16
Cardiovascular Disease: Answers to Common Questions from Medical Journalists

The purpose of this chapter is to summarise the key points of this report in simple terms using a question and answer format. Many of the questions used are commonly asked by medical journalists. The questions are grouped under headings, and generally follow the same order as the chapters in the report.

16.1 Definition of cardiovascular disease

Q1. What is cardiovascular disease?

Cardiovascular disease refers to disease of the arteries supplying the muscle of the heart (coronary heart disease, CHD), the brain (cerebrovascular and carotid artery disease) and the extremities, especially the legs (peripheral vascular disease). It involves the processes of atherosclerosis (lesions in the arterial wall) and thrombosis (blood clotting), as well as changes to the function of the arterial lining.

16.2 Epidemiology of cardiovascular disease

Q2. Does the UK have a high death rate from cardiovascular disease compared to other countries?

Cardiovascular disease is a major cause of death, particularly in the Western world, but increasingly so elsewhere. There are wide variations in death rates from cardiovascular disease around the world and, despite recent improvements, those in the UK are amongst the highest. Countries in Eastern and Central Europe, where mortality from cardiovascular disease has been rising rapidly recently, have higher rates than the UK. The lowest rates of cardiovascular disease amongst developed countries are found in Japan and the Mediterranean countries, such as France, Spain and Italy.

Q3. Are cardiovascular disease rates falling around the world?

On a global scale, the burden of cardiovascular disease is growing at an alarming rate. By 2020, it is projected that it will surpass infectious disease as the world's leading cause of death and disability. Mortality from cardiovascular disease is rising rapidly in developing countries as they become increasingly industrialised and urbanised, and their large populations adopt more westernised diets and lifestyles. This trend is particularly serious for low- and middle-income countries that already have to tackle the dual problem of infectious disease and undernutrition. Many countries of Central and Eastern Europe (most notably countries of the former Soviet Union) have also experienced an increase in cardiovascular disease mortality since the 1990s. This increase has been more pronounced in men than in women. Whilst the reasons for this rise in cardiovascular disease mortality are not clear, it is thought that alcohol, and binge drinking in particular, may be a major determinant in some countries (*e.g.* Russia).

In contrast, USA, Canada, Israel, Australia and New Zealand have seen large falls in premature cardiovascular disease mortality rates since the 1970s, while Western European countries, such as the UK, Belgium and Norway have seen smaller reductions.

For example, amongst men aged 35–74 years cardiovascular disease death rates fell by 39% between 1988 and 1998 in the UK. However, the number of people suffering from cardiovascular disease in the UK (for example, those with angina or having a heart attack) is not falling and may be rising in older age groups. This is probably associated with increased life expectancy and with improvements in treatment procedures.

Q4. Are there specific groups within the UK that are at high risk of cardiovascular disease?

Heart disease is more common in men than in women, and the prevalence increases with age in both sexes. Within the UK, mortality rates from heart disease are higher in Scotland and the north of England than they are in the south of England. They are also higher in manual than in non-manual social classes.

Certain ethnic groups seem to be particularly prone to cardiovascular disease. People from the Indian subcontinent are more likely to suffer from CHD than the white British population, while people of African and Caribbean descent living in the UK appear to be at greater risk of stroke.

Q5. Why are men at greater risk of heart disease than women?

Young men are more likely to die from heart disease than young women in most industrialised countries. Although female oestrogens are allegedly protective against heart disease, recent data go against this as a major explanatory factor. Rather, men's elevated risk may arise predominantly because they store more fat in central abdominal regions than do women. They have more to gain therefore from improving lifestyles, in particular by stopping smoking and increasing levels of physical activity, to prevent heart disease and diabetes. However, they are often less likely than women to seek medical advice or respond to health promotion activities.

Q6. So, is heart disease a man's disease?

Heart disease is far from being a man's disease. Although women do not seem to be as aware of the risk of heart disease as their risk of breast cancer (British Heart Foundation, 2003b), it is the single biggest killer of women in the UK. One in six women currently die from the condition, and in 2001 heart disease claimed the lives of over 54 000 women (British Heart Foundation, 2003a). This is more than four times the number of deaths from breast cancer.

Q7. How can individuals reduce their risk of cardiovascular disease?

The likelihood of an individual developing cardiovascular disease is influenced by some factors that are outside their control, such as genetic make-up, gender, race (see Chapter 1) and perhaps their early growth pattern (see Chapter 10). However, some of the detrimental effects can be counteracted by changes in behaviour, such as taking more exercise, stopping smoking, maintaining a healthy body weight and eating a varied and well-balanced diet.

Q8. Why has advice to reduce the risk of cardiovascular disease focused so much on fat in the diet?

Dietary fats have been regarded as having an important influence on cardiovascular disease because of their effects on blood cholesterol levels. Populations with a high intake of saturates (saturated fatty acids) have raised blood cholesterol levels and have a high prevalence of heart disease. Laboratory studies have also shown that blood cholesterol can be influenced by the balance of different types of fatty acids in the diet; blood levels of low-density lipoprotein cholesterol are lowered when some saturates (*e.g.* myristic and palmitic acid) are replaced by monounsaturates, polyunsaturates or carbohydrate. Fatty acids may also affect cardiovascular disease risk via other mechanisms (*e.g.* by influencing the tendency of blood to clot). Current recommendations recognise this by giving advice to cut down on the amount of fat in the diet, particularly saturates (principally found in spreads, full fat dairy products, fatty meat products and foods such as biscuits and cakes).

While blood cholesterol is an important risk factor for heart disease, a recent estimate suggested that even if the whole UK population managed to reduce their cholesterol levels below 6.5 mmol/l, there would only be around a 10% reduction in CHD deaths because CHD is a multifactorial disease. Thus advice to reduce risk of cardiovascular disease must also include advice to change other aspects of the diet

(*e.g.* to eat more fruit and vegetables, to reduce salt intake), as well as stopping smoking, taking more exercise and maintaining a healthy weight.

Q9. What is the most important risk factor for cardiovascular disease?

The risk of cardiovascular disease cannot be predicted from a single risk factor. Cardiovascular disease is a multifactorial disease, which arises out of interactive effects of different combinations of risk factors. The effect of different risk factors is also, to a greater or lesser extent, dependent upon individual susceptibility.

The 'classical' risk factors for cardiovascular disease are smoking, raised blood cholesterol, raised blood pressure, physical inactivity, obesity and diabetes; these are discussed in more detail in Chapters 1, 2, 11, 12 and 13. However, these cannot explain all cases of heart disease and this has led to a search for other risk factors, many of which have been discussed in detail in this report.

Q10. What might the novel risk factors described in this report add to what we know about cardiovascular disease?

Although cigarette smoking, elevated blood pressure and cholesterol account for many cases of cardiovascular disease, there is reason to believe that other risk factors may account for some of the differences in cardiovascular disease rates within and between populations. A better understanding of the role of these novel risk factors may help to identify other ways of establishing those at risk and additional approaches to tackle the condition.

