ABC of Nutrition

A STEWART TRUSWELL

REDUCING THE RISK OF CORONARY HEART DISEASE

Mortality (per 100 000) from coronary heart disease age 45-54 1978

	Men	Women
Finland	392	49
Scotland	353	79
England and Wales	272	50
USA	272	67
Sweden	160	26
Greece	102	14
Japan	28	7

National Heart Foundation of New Zealand -
Coronary Heart Disease. Auckland: National
Heart Foundation, 1983

Coronary heart disease, the leading cause of death in Britain and other Western countries, is only one tenth as common in industrial Japan and rare in most Third World communities. Its incidence is environmentally determined because immigrant groups soon take on the incidence of their host country and there have been large changes over time in mortality from coronary heart disease. It was uncommon before 1925 and then rose steadily except for a dip in Europe in the second world war. It is now declining in the USA and Australia, static in Britain and most of western Europe, and increasing in eastern Europe.

Coronary heart disease is a multifactorial disease, but diet is probably the fundamental environmental factor. The pathological basis is atherosclerosis, which takes years to develop. Thrombosis superimposed on an atherosclerotic plaque, which takes hours, usually precipitates a clinical event. Then whether the patient dies suddenly, has a classical infarct, develops angina, or has asymptomatic electrocardiographic changes depends on the state of the myocardium. Each of these three processes is likely to be affected by different components in the diet.

Atherosclerosis and cholesterol

The characteristic material that accumulates in atherosclerosis is cholesterol. This and other lipids in the plaque come from the blood, where they are carried on lipoproteins. In animals atheroma can be produced by raising plasma cholesterol concentrations with high fat diets.

People with familial increases in low density lipoprotein (LDL) cholesterol (type II hyperlipidaemia) tend to have premature coronary heart disease: this is accelerated in homozygotes, who all develop clinical coronary heart disease before 20 years of age.

Over 20 prospective studies in 14 countries all show that high plasma LDL (or total) cholesterol, arterial hypertension, and cigarette smoking are the big three risk factors for coronary heart disease.

The mean plasma total cholesterol of healthy adults ranges widely in different communities, from 3.0 mmol/l (120 mg/100 ml) in Bushmen and Masai to 7.2 mmol/l (280 mg/100 ml) in Finland a few years ago. None of the other common biochemical plasma variables show this variation. Only in countries whose average total cholesterol exceeds 5.2 mmol/l (200 mg/100 ml)—as in Britain—is coronary heart disease common.

Some countries where expert committees have recommended reductions in plasma cholesterol

Australia
Canada
France
Netherlands
New Zealand
Scandinavia
United Kingdom
United States of America
West Germany

Individuals at increased risk

Family history of coronary heart disease

Plasma cholesterol >6.4 mmol/l (250 mg/100 ml)

Hypertension

Smoking

Obesity

Angina

ECG abnormalities

To convert plasma cholesterol from mg/100 ml to mmol/l divide by 39. 1 mmol/l = 39 mg/100ml

Countries at increased risk—A major strategy of primary prevention for a whole country—or for the people in a district, or even in a practice—is to advise people to modify their diet to achieve a modest reduction in plasma total cholesterol. This can be done gently and inexpensively.

Individuals at increased risk—For individuals at increased risk of coronary heart disease a stricter cholesterol lowering diet should be prescribed and drugs may be indicated if hypercholesterolaemia does not fall satisfactorily.

Reduced saturated fat and cholesterol, partially replaced by polyunsaturated fat

Sample daily menu

Breakfast: Grapefruit or orange juice
Cereal or porridge with skim milk
Toast, polyunsaturated margarine, and marmalade or jam
Coffee or tea with skim milk (and sugar)

Mid-morning: Coffee or tea with skim milk (and sugar)

Lunch: Sandwiches made from bread and polyunsaturated margarine and filled with sardines, tuna, salmon, tomato, cucumber, Marmite, etc
Salad (no dressing)
Fruit
Tea with skim milk (and sugar)

Mid-afternoon: Tea with skim milk; piece of fruit

Dinner: Cooked poultry, fish, or lean meat, which may be fried in polyunsaturated oil
Boiled potatoes or rice or pasta
Vegetables, raw or cooked
Alcoholic drink if desired
Bread or plain roll
Fruit salad or allowed dessert
Coffee with skim milk (and sugar)

Bedtime: Tea with skim milk or juice

General guidelines

- Avoid butter and hydrogenated margarine, lard, and suet. Use moderate amounts of polyunsaturated margarines (P:S 2 or 3).
- Avoid cream and ice cream. Use skim or semiskim (2% fat) milk instead of standard, full cream milk.
- Eat less meat; more poultry (not the skin) and fish. Choose lean meat and remove visible fat. Grill rather than fry. Avoid sausages and meat pies.
- Reduce egg yolks.
- Keep cheese intake down. Use cottage cheeses.
- Restrict intake of cakes, pastries and biscuits (unless made at home with polyunsaturated fats), and chocolates.
- Use "polyunsaturated" (not just "vegetable") oils for cooking—for example, maize or sunflower oils.
- Eat more vegetables and fruits of all kinds.
- Cereals and products ad lib (bread, rice, pasta, breakfast cereals, oatmeal) and tapioca, cornflour, and sago.

