

DIETARY CHOLESTEROL: DISPELLING THE MYTHS



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The aim of this article is to briefly review the ways in which cholesterol is transported in the body and its relationship with the development of CHD. There will then follow a discussion which will seek to dispel the myths surrounding dietary cholesterol and the development of CHD, particularly in healthy individuals.

Cholesterol is an abundant fundamental lipid molecule in mammalian cells which also plays a critical role in the manufacture of steroid hormones, vitamin D and in the production of bile acids (14).

It is well documented that excessive cholesterol accumulation in the arterial intima can lead to the development of atherosclerosis (22) which is most commonly associated with an increased risk in the development of coronary heart disease (CHD) (8). However, it is also important to note that atherosclerosis can accumulate in many other arteries causing cerebral vascular accident (CVA), damage to the aorta and renal problems (23). High levels of cholesterol can also result in the formation of gall stones (6).

Given the plethora of data which supports the adverse relationship between dietary cholesterol and its role in the development of CHD (19), it is currently considered best practice in the UK and Europe for individuals who may be at risk of CHD to limit their intake of saturated fats and trans fat which are the major determinants of blood cholesterol concentrations (12). A recent review (11) has questioned the role of dietary cholesterol in the increased risks of developing CHD or increasing mortality from CHD.

CHOLESTEROL TRANSPORT AND DISEASE

Lipoproteins are particles which transport cholesterol and triglycerides, both of which are not soluble in aqueous solutions (5). Very Low density lipoproteins (VLDL) are produced by the liver with a primary function of supplying free fatty acids to tissues and are normally the predominant carriers of

circulating triglycerides. Low density lipoproteins (LDL) are by-products of VLDL metabolism and, in the normal state, are the primary carriers of plasma cholesterol which supply the body cells where required (23). VLDL and LDL are often referred to as 'bad cholesterol' (13) and high levels of both of these lipoproteins in the plasma are associated with an increased risk of CHD (28).

High density lipoproteins (HDL) are manufactured by the liver and on their release, this 'empty vessel' collects any excessive cholesterol in the peripheral tissues and transports this back to the liver (33). This is anecdotally referred to as 'good cholesterol' and data from several clinical trials suggests that raising HDL cholesterol may be beneficial in reducing the risks of CHD (25). Furthermore, HDL cholesterol confers antioxidant, anti-apoptotic, anti-inflammatory and anti proteolytic protection in endothelial cells (33). However, these lipoproteins should not be considered as separate entities

as it is well documented that it is the maintenance of a healthy LDL/HDL cholesterol ratio which is considered to be a key marker of CHD risk (11).

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SHOULD WE RESTRICT DIETARY CHOLESTEROL?

The current recommendation in the UK and Europe is that the individual total blood cholesterol levels should be four millimoles (mmol)/litre or less (9). The most recent dietary guidelines from the USA recommend an intake of 300mg or less of cholesterol per day in healthy individuals with a further restriction of less than 200mg per day in those individuals classified as a greater risk of heart

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Box 1: Cardio protective dietary guidelines adapted from the European Society of Cardiology (10)

- Saturated fats to account for <10% of total energy intake
- Limit trans fats to <1.0% of total energy from natural origin
- Avoid trans fats from processed meats and other foods
- <5.0g of salt per day
- Encourage at least 35g to 45g of fibre per day from wholegrain foods
- Two to three servings of fruit per day
- Two to three servings of vegetables per day
- At least one portion of oily fish a week
- Limit alcohol consumption to two glasses per day for men and one glass per day for woman

disease (34). However, according to Spence et al (32), dietary cholesterol should be restricted in all populations and not just in those with CHD. This advice is somewhat equivocal and confusing and begs the question as to whether there should be a 'carte blanche' dietary restriction of cholesterol applied to those who are otherwise healthy, or in those with an increased risk of developing CHD without a genetic predisposition.

For those individuals with familial hypercholesterolemia (FH) it is suggested that reducing dietary cholesterol is an effective adjunct when combined with statins (7). Given such individuals increased risk of developing premature CHD, this is a sensible safe practice (28). However, the data from the Spence report (32) was based on animals which were fed an equivalent of 9,500mg of cholesterol per day and therefore application of such results to a human population is both unethical and controversial (35). Other evidence considered in the report was from epidemiological studies in which no adjustments were made for the contribution of saturated fat in the diet which is a confounding factor, thus altering the reliability and validity of the study (11).

However, nutritional epidemiological studies form the scientific basis on which public health nutrition information is devised and implemented (20). Therefore, data from epidemiological studies per se, although not deemed to be the 'gold standard' of clinical research, plays an important role in informing evidenced based practice.

