European Nutrition Research

Update

A Periodic Summary of Nutrition Research

Issue 1
2007

The impact of diet on heart health

Prof Dr Ángeles Carbajal, Departamento de Nutrición, Facultad de Farmacia, Universidad Complutense de Madrid.

The human heart is a perfect machine. Each minute, hour and day, it beats without a break, being at the centre of life. At the age of 20 the average heart has already done about a billion beats. Yet, as the statistics show, many people do not take appropriate care of their hearts.

Risk factors and aetiology

Coronary heart disease (CHD) is the leading cause of death and disability in developed countries. Aging populations in Western nations and increasingly affluent Eastern nations may be bringing about a worldwide epidemic[1]. Key factors in this, and the rise in type 2 diabetes, are the numbers of people eating the wrong types of diets, smoking, exercising too little and becoming obese.

The underlying pathology of CHD is atherosclerosis, a generic term that encompasses arterial wall damage and subsequent build up of atheromatous plaques, i.e. clumps of blood cells and lipids, particularly oxidized LDL-cholesterol. These plaques narrow the arteries, setting the scene for a myocardial infarction (heart attack). It is believed that the process of atherosclerosis begins early in life, takes years to develop and is usually symptom-free in the early phases. The lag-time of CHD means that the benefits of modifying risk factors, especially in younger people, are not immediately apparent. This can make it a challenge to persuade people to change their behaviour[1].

Although the aetiology is not completely known, evidence suggests that multiple factors interact to create an overall risk[2]. Some of these are non-modifiable, e.g. family history of premature CHD, increasing age, or male gender. Others, such as dyslipidaemia (i.e. high total and LDL-cholesterol), hypertension, diabetes, and obesity are modifiable and, thus, help lower risk. Common public health responses include addressing dietary habits, physical activity levels, and smoking[3].

Dietary factors that impact on heart health

Since the early work of Ancel Keys and others in the 1960s, a vast literature has emerged suggesting that dietary factors play a major role in heart health[4]. The mechanisms range from improvements to lipid metabolism, thombotic tendency, blood pressure, inflammation, coagulation, insulin resistance, among others[1,5]. Diet can also lower CHD risk indirectly by addressing conditions such as obesity and diabetes.

Indeed, aside from smoking, diet is probably the most important modifiable factor. However, it is not easy to pinpoint the most effective measures out of those dietary modifications recommended. This is because, in epidemiological terms, dietary factors are frequently inter-related, tend to cluster, and display complex synergistic or antagonistic interactions. To be meaningful, individual dietary factors must be considered within overall lifestyles[5,6].

A good example of this is the clear benefits shown for 'prudent' or 'Mediterranean' diets. These are diets characterized by high intakes of fruits, vegetables, whole-grains, legumes, fish and poultry, and moderate intakes of low-fat dairy products and lean meat[4,7]. Adequate evidence is now available for the following:

Dietary fats

Saturated fatty acids (SFA), especially those with 12-16 carbons, raise blood levels of LDL, while a diet low in SFA can reduce the incidence of coronary events. The target is to restrict the intake of SFA to less than 7%,[2,5]. The major dietary sources of SFA are full-fat dairy products (whole milk, cheese, butter, cream), meat products, shortenings and tropical oils (i.e. palm, coconut and palm kernel oils).

Trans-fatty acids (TFA) have adverse effects on CHD through many different pathways

In this issue

including but not limited to increased LDL and reduced HDL cholesterol[1]. According to the evidence TFA are probably more atherogenic than saturated fat[4]. Natural sources include dairy products and meats but TFA also occur during processing, e.g. in the production of partially hydrogenated vegetable oils, deep-fried fast foods, vegetable shortenings, and some margarines[5,6]. An intake of less than 1% of daily energy intake is recommended[1,5].

Foods rich in long-chain n-3 polyunsaturated fatty acids, specifically eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids can lower the risk of CHD by preventing cardiac arrhythmia, lowering serum triglyceride levels, decreasing thrombotic tendency, and improving blood pressure control[5]. Key food sources are oily fish, (tuna, salmon, mackerel, sardines, herring), although fish oil supplements are also available. At least two servings of oily fish per week are recommended to confer cardioprotective effects[4]. It has been suggested that vegetable sources of n-3s (nuts, flaxseed, soya oil) are not as effective as the n-3s found in oily fish.