	Saturated					Other poly-unsaturated P:S*
	C4-12	C14-18 (myristic, palmitic, stearic)	Mono-unsaturated	Linoleic		
Butter, cream, milk	13	48	30	2	1	0.05
Cocoa butter	—	61	36	3	—	0.05
Beef	—	48	48	2	1	0.06
Coconut oil	58	31	8	2	—	0.1
Bacon and pork	—	42	50	7	1	0.2
Palm oil (used in ice cream)	—	45	45	9	—	0.2
Margarine (old style, hard)	3	37	33	12	1	0.3
Chicken	—	34	45	18	2	0.6
Olive oil	—	14	73	11	1	0.9
Groundnut oil	—	15	53	30	1	2.1
Fish oil	—	23	27	7	43†	2.2
Margarine, polyunsaturated (soft)	2	21	22	52	1	2.3
Corn (maize) oil	—	14	31	53	2	3.9
Soya bean oil	—	14	24	53	7	4.3
Sunflower seed oil	—	12	33	58	—	4.8

* These are in ascending order of ratio of polyunsaturated to saturated fats, but this is not the only consideration in choosing dietary fats and oils.

† Includes varying amounts of eicosapentaenoic acid (depending on species).

Fat—The major dietary influence on plasma LDL and total cholesterol is the amount and type of fat. The elevating effect comes mostly from saturated fatty acids, palmitic and myristic. (There is more palmitic in most diets.) Polyunsaturated fats, chiefly linoleic, tend to lower plasma LDL and total cholesterol but their effect is only half as strong as the elevating effect of palmitic acid. Monounsaturated fats have little effect either way. These effects take about 10 to 14 days to appear and last for years provided people stick to the diet.

Dietary cholesterol—Other dietary components have smaller effects. Dietary cholesterol is confused by the layman and the media with plasma

[Note that 37 out of 44 are ± 20 mg/100 ml, while 7 out of 44 appear to be outliers]

Bronsgeest-Schoute, *et al.* *Am J Clin Nutr* 1979;32: 2193-7

cholesterol. Eating cholesterol or eggs (the richest common source of cholesterol) in general has less effect on plasma cholesterol values than eating saturated fats, but there seems to be individual sensitivity. One egg a day (250 mg cholesterol) has little effect on plasma cholesterol in about 80% of people, but a minority respond. There is no easy way yet of predicting responders.

Reduced energy intake—Loss of weight from reduced energy (calorie) intake can lower plasma cholesterol concentrations, especially in people with an increase in both plasma cholesterol and triglycerides. But this response is not always seen: in anorexia nervosa the plasma cholesterol is often raised.

Dietary fibre—The effect of dietary fibre depends on the type. Wheat fibre (bran or wholemeal bread) does not decrease plasma cholesterol values but viscous types, like pectin and guar, in large doses produce major reductions. Even 5 g/day may reduce cholesterol concentrations by about 5%. Vegetables and fruits contain around 1% pectin, and so four portions would provide about 5 g/day. These foods are also likely to displace foods containing fat.

High density lipoprotein (HDL) cholesterol in the plasma is a protective factor. It helps to mobilise cholesterol deposits from the tissues. HDL does not explain the big differences between countries; its mean concentration is no higher in countries with little coronary heart disease. But in a country with a high incidence of coronary heart disease individuals with above average HDL have a reduced risk. Dietary fats have small effects on HDL. Concentrations of these lipoproteins are higher in women than men, decreased in obesity, and increased by alcohol consumption (though in moderate amounts this appears not to raise the protective subfraction HDL₂).

Thrombosis

It seems likely that diets which reduce the tendency to thrombosis may be similar in some ways—for example, low saturated fat—and different in others from those that lower plasma cholesterol. We know much less about the former but their potential importance is that they might reduce the risk of a first or a recurrent coronary attack after being eaten for only a few days. The best diet to prevent coronary heart disease in young adults is one that lowers plasma LDL, but in someone who already has manifest coronary disease and in old people the best diet—which now we can only see through a glass darkly—is one that makes thrombosis less likely.

The effects of diet on thrombosis can be studied only indirectly: in animals, epidemiologically, or by measuring components of the process in blood samples taken from patients, such as platelet stickiness, clotting factor concentration, or fibrinolysis.

Eicosapentaenoic acid (C20:5) and related very long chain marine polyunsaturated fats are the most potent inhibitors of thrombosis found in ordinary diets. Eicosapentaenoic acid appears to act as a competitive antagonist of thromboxane A₂ formation. It is found in fatty fish like pilchards and mackerel (and cod liver oil) and may explain the Eskimos' freedom from thrombotic diseases.

Other polyunsaturated oils—those found in seed oils like linoleic acid—have a smaller inhibiting effect.

