

# THE IMPORTANCE OF DIET, OBESITY AND TYPE II DIABETES FOR VASCULAR DISEASE<sup>1</sup>

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## Summary

Obesity, the metabolic syndrome, pre-diabetes and type 2 diabetes are important risk factors for the development of coronary artery disease and stroke (cardiovascular disease, CVD). Obesity, physical inactivity, diet composition, short sleep duration and smoking are the most important risk factors for type 2 diabetes; moderate alcohol and coffee consumption exert a weak protective effect. Excessive body weight together with inactivity can account for almost 90% of all new cases of type 2 diabetes. So prevention and treatment of weight gain, excessive body weight, and the metabolic syndrome, are the cornerstones of prevention of type 2 diabetes.

The major risk factors for weight gain and obesity are a sedentary lifestyle with little physical activity, impaired or short sleep duration, and an inappropriate diet. The dietary risk factors are large portion sizes, sugar-rich soft drinks, and high intakes of energy-dense foods poor in fibre and whole grain, including low intakes of fruit and vegetables. The optimal diet for prevention of weight gain provides 20–25% of energy from protein (low-fat meat, game, dairy products, fish, shellfish, and plant protein from peas, beans etc.), 25–30% of energy from fat (high ratio of polyunsaturated to saturated), and 45–55% from fibre-rich, whole-grain carbohydrates characterised by a low glycemic index. Moderate amounts of alcohol from beer and wine contribute to the prevention of type 2 diabetes and CVD, but should be recognised as contributors to total energy intake. The diet recommended for the prevention of obesity and type 2 diabetes is fortunately the same as that considered optimal for the prevention of CVD.

## Introduction

On a population level, epidemiological studies suggest that a healthy lifestyle with no tobacco smoking, avoidance of excessive alcohol consumption, adequate sleep, regular physical activity, an ideal body weight, and a

<sup>1</sup> Essential parts of the manuscript are based on a modification of a book chapter: Astrup A. A healthy lifestyle and cardiovascular risk reduction. In *Metabolic Risk for Cardiovascular Disease*. Ed. R.H. Eckel. American Heart Association 2010.

healthy diet, can reduce the risk of cardiovascular disease by up to 85–95%. Figure 1 (see p. 179) depicts the environmental factors that influences the risk of weight gain, obesity & type 2 diabetes.

Adequate treatment of risk factors would add ~221 million life-years and 244 million quality-adjusted life-years to the US adult population, or an average of 1.3 years of life expectancy for all adults. To achieve this, an intensive management is required, targeting the individual, as well as major changes in the toxic environment that tends to maintain unhealthy habits.

The present paper will focus on diet, smoking, impaired sleep and physical activity as risk factors for obesity and type 2 diabetes, which are major causes of CVD in this millennium. There is robust evidence that a large proportion of the cardiovascular disease seen today can be prevented by a generally healthier lifestyle in the population as a whole, and by targeting lifestyle change and medical management of cardiovascular risk factors in high-risk individuals (1). The high prevalence of obesity in most Westernized countries has important health consequences, and particularly abdominal obesity, increases the risk of several co-morbidities (Figure 2, see p. 179).

Prevention does not mean that CVD and death can be avoided, but rather that the onset of disease can be postponed and life expectancy prolonged. With the exception of smoking cessation, preventive lifestyle intervention will probably not save money on a societal level, as people will live longer and require medical care for other illnesses and extended social support. Effective prevention in the USA population could potentially reduce the incidence of MI by >60%, the incidence of stroke by ~30%, and increase life expectancy by an average of 1.3 years [1]. European studies have found that population-wide, best-practice interventions have the potential to reduce coronary heart disease mortality by 57%, and for primary prevention the corresponding reductions would be 75–85% [2]. This analysis did not, however, include the effect of weight reduction in the overweight section of the population, and prevention of weight gain by changed diet and exercise habits.