Monounsaturated fatty acids (i.e. oleic acid in olive and canola oil) when substituted for SFA lead to reductions of LDL and triglycerides, and small increases of HDL. They may also reduce the susceptibility of LDL to oxidative damage[2].

Dietary cholesterol can increase LDL levels, although to a lesser extent than SFA[1]. Foods high in SFA are often sources of dietary cholesterol and hence reduced intakes of such foods provide duel benefits. Dietary cholesterol is found in meat, eggs, high-fat dairy products, meat products, poultry and shellfish. However, some cholesterol-rich foods, such as egg yolks, are relatively low in SFA and do not need to be restricted, although some limitation remains prudent[2]. The target level for dietary cholesterol intake is <300 mg/d[2] but, for individuals with existing CHD risk factors, <200 mg/d is recommended.

Salt

High intakes of salt (sodium chloride) adversely affect blood pressure, a major risk factor of CHD in susceptible people. European recommendations for maximum salt intakes in adults range from 5-6 g/d[5].

Wholegrains & fibre

There is good evidence that regular consumption of whole-grain foods substantially lowers CHD risk[8] with protective effects seen at intakes of three servings per day[9]. Cereal grains are an excellent source of complex carbohydrates, fibre, vitamins, minerals and phyto-protective components that possibly work synergistically. The main food sources of whole-grains in the Western diet are bread and whole-grain breakfast cereals[9]. Some studies have showed the potential importance of breakfast consumption to insulin sensitivity and plasma cholesterol concentrations. An irregular meal frequency may have a deleterious effect on cardiovascular risk factors[10]. The specific type of fibre found in whole-grain products, especially cereals, has been consistently related to a lower risk of CHD[4,5]. Part of this reason may be the low glycaemic index of these foods[6]. The population intake goal is >25 g/d of total dietary fibre but average intakes fall way below this level[1]. Recent studies indicate that soluble (viscous) fibre, such as ß-glucan in oat bran, lowers serum cholesterol and LDL levels by several pathways (see article by Dr Welch in this issue). A daily intake of 5-10 g of ß-glucan is effective[5].

Fruit & vegetables

Many studies have shown an inverse association between consumption of vegetables and fruits and CHD risk[1,6]. The mechanisms vary as do the nutritional aspects that are implicated e.g. antioxidants, polyphenols, phytochemicals, fibre and low energy density[5]. Moreover, vegetables and fruits may displace from the diet other foods that are associated with increased CHD risk. Most Western countries recommend at least 400 g/d or five portions of fruits and vegetables, one of which can be pure juice[3].

Other factors

A number of other dietary factors impact on heart health. One of these is low to moderate alcohol consumption[1]. The optimal intake depends on age, gender, and presence of other risk factors or associated diseases, but it is generally recommended to limit alcohol intake to two drinks a day for men and one a day for women[2]. Upper limits are important as excessive intakes of alcohol cause multiple adverse health effects[5].

New evidence shows that plant stanols and sterols modify LDL levels by inhibiting cholesterol absorption[1]. The maximal lowering of LDL occurs at intakes of 2 g/d[5].

Other dietary factors have less clear effects on CHD risks and further research is needed. These include folate and other B-vitamins, some minerals, antioxidants, phytochemicals, flavonoids, the optimal ratio of n-3:n-6 fatty acids, soya protein, nuts, coffee, or tea[1,6].

Conclusion

It has been estimated that 80% of heart attacks could be prevented through maintaining healthy behaviours throughout life[4]. A central part of this is following a diet that is known to impact favourably on CHD risk factors. Combining a predominantly plant-based diet with an avoidance of smoking, mainte-

Table 1 - Ten tips for heart health

1.	Avoid use of and exposure to tobacco products.
2.	Achieve and maintain a healthy body weight (BMI < 25 kg/m2).
3.	Choose whole-grain and high-fibre foods. At least half of grain intake must come from whole-grains.
4.	Include a variety of fruits and vegetables.
5.	Modify food choices to reduce saturated fats, cholesterol, and trans fatty acids.
6.	Eat fish, preferably oily fish, at least twice a week.
7.	Use lower-fat dairy products, poultry (without skin) and lean cuts of meat.
8.	Limit salt (sodium chloride) intake to <6 g/d.
9.	If you drink alcohol, do so in moderation.
10.	Be moderately to vigorously physically active for at least 30 minutes each day (60 minutes per day for children, and overweight adults). Spend less time in sedentary activities.

