual bias when correlating a diet with the occurrence of cardiovascular events. The Mediterranean Lifestyle used to be more relaxed compared to the central European or American lifestyle.

2.2.10.1 Prognostic Benefits

In the mean time however the components of the Mediterranean diet have been analyzed in many countries and have been correlated with events in more than half a million persons. The Mediterranean diet is characterized by a high proportion of vegetables, legumes, fruits and cereals (primarily unprocessed), frequent fish consumption, less dairy products, rarely meat and a moderate consumption of alcoholic beverages mostly as wine and preferably with meals, a small amount of saturated fatty acids, but a high proportion of unsaturated fatty acids, particularly olive oil.

In middle aged persons, there was a significant inverse relation between the degree of compliance with the Mediterranean diet (as evaluated by a score) and mortality.[75] Because this score has been used extensively and repeatedly with only minor modifications in different studies and different countries it will be outlined in some detail here; this score can also be used as a checklist in advising patients to change their diet into the direction of a Mediterranean diet:

The traditional Mediterranean diet score includes nine components and results in values from 0 to 9 points (minimum to maximum conformity). One point each is given for intake at or above the gender-specific median intake for the six components considered to be healthy (fatty acid ratio, legumes, grains, fruits, vegetables [excluding potatoes] or fish), and one point if the consumption of the items considered to be less healthy (meat and dairy products) was below the gender specific median. One point is given for alcohol consumption within a specified range (5–25 g/day for women; 10–50 g/day for men).

Items of the Mediterranean Diet Score: one point for above (high intake) or below (low intake) the age and gender adjusted consumption of the corresponding nutritional item (Fig. 2.5).

1. High ratio of monounsaturated: saturated fatty acids,
2. High intake of legumes
3. High intake of grains
4. High intake of fruit and nuts
5. High intake of vegetables
6. High intake of fish
7. Low intake of meat and meat products
8. Low intake of milk and dairy products
9. Moderate consumption of alcohol (10–50 g/day for men, 5–25 g/day for women)

If participants met all the characteristics of the Mediterranean diet, their score was the highest (nine points), reflecting maximal conformance with a Mediterranean diet and if they met none of the characteristics, the score was zero reflecting minimal or no conformity with a Mediterranean diet.

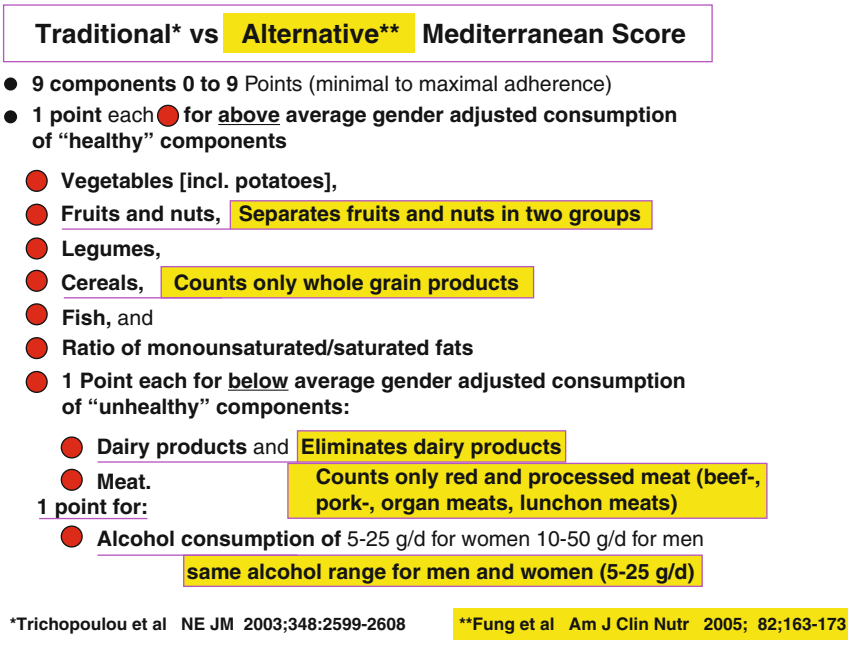

Fig. 2.5 The traditional and the alternative (marked with yellow background) Mediterranean Diet Score

In two cohorts of elderly persons the life prolonging effects of the Mediterranean diet could be observed:

In the HALE-Project among 2,339 apparently healthy men and women, aged 70–90 years, adherence to a Mediterranean diet was associated with a 23% lower rate of all-causes mortality.[34]

In the EPIC-Study of more than 74,000 above 60-year old European persons without coronary heart disease, stroke, or cancer at enrolment a two unit increment in the modified Mediterranean diet score was associated with a statistically significant reduction of overall mortality of 8%.[76]

Also in a prospective observational study of more than 380,000 Americans (age range 50–71 years), a 20% reduced total mortality and CV mortality could be seen as well as a 12–17% reduced cancer mortality in men and women who showed with 6–9 points a good conformity with a Mediterranean diet compared to persons with a score of 0–3. This relationship was seen in smokers and never smokers alike.[44] A similar Mediterranean diet score was used for nutritional evaluation in the HALE-Project,[34] the EPIC-Study[76] and in the American Study.[44] Thus the database for the primary preventive effects of the Mediterranean diet has been greatly strengthened.

The beneficial effects of the Mediterranean diet were recently confirmed in more than 74,500 women 38–63 years of age, without a history of cardiovascular disease and diabetes who were followed up from 1984 to 2004 in the Nurses' Health Study.