High fat meals (and lipaemia) favour thrombosis and so does a high plasma cholesterol concentration.

There are scattered reports suggesting that individual foods may promote fibrinolysis—for example, onions—or inhibit it—for example, fats—but a systematic re-examination of this area is overdue.

Myocardium

Most unrefined foods provide moderate amounts of potassium and magnesium. There are no rich sources of potassium. Potatoes are about the best, and legumes. Meat, fish, fruit, and vegetables all provide moderate amounts of potassium.

Magnesium occurs in moderate amounts in cereals (preferably wholegrain) and vegetables (in the chlorophyll). Fats and sugar contain negligible amounts of both elements.

The myocardium is more susceptible to damage, prone to arrhythmias, and sensitive to digoxin in patients with low plasma potassium concentrations. It has been suggested that sudden death after exercise might sometimes be due to the plasma potassium lowering action of catecholamines. More chronic hypokalaemia is usually secondary to increased losses—for example, from diuretics. An increased potassium diet may help in such potassium losing states, but hypokalaemia is rarely due to primary dietary deficiency.

Magnesium depletion likewise is nearly always due to abnormal losses—for example, through prolonged diarrhoea, malabsorption, excessive lactation, diabetic ketoacidosis, or thiazide diuretics. Hypomagnesaemia is usually accompanied by hypocalcaemia or hypokalaemia, or both. Patients with it are liable to various fast cardiac arrhythmias and it has been reported that a minority of patients presenting with acute myocardial infarction and ventricular arrhythmias had hypomagnesaemia.

Drinking water—Total cardiovascular mortality has, as a rule, been found to be lower in districts where the drinking water is hard than in soft water areas. In Britain the latter are mostly in the old industrial areas of the north and west so it is difficult to disentangle socioeconomic factors. If the water effect is real no component has been consistently associated in different countries. Calcium appeared protective in a large British study, not magnesium. The opposite was found in a large Canadian study. The only practical conclusion as yet is that people in hard water districts would be better not to install water softeners for their drinking water.

North Karelia

North Karelia is the most easterly county of Finland. In 1971 it had the highest (age standardised) death rate from coronary disease in the world. The people were worried and asked the government for help. A community programme was set up. Everyone was advised to stop smoking, eat less fat and more vegetables, avoid obesity, and have their blood pressure checked. By 1979 coronary mortality had fallen by 24% in men and 51% in the women, a significantly greater fall than the general decline in coronary deaths in Finland during the same period.

Coronary heart disease is not an inevitable consequence of either aging or industrialisation. It appears to be largely preventable and the World Health Organisation recommends preventive community action in countries with a high incidence. The role of personal physicians is vital here. Through talking to patients and by personal example and leadership they play a major part in increasing public awareness and in modifying attitudes and behaviour that affect health.

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Further reading
Truswell AS. End of static decade for coronary heart disease. *Br Med J* 1984;288:509-10.

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A patient who is slim, fit, and a normotensive non-smoker took an oral contraceptive until the age of 49 when she was changed to a progesterone only preparation (Femulen). Since taking this preparation she has not menstruated and is fearful of becoming pregnant. Will she be able to tell that she has reached the menopause? How effective is Femulen in this age group, and is it as effective as other preparations in preventing menopausal symptoms?

Bleeding irregularities with low dose progestogen only contraceptives are almost exclusively in the form of intermenstrual bleeding and "amenorrhoea does not present a problem" (though the question does not make clear for how long it had been present). The patient has probably therefore reached the menopause: this could be confirmed biochemically by finding a raised serum follicle stimulating hormone concentration. Broadly speaking, menopausal symptoms are related to oestrogen deficiency and will not therefore be helped by progestogen only preparations.—S L JEFFCOATE, professor of biochemical endocrinology, London.

1 Diczfalusy E, Landgren B-M. Low-dose progestogens and pituitary, ovarian and endometrial function. In: Benagiano G, Zulli P, Diczfalusy E, eds. *Progestogens in therapy*. Serono symposia publications, Vol 3. New York: Raven Press, 1983:133-56.

Recently three famous British actors have died of Parkinson's disease. Is this coincidence or could there be some common environmental and occupationally related factor?

The incidence of most neurological complaints such as parkinsonism, multiple sclerosis, and motor neurone disease among actors does not appear to differ appreciably from that of the general population. Parkinsonism accounts for between 1 and 1.6 per thousand deaths with the incidence rising with age. There could be a pre-Parkinsonian personality¹—for instance, a low incidence of cigarette smokers—but such personality patterns, as with migraine, can be misleading. Genetic factors may influence the disease but Calne and Langston suggest that in most cases the cause may include environmental factors, possibly toxic, superimposed on a background of slow, sustained neuronal loss due to advancing age.²—E M R CRITCHLEY, consultant neurologist, Preston.

1 Todes CJ, Lees AJ. The premorbid personality of patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1985;48:97-100.

2 Calne DB, Langston JW. Hypothesis: aetiology of Parkinson's disease. *Lancet* 1983;ii:1457-9.