### **The potential impact of a healthy lifestyle on CVD**

The most effective way to prevent CVD is to maintain a healthy lifestyle. Stampfer *et al.* defined low risk among adult women as those who were non-smokers, had a BMI below 25, consumed an average of half a unit of alcohol per day, engaged in moderate-to-vigorous physical activity for at least 30 min per day, and had a diet moderately high in cereal fiber, marine n-3 fatty acids, and folate, with a high ratio of polyunsaturated to saturated fat, and low in trans fat and glycemic load [3]. They found that only 3% of

the US population complied with this lifestyle, but that this 3% had a relative risk of coronary events of only 0.17 as compared to all the other women, and that 82% of coronary events could be attributed to lack of adherence to this healthy lifestyle.

## **General advice for the prevention of CVD and obesity**

### *The diet for the prevention of CVD*

A reduction of CVD can be achieved by modifying the diet to affect risk factors such as body weight and the components of the metabolic syndrome (LDL-cholesterol, postprandial triglycerides, blood pressure etc.). Diet plays an important role in achieving this goal. Previous analyses of the importance of saturated fat (SFA) for the development of CVD have overestimated its importance, and failed to compare foods rich in SFA with other relevant foods. From a recent consensus conference the following conclusions were reached [4]. Newer meta-analyses of observational studies have found that substituting polyunsaturated fat (PUFA) for SFA is associated with lower CHD risk, whilst substituting carbohydrate for SFA is associated with a moderately increased risk of CHD. Few studies have addressed the quality of carbohydrates. There is no evidence that monounsaturated fat (MUFA) is associated with CHD risk. Also the individual saturated fatty acids and the food matrix are important for cardiovascular risk. Despite having high contents of saturated fat, both dark chocolate and cheese have no negative impact on cardiovascular risk factors and can be part of a heart-healthy diet.

Industrially produced trans fat (TFA) is consistently associated with increased risk of CHD. On a gram for gram basis, TFA is associated with stronger risk than SFA, but the lowest risk is found for diets high in PUFA and low in TFA.

It is generally assumed that cheese, due to the high content of saturated fat, should be reduced in a diet that reduces cardiovascular risk. However, scientific evidence shows that the content of protein, calcium and other nutrients in the food matrix modifies the effect of saturated fat so the overall effect of cheese rather is cardioprotective [4] (Figure 3, see p. 180).

The evidence from epidemiological, clinical and mechanistic studies is consistent in finding that risk of CHD is reduced when PUFA replaces SFA. In countries following a 'western' diet, replacing 1% kJ of SFA with PUFA is likely to produce a 2-3% reduction in the incidence of CHD. Cohort studies find no evidence of lower risk of CHD when CHO replaces SFA. In fact, the evidence suggests a higher risk. The type of CHO is important. For ex-

ample, replacing SFA with CHO from wholegrain foods may lower the risk of CHD. The amount and type of carbohydrate are likely to have less effect on CHD risk in normally healthy and physically active individuals.

So what should we eat and drink to maintain a healthy body weight, and to prevent T2D & CVD? (Figure 4, see p. 180). The habitual diet should be low in industrially produced trans fat and saturated fats, sugar and salt, and contain plenty of fruit and vegetables (at least five portions a day), whole grain products and fish. Fish such as herrings, kippers, mackerel, pilchards, salmon, sardines and trout contain oils that can reduce the risk of thrombosis. Other foods that have a beneficial impact on CVD risk are beans, peas, lentils and oats, because they contain soluble fiber.

Regular aerobic (cardiovascular) exercise, for at least 30 minutes a day at least 3–4 times a week, has a beneficial impact on several CVD risk factors, and also helps to maintain a healthy body weight.