The authors used the Alternate Mediterranean Diet Score from self-reported dietary data collected through validated food frequency questionnaires administered six times

between 1984 and 2002. During 20 years of follow-up, 2,391 incident cases of CHD, 1,763 incident cases of stroke, and 1,077 cardiovascular disease deaths (fatal CHD and strokes combined) were ascertained. Women in the top Alternate Mediterranean Diet Score quintile were at 29% lower risk for CHD and 13% lower risk for stroke compared with those in the bottom quintile. Cardiovascular disease mortality was 39% lower among women in the top quintile of the Alternate Mediterranean Diet Score ($p < 0.0001$).[20]

The Lyon-Diet-Heart Study had shown already in 1999 in an interventional study that a strict Mediterranean diet in patients after MI is associated with a 45% reduction of the CV event rate. The Mediterranean diet is a class 1 recommendation (Evidence Level B) in the European Society of Cardiology recommendations for secondary prevention after transmural MI.[78]

This inverse relationship between consumption of "healthy" foods in the sense of the Mediterranean diet and risk of myocardial infarction was basically confirmed in the Interheart Study where dietary patterns were analyzed in patients after MI and controls in 52 countries. Three dietary patterns were identified and labeled as Oriental, Western, and prudent. The "Oriental" pattern had a high loading on tofu and soy and other sauces. The second dietary pattern was labeled "Western" because of its high loading on fried food, salty snacks, and meat intake. The third dietary pattern was labeled "prudent" because of its emphasis on fruit and vegetable intake.

The authors found significant, inverse, and graded associations between the intake of raw vegetables, green leafy vegetables, cooked vegetables, and fruits on the one hand and acute myocardial infarction on the other. Conversely, they observed a positive association between myocardial infarction and the intake of fried foods and salty snacks ($p < 0.001$) and a weaker association between quartiles of meat intake and AMI ($p = 0.08$).[26]

2.2.10.2 Effects of the Mediterranean Diet on Risk Indicators and Risk Factors

The exact mechanisms leading to decreased myocardial infarction, cardiovascular deaths and all cause deaths are not clear, but several indicators of risk such as indicators of inflammation and established CV risk factors are decreased by the Mediterranean diet.

Estruch et al. examined in the randomized controlled PREDIMED-trial the effects of a Mediterranean diet supplemented with 1 L olive oil per week or with 30 g of nuts/day in comparison to a low fat diet in 772 asymptomatic persons 55–80 years of age at high cardiovascular risk.

Compared with a low-fat diet after 3 months, both Mediterranean diets lowered plasma glucose levels, systolic blood pressure, and the cholesterol/HDL- cholesterol ratio. The Mediterranean diet supplemented by olive oil also reduced C-reactive protein levels compared with the low-fat diet.[14]

In the same study the effects of the Mediterranean diet on in vivo lipoprotein oxidation were assessed. After the 3-month interventions, mean oxidized low-density lipoprotein (LDL) levels decreased in the traditional Mediterranean diet group supplemented by virgin olive oil significantly and to a lesser degree also in the group supplemented by nuts – without significant changes in the low-fat diet group. Change in oxidized LDL levels in the

traditional Mediterranean diet virgin olive oil group reached significance vs that of the low-fat group ($p = 0.02$).

A Mediterranean diet supplemented with nuts (30 g/day) or olive oil (135 mL/day), resulted within 3 months in lower blood pressure, fasting blood sugar and inflammatory markers as compared to a low fat diet.[17]

2.2.10.3 Mediterranean Diet and Inflammation

The inflammatory reaction of the body in relation to adherence to the Mediterranean diet was assessed in more than 300 middle-aged male twins using the mentioned diet score. A one-unit absolute difference in the diet score was associated with a 9% (95% CI, 4.5–13.6) lower interleukin-6 level – an established marker of inflammation related to progression of atherosclerotic disease.

Thus reduced systemic inflammation appears to be an important mechanism linking Mediterranean diet to reduced cardiovascular risk.[9] A recent study presented the first in vivo functional evidence to support the hypothesis that inflammation impairs reverse cholesterol transport at numerous steps in the pathway from initial macrophage efflux to HDL acceptor function and the final step of cholesterol flux through liver to bile and feces. The anti-inflammatory effect of the Mediterranean diet could to some degree contribute to its beneficial effects on cardiovascular but possibly also cancer incidence.[42]

However, it is not only the arterial system that benefits from a high intake of plant foods and fish and less red and processed meat: also the risk for venous thromboembolic events is reduced! In a prospective study as part of the Atherosclerosis Risk in Communities (ARIC) Study, almost 15,000 middle-aged adults participating were followed up over 12 years for incident venous thrombo embolism. At baseline the average age of study participants was 54 years. A food frequency questionnaire assessed dietary intake at baseline and after 6 years. The risk of venous thrombo embolism was assessed in quintiles of fruit and vegetable intake.

There was a significant risk reduction of venous thrombo embolism incidence of 40–50% in quintiles 3–5 compared with quintile 1.

Eating fish 1 or more times per week was associated with 30–45% lower incidence of venous thrombo embolism for quintiles 2–5 compared with quintile 1, suggestive of a threshold effect. High intake of red and processed meat (quintile 5) doubled the risk (p trend = 0.02). Hazard ratios were attenuated only slightly after adjustment for factors VIIc and VIIIc and von Willebrand factor.[71]

2.2.10.4 Mediterranean Diet and Diabetes

Considering the components of the Mediterranean diet it may come as no surprise that the Mediterranean diet has a preventive effect for the development of diabetes. In a recent prospective cohort study from Spain a relation between adherence to a Mediterranean diet

and the incidence of diabetes among initially healthy participants (university graduates) could be shown – after adjustment for covariables such as sex, age, years of university education, total energy intake, body mass index, physical activity, sedentary habits, smoking, family history of diabetes, and personal history of hypertension. Participants who adhered closely to a Mediterranean diet had a lower risk of diabetes. The incidence rate ratios in the fully adjusted analyses showed that a two point increase in the score was associated with a 35% relative reduction in the risk of diabetes with a significant inverse linear trend ($p = 0.04$) in the multivariate analysis. A high adherence (7–9 points) was associated with an 80% reduced incidence rate of diabetes compared with a low score of 0–2.[41]Thus the traditional Mediterranean diet may have considerable protective effects against diabetes.

Similar results were obtained in patients after myocardial infarction.[48]

In prospectively obtained data of 8,291 Italian patients with a recent (<3 months) myocardial infarction, who were free of diabetes at baseline the incidence of new-onset diabetes (new diabetes medication or fasting glucose ≥7 mmol/L) and impaired fasting glucose (fasting glucose ≥6.1 mmol/L and <7 mmol/L) were assessed up to 3.5 years. A Mediterranean diet score was assigned according to consumption of cooked and raw vegetables, fruit, fish, and olive oil. Associations of demographic, clinical, and lifestyle risk-factors with incidence of diabetes and impaired fasting glucose were assessed with multivariable Cox proportional hazards regression analysis.

