The science behind lifestyle risk factors for **cardiovascular disease**

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When it comes to providing advice to patients about cardiovascular risk, most doctors understand that the cornerstones of lifestyle advice are: stopping smoking, improving diet, exercise and losing weight. What might be less well known is how each of these factors contributes to cardiovascular risk.

This article examines how modifiable lifestyle factors contribute to cardiovascular risk, and some of the benefits of lifestyle intervention.

Smoking

Some of the advantages of smoking cessation are almost immediate, while others take time. After a myocardial infarction stopping smoking is potentially the most effective of all preventative measures.[1] In some studies platelet aggregation has been demonstrated to improve in two weeks following smoking cessation. For a person who stops smoking, after five years their personal CVD risk is the same as a person who has never smoked.

Smoking and CVD

Approximately 22% of New Zealanders aged 15 to 64 years are cigarette smokers.[3] Cigarette smoking is estimated to cause approximately 5000 deaths per year with about 70% of these being from CVD. There are approximately another 400 deaths per year attributable to second hand smoke.[4] Cigarette smoking is associated with an increased rate of myocardial infarction and fatal CVD. Active smokers have approximately an 80% higher risk of CVD compared to non-smokers, while non-smokers who are regularly exposed to environmental smoke have a 30% increase in CVD.[5]

Smoking increases the risk of CVD in two key ways: by increasing the rate of atherosclerosis and by increasing the incidence of thrombosis. It is widely believed that these two mechanisms work together, with coagulation being initiated after exposure of blood to tissue factors present in artheromatous plaques.

There is a range of pathophysiology caused by the toxic effects of smoking. These changes do not occur independently of each other, and in some cases effects can cascade. The effects are dose related, depending on the degree of cigarette exposure. When a person stops smoking the decrease in thrombotic risk is almost immediate, whereas the effect on atherosclerosis takes some years to reverse.

Some of the pathological consequences of smoking include:[2]

- Damage to the endothelium in vessel walls. This has been demonstrated in the endothelium of the macrovascular bed of the coronary arteries.
- Altered lipid profile (increases cholesterol, triglyceride and LDL, but decreases HDL). Smoking has been shown to immediately increase serum free fatty acids,[6] with long term smoking leading to hyperlipaemia.
- Inflammation in the blood and vessel walls. The leucocyte count of smokers is estimated to be 20 – 25% higher than non smokers.
- Increased prothrombotic factors and decreased fibrinolytic factors. People who smoke have been demonstrated to have higher fibrinogen (prothrombotic) levels than non-smokers, but these will return to normal if the person stops smoking.
- Platelet dysfunction (increased aggregability). People who smoke show increased potential for platelet aggregation, lower platelet survival rate and increased excretion of thromboxane metabolites (involved with platelet release).

Composition of cigarette smoke

Cigarette smoke can be divided into two phases: a tar phase and a gas phase. The tar phase is the material predominantly trapped in the filter. The gas phase is the material that passes through.[2]

Cigarette smoke intentionally inhaled is known as mainstream smoke, while sidestream smoke is the smoke emitted from the burning end. Sidestream smoke has not passed through a filter and therefore contains a higher proportion of the tar phase, which contains more harmful free radicals than the gas phase, therefore is more hazardous.

It is estimated that two thirds of the smoke from a burning cigarette is not inhaled and enters the environment. Environmental smoke is a mixture of sidestream smoke and exhaled main stream smoke and inhalation of this is known as passive or second-hand smoking.

Nutrition

Most guidelines advise overall healthy eating plans that include a variety of fruits, vegetables, grains, low-fat or non-fat dairy products, fish, legumes, poultry and lean meats. There is a wealth of information of the health benefits in following this type of diet, including reductions in cardiovascular risk but there is a lack of information about the biological mechanisms by which this type of diet contributes to decreased risk.

Fats

The traditional New Zealand diet is high in saturated and total fat and this is considered a key contributor to CVD.[7] A diet rich in saturated and trans fats is associated with adverse changes in lipid profile, including increased levels of LDL and decreased levels of HDL (Table 1).