### *Diet for the prevention of obesity*

Overweight and obesity, and in particular abdominal fat distribution, adversely affect several risk factors of CVD, and generate numerous other comorbidities (Figure 2, see p. 179), and weight reduction has been shown to decrease incidence and cardiovascular mortality in severely obese subjects. Obese individuals are known to be capable of losing weight, but weight maintenance is more problematic, so prevention of weight gain is a primary focus. The major risk factors for weight gain and obesity are a sedentary lifestyle with little physical activity, impaired or short sleep duration, and an inappropriate diet. The dietary risk factors are large portion sizes, sugar-rich soft drinks, high intakes of energy-dense foods poor in fibre and whole grain, and low intakes of fruit and vegetables (Figure 4, see p. 180). The optimal diet for prevention of weight gain provides 20–25% of energy from protein (low-fat meat, dairy, fish, shellfish, game, protein from plants; peas, beans etc.), 25–30% of energy from fat (high ratio of polyunsaturated to saturated), and 45–55% from fibre-rich, whole-grain carbohydrates characterised by a low glycemic index [5]. Moderate amounts of alcohol from beer and wine contribute to the prevention of type 2 diabetes and CVD, but should be recognised as contributing to total energy intake. The diet recommended for the prevention of obesity and type 2 diabetes is fortunately the same as that considered optimal for the prevention of CVD.

### *Smoking and alcohol*

For smokers, giving up will reduce the risk of developing coronary heart disease by 50%. Smoking causes the majority of cases of coronary throm-

basis in people under the age of 50. Small amounts of alcohol may help to reduce CVD. However, excessive alcohol consumption increases blood pressure and risk of stroke, as well as several other health risks, and it is therefore advisable that individuals abide by public health recommendations regarding alcohol consumption. Health guidelines in the UK recommend that men consume no more than three to four units of alcohol a day, and that women do not exceed two to three units. Binge drinking should be avoided.

Blood pressure can be controlled by eating a healthy diet that is low in saturated fat, and high in fruit, vegetables, and low-fat dairy products, and by exercising regularly. In addition, avoiding smoking and excessive alcohol consumption assists in maintaining a normal blood pressure. Diabetics have a greater risk of developing CVD. The risk of developing type 2 diabetes can be reduced dramatically by maintaining a healthy body weight, being physically active, and by eating a healthy diet.

### **Multifactor interventions**

For clinicians and their patients, studies addressing the total reduction in risk of CVD that can be achieved by a healthy lifestyle are more relevant than addressing each of the lifestyle components individually. However, the flip side of the coin is that these studies leave the contribution of each of the components uncertain. Regarding the primary prevention of CVD, larger intervention studies are obviously required to demonstrate a risk reduction, but observational epidemiological studies have suggested that dramatic health gains can be achieved. An analysis by Asaria *et al.* [6] found that 13.8 million deaths could be prevented over 10 years if measures to reduce tobacco and salt exposure were implemented. About three out of four deaths averted would be from cardiovascular diseases [6]. Hu *et al.* found that the incidence of coronary disease declined by 31% from 1981 to 1992, while the proportion of smokers declined by 41%, the proportion of postmenopausal women using HRT increased by 175%, and the prevalence of overweight increased by 38%. These factors could explain a 21% decline in the incidence of coronary disease, representing 68% of the overall decline. The reduction in smoking could account for a 13% decline in the incidence, improved diet could account for a 16% decline; and increase in HRT could explain a further 9% decline. However, the increase in overweight could account for an 8% increase in incidence in coronary disease [7].

Major benefits could also be achieved from secondary prevention of CVD. Ornish *et al.* [8] showed that intensive lifestyle changes (fat-reduced diet, aerobic exercise, stress management training, smoking cessation, group psychosocial support) for 5 years produced greater regression of coronary

atherosclerosis and fewer cardiac events than in a control group. An effect of comprehensive cardiac rehabilitation sessions was recently assessed in the randomized controlled GOSPEL trial in post-MI patients, comparing a long-term, reinforced, multi-factorial educational and behavioral intervention with usual care, including supervised aerobic exercise and lifestyle modification consisting of risk factor counseling about a Mediterranean diet, smoking cessation, and stress management [9]. The targets of the intervention strategy were smoking cessation, adoption of a healthy Mediterranean diet, increase in physical activity up to at least 3 h/wk at 60% to 75% of the mean maximum heart rate, and maintenance of BMI at <25 kg/m and blood pressure of <140/85 mmHg. After 3 years of observation there were only small improvements in most of the lifestyle variables, and these did not significantly reduce the primary end-point [9], though several secondary end-points were decreased: CV mortality plus nonfatal MI and stroke (3.2% vs 4.8%; HR, 0.67), cardiac death plus nonfatal myocardial infarction (2.5% vs 4.0%; HR, 0.64), and nonfatal MI (1.4% vs 2.7%; HR, 0.52). The intervention group still had plenty of room for further improvement in lifestyle, such as smoking cessation and weight gain prevention, and this emphasizes the potential for health benefits that can be achieved even in patients with established CVD.

