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Original article

Clinical Nutrition University. The place of nutrition in the prevention of cardiovascular diseases (CVDs)

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SUMMARY

CVDs, including coronary heart disease (CHD) and stroke, currently represent the major causes of mortality and morbidity all over the world. In Europe, CVDs are responsible for 43% of deaths in men and 55% in women and for 30% of all deaths before the age of 65 years. CVD burden could be substantially reduced by early diagnosis and appropriate measures, since atherosclerotic lesions may be substantially improved in response to measures taken.

CVD results from a combination of genetic and environmental factors; some factors vary between different ethnic groups. Plasma lipid profile is an important, but certainly not the only, risk factor for CVD.

Prevention includes healthy lifestyle: no smoking, weight control, physical activity, and healthy dietary intake; control of blood pressure, plasma glucose, and inflammation is important.

The Mediterranean diet is a good example of healthy dietary pattern. Components of the Mediterranean diet may be adapted to nutritional habits of different countries, taking into account differences of taste and culture. The benefits of a healthy lifestyle exceed, but are additive to, those of medical treatment.

Learning objectives

- To demonstrate the importance of cardiovascular diseases (CVD) in morbidity and mortality in the world.
- To review the major risk factors for CVD.
- To give an overview on the importance of lifestyle components, particularly diet, in the modification of different risk factors, and in the prevention of CVD.
- To review the mechanisms through which nutrition may affect CVD.
- To provide global and specific recommendations on healthy diets.

1. Rationale for prevention of CVD

CVDs, including coronary heart disease (CHD) and stroke, currently represent the major causes of mortality and morbidity all over the world. In Europe, CVDs are responsible for 43% of deaths in men and 55% in women and for 30% of all deaths before the age of 65 years.[1]

In 2000, CVDs also accounted for 22% of all disability adjusted life years (DALY's) lost in Europe.

Eighty percent of CV accidents could probably be avoided by lifestyle adjustment (weight control, smoking abstinence, physical activity, and a healthy diet), together with proper management of clinical and biological risk factors. Fig. 1 (data taken from European statistics[2,3]) represents the evolution of cardiovascular mortality in several European countries. In developed countries, there is clearly a decreasing trend as a reflection of appropriate measures. However, in countries with a more recent access to a Westernized way of life, the tendency is towards an increase. This corresponds to the trend observed all over the world. In developing countries, there is a sharp contrast between a high CVD incidence in cities in relation to urbanisation, and a lower CV mortality rate in the rural areas.[4] Most of the current research efforts have been aiming to identify and treat individual-level risk factors of CVD. Despite important achievements, the inequalities continue to persist.

The projection for the major causes of deaths all over the world in 2020 suggests a further rise in CVD mortality and morbidity, mainly in developing countries.[5] In addition, the epidemics of obesity and the frequently associated metabolic syndrome raise

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major concerns for the immediate and mid-term future, even in developed countries.[6,7]

2. The atherosclerotic lesion

The major typical feature of CVD is the atherosclerotic plaque, an inflammatory lesion which develops insidiously around cholesterol depots in the intima of the arterial wall over many (20–30) years.[8–10] This is due to the fact that particles carrying cholesterol in plasma may cross the endothelium (inner unicellular layer of the vessel) and enter the intimal space of the arterial wall[11]; while low-density lipoproteins (LDL) may in some conditions be endocytosed by intimal macrophages and deposit cholesterol in the intima. This is associated with further recruitment, in the intima, of macrophages and lymphocytes, and increased production of inflammatory mediators. Another important factor in the initial phase of the process is a dysfunction of the endothelium induced by atherogenic lipoproteins and hypertension; the endothelium loses its capacity to produce nitric oxide (which induces vasodilatation, but also protects against arterial remodelling and platelet aggregation) and secretes free radicals and inflammatory mediators. The inflammation may thicken the arterial wall locally and reduce the lumen (creating a stenosis), but may also erode the lesion; interaction between blood platelets, lipids, and different mediators quickly induce thrombus formation at the site of erosion. In fact, coronary artery occlusion is much more often caused by a thrombus after rupture of a fragile lesion than by progressive stenosis.

Cholesterol deposition and development of inflammatory lesions are prevented by high-density lipoproteins (HDL) which ensure a reverse cholesterol transport to the liver (and glands producing steroid-derived hormones) and reduce inflammatory and peroxidative reactions.

It is important to understand that endothelial dysfunction and inflammatory reactions may be corrected by appropriate lifestyle and therapeutic measures, causing the atherosclerotic lesion to be reversed, modified or stabilised. As a corollary, the vast majority of CVD accidents can and should be prevented.