Q11. Are changes in diet and lifestyle becoming less important as treatment for cardiovascular disease and its risk factors improves?

A number of highly effective drugs (such as lipid lowering agents, ACE-inhibitors, aspirin and beta blockers) are now available in developed countries, such as the UK, and have had a substantial impact on reducing cardiovascular disease mortality. However, the treatment of cardiovascular disease by drugs or medical intervention has important cost implications. In 2001, the cost of prescriptions for lipid lowering drugs alone (including statins) in the UK was just under £440 million (British Heart Foundation, 2003a). Encouraging changes to diet and lifestyle can prevent heart disease without the side effects associated with surgery and drug therapy. Being physically active and eating a balanced diet can also tackle several cardiovascular risk factors simultaneously. Thus lifestyle changes remain critical to reducing the prevalence of heart disease and stroke.

Q12. Why do the French have low rates of heart disease compared to the UK?

Around the Mediterranean, CHD rates are uniformly low, but in many of these countries this can be explained by lower blood cholesterol levels. The French, however, have a high intake of saturates, and their cholesterol levels, blood pressure and the proportion of those who smoke are similar to those in other Western industrialised nations. Yet, the risk of dying from a heart attack for a man in France is only around a third of that of a man in the UK, and a French woman's risk is one-fourth of that for a woman in the UK. This has been termed 'the French Paradox'. By contrast, life expectancy is not better in France than in the UK, owing to higher rates of cancer and violent deaths.

Although there may be some under-reporting of heart disease mortality in France, this is unlikely to be the whole explanation. The relative immunity of the French to heart disease has been attributed mainly to their red wine consumption. Regular consumption of moderate amounts of alcohol (around 2 units per day) can reduce risk of heart disease, and this has been attributed in part to increased blood levels of high-density lipoprotein (HDL) cholesterol (see Chapters 1, 3), an inhibition of platelet aggregation (see Chapter 6) and improved endothelial function (see Chapter 4). Many investigators have claimed that red wine is particularly beneficial due to the presence of flavonoids which may act as antioxidants in the body, but this remains to be substantiated. Other potential contributors suggested to explain the French Paradox have included the cardioprotective effects of a Mediterranean diet (that emphasises olive oil, fibre, fruits, vegetables and fish) and various measures implemented in the early 1900s to improve the nutrition and health of mothers and children in France (see Chapter 10).

16.3 Definitions and prevalence of diabetes, the insulin resistance syndrome and obesity

Q13. What is insulin?

Insulin is a hormone produced by the pancreas that allows glucose (obtained from foods containing carbohydrate) to be absorbed from the bloodstream into the cells. This is very important because glucose is the main source of energy that the body needs in order to function properly.

Q14. What is diabetes?

Diabetes, properly known as diabetes mellitus, is a chronic disorder in which the body's sugar (glucose) level is too high. There are two main types of the disease. Type 1 diabetes (also known as insulin dependent diabetes) occurs when the pancreas does not produce the hormone insulin, which is needed to control blood sugar levels. Type 1 sufferers usually develop the disease during childhood or adolescence. Type 2 diabetes (or non-insulin dependent diabetes) is the most common form of the disease and occurs mostly in adults who are overweight. It arises when the body does not produce enough insulin or the body's tissues become resistant to insulin, which causes the blood glucose levels to rise (see Chapter 2).

Q15. How many people suffer from type 2 diabetes?

About 1.4 million people in the UK have been diagnosed with diabetes, but experts estimate that about the same number have the condition without knowing it.

Q16. What are the possible complications?

People with diabetes have a higher chance of developing heart disease, strokes, high blood pressure, circulation problems, nerve damage, and damage to the kidneys and eyes. The risk is particularly high for sufferers who are obese, who smoke or who are not physically active.

Q17. How is diabetes linked to cardiovascular disease?

People with diabetes are at greater risk of cardiovascular disease, although the reasons for this are only partly understood. It is thought that if blood glucose levels are higher than normal and not controlled, this may affect the lining of the body's arterial walls, increasing susceptibility to atherosclerosis (furring up of the arteries).

As well as insulin resistance and high glucose levels, people with type 2 diabetes tend to have central obesity, high blood pressure and abnormal lipid concentrations. All of these are risk factors for cardiovascular disease and when they occur together they are often called the metabolic syndrome (see Chapter 2).

Q18. What are the main risk factors for type 2 diabetes?

Risk factors include increasing age, family history and being overweight, particularly with central obesity (see Q75). People of Asian or African-Caribbean origin and women who have developed diabetes during pregnancy are also at greater risk. Low birthweight and diabetes in the mother during pregnancy have recently been identified as risk factors for type 2 diabetes (see Chapter 10).

Q19. Can lifestyle changes reduce the risk of developing diabetes?

Being physically active, maintaining a healthy body weight and eating a healthy diet that is low in fat, particularly saturates, and high in fibre-rich foods, such as whole-grain cereals and fruit and vegetables, can lower the risk of developing the condition.

Q20. What is insulin resistance?

A person who is insulin resistant has cells that respond sluggishly to the action of insulin. This means that the body's cells cannot take up enough glucose and the level of glucose in the bloodstream remains high. This signals yet more insulin to be released from the pancreas until the glucose is taken up by the cells. If the pancreas fails to sustain this increase in insulin secretion, type 2 diabetes develops.

Q21. How common is insulin resistance?

Experts suggest that 10–25% of the adult population may be resistant to insulin to some degree. People

who are overweight, those who have a parent or sibling with type 2 diabetes, women who developed diabetes during pregnancy and some ethnic groups (*e.g.* South Asians) are at increased risk of insulin resistance and the insulin resistance syndrome.

Q22. What causes it?

Insulin resistance is thought to be caused by both genetic and lifestyle factors. Physical inactivity, a high fat diet, excess alcohol consumption and increased body weight, high blood pressure and raised cholesterol are linked to the incidence of insulin resistance, and may trigger the condition in people who are genetically prone to it. All these factors are also linked to an increased risk of cardiovascular disease, and this may be one reason why there is a high incidence of heart disease and stroke among people with type 2 diabetes.

Q23. What do we mean by the insulin resistance syndrome?

The insulin resistance syndrome (also referred to as the metabolic syndrome or syndrome X) refers to a combination of health problems, including insulin resistance, abnormal levels of blood fats (high triglycerides and low HDL or 'good' cholesterol), central obesity and high blood pressure. These are all risk factors for type 2 diabetes and heart disease. It is estimated that people with this syndrome are around three times more likely to die from cardiovascular disease, even after controlling for other risk factors (see Chapter 2, Section 2.3.2).

Q24. How common is the insulin resistance syndrome?

To some extent this will vary depending on the definition used (see Chapter 2, Section 2.3.2), but it is likely that as the population ages and the problem of overweight and obesity continues to rise, the number of people with the syndrome will increase.

Q25. What can be done to prevent it?