nance of a healthy body weight, moderate alcohol consumption and regular physical activity can offer significant benefits (Tabel 1).■

References

1. *WHO (2003). Diet, nutrition and the prevention of chronic diseases. Report of a Joint WHO/FAO Expert Consultation. WHO Technical Report Series No. 916 Geneva: World Health Organization.*
2. *Reddy KS & Katan MB (2004). Diet, nutrition and the prevention of hypertension and cardiovascular diseases. Public Health Nutrition 7 (Suppl 1), 167-186.*
3. *Lichtenstein AH et al (2006). Diet and lifestyle recommendations: a scientific statement from the American Heart Association Nutrition Committee. Circulation 114, 82-96.*
4. *Willett WC (2006). The Mediterranean diet: science and practice. Public Health Nutrition 9, 105-110.*
5. *National Cholesterol Education Program (2002). Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report. Circulation106, 3143-3421.*
6. *Hu FB & Willett WC (2002). Optimal diets for prevention of coronary heart disease. Journal of the American Medical Association 288, 2569-2578.*
7. *Knoops KT et al (2004). Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. Journal of the American Medical Association 292, 1433-1439.*
8. *Flight I & Clifton P (2006). Cereal grains and legumes in the prevention of coronary heart disease and stroke: a review of the literature. European Journal of Clinical Nutrition 60, 1145-1159.*
9. *Richardson DP (2003). Wholegrain health claims in Europe. Proceedings of the Nutrition Society 62, 161-169.*
10. *Farshchi HR et al (2005). Deleterious effects of omitting breakfast on insulin sensitivity and fasting lipid profiles in healthy lean women. American Journal of Clinical Nutrition 81, 388-396.* ■

Oat β-glucan and heart health

Robert W Welch, Northern Ireland Centre for Food and Health, School of Biomedical Sciences, University of Ulster, Coleraine, UK

Although heart disease is a multi-factorial condition, elevated plasma cholesterol, particularly LDL-cholesterol, are major risk factors[1]. Various pharmacological and dietary strategies have been shown to lower cholesterol, and reduce the risk of heart disease. Currently, statin drugs are a favoured pharmacological approach, while evidence-based dietary strategies include decreasing fat intake, substituting saturated fat with unsaturated fats, and increasing soluble fibre[2]. Fruits and vegetables contain soluble fibre but a particularly concentrated source is oats. The main type of soluble fibre in oats is β-glucan, which is also known as oat gum. Traditional porridge oats (oatmeal) are whole grains and contain ~ 4% β-glucan, while oat bran, a coarse milling fraction of oats, has at least 5.5% β-glucan[3]. Both of these foods have been shown to lower plasma cholesterol in a number of human trials.

What are β-glucans?

Glucans is another term for glucose polymers, i.e. long chains of glucose molecules. There is a diverse range of these in nature, varying in size (degree of polymerisation), structure and solubility (Table 1)[2]. β-glucans can be hydrolysed by digestive enzymes to yield maltose and, ultimately, glucose. Examples include *amylose* (linear) and *amylopectin* (branched), which are constituents of starch. In most other glucose polymers, the glucose units are joined by β-linkages, which are not susceptible to digestive enzymes. Thus, β-linked polymers are termed 'indigestible carbohydrates' and form part of the dietary fibre fraction. *Cellulose*, which comes primarily from plant cell walls, has only β-linkages and is the most common indigestible glucose polymer. Other examples of β-linked glucose polymers include the insoluble, bacterial-derived *curdlan,* which has food applications, soluble *laminarin* found in seaweed species, and *oat β-glucan* (Figure 1). Oat β-glucan is concentrated in the outer bran layers of the oat groat, while a similar type of β-glucan is found in barley. As a consequence of its structure, oat β-glucan is generally soluble in water, where it forms viscous solutions. Partly due to its contribution to overall fibre intakes, and partly due to other properties, oat β-glucan is now believed to offer heart health benefits.