3. Risk factors for cardiovascular disease events

Plasma cholesterol concentration has long been considered as the major (if not the sole) risk factor for CVDs. Indeed, Fig. 2 (adapted from [12]) is often used to confirm the strong correlation between total cholesterol concentration and coronary heart disease.

However, one should also consider the huge differences in the rate of CVD mortality between countries with similar average cholesterol levels (e.g. ~220 to 230 mg/dl or 5.5–5.6 mmol/l). This supports the role of other factors. Indeed, the initiation and development of the atherosclerotic lesion are related to a combination of several independent risk factors.

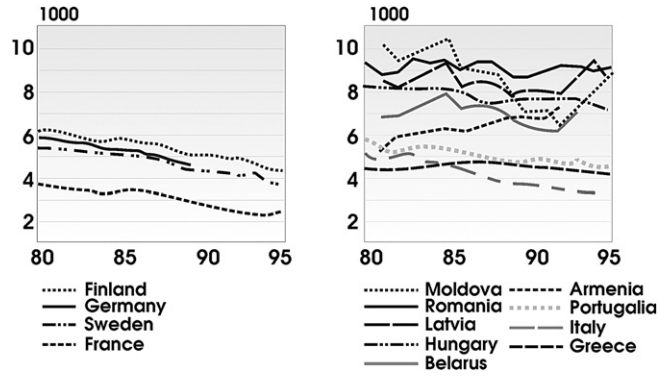

Fig. 1. Mortality from CVD in Europe (1980–1995) (data taken from refs. [2,3]).

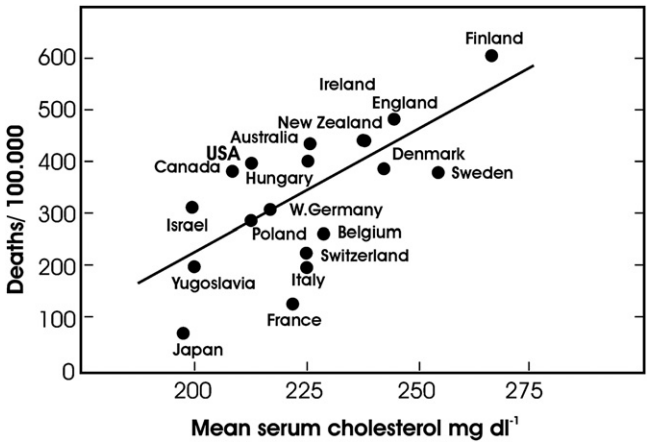

Fig. 2. Relationship between coronary death rates and mean serum cholesterol in 19 European countries and Japan.[12]

Some factors (genetic background, familial and personal history, gender) may not be modifiable while others may be corrected or improved (Table 1).

3.1. Serum lipids

Since cholesterol deposition in the arterial wall is a key and early step in the initiation of the atherosclerotic process,[13,14] decreasing the number of LDL particles (and/or avoiding their retention in the intimal space) has been a primary target (Table 2).[15]

This is achieved by improving the number and activity of LDL receptors largely located in the liver; drugs such as statins efficiently stimulate LDL removal, but nutrition may also activate or impede receptor activity. Also, evidence that some LDL, modified by peroxidation, glycation, addition of adducts, or presence of additional apoprotein(a) have a markedly increased atherogenic potential[16,17] has drawn attention to the role of LDL size and composition, as well as to the impact of associated pathologies such as diabetes mellitus, chronic renal failure, chronic inflammatory diseases.[18] Tobacco smoking is a major risk factor and air pollution has also been implicated; again, nutrition may affect LDL atherogenicity.

Attention is currently paid not only to decreasing LDL-cholesterol, but also to increasing the number of HDL particles; this can be achieved by lifestyle adjustments: weight[19] control, physical activity, non-smoking and proper nutrition.[20,21] A high concentration of plasma triglycerides is recognised as a direct risk factor (increased coagulation, impaired endothelial function, etc)[22] but may also have indirect effects by decreasing HDL level and increasing LDL atherogenicity via formation of atherogenic small dense (sd) LDL.

3.2. Hypertension

Hypertension increases the entry of LDL particles into the intima and impedes their return into the circulation; it also raises peroxidative damage, inducing endothelial dysfunction and LDL modifications; dietary manipulation may help reducing blood pressure, e.g. by reducing weight and salt intake.

3.3. Inflammatory processes

In different organs and at distant sites from atherosclerotic lesions, inflammation is now recognised as an aggravating factor,