Adopting a healthy lifestyle, which means eating a healthy diet, maintaining a healthy body weight, not smoking, being physically active and drinking alcohol in moderation, can help to avoid all of the features of the syndrome. Because the conditions occur in a cluster, steps taken to bring one of them into the healthy range will probably improve the others. For example, if you're overweight, losing up to 10–15% of your body weight will bring down blood pressure and increase cells' sensitivity to insulin. Similarly, being more physically active can promote weight loss and raise HDL-cholesterol levels.

Q26. How is obesity usually defined?

Obesity is generally defined as a body mass index (BMI) of more than 30 kg/m^2 (see Chapter 2, Section 2.4.1). Waist circumference (which indicates central or abdominal obesity) is also thought to be important in determining risk of cardiovascular disease (see Q75). An increased risk to health is associated with a waist circumference of over 94 cm (34 in) in men and over 80 cm (32 in) in women. A substantial risk to health is associated with a waist circumference of over 102 cm (40 in) in men and over 88 cm (35 in) in women (see Chapter 2, Section 2.4.2).

Q27. Why is the worldwide increase in obesity of concern in relation to cardiovascular disease?

People who are obese are two to three times more likely to suffer from cardiovascular disease. Obesity is associated with other features of the insulin resistance syndrome (*e.g.* it increases the risk of high blood pressure, insulin resistance and abnormal levels of blood fats). There is also a strong link between obesity and type 2 diabetes, even with modest degrees of overweight. For example, women with a BMI of just 25 kg/m^2 have a more than five-fold increased risk, while those with a BMI of more than 35 kg/m^2 have more than fifty-fold increased risk compared to women with an ideal body weight (BMI 20–25 kg/m^2).

Q28. How many adults in the UK are currently overweight or obese?

The prevalence of obesity in Britain has trebled since the mid-1980s. Figures from the National Diet and Nutrition Survey suggest that 25% of men and 20% of women are obese (BMI > 30.0 kg/m^2) and a further 42% of men and 32% of women are overweight (BMI 25–30 kg/m^2). If these trends continue, it is estimated that 30% of the adult population will be obese by 2010.

Q29. What are the health implications of being overweight during childhood?

Apart from the social and psychological problems experienced by overweight children, there are also long-term risks. Compared with normal weight children, obese children have higher blood pressure and insulin levels, and a lipid pattern that is associated with heart disease. Children who are overweight in their teens are more likely to be overweight as adults and this 'tracking' effect is accompanied by an increase in cardiovascular risk factors during adulthood. There is also some evidence that adolescents, regardless of whether they remain obese as adults, are more likely to develop heart disease (see Chapter 2, Section 2.4.10).

Q30. How much weight loss can reduce cardiovascular risk?

Long-term studies have shown that for obese people a sustained modest weight loss of 5% to 10% of body weight can improve a number of risk factors for heart disease (*e.g.* lower blood pressure, blood glucose concentrations and cholesterol levels). Decreases of just 4 kg over a 4-year period have been shown to cut the risk of diabetes by more than a half.

Q31. Why is being physically active important in reducing cardiovascular disease risk?

Regular physical activity can reduce the risk of diabetes and cardiovascular disease by improving lipid levels, aiding weight loss and lowering blood pressure. Physical activity also has multiple beneficial effects on the emerging risk factors discussed in this Report (see Chapter 2, Section 2.8 and Chapter 12, Section 12.2).

Q32. How much can genes influence the risk of insulin resistance and cardiovascular disease?

There is much ongoing research into this very question. In some rare families, there are powerful and dangerous genes which make it almost certain that an individual will get insulin resistance or cardiovascular disease. However, such families and genes are uncommon and in most people there are only weaker genetic effects. Our present estimate is that about 30–50% of an average individual's chance of getting insulin resistance and cardiovascular disease will come from his or her genes, whilst the rest will depend upon their lifestyle.

16.4 Lipid-related factors

Q33. What is cholesterol?

Cholesterol is a fatty substance (a lipid) that is found in the bloodstream and in all cells. It has an important role as part of the walls or membranes of each cell. It is also a key component in the manufacture of hormones (chemical messengers in the body) and bile acids (that promote the absorption of fat from the diet).

Some foods, such as meat, poultry, shellfish and dairy products, contain dietary cholesterol. Organ meats, such as liver, are especially high in cholesterol, while it is not found at all in foods of plant origin. However, only a small proportion of cholesterol in the body comes from the cholesterol in food. It is made mostly in the liver and this process is stimulated by saturates (see Chapter 13, Table 13.5 for the main sources of saturates in the UK diet).

Q34. What are 'good' and 'bad' cholesterol?

There are two main types of blood cholesterol, which are often referred to as 'good' and 'bad' cholesterol.

Low-density lipoprotein (LDL) is the main cholesterol carrier in the blood. If too much LDL-cholesterol circulates in the blood, it can build up in the lining of the arteries and form atheromas or fatty deposits. These can cause the arteries to narrow in a process called atherosclerosis. An elevated level of LDL-cholesterol is associated with increased risk of heart disease, and is therefore referred to as 'bad' cholesterol.

High-density lipoprotein (HDL) is called 'good' cholesterol as it carries cholesterol away from the arteries and back to the liver. High levels of HDL-cholesterol help to protect against the development of atheroma in the arteries, while a low level of HDL-cholesterol may increase risk of atherosclerosis

Thus, a high level of LDL- and low level of HDL-cholesterol (known as a high LDL/HDL ratio) increases the risk of atherosclerosis, while a low level of LDL- and high level of HDL-cholesterol (a low LDL/HDL ratio) is desirable.

Q35. What is Lp(a) cholesterol?

Lipoprotein(a) or Lp(a) is a lipoprotein particle found in the bloodstream. Lp(a) levels appear to be largely genetically determined. High levels are associated with an increased risk of heart disease (see Chapter 3).

Q36. What are triglycerides and how are they linked with heart disease?

Fat in food and fat stored in the body is in the form of triglycerides. A triglyceride is a substance that is composed of three fatty acids attached to a single glycerol molecule. Triglycerides are carried in the blood bound to proteins forming high- and low-density lipoproteins. Like cholesterol, triglyceride in the blood comes either from the diet or from its synthesis in the liver. High triglyceride levels are associated with increased risk of heart disease and stroke.

Q37. What are apolipoproteins?

Apolipoproteins are particles of protein that are mostly formed in the liver and intestine. They play an important role in the production and transport of cholesterol around the body. There are at least nine types of apolipoprotein, including apoA-1, apoB and apoE. Each type bonds with cholesterol in the blood to form either the protective cholesterol, HDL, or the more harmful cholesterol, LDL (see Q38 and Chapter 3).

Q38. What part do they play in coronary heart disease?

High levels of certain apolipoproteins may increase risk of heart disease, diabetes or stroke. For example, high levels of apoB are associated with higher risk of heart disease as it is the main protein in LDL-cholesterol. In contrast, high levels of other apolipoproteins can protect against coronary disease. For example, ApoA-1 is the major protein in the more protective HDL-cholesterol (see Chapter 3).