Saturated fats are found in animal products such as butter and fatty meat. Leaner cuts of meat should be used and butter should be replaced with margarines and oils which contain unsaturated, polyunsaturated and monounsaturated fats. Olive and canola oils and nuts are monounsaturated while soybean and sunflower oils are sources of polyunsaturated fat.

Trans fats have the most adverse effect on the lipid profile. They are oils that have been hydrogenated to turn them into semi-hard fats. As awareness of the adverse effects of trans fat has increased over recent years, a number of food manufacturers in New Zealand have voluntarily lowered the trans fat in their food production.

Omega-3 and omega-6 are essential fatty acids, which may have a beneficial effect on lipid profile. They are obtained from polyunsaturated fats. Fish is the best source of omega-3, while omega-6 is found in corn, soybean and safflowers oils.

Sterols are a component of the structure of plant cell membranes. When consumed they block absorption of cholesterol into the blood stream and can be useful in lowering LDL levels.

Fruit and vegetables

Probably the most consistent dietary advice given over recent years is the “5+ a day” campaign to encourage consumption of five or more portions of fruit or vegetables per person per day.

Table 1: Fats and their effect on lipid profile

Type of fat	Dietary sources	Effect on LDL	Effect on HDL	Effect on triglyceride
Trans	Commercially fried foods, prepared snacks and baked goods	Increases	Slight decrease	No effect
Saturated	Red meat, cheese, butter, palm oil	Increases	No effect	No effect
Monounsaturated	Nuts, olives, avocado, olive and canola oils	Decreases	No effect	No effect
Polyunsaturated **Omega-6**	Corn, soybean and safflower margarine and oils	Decreases	May decrease	Unknown
Polyunsaturated **Omega-3**	Salmon, mackerel, herring, flax seed, walnuts, soybean	Variable	No effect	Decreases
Sterols	Margarine with added plant sterols	Decreases	No effect	No effect

The beneficial effect of fruit and vegetables is thought to be associated with components such as fibre, antioxidants, potassium and folate. Higher fruit and vegetable consumption can often be associated with other healthy behaviours such as not smoking and exercising more frequently.

There is a lack of consensus on whether fruit juice can be included as a serve of fruit or vegetables. Fruit juice offers no nutritional advantage over whole fruit, and may lack the fibre contained in whole fruit.

Antioxidants have been shown to protect cells from the effects of free radicals. The main danger of free radicals is the damage they can do when they react with important cellular components such as DNA, or the cell membrane. A higher intake of certain antioxidants has been shown to lower the incidence of heart disease. Polyphenols are the most abundant antioxidants in the diet and include vitamin E, beta-carotene and vitamin C. The main dietary sources are fruits, dry legumes, cereals, chocolate and plant-derived beverages such as fruit juices, tea, coffee, red wine.

Salt

Hypertension is a well recognised risk factor for cardiovascular disease. There is a strong association between hypertension and salt intake. Controlling salt intake can lower blood pressure, as well as lowering the risk of a cardiovascular event.[8] It is thought that increased sodium intake also adversely effects the cardiovascular system independently of blood pressure.

The way that food products are labelled may make it more difficult for people to understand how much salt they are eating.[9] Dietary recommendations are given in terms of maximum levels of salt (sodium chloride) intake, whereas the nutritional information on most food packaging tends to be indicated by sodium content. To estimate sodium chloride content, the sodium content has to be multiplied by 2.5. People should not consume more than 6g of salt per day.

Fibre

Dietary fibre is either soluble or insoluble, with both types derived from plants. Insoluble fibre (e.g. wheat, bran, potato skin) passes through the body mostly unchanged but absorbs water and swells which helps to soften stool and increase bulk, and reduce gut transit time. Soluble fibre (e.g. peas, apples, carrots, oats) is broken down once it reaches the large bowel where gut flora feed and multiply contributing to softer, bulkier stools.

The protective mechanism of dietary fibre in CVD risk is unknown, but individuals that consume higher levels of dietary fibre have:[10]

- Lower BMI and less likelihood of being overweight
- Reduced risk of hypertension
- Decreased levels of apolipoprotein (apo) B, cholesterol and homocysteine.