Cholesterol-lowering effects

The first report that oats could lower plasma cholesterol came in 1963 from the Netherlands[4]. In an uncontrolled 3-week experiment, subjects consumed 140 g/d of rolled oats incorporated into bread, which lead to an impressive 11% decrease in cholesterol. Many subsequent controlled trials with lower doses of rolled oats also showed decreases in cholesterol, but the cholesterol-lowering effects

Figure 1 - Structures of glucose polymers (glucans) found in plant food sources

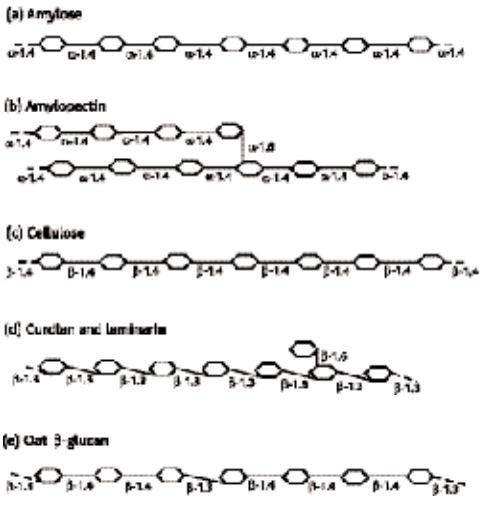
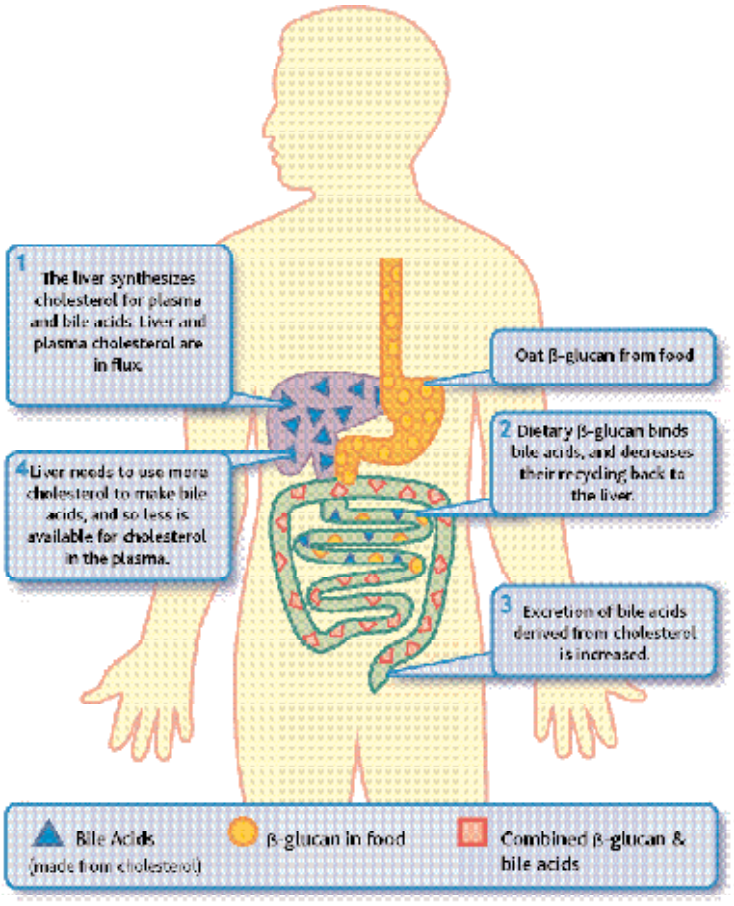


Figure 2 - How β-Glucan lowers blood cholesterol



were not always statistically significant. However, the onset of the effect was rapid. Consumption of oat bran for as little as three weeks resulted in substantial reductions in cholesterol[5]. By 1997, considerable experimental evidence had accumulated from these human studies to show that oats lowered plasma cholesterol, and in particular LDL-cholesterol. This led the US Food and Drugs Administration (FDA) to permit a health claim for oats to the effect that diets high in oatmeal or oat bran may reduce the risk of heart disease. The FDA claim acknowledged that oat β-glucan soluble fibre was the cholesterol-lowering agent, and that a daily minimum intake of about 3 grams of oat soluble fibre was needed in order to achieve significant cholesterol-lowering effects. To qualify for the claim, a single portion of oat food needed to provide at least 0.75 g of oat soluble fibre. Similar health claims were subsequently permitted in the UK and in Sweden

Mechanisms

A number of potential mechanisms have been proposed for the cholesterol-lowering properties of oat β-glucan. These include (a) less efficient absorption of dietary lipids from the gut (e.g. triacylglycerols and cholesterol); (b) hormonal effects induced by improved insulin responses, or via beneficial changes to gut hormones; (c) inhibition of cholesterol synthesis in the liver by circulating short chain fatty acids. These fatty acids are by-products of β-glucan fermentation in the colon[6].