High levels of apoE can affect the breakdown of cholesterol in the blood, and also influence the progression of heart disease, depending on the subtype of the apolipoprotein. ApoE2 appears to play a protective role as it goes hand in hand with lower LDL levels, while apoE4 slows the removal of LDL from the circulation, and may increase the risk of heart disease.

Q39. What causes high cholesterol?

There are several factors that may contribute to high blood LDL-cholesterol and/or low HDL-cholesterol levels. These include a diet that is high in saturates, lack of physical activity, family history, being overweight, drinking alcohol excessively and smoking. Rarely, high blood cholesterol can also be caused by a condition that runs in the family called familial hypercholesterolaemia.

Q40. Do low fat diets have beneficial effects on blood lipids?

Consuming a diet that is low in fat can lower blood levels of total and LDL ('bad') cholesterol. As diets that are low in fat, particularly those that are high in complex carbohydrate, are usually lower in energy (calories) than high fat diets, they can also improve body weight (see Chapter 3, Section 3.2.1 and Chapter 11, Section 11.3.2). However, there is accumulating evidence that the type of fat is of even greater importance for cardiovascular disease than the total amount of fat in the diet. In particular, lowering the amount of saturates in the diet is important in reducing blood cholesterol levels (see Q87 and Chapter 11, Section 11.8).

Q41. What sorts of fats are in foods?

Fat in food is composed of two main types of fatty acids, *saturates* and *unsaturates.* Unsaturated fatty acids can be either *polyunsaturates* or *monounsaturates* (see Chapter 1)**.** There are two main types of polyunsaturates: *n*-6 fatty acids obtained predominantly from the seeds of plants, such as sunflower oil or soya oil, and *n*-3 fatty acids, some of which are predominantly from seeds (*e.g.* rapeseed and linseed), and others that are present in large amounts in fish oils and have attracted particular attention. All foods contain a combination of saturates, polyunsaturates or monounsaturates, but the proportion of each varies greatly with different foods (see Chapter 13, Table 13.5).

Q42. What effects do these fatty acids have on blood lipids?

Saturates raise blood cholesterol levels more than anything else in the diet. *n*-6 polyunsaturates lower LDL-cholesterol levels associated with CHD, but also appear to lower HDL-cholesterol protective to the heart. Monounsaturates have been found to help lower the amount of LDL-cholesterol in the blood but maintain HDL-cholesterol levels (see Chapter 13, Table 13.5 for dietary sources). This is likely to be a factor contributing to the ability of Mediterranean-style diets, which are rich in monounsaturates, to protect against cardiovascular disease.

One of the ways in which the long-chain *n*-3 polyunsaturates found in oil-rich fish may protect against heart disease is via their ability to lower blood triglyceride levels. There is also some evidence that high intakes of these fatty acids may additionally protect against heart disease by positive effects on other blood lipids (*e.g.* by lowering small, dense LDLs and reducing remnant lipoproteins; see Chapter 3). Their effect on blood cholesterol levels, however, is less clear, and it may be that individuals react differently to these fatty acids. The extent to which the *n*-3 fatty acids present in seeds can simulate the effects of those from oil-rich fish remains to be clarified.

Q43. What about *trans* fatty acids?

Trans fatty acids are produced during the process of hydrogenation of unsaturated fats and are principally found in manufactured confectionery products (*e.g.* biscuits, cakes and chocolates) and some margarines. They have a particularly adverse effect on lipoproteins (they increase LDL-cholesterol and Lp(a) and decrease HDL-cholesterol), and have been shown to increase risk of heart disease. In the UK, however, they contribute a relatively small proportion of total energy compared with saturates, and current average intakes are well below the dietary reference value (see Chapter 3 and Chapter 11, Section 11.8.6).

Q44. Is eating between meals a problem?

While there is little evidence to date that snacking has negative metabolic consequences or any effect on body weight after controlling for total energy intake, more research is needed to clarify the effects of meal frequency on blood lipids and body weight (see Chapter 3). Nevertheless, some highly palatable snack foods are high in fat and energy-dense, which may encourage over-eating. Irregular eating habits can also create difficulties for those trying to control their weight.

Q45. How does exercise affect blood lipids?

Exercise has a number of beneficial effects on the blood fats cholesterol and triglycerides. Regular exercise is associated with an increased ability to clear fat particles from the bloodstream after meals. This is because the exercised muscles need more energy from fat and thus utilise the fat quickly so that it is cleared from the bloodstream. Exercise also affects blood cholesterol levels by increasing HDL-cholesterol ('good' cholesterol) levels. Long-term exercise programmes may also reduce LDL-cholesterol ('bad' cholesterol) concentrations. In addition, being physically active can help to control weight, diabetes and high blood pressure, all of which would reduce the risk of heart disease (see Chapter 12).

Q46. Can the effect of different diets on blood lipid levels vary between individuals

Yes, genetic make-up can have a large effect on blood lipid levels and the way in which the body deals with different diets. For example, two healthy individuals eating the same food and with similar lifestyles can have quite different blood cholesterol levels. Also, some individuals may experience greater benefit from changing their diet and lifestyles than others (see Chapter 3).

16.5 The role of the endothelium

Q47. What is the endothelium and what is its main role?

The endothelium is the layer of cells lining various blood vessels of the body. It regulates the normal functioning of blood vessels and plays a role in functions as diverse as: expansion and contraction of blood vessels, repair of damage, formation of new vessels, and immune responses to infection. Due to its key position, the endothelium also forms a selective

barrier between the blood and the underlying tissue, allowing oxygen and nutrients to cross, but not dangerous substances. The range of functions that the endothelium is involved in means that its damage can have serious consequences to the vascular system. These include the formation of atherosclerotic plaques, the build-up of fatty materials within the walls of the arteries, and potentially angina, stroke and heart attacks.

Q48. What is endothelial dysfunction?

Any abnormality in the function of the blood vessel lining which, when functioning properly, helps prevent the process of blood vessel narrowing and fat accumulation.

Q49. How does endothelial dysfunction affect cardiovascular disease risk?

In this circumstance, rather than preventing the process of blood vessel narrowing and thus heart disease, the lining of the blood vessels can actually contribute to this process by allowing more cholesterol to enter blood vessels and more harmful inflammatory processes to occur at the lining of blood vessels. In other words, when the endothelium is 'dysfunctional' it can actually accelerate many of the processes leading to blood vessel narrowing and thus increase risk of cardiovascular disease.

Q50. Does diet influence endothelial function?

Some dietary factors do appear to alter the function of the blood vessel lining. For example, long chain *n*-3 fatty acids (found predominantly in oil-rich fish), Mediterranean-style diets (rich in monounsaturates) and some vitamins (*e.g.* folic acid) appear to enhance the ability of the blood vessel lining to prevent heart disease. In contrast, other agents, either acutely or chronically (*e.g.* high fat levels), can harm blood vessel function.

Q51. Can other lifestyle factors influence endothelial function?

Yes, other lifestyle factors are important. Physical activity (see Chapter 12), moderate weight loss and smoking cessation can all improve endothelial function. There is also some evidence to suggest that a moderate alcohol intake may have beneficial effects on the endothelium (see Chapter 4).