These patients had a 15-fold higher annual incidence rate of impaired fasting glucose and a more than twofold higher incidence rate of diabetes during a mean follow-up of 3.2 years (26,795 person-years) compared with population-based cohorts. Consumption of typical Mediterranean foods, smoking cessation, and prevention of weight gain were associated with a lower risk.

2.2.10.5 Meta Analysis of Mediterranean Diet Studies

In a recent meta analysis the benefits of the Mediterranean diet pattern were evaluated in 514,816 subjects on the basis of 33,576 deaths occurring during the respective observation time. The overall mortality in relation to adherence to a Mediterranean diet showed that a two point increase in the adherence score was significantly associated with a 9% reduced risk of all causes mortality and likewise a 9% reduction on cardiovascular mortality as well as a 6% lower incidence of mortality from cancer. The message from these studies is that it is the completeness of adherence to the Mediterranean diet rather than the consumption of individual components, which is effective in improving the prognosis. Unexpectedly also the incidence of Parkinson's disease and Alzheimer's disease was significantly reduced by 13%.[70]

Thus a greater adherence to a Mediterranean diet is not only associated with a significant reduction in mortality from arterial cardiovascular diseases but also from a reduced incidence of venous thromboembolism. In addition other diseases that are a threat to the well being and quality of life in the later years are decreased: cancer, Parkinson's disease and

Alzheimer's disease – diseases for which no specific strategies of prevention have been established. This makes it easy for the physician to recommend this type of diet to the cardiovascular patient after myocardial infarction: the side effects of this diet are most welcome.[43]

2.2.10.6 Dietary Risk Score (DRS) and Acute Myocardial Infarction (AMI)

The authors from the Interheart Study computed from their data a Dietary Risk Score and observed a graded and positive association between this Dietary Risk Score and risk of AMI. Food items that were considered to be predictive (meat, salty snacks, and fried foods) or protective (fruits and green leafy vegetables, other cooked vegetables, and other raw vegetables) of CVD were used to generate a DRS. The authors used a point system. Compared with the lowest quartile, odds ratios (adjusted for age, sex, and region) varied from 1.29 in the second quartile of Dietary Risk Score to 1.92 in the fourth quartile. The association of the score with AMI varied by region ($p<0.0001$) but was directionally similar in all regions. The Population Attributable Ratio for this score was 30% (95% CI 0.26–0.35) in participants in the INTERHEART Study (Fig. 2.6).

Thus the Interheart Study confirmed in a case control study the relationship obtained from observational studies between healthy (prudent) food intake and lower risk for myocardial infarction. However, randomized interventional studies are necessary to develop

Population Attributable Fraction	
Overall[a]	0.30 (0.26-0.35)
Male[b]	0.28 (0.23-0.33)
Female[b]	0.39 (0.30-0.49)
N. America, W.Europe and Australia[c]	0.30 (0.17-0.42)
Central Europe[c]	0.31 (0.18-0.44)
Middle East[c]	0.28 (0.17-0.40)
Africa[c]	0.10 (-0.14-0.35)
South Asia[c]	0.29 (0.18-0.40)
China[c]	0.18 (0.07-0.29)
Southeast Asia[c]	0.58 (0.45-0.71)
S. America[c]	0.15 (-0.03-0.32)

[a] adjusted for age, sex and regions
[b] adjusted for age and regions
[c] adjusted for age and sex

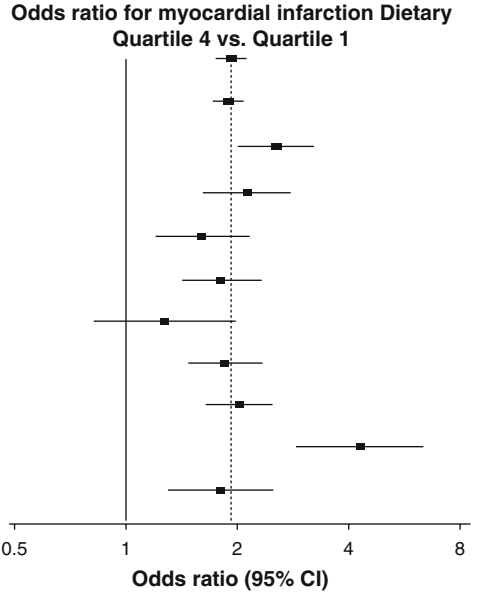

Fig. 2.6 Population attributable risk and odds ratios for acute myocardial infarction associated with dietary risk score. Modified from *Iqbal et al. 2008*[26]

specific dietary recommendation for patients with CV disease and with different metabolic problems. (Figs.2.7–2.10).

2.2.11 Drinks

The fluid requirements of the body vary depending on the environment and the physical activity. The type of fluid preferred to fulfill the requirements depend on tradition and environment.

2.2.11.1 Coffee or Tea Consumption and Cardiovascular Events

Coffee and tea and to a lesser degree chocolate have been the most widely used drinks during the course of the day for decades if not centuries, but their relationship to the risk of coronary disease has been examined only in recent years.