## Smoking

A smoke free environment is an important part of a healthy lifestyle. The first major epidemiological study showing a strong correlation between smoking and cardiovascular disease was published around 1960, and although observational studies could not provide definitive evidence that tobacco smoke is responsible for increased coronary risk, it prompted the first anti-smoking measures by the US Surgeon General in his 1964 report. Smoking is a highly addictive habit, and despite major reductions in smoking prevalence in most Western countries over the last 50 years, tobacco use continues to grow in global importance as a leading preventable cause of cardiovascular disease. Tobacco smoking exerts both prothrombotic and atherogenic effects, and it increases the risk of acute myocardial infarction, sudden cardiac death, stroke, aortic aneurysm and peripheral vascular disease [10]. It has been found that even low-level exposure (e.g. passive smoking) increases the risk of acute myocardial infarction. Figure 5 (see p. 181) shows the number of years of life lost being obese at age 40 ~ the effects of smoking [11].

Fortunately, smoking cessation and avoidance of passive smoking rapidly reduces this risk. A major problem with smoking cessation is that the majority of smokers experience a weight gain when they stop smoking which,

in many cases, leads to taking up the habit again. It has been estimated that more people in the USA die from obesity-related complications than from tobacco. Only a small proportion of the weight gain observed in the population can be attributed to smoking cessation [12], but the observation emphasizes the need to include advice on prevention of weight gain in the package of smoking cessation tools.

### Physical inactivity

Regular physical activity is an important part of a healthy lifestyle, and sedentary behavior can account for a substantial risk of CVD. Individuals who regularly undertake physical activity have a decreased risk of obesity, heart disease, hypertension, diabetes, and premature mortality. Systematic reviews and meta-analyses have consistently found that physical activity is associated with a marked decrease in cardiovascular and all-cause mortality in both men and women, even after adjusting for other relevant risk factors.

Nocon *et al.* recently reported a meta-analysis including 33 studies with 883,372 participants, with follow-up ranging from 4 years to over 20 years [13]. Physical activity was associated with a risk reduction of 35% in cardiovascular mortality (95% CI: 30–40%). All-cause mortality was reduced by 33% (28–37%). Oguma *et al.* examined the dose-response relationship in women based on a meta-analysis of 30 studies and found, when studies were combined according to relative levels of physical activity, that the risk reduction showed a dose-response relationship for CHD (RR=1 [reference], 0.78, 0.53, 0.61, respectively) for studies with four PA levels,  $n = 5$ ); for stroke (RR=1 [reference], 0.73, 0.68) (14). For overall CVD the reduction was also substantial (RR=1 [reference], 0.82, 0.78). When studies were combined by absolute walking amount, even 1 hour/week walk was associated with reduced risk of CVD outcome.

Analyses from the Health Professionals' Follow-up Study of the importance of levels of leisure-time physical activity for incidence of CHD found that total physical activity, running, weight training, and rowing were each inversely associated with risk of CHD. Men who ran for an hour or more per week had a 42% risk reduction (RR, 0.58; 95% CI, 0.44–0.77) compared with men who did not run (16). Men who trained with weights for 30 minutes or more per week had a 23% risk reduction and those who rowed for 1 hour or more per week had an 18% risk reduction compared to those who did not undertake these activities. Average exercise intensity was also associated with reduced CHD risk, independent of the total volume of physical activity. Half an hour or more of brisk walking per day was associated with an 18% risk reduction, but walking pace was associated with

reduced CHD risk independent of the number of walking hours. These results show that various activities can be used, and that exercise intensity and fitness should be considered, as well as the amount of time spent on exercise activities, when calculating the health benefits of physical activity.