It is recommended that people should consume at least 25 g/day of dietary fibre, although levels of up to 35g/day can be expected to provide even more benefit.

Alcohol

Small amounts of alcohol may protect against CVD. This is distinct from the effect of antioxidants in some drinks such as red wine. The protective effect of alcohol is primarily explained by an increased HDL, decreased platelet aggregability and promotion of fibrinolysis, although there are probably a number of other mechanisms. This may include the possibility that people who only drink moderately have generally healthier lifestyles.[11]

Detrimental alcohol-related effects begin to counteract the benefits from alcohol consumption above an intake of around 10g of alcohol per day (one standard drink).[12] Therefore while light to moderate drinking is unlikely to cause significant harm, non-drinkers should not be advised to take up drinking to improve their cardiovascular health.

Exercise

Physical exercise has been shown to provide a wide variety of benefits for all individuals, whether or not they have had a previous cardiac event. A sedentary lifestyle is said to carry approximately the same risk for the development of coronary artery disease as the more traditional risk factors of cigarette smoking, hypertension and hypercholesterolemia.[13] Although the precise mechanism by which exercise reduces coronary risk remains unknown, exercise training induces physiological changes that may be cardioprotective and also favourably modifies other coronary risk factors.

In the Harvard Alumni Study, men who were physically active had a 25% lower risk of death from any cause and a 36% lower rate of death from coronary heart disease compared to less active men.[14]

The most constant benefit of exercise training in both healthy individuals and people with coronary artery disease is an improvement in exercise tolerance. This results in:

- An increase in maximal oxygen uptake
- Higher resting and exercise stroke volumes
- Lower resting heart rate
- Beneficial adaptations in skeletal muscle
- Slowed age related cardiac decline