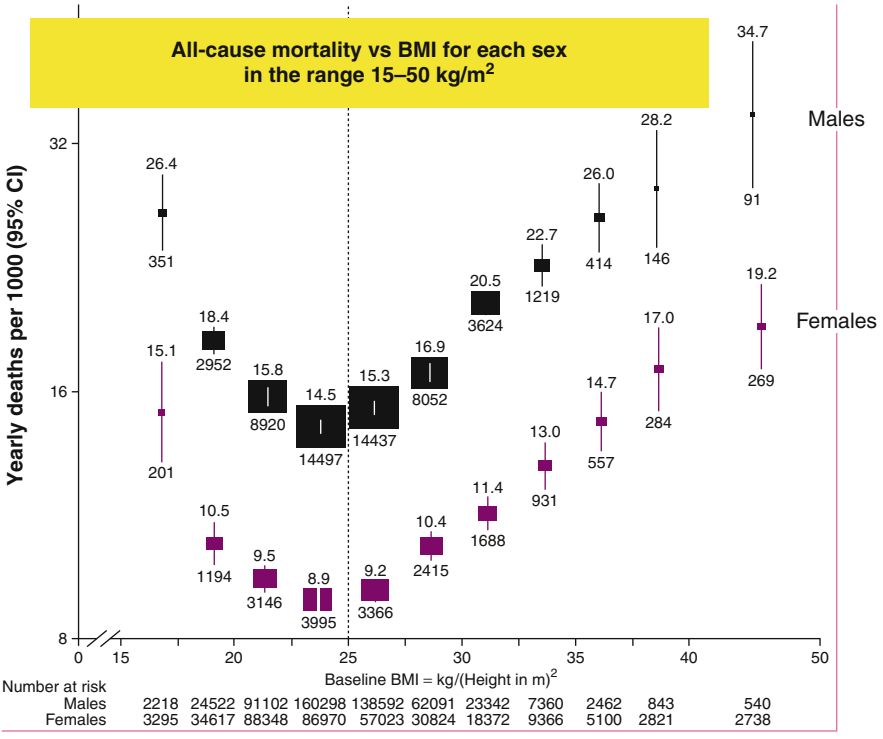

Fig. 2.7 Modified after Whitlock et al. 2009[80]

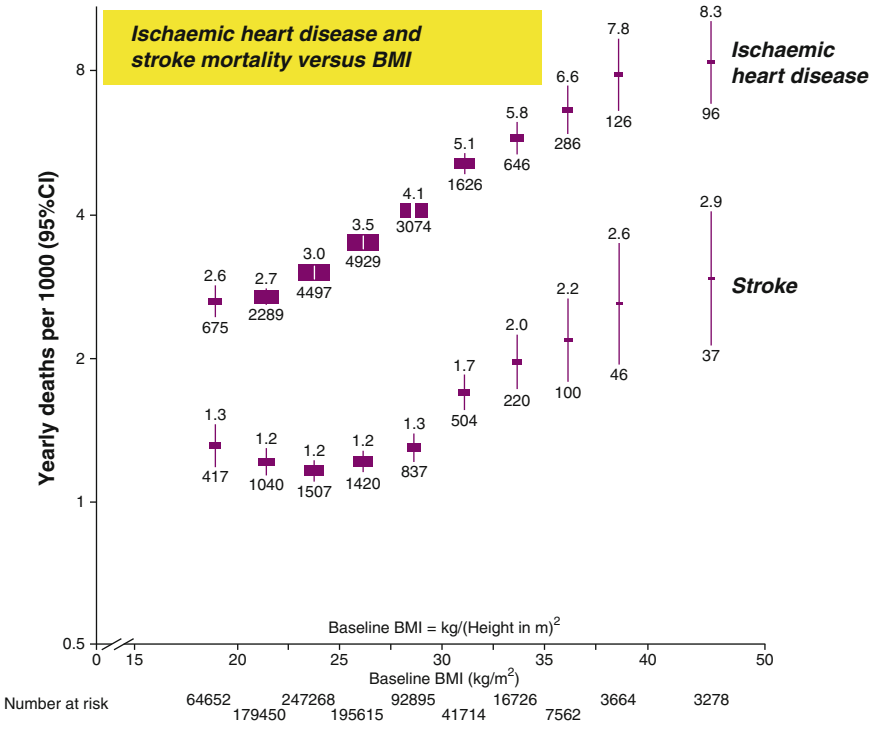

Fig. 2.8 Modified after Whitlock et al. 2009[80]

Fig. 2.9 Key components of the Mediterranean Diet

Fig. 2.10 A glimpse of the mediterranean scenery – a low risk area for cardiovascular disease (here: Isle of Mykonos)

2.2.11.2 Coffee

Coffee consumption has been associated with an increased risk in patients with coronary artery disease. The influence of coffee on cholesterol levels was already examined in 1989. After 9 weeks of coffee consumption boiled coffee increased LDL-cholesterol by 10% whereas filtered coffee showed no difference compared to a "no-coffee" group.[1]

In the Health Professionals' Follow-Up Study almost 42,000 male employees in the hospital (age range 40–75 years) were asked about their coffee consumption, lifestyle and risk factors every 2 years for a total of 12 years. Similarly in the Nurses' Health Study, more than 84,000 nurses in the age range of 30–55 years were asked about their coffee consumption every 2 years for a total of 18 years. In both studies the prevalence of diabetes mellitus was examined: coffee consumption of four to five cups per day reduced the prevalence of diabetes mellitus by 29–30% in males and females similarly after multivariate analysis. In males, even a consumption of coffee of more than six cups per day reduced the risk of diabetes by 46% whereas in females there was no further decrease of the diabetes prevalence beyond the consumption of five cups of coffee.[65]

In a systematic review habituary coffee consumption was associated with a substantial lower risk of type II diabetes which was also observed for decaffeinated coffee in postmenopausal women[59] as well as middle-aged and younger US women.[77]Thus there may be ingredients in the coffee – other than caffeine – that protect from diabetes.

Coffee consumption in two observational studies showed no increased risk for the development of coronary artery disease. The consumption of up to five cups of coffee per

day is without harm for the coronary patient and possibly beneficial by preventing or delaying the occurrence of diabetes – but beware of the sugar and cream!

Coffee consumption decreased the relative risks of stroke across categories of coffee consumption in the more than 83,000 women of the Nurses' Health Study. After adjustment for high blood pressure, hypercholesterolemia, and type 2 diabetes, the relative risk reduction was 43% among never and past smokers (RR for > 4 cups a day versus < 1 cup a month; $p < 0.001$), but not significant among current smokers. Similarly there was a protective effect among non hypercholesteremics (HR 0.77; $p < 0.003$), non diabetics (HR 0.79; $p = 0.009$), and nonhypertensives (HR 0.72; $p = 0.001$). However, no protective effect was seen in women with diabetes, hypertension or hypercholesterolemia, suggesting that the moderate beneficial effects of coffee consumption cannot override the detrimental effects of these important risk factors. The authors also observed a slightly lower risk of stroke in women who drank moderate amounts of decaffeinated coffee (2–3 cups/day vs. < 1 cup/month; HR 0.84; $p = 0.002$) suggesting that components in coffee other than caffeine may be responsible for the potential beneficial effect of coffee on stroke risk.[38]