### *Is “fat but fit” a sufficient goal?*

With an increasing proportion of the population being either overweight or obese, the question has been raised whether physical activity can eliminate the adverse health effect of obesity. In a recent meta-analysis of observational studies Blair and Brodney concluded that: 1) regular physical activity clearly attenuates many of the health risks associated with overweight or obesity; 2) physical activity appears to not only attenuate the health risks of overweight and obesity, but active obese individuals actually have lower morbidity and mortality than normal weight individuals who are sedentary; and 3) inactivity and low cardio-respiratory fitness are as important as overweight and obesity as mortality predictors [17]. Notably, their findings were entirely based on observational studies, and randomized clinical trials addressing these questions should be undertaken. Patients with established CVD will benefit from more exercise and increasing fitness, and the risk of sudden death induced by exercise is negligible compared with the benefits.

In order to reduce the ill health caused by physical inactivity the CDC and the American College of Sports Medicine recommend that adults engage in at least 30 minutes of moderate physical activity on most days, and preferably on all days [18]. Recently, the CDC analyzed data from the Behavioural Risk Factor Surveillance System (BRFSS) and found that from 2001 to 2005 the prevalence of regular physical activity increased 8.6% among women overall (from 43.0% to 46.7%) and 3.5% among men (from 48.0% to 49.7%) [18]. These figures may be due to over-reporting bias, but they suggest that efforts to increase the proportion of individuals complying with the recommendations is increasing. However, they also demonstrate that the majority of the adult population does not meet the recommendations for physical activity, and there is obviously potential for further reductions in the incidence of CVD by activating the sedentary section of the population. State and local public health agencies, and other public health stakeholders, should continue to implement evidence-based, culturally appropriate initiatives to further increase physical activity and fitness levels in all adults.

### **Sleep**

Sleep of sufficient duration and quality is inherent as part of a healthy lifestyle, and too little high-quality sleep promotes CVD through a dis-

rupted appetite control, and through effects on metabolism that increase the risk of type 2 diabetes. Too little sleep has become a common health problem in western societies. The average duration of sleep has decreased by approximately one hour compared to 30 years ago [19], with more severely impaired sleep seen in certain individuals and/or groups. Sleep loss, widespread in modern societies, is an under-recognized public health problem that has a cumulative effect on physical and mental health. Epidemiological studies support the hypothesis that there are links between impaired sleep and overweight, especially in the young [20–22]. A number of intervention studies also imply that disturbed sleep has an impact on numerous physiological functions, such as appetite regulating hormones, substrate metabolism, and blood pressure [22–26]. In addition, numerous reports have found too little or impaired sleep to be a risk factor for mental distress, depression, anxiety, obesity, hypertension, diabetes, high cholesterol levels, and premature CVD death [27,28]. Though confounding by other adverse health behaviors such as cigarette smoking, physical inactivity, and heavy drinking may be important, there is accumulating evidence that too little and impaired sleep per se increase risk of CVD, partly directly and partly through weight gain and insulin resistance. Recently, King *et al.* [27] found a robust association between objectively measured sleep duration and 5-year incidence of coronary artery calcification, a subclinical predictor of future coronary heart disease events. They found that one more hour of sleep decreased the estimated odds of calcification incidence by 33%. The magnitude of the observed effect was similar to that of other important CVD risk factors (e.g. one additional hour of sleep reduced risk similarly to a reduction of 16.5 mmHg in systolic blood pressure) [27].

Recent intervention studies have provided a mechanistic explanation for the deleterious effects of sleep deprivation on health. These physiological data suggest that short-term partial sleep restriction leads to striking alterations in metabolic and endocrine functions, such as insulin resistance, increased sympathetic tone, elevated levels of cortisol and pro-inflammatory cytokines, and decreased leptin and increased ghrelin levels [22]. Furthermore, abnormal sleep-wake patterns probably alter intracellular circadian clocks, which may potentiate disrupted metabolism [28]. Chronic lack of sleep is stressful and biologically demanding and must be avoided if good health is a goal.