Extensive research originating from early 20th century epidemiological studies, including the Framingham study (21) and the Nurses study (18), does not support a relationship between dietary cholesterol and CHD. More recently, a review from Fernandez (11), which analyses both epidemiological studies and data from randomised clinical controlled trials, noted to be the gold standard in research (27), also reports that dietary cholesterol has no effect on blood cholesterol levels CHD risk or CHD mortality. But these results should be interpreted with caution as there is now a growing body of evidence which highlights a significant association with dietary cholesterol and an increased risk of CHD in the diabetic population (17).

WHAT IS BEST PRACTICE?

Hayward and Krumholz (15) suggest that there is no firm data to support the theory that patients at risk of CHD should be treated according to LDL targets. In contrast,

the British Dietetic Association (3) argues that it is better to encourage individuals at risk of CHD to decrease the amount of saturated fat in their diet as opposed to concentrating solely on the reduction of LDL cholesterol. This is because diets high in saturated fat are said to not only increase the amounts of LDL cholesterol but can also decrease the levels of HDL cholesterol, suggesting that saturated fats are potentially more atherogenic than cholesterol alone (1). However, the evidence surrounding the atherogenicity of saturated fat is equivocal (30) with recent studies suggesting that some dairy products, for example cheese which is high in saturated fat, can actually lower LDL cholesterol, when compared to butter with the same amount of saturated fat (16).

Moreover, as egg yolk has a high cholesterol concentration, limited egg consumption has been recommended in the past to lower the risk of ischaemic heart disease (24). But there is now a growing body of evidence refuting this atherogenic relationship with egg consumption and more recent data from a randomised controlled trial asserts that eating eggs on a daily basis can lead to increased levels of plasma HDL and improvements in HDL profiles in those with metabolic syndrome when compared to those consuming a yolk-free egg substitute (2). Furthermore, consuming eggs on a daily basis is not thought to be associated with an increased risk of CHD or stroke in healthy individuals (29). More research is required, however, before this recommendation is made for diabetic individuals (26). Healthcare professionals should also be aware that, although some foods such as shellfish and in particular prawns and other cuts of offal, contain high amounts of cholesterol, these foods have a lesser effect on raising plasma cholesterol levels when compared to a diet which is high in foods containing saturated fat (3).

It is also important to recognise that there are some individuals in whom blood cholesterol rises as a response to a high cholesterol intake by increasing both LDL and HDL cholesterol, but with no subsequent changes to the LDL/HDL cholesterol ratio (11), which, as previously stated, is a more sensitive marker and predictor of increased risk of CHD (31). The European Society of Cardiology (10), however, asserts that a 'healthy diet' is the cornerstone of CHD prevention and has produced some dietary guidelines for healthcare professionals which are summarised in Box 1.

Diet is an integral part of lifestyle and although this will contribute towards improving heart health, it is recommended that adults of all ages should be encouraged to participate in physical activity in conjunction with a healthy eating plan (4). Moreover, the British

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Dietetic Association (3) suggests that other lifestyle factors, including overweight, obesity and smoking should also be addressed in order to promote a healthy cardiovascular system.

CONCLUSION

Despite these equivocal findings, lowering dietary cholesterol might reduce the risk of CHD considerably in a subgroup of individuals who are highly responsive to changes in cholesterol intake (19). Certainly, this advice applies to those individuals who have a genetic susceptibility to hypercholesterolemia and for

those individuals with diabetes who also have an increased risk of developing CHD.

However, the most recent evidence consistently indicates that dietary cholesterol does not increase the risk for heart disease in a healthy population. Therefore, it is best practice to advise these individuals to maintain a healthy weight by following a balanced diet incorporated with physical exercise. For any person who wishes to consider restriction of dietary cholesterol, it is advisable to consult their own GP or a qualified dietitian prior to making any dietary changes.

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Questions relating to: <i>Dietary cholesterol: dispelling the myths.</i>	
Type your answers below and then print for your records . Alternatively print and complete answers by hand.	
Q.1	What is cholesterol?
A	
Q.2	What are the dangers of high levels of arterial cholesterol?
A	
Q.3	What are lipoproteins and why are VLDLs referred to as 'bad cholesterol'?
A	
Q.4	Why are high density lipoproteins considered good cholesterol?
A	
Q.5	Explain the BDA's advice on LDL targets for individuals at risk of CHD.
A	
Q.6	What is the current thinking on egg consumption and its effect on cholesterol levels?
A	
Q.7	The European Society of Cardiology has produced guidance on diet and its cardio protection. Describe at least six of the dietary recommendations in these guidelines.
A	
Q.8	What is best practice advice for reducing the risk of CHD for all individuals?
Please type additional notes here . . .	