16.6 Diet and oxidative stress

Q52. Why do nutritionists and dietitians promote the 'five-a-day' message?

There is now a considerable body of evidence that has shown diets rich in fruit, vegetables and other plant foods to be associated with a reduced risk of suffering or dying from a number of diseases, in particular cardiovascular disease and some cancers, but also age-related eye conditions such as cataract and macular degeneration, and chronic lung disorders. This has led to the recommendation to eat at least 400 g (five 80 g portions) of fruit and vegetables every day.

Q53. What constitutes a portion?

The recommended 'five portions a day' excludes potatoes and nuts, but includes pulses (*e.g.* baked or red kidney beans and lentils), although they collectively count only as one portion per day no matter how often they are eaten. Similarly, unsweetened fruit juice can also constitute a portion, but only one per day however much is drunk. Fruit and vegetables do not have to be raw – frozen, canned, dried and cooked versions all count towards the five-a-day target. Fruit- and vegetable-based dishes (*e.g.* fruit crumble, vegetable soup, curry, pizza) will also count as long as they contain at least one portion of fruit and vegetables per serving.

A portion is generally considered to be around 80 g. This corresponds to:

- a piece of a large fruit (*e.g.* half an avocado or grapefruit, a large slice of melon, a couple of rings of pineapple)
- one medium sized vegetable or fruit (*e.g.* apple, orange, banana)
- a couple of small fruits (*e.g.* two plums)
- one cup of very small fruit (*e.g.* grapes)
- 1/2–1 tbsp dried fruits (*e.g.* dates, sultanas)
- 2–3 tbsp cooked or canned fruit
- two tbsp raw, cooked, frozen or canned vegetables
- a bowl of salad
- three heaped tbsp beans or lentils (but only counts once per day)
- a glass of fruit juice (but only counts once per day).

Q54. What is so good about fruit and vegetables?

Fruit and vegetables collectively contain a wide range of nutrients, including vitamins with antioxidant properties (*e.g.* vitamins C and E, and carotenoids such as beta-carotene), folate and other B vitamins, a wide range of minerals including potassium and iron, essential fatty acids and dietary fibre. In addition, there are known to be tens of thousands of bioactive compounds (or phytochemicals), including flavonoids, glucosinolates and phyto-oestrogens, which have been suggested to have beneficial properties with respect to human health. The range of nutrients and phytochemicals present varies considerably between different types of fruit and vegetables. So, the advice with regard to consumption of these foods should focus on variety, to ensure that a wide array of nutrients and bioactive substances are consumed.

There is also likely to be a displacement effect – that is, by eating more fruit and vegetables we tend to eat less of the high fat and energy-dense foods that may increase the risk of obesity, heart disease and diabetes.

Q55. Do antioxidant nutrients reduce the risk of heart disease?

The search for the specific constituents of fruit and vegetables that might protect against heart disease has led to considerable interest in the role of the antioxidant nutrients found in these foods, *e.g.* vitamins E and C, and beta-carotene. Free radical damage has been implicated as a factor in the development of heart disease and stroke, and a number of antioxidant nutrients are important in the body's defence systems (see Chapter 5). However, whilst promising and consistent results have been reported in animal and *in vitro* studies, *e.g.* tissue culture, human intervention trials have not generally supported a role for these nutrients in heart disease prevention. The contribution of other plant constituents with antioxidant properties, such as flavonoids and sulphur-containing compounds, remains to be established.

Q56. Can supplements be as effective as fruit and vegetables in reducing your risk of cardiovascular disease?

Intervention trials have not supported the notion that supplements provide the same protection against chronic diseases as increasing fruit and vegetable intake. This might be because it is the cocktail effect of the many substances present in whole fruit and vegetables that confer the health properties or that the substances tested are not those responsible.

Q57. Is it true that chocolate contains substances that might be good for the heart?

There is evidence that chocolate, which contains a range of bioactive substances including flavonoids, can increase the antioxidant capacity of the blood and reduce the oxidation of LDL-cholesterol, which is a pre-requisite step for cholesterol being deposited in the arterial wall. The consumption of cocoa-rich products has also been shown to have beneficial effects on other processes associated with cardiovascular disease, *e.g.* helping to prevent blood platelets from clumping together and forming a clot, and increasing blood vessel flexibility. However, much of this work has been done in a test tube and needs to be supported by human studies. It should also be remembered that chocolate and products containing it tend to be energy-dense and contain relatively high amounts of fat and sugar, and thus should be eaten in moderation.

16.7 Diet and blood clotting

Q58. How and why does blood clot?

Blood is usually a liquid as it is being pumped around the body by the heart, but under some circumstances (*e.g.* following a cut) it needs to solidify and form a clot. Blood clotting (also known as coagulation) is triggered by a protein called tissue factor, found on cells in the deeper layers of the blood vessels and not normally exposed to blood until there is an injury. Tissue factor reacts with a blood protein called factor VII, and with the aid of small blood cells called platelets initiates a series of complex chemical reactions to produce a substance called thrombin. Thrombin converts a blood protein fibrinogen to fibrin at the site of the wound. These strands of fibrin trap platelets and other blood cells, forming a blood clot (see Chapter 6). This process protects the body from excessive bleeding, ensuring that a clot forms at the site of a wound or injury – either internally or on the body surface. As part of the body's natural healing mechanism, clots are usually dissolved and reabsorbed by the body.

Q59. What happens when the clotting mechanism goes wrong?

The clotting process (also known as haemostasis) functions as a careful balance between blood flowing and stopping, and between clotting and dissolving/reabsorbing clots. The consistency of the blood is regulated and maintained by a large number of different proteins, some of which are involved in clot formation (coagulation), whilst others are involved in the prevention of clot formation (anticoagulation) and dissolving of formed clots (fibrinolysis). Abnormalities in the amounts of either these coagulation, anticoagulation or fibrinolytic proteins can cause problems. If the blood is prone to clot too little, then there is a risk of haemorrhage; too much and there is a risk of clots forming where they are not wanted. For example, clots that develop in an artery supplying blood to the heart or brain can cause a heart attack or stroke.

Q60. What markers suggest that the blood might be prone to clotting?

Raised concentrations of some of the substances involved in blood clotting or the removal of blood clots from the circulation seem to predict cardiovascular disease risk. For example, high blood levels of the blood clotting protein fibrinogen seem to be associated with increased risk. Emerging research is also identifying a number of other markers (*e.g.* PAI-1, tPA, von Willebrand factor) that may help in the assessment of patients at risk for CHD (see Chapter 6).

Q61. What can be done to prevent clots forming in the arteries?

There are a number of widely used drugs that act as anticoagulants (which help to prevent blood clots) or clot busters (that help to dissolve blood clots), which can be prescribed to people who are at high risk of cardiovascular disease. One of the most commonly used antithrombotics is aspirin.