While there is evidence for all of these, the major mechanism appears to relate to the ability of oat β-glucan to decrease the enterohepatic circulation of bile acids (Figure 2)[7,8]. The enterohepatic circulation involves the absorption of bile acids from the small intestine, their transport to the liver, and subsequent recycling in the bile. Bile acids are initially synthesised in the liver from cholesterol intermediates, and stored in the gall bladder before secretion into small intestine where they facilitate fat digestion and absorption. Bile acids are conserved by being re-absorbed from the distal small in-testine, and returned to the liver for recycling. Oat β-glucan remove bile acids (and thus cholesterol) from the enterohepatic circulation by binding them in the small intestine. The bile acids are then expelled in the faeces. These losses prompt the liver to take up more cholesterol from plasma in order to create new bile acids since there is less recycling. The end result is lower levels of plasma cholesterol.

Other health effects

In addition to its cholesterol-lowering properties, there is evidence that oat β-glucan may exert other beneficial physiological changes, e.g. normalising glucose metabolism (thus impacting on insulin resistance), or acting as a pre-biotic by promoting numbers of beneficial bacterial in the colon[9]. Oats contain other potentially bio-active components that could impact on heart disease and other conditions. These include avenanthramides, phenolic phytochemicals unique to oats, which may act as anti-oxidants[10]. Oats also have a good nutrient profile, and have long been viewed as valuable for the nutrition of infants and adults.

Conclusions

The accumulating evidence that oats impact favourably on heart disease risk enables products containing oats to be classified as 'functional foods'. Furthermore, although more research is required, the probability that oats may exert other beneficial effects has led to them being described as a multi-functional food[9].

Table 1 - Characteristics of glucose polymers (glucans) found in plant food sources

	Digestible[a]	Water solubility	Size (DP)[b]	Structure	Major sources
Amylose (α-glucan)	Y	Forms gels	Up to 50,000	Linear	Component of starch
Amylopectin (α-glucan)	Y	Forms gels	Up to 6,000,000	Branched	Component of starch
Cellulose (β-glucan)	N	Insoluble	Up to 10,000	Linear	Plant cell walls
Curdlan (β-glucan)	N	Insoluble	~ 450	May be branched	Alcaligenes faecalis
Laminarin (β-glucan)	N	Soluble	20-30	May be branched	Laminaria digitata
Cereal-glucan (β-glucan)	N	Soluble, but can vary	Up to 120,000	Linear	Oats and barley

a: Y = yield glucose for absorption; N = not digested by gut enzymes

b: DP = degree of polymerisation (number of glucose units per molecule)

References

1. *Genser B & Marz W (2006). Low density lipoprotein cholesterol, statins and cardiovascular events: a meta-analysis. Clinical Research in Cardiology 95, 393-404.*
2. *Warrand J (2006). Healthy Polysaccharides: the next chapter in food products. Food Technology and Biotechnology 44, 355–370.*
3. *Welch RW (1995). The chemical composition of oats. In: The Oat Crop - Production and Utilisation (Ed. RW Welch). London: Chapman and Hall, pp 279-320.*
4. *de Groot et al (1963). Cholesterol-lowering effect of rolled oats. Lancet ii, 303-304.*
5. *Brown L et al (1999). Cholesterol-lowering effect of dietary fiber: a meta analysis. American Journal of Clinical Nutrition 69, 30-42.*
6. *Anderson JW et al (1990). Soluble fiber: hypocholesterolemic effects and proposed mechanisms. In: Dietary Fiber: Chemistry, Physiology and Health Effects. Ed. D Kritchevsky et al. New York: Plenum Press, pp 339-363.*
7. *Marlett JA et al (1994). Mechanism of serum-cholesterol reduction by oat bran. Hepatology 20, 1450-1457.*
8. *Andersson M et al (2002). Oat bran stimulates bile acid synthesis within 8 h as measured by 7-hydroxy-4-cholestone-3-one. American Journal of Clinical Nutrition 76, 1111-1116.*
9. *Welch RW (1998). Oats - a multifunctional food. In: Functional Foods: The Consumer, The Products and the Evidence. Ed. MJ Sadler & M Saltmarsh. Cambridge: Royal Society of Chemistry, pp. 99-105.*
10. *Peterson DM (2001). Oat antioxidants. Journal of Cereal Science 33, 115-129.* ■

Benefits of a Mediterranean diet

Since the influential studies of Keys et al, the Mediterranean diet has represented a type of panacea for heart health. Yet, much of the evidence rests on the findings of observational studies and subjective reviews.