2.2.11.3 Tea

Tea has traditionally a better image concerning the development of cardiovascular disease. The relationship between tea consumption and mortality after acute myocardial infarction was examined in a prospective study. The self-reported tea consumption in the year before myocardial infarction was associated with a lower mortality after myocardial infarction.[50]

Short-term and long-term black tea consumption has a potential to reverse endothelial dysfunction in patients with coronary artery disease which may partly explain the association between tea consumption and decreased cardiovascular disease events in primary and secondary prevention.[13] The addition of milk however counter-acted the favorable health effects of tea on flow-mediated dilatation.[39] Similarly, green tea consumption is associated with reduced mortality due to all causes and due to cardiovascular disease, but primarily because of a decreased risk of stroke. The hazard ratios of cancer mortality were not significantly different from non tea consumers.[37] Drinking a cup of tea is frequently associated with relaxation and recovery from stress. *Steptoe and coworkers* reported in a double-blind (!) randomized trial that regular drinking of tea is associated with less platelet leucocytes aggregates, platelet monocytes aggregates and platelet neutrophile aggregates. They also found lower post-stress cortisol levels and a stronger subjective feeling of relaxation under the artificial test conditions. The consumption of black tea may have its potential health effects mediated through better recovery from a stress via psycho endocrine and inflammatory mechanisms.[72]

Thus, in summary, coffee has no unfavorable effect on the coronary risk and probably decreases the risk for stroke and diabetes mellitus, and this also applies to decaffeinated coffee. Black tea (without milk) has a favorable effect on the flow-dependent vasodilatation. Tea consumption is associated with decreased mortality after myocardial infarction and green tea reduces the stroke risk, but probably has no effect on the coronary artery risk.

2.2.11.4 Alcohol after Myocardial Infarction

A large number of epidemiological studies of both community, and clinical cohorts have associated moderate alcohol consumption with decreased risk for subsequent cardiovascular morbidity and mortality.[33]

The consumption of wine, particularly of red wine, has been associated in cross-sectional studies with a better prognostic outcome than the consumption of beer.

This was partially attributed to the content of polyphenols and oligomeric procyanidins in red wine, which have favorable effects on endothelial function.[6] Johansen et al. examined in a cross-sectional study the food buying habits of people who buy wine or beer. People buying wine significantly more often also bought olive oil and low fat milk products or low fat meat whereas people buying beer favored sausages, cold cuts and pork. Thus there is a significant potential for social selection bias by just evaluating the type of alcohol consumed.[28]

In the setting after myocardial infarction only the ONSET, the Lyon Diet Heart Study, Survival and Ventricular Enlargement trial and recently the Stockholm Heart Epidemiology Program (SHEEP)[27] have prospectively compared mortality across alcohol consumption categories in survivors of a recent AMI.

The favorable effects of moderate alcohol consumption are reproduced in most of these studies and the possibility of a "social selection bias" has largely been excluded by multivariate analysis, although unknown confounders in the absence of randomized studies are still a possibility. In the SHEEP-Study there was no difference in the beneficial effects between wine and beer consumption and the benefit started already at a low doses of less than 5 g of alcohol per day.[27]

Alcoholic beverages in small amounts – and it is probably the ethanol itself – have anti-inflammatory effects and reduce fibrinogen. In interventional studies, a significant reduction of CRP concentrations and fibrinogen after 3 weeks of diet-controlled consumption of three glasses of beer/day in women or four glasses of beer/day in men have been demonstrated. Moreover, a 4-week consumption of 30 g/day of red wine led to a significant decrease in CRP (21%) in healthy adult men.[14]

Alcohol also increases HDL-Cholesterol, endothelial function, antioxidative effects, and fibrinolysis, and leads to a decrease in plasma viscosity and platelet aggregation. The combination of these effects may explain part of the beneficial effects of alcohol on the incidence of CV events.[33]

Moderate alcohol consumption in primary prevention had a beneficial effect on the incidence of ischemia related heart failure in the Physicians' Health Study,[12] where the authors concluded that modest alcohol consumption may lower the risk of heart failure and that this possible benefit may be mediated through beneficial effects of alcohol on coronary artery disease but the study also showed that moderate alcohol consumption does not prevent nonischemic cardiomyopathy. In women who consume more than two drinks per day[5] and in men who consume more than five drinks per day, the risk of atrial fibrillation increases. A consumption of 1–2 drinks on 3–4 days per week was not associated with increased hazard.[51] Patients with heart failure should avoid alcohol to prevent atrial fibrillation (so-called holiday-heart syndrome) and be abstinent in alcoholic cardiomyopathy to improve prognosis!

Women tolerate less alcohol or have similar benefits at a lower dosage of alcohol, probably because of the lower activity of the gastric alcohol-dehydrogenase. In women the beneficial effects of alcohol consumption on the heart are mitigated by an increase of breast cancer risk.[10]

There are also some dangers of alcohol consumption: alcohol consumption appears to be associated with a higher risk for ischemic stroke among men who consumed >2 drinks per day.[49]

Despite the theoretically favorable effects of alcohol in men each year e.g., in Germany 40,000 persons die from alcohol consumption and 2,000 children are born with alcohol induced malformations.[69] Therefore, a recommendation for alcohol consumption appears at present not sensible and possibly hazardous – although a recent observation from the ARIC-Study showed that people who spontaneously begin consuming alcohol in middle age rarely do so beyond recommended amounts. Those who begin drinking moderately experience a relatively prompt benefit of lower rates of cardiovascular disease morbidity with no change in mortality rates after 4 years.[32]

In nine nationally representative samples of U.S. adults, light and moderate alcohol consumption were inversely associated with CVD mortality, even when compared with lifetime abstainers, but consumption above recommended limits was not.[52]

The findings also support the safety of continued light alcohol consumption among adults who have been able to appropriately regulate the quantity, type, and timing of their alcohol use: thus the decision for alcohol consumption has to be individualized.