However, there are several diet and lifestyle factors that may increase the risk of a blood clot forming. Being overweight, inactive, smoking and consuming a diet that is high in fat (particularly saturates) increases the risk. In contrast, a moderate amount of alcohol may reduce the tendency of blood cells to stick together and form a clot. Studies have also demonstrated longer bleeding times and a reduced tendency for platelets to stick together and form a clot in subjects consuming high doses of *n*-3 fatty acids, found primarily in oil-rich fish (see Chapter 6).

Q62. Are some people genetically more prone to blood clots?

A number of changes (mutations) in the genes that code for some of the factors involved in coagulation or anticoagulation have been identified, and some of these have been shown to increase the risk of blood clots forming in veins inappropriately. Individuals who inherit these mutations may have an increased risk or 'predisposition' to developing blood clots compared to the rest of the population.

16.8 Inflammation

Q63. What is inflammation and what effect does it have on the risk of heart disease?

There are two types of inflammation: acute and chronic. Acute inflammation is a normal process whereby white blood cells are recruited into tissues in response to an injury, infection or an allergen. This is accompanied by the common signs of acute inflammation – pain, swelling, redness and heat. These symptoms are caused by blood vessels dilating around the affected area, bringing substances involved in the inflammatory process to dispense or disperse the damaged tissue or bacteria.

In contrast, chronic inflammation is a longer-term problem. This occurs when the body's inflammatory response is prolonged beyond normal and can occur because the immune system becomes overstimulated, overactive or fails to switch off its response (or any combination of the three). This type of inflammation can be detrimental as it may injure body tissues; it has been implicated in the aetiology of inflammatory diseases, such as rheumatoid arthritis, psoriasis and inflammatory bowel disease. It has also been suggested that prolonged inflammation of the arteries may be one of several factors that contribute to atherosclerosis (narrowing and hardening of the arteries). The result is that the arteries become constricted, their elasticity and flexibility disappears and the volume of blood able to travel through them at any given time is reduced. This 'hardening' can lead

to elevated blood pressure and increase the risk of a heart attack or stroke (see Chapter 1).

Q64. What causes the type of inflammation associated with increased risk of heart disease?

It is not yet known exactly what causes the inflammation that might increase risk of cardiovascular disease. Some researchers suggest that inflammatory cells are recruited to the sites of developing atherosclerotic plaques in response to deposits of cholesterol or oxidised lipid products on the artery wall. Other possible factors that may cause an inflammatory response in the body are smoking, uncontrolled diabetes mellitus and high blood pressure. Others suggest that infection, possibly caused by a bacteria or a virus, might contribute or even start the narrowing process. Possible infectious bacteria include *Chlamydia pneumoniae* and *Helicobacter pylori* (see Chapter 7).

Q65. Can markers of inflammation be used to identify people at risk of heart disease?

Levels of some markers of inflammation (*e.g.* C-reactive protein, fibrinogen and lipoprotein-associated phospholipase A2) have been linked with risk of cardiovascular disease. For example, researchers have found that people with high blood levels of C-reactive protein, which indicates underlying levels of inflammation, are more likely to develop heart disease and stroke than those with lower levels of this protein. This increased risk has been shown to be independent of other risk factors, such as high cholesterol levels, smoking, high blood pressure and obesity. As sensitive tests for C-reactive protein are now available, the protein could be measured to identify those at high risk of developing a first heart attack or stroke (it is likely to be of limited value in secondary prevention as a heart attack increases C-reactive protein levels). However, it is not yet known whether C-reactive protein is a causative agent or a consequence of heart disease, or whether reducing circulating levels of this protein can actually lower cardiovascular disease risk (see Chapter 7).

Whether markers of inflammation, such as C-reactive protein, add anything of value over conventional markers of heart disease as yet remains unclear. The best marker of inflammation to measure is also still being debated.

Q66. Can inflammation be reduced by drugs or diet?

There are a number of pharmacological agents available to reduce inflammation, but we need further research to predict their ability to reduce cardiovascular risk. Some aspects of the diet also seem to influence the inflammatory response. For example, the long chain *n*-3 fatty acids (found primarily in oil-rich fish) and some antioxidant nutrients (*e.g.* vitamin C and beta-carotene) seem to have anti-inflammatory effects, but we need more research to determine whether these nutrients influence cardiovascular disease risk in this way.

16.9 Homocysteine

Q67. What is homocysteine?

Homocysteine is an amino acid in the blood that is produced as the body digests and breaks down protein. It is normal to have a certain level of homocysteine in the blood. However, during the past few years, researchers have suspected that high levels of homocysteine increase the risk of cardiovascular disease (see Chapter 8).

Q68. Should people at high risk of cardiovascular disease be screened for elevated homocysteine levels?

The link between high levels of low-density lipoprotein (LDL) cholesterol (the 'bad' form of this fatty substance) and cardiovascular disease is well known, and doctors routinely measure cholesterol to gauge a person's risk. In the past few years, several studies have suggested that elevated levels of homocysteine may also increase the risk of heart disease and stroke. However, this link has not yet been proved, and recent data suggest that homocysteine levels may be a less important risk factor for heart disease than expected. Therefore, unlike cholesterol, homocysteine is not routinely measured and most people who suffer from heart disease will not have their levels measured at routine check-ups. For most people, measuring homocysteine or screening for a genetic mutation that predisposes to high homocysteine is not warranted right now. However, targeted testing is recommended, such as for young people who develop unexpected heart disease or stroke.

Q69. What aspects of the diet and lifestyle can alter homocysteine levels?

Several of the B vitamins are involved in homocysteine metabolism (folic acid, vitamin B_6, vitamin B_{12} and riboflavin) and supplementation with these vitamins, particularly folic acid, can lower homocysteine levels. Homocysteine levels are higher in current smokers than in non-smokers and in people who consume very high amounts of coffee, but are lower in individuals who are moderate alcohol drinkers.

Q70. Should people take vitamin pills containing folic acid to reduce risk of heart disease?

No, taking folic acid to prevent risk of heart disease or stroke is not currently recommended. Whether having extra folic acid lowers the risk of heart attack and stroke must first be put to the test in clinical trials, which are currently underway.

Q71. Why don't we fortify foods with folic acid in the UK when it is mandatory in the USA?

There is concern that fortification of flour with folic acid for the prevention of birth defects (neural tube defects such as spina bifida) could be hazardous for older people due to delay in the diagnosis of vitamin B_{12} deficiency or exacerbation of either peripheral neuropathy (a disease affecting the nerves) or other neuropsychiatric complications associated with vitamin B_{12} deficiency. Experts believe that fortification of flour with folic acid should be accompanied by screening of people aged 75 years or over for vitamin B_{12} deficiency, or there should be combined fortification of flour with folic acid and vitamin B_{12}.

16.10 Factors relating to adipose (fat) tissue

Q72. Why is being obese a risk factor for cardiovascular disease?

Being overweight increases the chance of having a heart attack. This is partly due to the strain placed upon the heart by excess weight, but obese individuals are also more likely to experience a number of cardiovascular risk factors, including diabetes, high blood pressure (hypertension), an adverse blood lipid profile and abnormalities of blood clotting factors. Emerging research has also shown that fat (adipose) tissue secretes a number of substances (collectively referred to as adipokines) that may themselves increase the risk of cardiovascular disease. The good news is that all of these risk factors are reduced when obese people lose weight.