A systematic review by Serra-Majem[1] has now brought together 35 human intervention studies to evaluate the direct effect of Mediterranean diets on CVD. Thirty of these looked at CVD risk factors, while the rest had incidence or mortality as their end points. Sample sizes ranged from 11 to 13,000, with the majority involving less than 100 subjects. There was some difficulty in comparing studies because the definition of a Mediterranean diet varied.

At an international conference in 1993, food patterns reflecting the Mediterranean diet were defined (Table 1). To this is often added "regular consumption of oil-rich fish".

The results of the review showed that study findings were consistent. Mediterranean diets lowered triglycerides, total cholesterol and LDL cholesterol, and increased HDL cholesterol. In some studies, but not others, there were improvements in endothelial function, plasma antioxidant capacity, insulin resistance and metabolic syndrome. Despite Mediterranean diets providing more total fat than traditional low fat diets, body weight in the intervention groups did not appear to be adversely affected.

There have been concerns that Western dietary recommendations, based on the low fat, high starch model, are unable to replicate the benefits attributed to Mediterranean diets. Indeed, in some interventions, low fat diets have caused worse lipid profiles e.g. higher triglycerides and lower HDL cholesterol. Willett[2] argues that the accepted heart health diet fails to take proper account of the adverse effects of trans fats or the beneficial effects of monounsaturated fats, fruits and vegetables, fish and wholegrains & fibre.

Studies that have compared Mediterranean diets with low fat diets have found significant differences. In a Spanish multi-centre randomised trial with 770 adults[3] three diets were compared;

- low fat diet
- Mediterranean diet with nuts (30g/d)
- Mediterranean diet with olive oil (1L/wk)

Subjects following either Mediterranean diet demonstrated reductions in fasting glucose, C-reactive protein, blood pressure and the LDL-HDL ratio. Body weight reduced slightly on all diets and there were modest reductions in daily energy intake.

In a French trial[4] – the randomised *Lyon Heart Diet Study* – a Mediterranean-style diet was compared with a low fat diet in survivors of acute myocardial infarction (AMI). The Mediterranean diet contained around 0.6% energy from alpha-linolenic acid. After 4 years of follow-up, there was a 50-70% lower risk in the recurrence of AMI in patients following the Mediterranean diet compared with the low fat diet. Willett used data from the North American prospective Nurses' Health Study to estimate the benefit of widespread adherence to a Mediterranean diet. The findings suggested that over 80% of premature CVD deaths could have

Table 1 - Definition of a Mediterranean diet

Abundant intake of plant foods, e.g. cereals, beans, fruits, vegetables, nuts and seeds
Minimally processed, seasonal, fresh foods
Olive oil as principle source of dietary lipids
Dairy products consumed in low to moderate amounts
Fewer than four eggs per week
Red meat consumed infrequently and in small amounts
Wine consumed in low to moderate amounts, generally with meals
Sweets based on olive oil, nuts and honey consumed infrequently

International Conference on the Diets of the Mediterranean (1993).

been prevented if all subjects had followed this regime. This seems an unrealistic figure until one takes into account the very low prevalence of CVD in traditional Cretan communities; 80-90% lower than in the US. Taken together, these studies endorse more widespread use of the Mediterranean diet. The heart health benefits relate to various nutrients and plant compounds working synergistically rather than to one specific food or nutrient. Some authors appear convinced that Western dietary guidelines need to be better aligned with the Mediterranean model.

Comment

Changing to a Mediterranean diet, as detailed in Table 1, seems to offer clear heart health benefits. However, for the large majority of the people in Europe, this would require a complete change of the diet, which may be very difficult to achieve. It might be useful to change to a Mediterranean diet using a step wise approach. Research is needed to help to estimate the likely impact of individual aspects of the Mediterranean diet. This will assist health professionals in giving appropriate evidence-based advice, which takes into account feasible dietary changes.