2.2.11.5 Soft Drinks

Soft drinks are an American development and represent an important nutritional problem worldwide but particularly in the USA. The problem has been analyzed in several studies. Sugar-sweetened soft drinks contribute 7.1% of total energy intake and represent the largest single food source of calories in the US diet. In children and adolescents beverages now even account for 10–15% of the calories consumed. For each extra can or glass of sugared beverage consumed per day, the likelihood of a child's becoming obese increases by 60%.[2]

The regular consumption of soft drinks has been associated with overweight, the metabolic syndrome, and Diabetes.

The rise of obesity and type 2 diabetes in the United States paralleled the increase in sugar-sweetened soft drink consumption.[40]

In the longitudinal observation of the Nurses' Health Study women consuming one or more sugar-sweetened soft drinks or fruit punch per day had an almost twofold risk to develop a type 2 diabetes compared with those who consumed less than one of these beverages per month.

Similarly regular consumption of sugar-sweetened beverages, i.e., one serving per month vs. two servings per day during 24 years of follow-up was associated with a 35% (CI 7–69%) higher risk of CHD in women, even after other unhealthful lifestyle or dietary factors are accounted for, whereas artificially sweetened beverages were not associated with CHD.[19]

2.3 Concluding Remarks

Thus there is strong evidence supporting a protective cardiovascular effect for intake of vegetables, nuts, mono unsaturated fatty acids and Mediterranean diet as well as high-quality or a "prudent" dietary pattern. There is good evidence supporting a protective effect for intake of fish, Omega-3 fatty acids, whole grains, fruits, and fibers and a low dose of alcohol.

There is indirect evidence for the beneficial effect of a low ratio of saturated/monounsaturated fatty acids mostly through the evidence from the Mediterranean diet.

There are negative associations for a western dietary pattern, trans-fatty acids, and for foods (or drinks) with a high glycemic index or load.

References

1. Bak AA, Grobbee DE. The effect on serum cholesterol levels of coffee brewed by filtering or boiling. *N Engl J Med*. 1989;321:1432-1437.
2. Brownell KD, Frieden TR. Ounces of prevention – the public policy case for taxes on sugared beverages. *New Engl J Med*. 2009;360:1805-1808.
3. Büttner HJ, Mueller C, Gick M, et al. The impact of obesity on mortality in UA/Non-ST-segment elevation myocardial infarction. *Eur Heart J*. 2007;28(14):1694-1701.
4. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *N Engl J Med*. 2003;348:1625-1638.
5. Conen D, Tedrow UB, Cook NR, Moorthy MV, Buring JE, Albert CM. Alcohol consumption and risk of incident atrial fibrillation in women. *JAMA*. 2008;300:2489-2496.
6. Corder R, Mullen W, Khan NQ, et al. Oenology: red wine procyanidins and vascular health. *Nature*. 2006;444:566.
7. Corti R, Flammer AJ, Hollenberg NK, Lüscher TF. Cocoa and Cardiovascular Health. *Circulation*. 2009;119:1433-1441.
8. Covas M-I, Nyyssönen K, Poulsen HE et al for the EUROLIVE Study Group. The effect of polyphenols in olive oil on heart disease risk factors. A randomized trial. *Ann Intern Med*. 2006;145:333-341.
9. Dai J, Miller AH, Bremner JD, et al. Adherence to the Mediterranean Diet Is Inversely Associated With Circulating Interleukin-6 Among Middle-Aged Men. A Twin Study. *Circulation*. 2008;117:169-175.
10. Di Castelnuovo A, Costanzo S, Bagnardi V, Donati MB, Iacoviello L, de Gaetano G. Alcohol dosing and total mortality in men and women. *Arch Intern Med*. 2006;166:2437-2445.
11. Digenio AG, Mancuso JP, Gerber RA, Dvorak RV. Comparison of methods for delivering a lifestyle modification program for obese patients – a randomized trial. *Ann Intern Med*. 2009;150:255-262.
12. Djoussé L, Gaziano JM. Breakfast cereals and risk of heart failure in the physicians' health study. *Arch Intern Med*. 2007;167:2080-2085.
13. Duffy SJ, Keaney JF Jr, Holbrook M, et al. Short- and long-term black tea consumption reverses endothelial dysfunction in patients with coronary artery disease. *Circulation*. 2001;104:151-156.