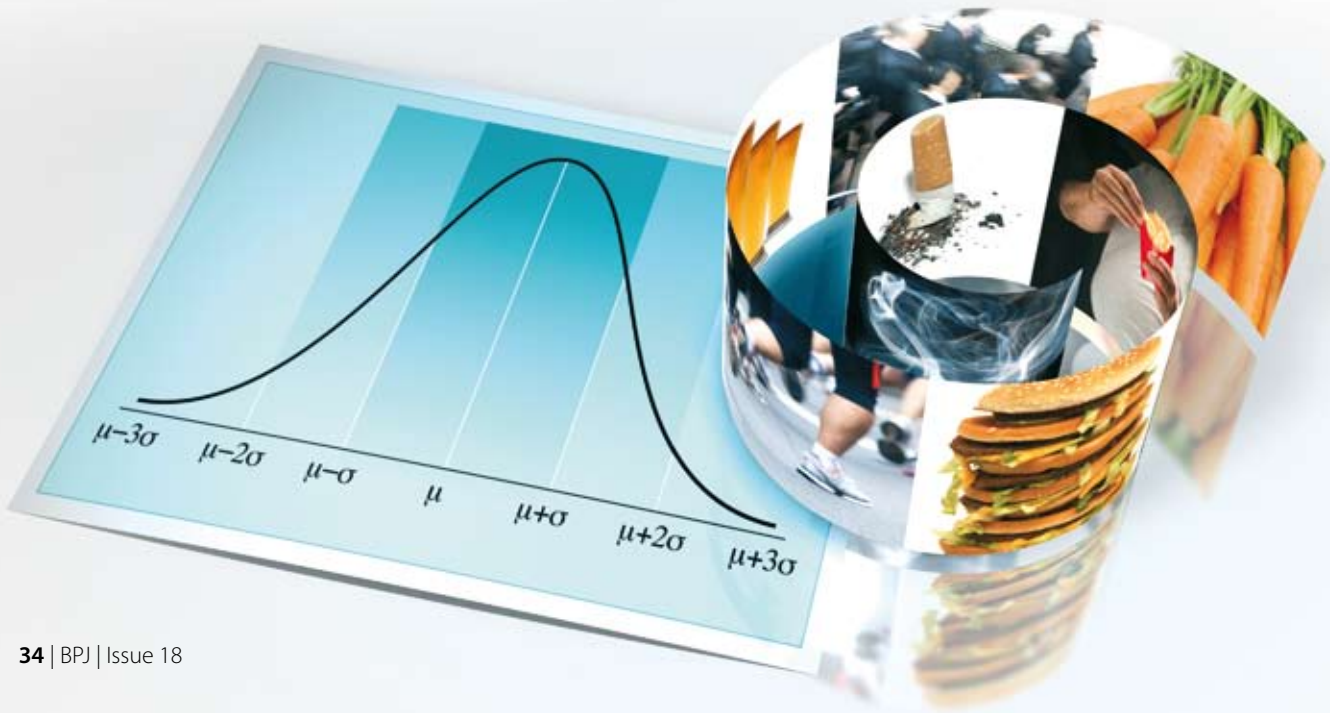
Obesity

Obesity is influenced by a combination of genetic, behavioural and environmental factors. The incidence of obesity is increasing world wide, mainly attributable to an increase in sedentary lifestyles and high fat diets.

Recent studies have demonstrated that the distribution of adipose tissue is more important for determining cardiovascular risk than total body weight. Increased intra-abdominal fat (see sidebar) has been demonstrated to be strongly associated with increased cardiovascular risk.

Tools for estimating obesity include body mass index, waist circumference or waist-hip circumference ratio.[15] Although BMI is less prone to measurement error, increasing waist circumference and waist-hip circumference ratio have been shown to be more strongly associated with increased cardiovascular risk. The World Health Organisation and the American Heart, Lung and Blood Institute recommend the use of waist circumference as an additional indicator of CVD risk.

To reduce measurement error, it is important waist circumference is measured midway from the lower rib margin to the anterior superior iliac crest.



The impact of increased adipose tissue mass on CVD

There are a number of mechanisms by which being overweight or obese contributes to increased cardiovascular risk.[17] Although adipose tissue tends to be less vascular than other tissues, it is surrounded by a significant capillary network, thereby increasing overall fluid levels in an overweight person. This increased blood volume along with the increased metabolic demand, caused by the extra weight leads to an increased cardiac output. The increased cardiac volume is produced by increased stroke volume, rather than an increase in heart rate and this may eventually lead to ventricular chamber dilation and left ventricular hypertrophy.

Left ventricular hypertrophy is often associated with ventricular diastolic dysfunction, particularly in people who are morbidly obese. In addition, longer periods of obesity are associated with poorer left ventricular systolic and diastolic function.

In obesity, fat deposits can occur in a number of organs, including the heart. Organ function can be reduced due to cell dysfunction or cell death, a phenomenon known as lipotoxicity. If this occurs in the heart, this may contribute to obesity cardiomyopathy.

Fat, particularly visceral fat, is capable of synthesising a number of compounds, such as angiotensin II, C-reactive protein, fibrinogen, which can exert a negative effect on the cardiovascular system.

Individual variability

It is worth remembering that these lifestyle factors occur on a background of inherited individual susceptibility. There will always be exceptions, people who ignore this advice and live to an old age. These exceptions should not be accepted as evidence that change is not needed.

Types of adipose tissue

Adipose tissue is present in two main forms: visceral (or intraabdominal) and subcutaneous. Men tend to gain weight in the classic “apple” shape as a result of accumulation of both subcutaneous and visceral fat abdominally. Women tend to develop the classical “pear” shape, due mainly to the accumulation of subcutaneous fat in buttocks and thighs. Following menopause, women are more likely to accumulate more visceral fat.

Obesity in New Zealand

The New Zealand Health Survey (2006/2007) reported the age-standardised obesity prevalence rate for the population aged 15 years and over as 25%. This was similar to the 2002/2003 rate of 24% but a significant increase from the 1997 rate of 19%.[3]

The study also reported that 8% of children aged 5 to 14 years were obese, a prevalence rate similar to that of 2002 (9%). Obesity in adolescence is a strong predictor of adulthood obesity.[16]

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