Q73. What are adipokines?

Adipokines are a group of substances, including enzymes, cytokines and hormones, which are produced in, or released into the blood from, fat tissue. Circulating levels of many of these substances are affected by obesity and have been shown to regulate, directly or indirectly, a number of the processes that contribute to the development of heart disease, including hypertension, endothelial dysfunction, chronic inflammation and insulin resistance. Whilst a lot more research is needed, it is hoped that a greater understanding of the role of these substances and how their levels are regulated might help to combat obesity and cardiovascular disease in the future (see Chapter 9).

Q74. Does diet influence levels of these adipokines?

As weight loss leads to a reduction in the amount of fat in the body, it is associated with a fall in levels of several of the adipokines. Similarly, weight gain and excess energy intake is associated with a rise in many of these substances. At the moment there is insufficient evidence of the effect on adipokines of individual nutrients, but this remains an active area of research.

Q75. Does where you carry excess fat influence your risk of heart disease?

The medical risks from being overweight are increased if excess fat is distributed abdominally (*i.e.* around the stomach). Men and post-menopausal women tend to lay down fat around the abdomen and have an 'apple-shaped' distribution of fat, while pre-menopausal women tend to have a 'pear-shaped' distribution, with fat on the thighs and buttocks. 'Apples' generally carry a higher risk of developing heart disease and diabetes than 'pears'. Measuring your waist circumference is the quickest and simplest way to estimate whether abdominal fat is increasing

your risk of cardiovascular disease (see Chapter 2, Section 2.4.2 and Q26).

Q76. What is leptin?

Leptin is a hormone produced by the fat cells, that is believed to control appetite and regulate body weight via receptors in the brain. A decrease in body fat leads to a decrease in the hormone, which in turn stimulates food intake by increasing appetite. Increased body fat leads to increased levels of the hormone, which act to reduce food intake by turning off the feeling of hunger in the brain. By this mechanism, weight is usually maintained within a relatively narrow range.

Q77. Can leptin be used to treat obesity?

Around 10 years ago, researchers gave leptin to obese mice that were genetically leptin deficient and found that they lost a substantial amount of weight. This led to great excitement both in the media and the scientific community about the possible role of leptin as a treatment for human obesity. However, researchers quickly discovered that most obese humans are, in fact, very unlikely to be deficient in leptin. Whilst rare cases of leptin deficiency have been identified in a small number of obese children, most obese people appear to have much higher levels of the hormone than non-obese people, so giving them more would have little effect. This is not completely unexpected since obese people have more fat and so will produce more leptin, but it seems that they may be resistant to high levels of the hormone so that it does not signal effectively to the brain to eat less. We need to discover more about the functions of leptin and the role it plays in weight gain before we can estimate its potential role in helping to treat obesity.

16.11 Nutrition during pregnancy and fetal growth

Q78. Are small babies at greater risk of developing cardiovascular disease in later life?

Babies who are born small for gestational age (not those born small because of premature delivery) are at increased risk of cardiovascular disease, hypertension and type 2 diabetes in later life (see Chapter 10). Babies born excessively large because of maternal diabetes during pregnancy also have an increased risk of adult type 2 diabetes.

Q79. Are small babies more likely to become obese in adult life?

There is some evidence that they have more central obesity in adult life (fat around the upper body and in the abdomen, which is a risk factor for cardiovascular disease) (see Chapter 2, Section 2.4.10, Q26 and Q75). There is no evidence that small babies develop increased *total* body fat. However, other effects of low birthweight on the body's metabolism, for example insulin resistance, may lead to increased risk of obesity-related disease at relatively low levels of total body fat. Lower birthweight babies have reduced adult lean body mass.

Q80. What is the 'fetal origins of adult disease' (or 'thrifty phenotype') hypothesis?

This hypothesis, put forward by Professor David Barker, proposes that undernutrition during critical periods of development in fetal life and infancy has permanent effects on body build, on the structure of individual organs and tissues, and on hormone systems and metabolism, which lead to an increased susceptibility to adult diseases, including cardiovascular disease, type 2 diabetes and osteoporosis.

Q81. Is growth during early childhood important in terms of cardiovascular disease risk?

The effects of linear (height) growth in childhood are unclear, although shorter adult height is a risk factor for cardiovascular disease. Accumulation of body fat during childhood has a strong effect on later disease risk. Children who cross centile lines upwards for body mass index are more likely to become fatter adults and to develop cardiovascular disease. This effect appears to be strongest in people who were born small.

Q82. Do we know what aspects of a mother's diet are associated with low birthweight?

Mothers of low past or current nutritional status, as measured by height and body mass index, or blood

levels of micronutrients, have lower birthweight babies. However, intervention trials of nutritional supplements in Western countries have shown little or no increase in birthweight. Most trials have used energy and/or protein, or single micronutrients, and started in mid-pregnancy. There is a need for more research in this area, especially of pre-conceptual effects.

Q83. Are early influences on susceptibility to later disease irreversible or is lifestyle during adulthood still important?

We do not know if adverse metabolic programming in early life is reversible or not. We do know that low birthweight interacts with childhood adiposity and with adult lifestyle risk factors for cardiovascular disease, including obesity and smoking; thus adult lifestyle is very important.

16.12 The effect of different diets on heart disease risk

Q84. Should low carbohydrate, high fat diets be encouraged as a method of weight loss?

For those who need to lose weight, the key aspect is reducing energy intake below energy expenditure. This is best done through a combination of increased physical activity and reduced energy intake. The macronutrient source of the energy (*i.e.* fat versus carbohydrate) is generally of secondary importance. The low carbohydrate approach to dieting has attracted considerable publicity, but a systematic review of the evidence has demonstrated that weight loss whilst on such diets is primarily the result of a decrease in energy intake, rather than being associated with reduced carbohydrate *per se.* The review also advises that such diets have been popularised in the absence of detailed information on the possible adverse effects of very low carbohydrate intakes, and in particular their effect in people with cardiovascular disease, dyslipidaemia, type 2 diabetes or hypertension (see Chapter 11, Section 11.3.3). Concern has been expressed about the limited information available on the effects of carbohydrate intakes below 20 g per day, about use of the diets for periods longer than 90 days, about use in people over the age of 50 years, and about the suitability of the dietary approach for people with diabetes who need to lose weight.

Q85. What does glycaemic index mean?

Glycaemic index (GI) is a measure of the rate at which sugar is absorbed into the bloodstream after eating a specific food. It is determined by comparing the blood glucose response after ingestion of a portion of the test food providing 50 g of carbohydrate with the response to a reference food proving an identical amount of carbohydrate (usually either glucose or white bread). Whole-grain foods, pasta, apples and pulses are examples of low GI foods, whereas potatoes, white bread, rice and bananas all have a higher GI. The GI of a food or food ingredient is moderated by how it is prepared, by its degree of ripeness (*e.g.* fruit) and by the other foods consumed with it (see Chapter 11, Section 11.3.2).