14. Estruch R, Martinez-Gonzalez MA, Corella D, Salas-Salvado J, Ruiz-Gutierrez V, Covas MI, Fiol M, Gomez-Gracia E, Lopez-Sabater MC, Vinyoles E, Aros F, Conde M, Lahoz C, Lapetra J, Saez G, Ros E, For the PREDIMED Study Investigators. Effects of a mediterranean-style diet on cardiovascular risk factors – a randomized trial. *Ann Intern Med*. 2006;145:1-11.
15. Ezzati M, Vander Hoorn S, Rodgers A, Lopez AD, Mathers CD, Murray CJL, The Comparative Risk Assessment Collaborating Group. Estimates of global and regional potential health gains from reducing multiple major risk factors. *Lancet*. 2003;362:271-280.
16. Farzaneh-Far R, Lin J, Epel ES, Harris WS, Blackburn EH, Whooley MA. Association of marine omega-3 fatty acid levels with telomeric aging in patients with coronary heart disease. *JAMA*. 2010;303(3):250-257.
17. Fitó M, Guxens M, Corella D, Sáez G, Estruch R, de la Torre R, Francés F, Cabezas C, del Carmen M, López-Sabater, Marrugat J, García-Arellano A, Arós F, Ruiz-Gutierrez V, Ros E, Salas-Salvadó J, Fiol M, Solá R, Covas M-I, For the PREDIMED Study Investigators. Effect of a traditional mediterranean diet on lipoprotein oxidation: a randomized controlled trial. *Arch Intern Med*. 2007;167:1195-1203.
18. Flammer AJ, Hermann F, Sudano I, et al. Dark chocolate improves coronary vasomotion and reduces platelet reactivity. *Circulation*. 2007;116:2376-2382.
19. Fung TT, Malik V, Rexrode KM, Manson JAE, Willett WC, Hu FB. Sweetened beverage consumption and risk of coronary heart disease in women. *Am J Clin Nutr*. 2009;89:1-6.
20. Fung TT, Rexrode KM, Mantzoros CS, et al. Mediterranean diet and incidence of and mortality from coronary heart disease and stroke in women. *Circulation*. 2009;119:1093-1100.
21. Gruppo Italiano per lo Studio della Sopravivenza nell'Infarto miocardico. Dietary supplementation with n-3 polyunsaturated PUFAs and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. *Lancet*. 1999;354:447-455.
22. Hajer GR, van Haeften TW, Visseren FLJ. Adipose tissue dysfunction in obesity, diabetes, and vascular diseases. *Eur Heart J*. 2008;29:2959-2971.
23. Hammer S, Snel M, Lamb HJ, et al. Prolonged caloric restriction in obese patients with type 2 diabetes mellitus decreases myocardial triglyceride content and improves myocardial function. *JACC*. 2008;52:1006-1012.
24. Harris WS, Mozaffarian D, Rimm E, et al. Omega-6 fatty acids and risk for cardiovascular disease. a science advisory from the American Heart Association Nutrition Subcommittee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Cardiovascular Nursing; and Council on Epidemiology and Prevention. *Circulation*. 2009;119:902-907.
25. Hu FB, Bronner L, Willett WC, et al. Fish and omega-3 fatty acid intake and risk of coronary heart disease in women. *JAMA*. 2002;287:1815-1821.
26. Iqbal R, Anand S, Ounpuu S, Islam S, Zhang X, Rangarajan S, Chifamba J, Al-Hinai A, Keltai M, Yusuf S, On behalf of the INTERHEART Study Investigators. Dietary patterns and the risk of acute myocardial infarction in 52 countries: results of the INTERHEART Study. *Circulation*. 2008;118:1929-1937.
27. Janszky RL, Ahnve S, Hallqvist J, Bennet AM, Mukamal KJ. Alcohol and long-term prognosis after a first acute myocardial infarction: the SHEEP study. *Eur Heart J*. 2008;29:45-53.
28. Johansen D, Friis K, Skovenborg E, Ml G. Food buying habits of people who buy wine or beer: cross sectional study. *Br Med J*. 2006;332:519-524.
29. Joshipura KJ, Hu FB, Manson JE, et al. The effect of fruit and vegetable intake on risk for coronary heart disease. *Ann Intern Med*. 2001;134:1106-1114.
30. Katan MB. Weight loss diets for prevention and treatment of obesity. *N Engl J Med*. 2009;360:923-925.
31. Khaw K-T, Bingham S, Welch A, et al. Relation between plasma ascorbic acid and mortality in men and women in EPIC-Norfolk prospective study: a prospective population study. *Lancet*. 2001;357:657-663.

32. King DE, Mainous AG, Geesey ME. Adopting moderate alcohol consumption in middle age: subsequent cardiovascular events. *Am J Med*. 2008;121:201-206.
33. Kloner RA, Rezkalla SH. To drink or not to drink? that is the question. *Circulation*. 2007;116:1306-1317.
34. Knoops KT, de Groot LC, Kromhout D, et al. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. *JAMA*. 2004;292:1433-1439.
35. Kris-Etherton PM, Hu FB, Ros E, Sabaté J. The role of tree nuts and peanuts in the prevention of coronary heart disease: multiple potential mechanisms. *J Nutr*. 2008;138:1746S-1751S. Supplement: 2007 Nuts and Health Symposium.
36. Kumanyika SK, Obarzanek E, Stettler N, et al. Population-based prevention of obesity: the need for comprehensive promotion of healthful eating, physical activity, and energy balance: a scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the Expert Panel on Population and Prevention Science). *Circulation*. 2008;118:428-464.
37. Kuriyama S, Shimazu T, Ohmori K, et al. Green tea consumption and mortality due to cardiovascular disease, cancer, and all causes in Japan: the Ohsaki study. *JAMA*. 2006;296:1255-1265.
38. Lopez-Garcia E, Rodriguez-Artalejo F, Rexrode KM, Logroscino G, Hu FB, van Dam RM. Coffee consumption and risk of stroke in women. *Circulation*. 2009;119:1116-1123.
39. Lorenz M, Jochmann N, von Krosigk A, et al. Addition of milk prevents vascular protective effects of tea. *Eur Heart J*. 2007;28:219-223.
40. Malik VS, Popkin BM, Bray GA, Despres JP, Hu FB. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. *Circulation*. 2010;121:1356-1364.
41. Martınez-Gonzalez de la Fuente-Arrillaga C, Nunez-Cordoba JM, Basterra-Gortari FM, et al. Adherence to Mediterranean diet and risk of developing diabetes: prospective cohort study. *Br Med J*. 2008;336:1348-1351.
42. McGillicuddy FC, de la Llera MM, Hinkle CC, et al. Inflammation impairs reverse cholesterol transport in vivo. *Circulation*. 2009;119:1135-1145.
43. Mente A, de Koning L, Shannon HS, Anand SS. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. *Arch Intern Med*. 2009;169:659-669.
44. Mitrou PN, Kipnis V, Thiébaut ACM, et al. Mediterranean dietary pattern and prediction of all-cause mortality in a US population. Results from the NIH-AARP diet and health study. *Arch Int Med*. 2007;167:2461-2468.
45. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA*. 2004;291:1238-1245.
46. Mozaffarian D, Ascherio A, Hu FB, et al. Interplay between different polyunsaturated fatty acids and risk of coronary heart disease in men. *Circulation*. 2005;111:157-164.
47. Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ, Willett WC. Trans-fatty acids and cardiovascular disease. *N Engl J Med*. 2006;354:1601-1613.
48. Mozaffarian D, Marfisi R, Levantesi G, et al. Incidence of new-onset diabetes and impaired fasting glucose in patients with recent myocardial infarction and the effect of clinical and lifestyle risk factors. *Lancet*. 2007;370:667-675.
49. Mukamal KJ, Ascherio A, Mittleman MA, et al. Alcohol and Risk for Ischemic Stroke in Men: The Role of Drinking Patterns and Usual Beverage. *Ann Intern Med*. 2005;142:11-19.
50. Mukamal KJ, Maclure M, Muller JE, Sherwood JB, Mittleman MA. Tea Consumption and Mortality After Acute Myocardial Infarction. *Circulation*. 2002;105:2476-2481.
51. Mukamal KJ, Psaty BM, Rautaharju PM, et al. Alcohol consumption and risk and prognosis of atrial fibrillation among older adults: the cardiovascular health study. *Am Heart J*. 2007;153:260-266.