References

1. *Scientific evidence of interventions using the Mediterranean diet: A systematic review. Serra-Majem et al (2006). Nutrition Reviews 64, S27-S47.*
2. *The Mediterranean diet: science and practice. Willett (2006). Public Health Nutrition 9,105-110.*
3. *Effects of a Mediterranean-style diet on cardivascular risk factors: A randomized trial. Estruch et al (2006). Annals of Internal Medicine 145, 1-11.*
4. *he Mediterranean-style diet for the prevention of cardiovascular diseases. Lorgeril et al (2006). Public Health Nutrition 9, 118-123.*

Fibre lowers cardiovascular disease risk

Cardiovascular diseases (CVD) are the leading cause of death in developed countries and are increasingly affecting the developing world. A number of dietary factors are known to affect CVD risk, including saturated fats, long-chain omega-3 fats, fruit, vegetables, trans fatty acids and fibre.

A review has focussed on the impact of fibre on CVD[1]. Fibre is expressed analytically as that which is insoluble in water, i.e., cellulose, hemi-cellulose and lignin, or soluble in water, i.e. pectins, gums, storage polysaccharides and ß-glucan. The major sources of fibre in Western diets are cereal products, fruits, vegetables and legumes. There is consistent evidence that high fibre diets have a positive impact on CVD. More specifically, it is found that soluble fibre lowers total and LDL cholesterol. One review suggested that 3g/d of soluble fibre was associated with a 0.13 mmol/l reduction in total cholesterol concentration. Soluble fibre has also been found to decrease small, dense LDL particles, which are thought to be particularly atherogenic. The likely mechanism involves the gel-forming properties of soluble fibre which help to trap bile acids in the small intestine. Excretion of bile acids then increases, resulting in bile acid synthesis and greater cholesterol uptake from plasma. The accumulated evidence led the FDA to authorize a health claim for oat fibre in 1997.
Interestingly, the data for the CVD benefits of soluble fibre are more consistent than those supporting the benefits of fruits and vegetables. Insoluble fibre intake has also been linked to improved lipid profiles. The mechanism behind this effect could be explained by diet compensation, i.e. high fibre foods replacing foods high in fat and saturates.

Another risk factor that may be ameliorated by dietary fibre is insulin resistance. Fibre appears to delay gastric emptying and slow the rate of post-prandial glucose absorption. Less insulin is then produced in response. One study on soluble fibre suggested greater activity of GLUT4 receptors, while another found increases in pro-glucagon circulation. Both of these metabolic factors would improve glucose tolerance. In order to achieve a consistent effect on glucose metabolism, around 4-14g soluble fibre per meal may need to be consumed. This is higher than current intakes for the majority of people.

Ongoing research is revealing additional metabolic benefits linked to fibre consumption but the evidence for these relies upon observational studies. While dietary fibre intake is often found to relate inversely to blood pressure, interventions with fibre supplements have failed to find an effect. The same is true for clotting factors. One area of interest is C-reactive protein, a marker of inflammation that relates to CVD risk. In one large prospective, observational study[2], diets high in soluble and insoluble fibre related significantly to lower odds ratios for C-reactive protein. The relationship was dose dependent and the best effect was seen for soluble fibre intakes over 7.8g/d. A trial of soluble fibres[1] found lower levels of C-reactive protein at daily intakes of 9.8g/1000 kcal.

The authors of these papers conclude: ' while soluble fibre has beneficially affected CVD risk factors, food sources of mainly insoluble fibre, primarily contributed by cereal products, have been the most consistently associated with lower incidence rates of CVD. It is suggested that, to support CVD prevention, a high fibre diet should be consumed providing a mixture of different fibre types'.

Comment

Epidemiological data have been consistent in reporting associations between fibre and blood lipid profiles. Intervention and mechanistic studies have backed this up, particularly for soluble fibre. A major step towards a heart health diet is increasing daily fibre intake to the recommended 25-30g, something which the majority of people have yet to achieve. As a second step increasing the consumption of soluble fibre would be expected to help control cholesterol levels.

References

1. *Fiber and cardiovascular disease risk: How strong is the evidence? Erkkilä & Lichtenstein (2006). Journal of Cardiovascular Nursing 21, 3-8.*
2. *Association between dietary fiber and serum C-reactive protein. Ma et al (2006). American Journal of Clinical Nutrition 83, 760-766.*