Q86. Are low glycaemic index diets useful for people with diabetes or those trying to lose weight?

The effect of low GI foods is to regulate the rate at which sugars are absorbed; hence inclusion of such foods is often recommended as part of the dietary regimen for diabetics. There is also evidence of beneficial effects on blood lipids (particularly in the context of the insulin resistance syndrome, *e.g.* triglycerides) in diabetic patients. However, whether the GI of foods has a role to play in appetite and body weight regulation in the general population is much more controversial. There is currently no evidence that low GI diets are superior to high GI diets with regard to long-term body weight regulation (see Chapter 11, Section 11.3.2). Also, use of GI may be confusing for the public, as many of the starchy foods recommended as important components of a balanced diet (*e.g.* potatoes and rice) have a high GI, and conversely common dietary carbohydrates that are primarily sugars may have a low GI, because about half the carbohydrate is present as fructose (*e.g.* in the case of ordinary sugar, sucrose) or as galactose (as in the case of the carbohydrate source, lactose, in milk).

Q87. Is a low fat diet the best approach to reduce heart disease risk?

Current dietary recommendations in the UK advise that the population average fat intake should be reduced to 35% of energy, a level that has more-

or-less been achieved, and that intake of saturates should be reduced to 11% of energy, and *n*-6 polyunsaturates increased to 6.5% of energy. In the past, other countries have adopted even lower targets for total fat (*e.g.* 30% of energy) and for saturates. This strategy is primarily aimed at reducing LDL-cholesterol levels. In recent years, however, evidence has accumulated about the potential of low fat/high carbohydrate diets to adversely affect other blood lipid markers (*e.g.* triglycerides and small, dense LDL particles), and on the potential benefits of other families of fatty acids on other cardiovascular risk factors, particularly insulin resistance. This evidence points to the need for more attention to be paid to the positive attributes of the long chain *n*-3 fatty acid found in oil-rich fish and to monounsaturates (as found in olive oil and rapeseed oil, for example).

In order to include such fatty acids in adequate amounts, some experts have argued strongly for a more flexible approach to total fat recommendations, especially for subjects with the insulin resistance syndrome (syndrome X). But the answer is not yet clear cut and more evidence is needed before conclusions can be drawn (see Chapter 11, Sections 11.6–11.8). What is known, however, is that regular moderate intensity physical activity can counteract the impact of a low fat diet on increased triglycerides levels, as can inclusion in the diet of long chain *n*-3 fatty acids present in oil-rich fish (see Chapter 13).

16.13 Physical activity

Q88. How much physical activity should we be doing?

It seems that there is no threshold for the minimal amount of exercise necessary to decrease cardiovascular risk, and that any increase in daily energy expenditure is beneficial. However, to gain real benefit, adults (including older adults) should take at least 30 minutes of at least moderate intensity activity on five or more days each week. Moderate intensity activity should lead to an increase in breathing rate, an increase in heart rate and to a feeling of increased warmth, possibly accompanied by sweating. The good news is that shorter bouts of activity can be accumulated during the day and still count towards the 30-minute minimum.

Children and young people should achieve a total of at least 60 minutes of at least moderate intensity physical activity each day. At least twice a week this should include activities to improve bone health (activities that produce high physical stress on the bones), muscle strength and flexibility.

Taking 30 minutes of moderate intensity activity on at least five days a week will limit health risks for diseases such as heart disease and diabetes, but 45–60 minutes per day is required to prevent the transition to overweight or obesity, and people who have been obese and who have lost weight may need to do 60–90 minutes of activity a day to maintain their weight loss.

Q89. What types of activities count?

All types of activity, provided they are of at least moderate intensity, contribute towards the 30 minute minimum, and different types of physical activity benefit different body systems, *e.g.* aerobic activity for heart, or weight bearing activities for bones. For many people, the easiest way of increasing their daily activity levels may be to incorporate more brisk walking, stair climbing or cycling into their daily routine, or to take up active hobbies such as gardening. Others will find it easier to join a gym or to make use of local sports centre facilities.

16.14 Public health recommendations to reduce cardiovascular disease risk

Q90. Are we meeting the current dietary targets for cardiovascular disease prevention in the UK?

The good news is that the target to adjust the population average fat intake down to 35% of energy has more-or-less been met in both men and women, and good progress has been made towards the 11% of energy target for saturates, although there is still room for improvement; average intake of *trans* fatty acids is now well below the 2% of energy target at 1.2% of energy in both men and women. In contrast, since the targets were last revised in 1994, salt intakes have, if anything, risen slightly and so are still well above the 6 g/day target, at 9.5 g/day, and the majority of the public still fail to meet the five-a-day fruit and vegetable target and the 'at least five-a-week' physical activity target (30 minutes of at least moderate intensity activity on at least five days per week). In the light of the new evidence summarised in this Task Force Report, an important question to be

addressed is whether the targets now need some adjustment, especially in terms of fatty acid profile, paying more attention to intakes of monounsaturates and *n*-3 polyunsaturates.

Q91. It is recommended that people increase their intake of oil-rich fish, but what about the risk from contaminants such as dioxins?

A weekly serving of oil-rich fish is recommended because, for most people, the impact of the *n*-3 fatty acids (also known as omega 3) provided will far outweigh any risk from contaminants such as dioxins and mercury. A particular role of oil-rich fish has been identified for preventing death from heart disease in those who have already suffered a heart attack (see Chapter 11, Section 11.8.3), although average intakes in the UK population remain low at a third of a portion per week.

The Scientific Committee on Nutrition has recently reviewed the evidence and made recommendations on the maximum level at which the health benefits of preventing heart disease clearly outweigh the possible risks from dioxins. Men, boys and women past child-bearing age are advised to eat up to four portions of oily fish per week, while women of child-bearing age, including pregnant and breast-feeding women, and girls, can eat up to two portions a week (www.food.gov.uk). In addition, the Food Standards Agency (FSA) has advised pregnant and breast-feeding women, and those intending to become pregnant, to avoid eating shark, swordfish and marlin because the mercury present in these fish can potentially harm an unborn child's nervous system. They also advise pregnant women to limit the amount of tuna eaten to no more than four medium-sized cans of tuna or two fresh tuna steaks a week.

Q92. Do the dietary recommendations need to be revised in light of new research into the links between diet and heart disease?

It is a recommendation of this Task Force that current dietary recommendations for the prevention of cardiovascular disease and related risk factors need to be reviewed in the light of new research now available, particularly that concerning the influence of the fatty acid profile of the diet, but also new information about other dietary factors that influence blood lipids and other risk factors (see Chapter 13).

16.15 Key references

British Heart Foundation (2003a) *Coronary Heart Disease Statistics.* British Heart Foundation, London (www.bhf.org.uk).

British Heart Foundation (2003b) *Take Note of your Heart: A Review of Women and Heart Disease in the UK 2003.* British Heart Foundation, London.