52. Mukamal KJ, Chen CM, Rao SR, Breslow RA. Alcohol consumption and cardiovascular mortality among U.S. adults, 1987 to 2002. *JACC*. 2010;55:1328-1335.
53. Nicholls SJ, Lundman P, Harmer JA, et al. Consumption of saturated fat impairs the anti-inflammatory properties of high-density lipoproteins and endothelial function. *JACC*. 2006;48:715-720.
54. Nöthlings U, Schulze MB, Weikert C, et al. Intake of vegetables, legumes, and fruit, and risk for all-cause, cardiovascular, and cancer mortality in a European diabetic population. *J Nutr*. 2008;138:775-781.
55. O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health. *JACC*. 2008;51:249-255.
56. Oh K, Hu FB, Manson JE, Stampfer MJ, Willett WC. Dietary fat intake and risk of coronary heart disease in women: 20 years of follow-up of the Nurses' Health Study. *Am J Epidemiol*. 2005;161:672-679.
57. Okie S. New York to trans fats: you're out! *N Engl J Med*. 2007;356:2017-2021.
58. Pereira MA, O'Reilly E, Augustsson K, et al. Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. *Arch Intern Med*. 2004;164:370-376.
59. Pereira MA, Parker ED, Folsom AR. Coffee consumption and risk of type 2 diabetes mellitus: an 11-year prospective study of 28 812 postmenopausal women. *Arch Intern Med*. 2006;166:1311-1316.
60. Pischon T, Boeing H, Hoffmann K, et al. General and abdominal adiposity and risk of death in Europe. *N Engl J Med*. 2008;359:2105-2120.
61. Retterstøl K, Hennig CB, Iversen PO. Improved plasma lipids and body weight in overweight/obese patients with type III. *Clin Nutr*. 2009. doi:10.1016/j.clnu.2009.01.018.
62. Romon M, Lommez A, Tafflet M, et al. Downward trends in the prevalence of childhood overweight in the setting of 12-year school- and community-based programmes. *Public Health Nutr*. 2009;12:1305-1306.
63. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight loss diets with different compositions of fat, protein, and carbohydrates. *N Engl J Med*. 2009;360:859-873.
64. Salas-Salvado J, Fernandez-Ballart J, Ros E, et al. Effect of a Mediterranean diet supplemented with nuts on metabolic syndrome status-one-year results of the PREDIMED randomized trial. *Arch Intern Med*. 2008;168:2449-2458.
65. Salazar-Martinez E, Willett WC, Ascherio A, et al. Coffee consumption and risk for type 2 diabetes mellitus. *Ann Intern Med*. 2004;140:1-8.
66. Shai I, Schwarzfuchs D, Henkin Y, Shahar DR, Witkow S, Greenberg I, Golan R, Fraser D, Bolotin A, Vardi H, et al For the Dietary Intervention Randomized Controlled Trial (DIRECT) Group. Weight loss with a low-carbohydrate, mediterranean, or low-fat diet. *N Engl J Med*. 2008;359:229-241.
67. Shai I, Spence JD, Schwarzfuchs D, Henkin Y, Parraga G, Rudich A, Fenster A, Mallett C, Liel-Cohen N, Tirosh A, Bolotin A, Thiery J, Fiedler GM, Buher M, Stumvoll M, Stampfer MJ For the DIRECT Group. Dietary intervention to reverse carotid atherosclerosis. *Circulation*. 2010;121:1200-1208.
68. Shinmura K, Tamaki K, Saito K, Nakano Y, Tobe T, Bolli R. Cardioprotective effects of short-term caloric restriction are mediated by adiponectin via activation of AMP-activated protein kinase. *Circulation*. 2007;116:2809-2817.
69. Singer MV, Teyssen S. Alcohol associated somatic hazards. *Dtsch Ärztebl*. 2001;98:A2109-A2120.
70. Sofi F, Cesaro F, Abbate R, Gensini GF, Casini A. Adherence to Mediterranean diet and health status: meta-analysis. *Br Med J*. 2008;337:a1344.
71. Steffen LM, Folsom AR, Cushman M, et al. Greater fish, fruit, and vegetable intakes are related to lower incidence of venous thromboembolism. The longitudinal investigation of thromboembolism etiology. *Circulation*. 2007;115:188-195.

72. Steptoe A, Gibson EL, Vounonvirta R, et al. The effects of tea on psychophysiological stress responsivity and post-stress recovery: a randomized double-blind trial. *Psychopharmacology*. 2007;190:81-90.
73. Streppel MT, Ocke MC, Boshuizen HC, Kok FJ, Kromhout D. Long-term fish consumption and n-3 fatty acid intake in relation to (sudden) coronary heart disease death: the Zutphen study. *Eur Heart J*. 2008;29:2024-2030.
74. Sun Q, Ma J, Campos H, et al. A prospective study of trans fatty acids in erythrocytes and risk of coronary heart disease. *Circulation*. 2007;115:1858-1865.
75. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med*. 2003;348:2599-2608.
76. Trichopoulou A, Orfanos P, Norat T, et al. Modified Mediterranean diet and survival: EPIC-elderly prospective cohort study. *Br Med J*. 2005;330:991-997.
77. Van Dam RM, Willett WC, Manson JE, Hu FB. Coffee, caffeine, and risk of type 2 diabetes. A prospective cohort study in younger and middle-aged U.S. women. *Diab Care*. 2006;29:398-403.
78. Van de Werf F. The European Heart Journal – the challenge of the next years. *Eur Heart J*. 2003;24:28-66.
79. von Schacky C, Harris WS. Cardiovascular benefits of omega-3 fatty acids. *Cardiovasc Res*. 2007;73:310-315.
80. Whitlock G, Lewington S, Sherliker P, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies Prospective Studies Collaboration. *Lancet*. 2009;373:1083-1096.
81. Committee WHO-Expert. Physical status: the use and interpretation of anthropometry: report of a WHO expert committee. *World Health Organ Tech Rep Ser*. 1995;854:1-452.
82. www.chocosuisse.ch facts and figures – per capita consumption of chocolate (accessed on 21.11.2010).