There seems to be good reason to question patients about sleep duration and sleep quality, and to recommend efforts to ensure that they get sufficient amounts of high quality sleep. The optimal sleep duration for adults appears to be 7–8 hours. Sleep of longer duration is a strong risk factor for increased mortality [29], but it is unclear whether the association is confounded by

other adverse health behaviors. More than 7.5 hours of sleep may increase the risk of cerebrovascular deaths, both in women and men [230]. It is not clear why sleep exceeding 7.5 hours should be associated with excess mortality, and the observation has not been confirmed by all studies. Long sleep duration may not be a causal factor for the increased mortality in itself, and sleep apnea may be an underlying confounding factor.

It should be kept in mind that there are still research questions to be answered, and there is obviously a need for RCTs to demonstrate that improved sleep duration and quality can reduce CVD risk. However, there is no risk in taking a pragmatic approach and encouraging a good night's sleep as an adjunct to other health measures. Sleep is a modifiable risk factor and the benefit of increasing sleep duration in at-risk individuals outweighs the harms.

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Figure 1.



Figure 2.



Figure 3.

**So what should we eat and drink to maintain a healthy body weight, and to prevent T2D & CVD ?**

- ❑ Reduce fat to 30-35 % of energy
  - Less Saturated fat (except from cheese and dark chocolate), and more polyunsaturated
- ❑ Increase protein to 20-25 %
  - Lean unprocessed meat, dairy products, fish, and vegetable proteins
- ❑ Keep carbohydrate ~ 40-50 %
  - with emphasis on whole grain food, fruit and vegetables
- ❑ Reduce sugar-rich soft drinks
- ❑ Alcohol in moderation
  - Wine and beer preferable
- ❑ Daily physical activity

Figure 4.

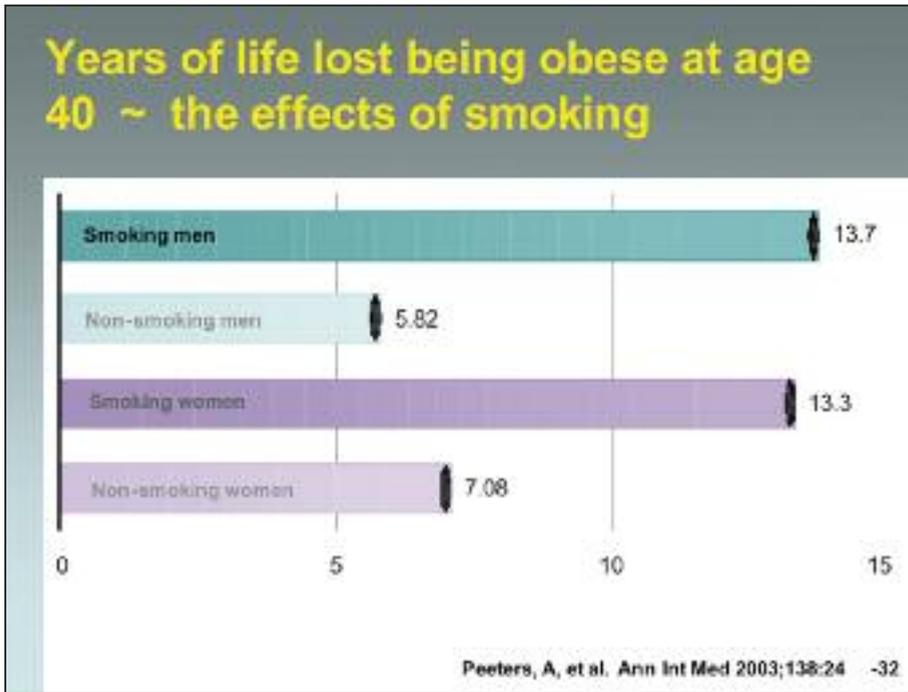


Figure 5.