The beneficial role of peanuts in the diet – an update and rethink!

Peanuts and their role in CHD

Jennette Higgs

Introduction

Surprising as it may seem and despite their fat content, in the last few years, research has indicated firmly that nuts and peanuts are in fact positively beneficial to our health. Peanuts have been and remain an important staple food for many Asian, African and American populations, where they make a significant nutritional contribution to the diet. Yet the last few decades of education on eating less fat in Britain helped to label certain foods as good or bad simply because of their fat content. Peanuts were one of those casualties, being some 46 per cent fat, they were swiftly labelled as a “baddy”. However, recent epidemiological, prospective and clinical studies have illustrated that regular nut consumers have a reduced risk of heart disease. Furthermore, research supports a positive role for nuts in the battle against cancer, obesity and type II diabetes (Awad et al., 2000; McManus et al., 2001; Jiang et al., 2002).

Background

Although peanuts are classed with other nuts for the purposes of dietary studies, they are, in fact, not nuts at all. Peanuts are legumes and grow underground, often being referred to as groundnuts. They have many characteristics of both (tree) nuts and pulses and as such are quite unique. At 46g/100g fat, peanuts contain slightly less fat than most other nuts. More than three-quarters of the fat in peanuts is unsaturated, with 48 per cent monounsaturated, oleic acid. Peanuts and peanut butter provide a valuable nutritional supplement, being high in plant protein, the amino acid arginine, energy and fibre, as well as offering a generous supply of vitamins, minerals and phytochemicals. A part from their role in the prevention of CHD, peanuts have the potential to make a significant nutritional contribution to our health. This is the subject of a separate paper in Nutrition and Food Science Journal.

Being a plant food, peanuts contain no cholesterol, and peanut butter contains no butter!
Peanuts and their role in coronary heart disease (CHD)

It is now widely recognised and supported by both metabolic and epidemiological studies, that it's the type of fat that influences cholesterol levels, not the total fat levels. Replacing saturated fat with unsaturated fat is more effective at lowering the risk of CHD than simply reducing total fat intake. Furthermore, secondary prevention trials have effectively demonstrated a strong protective effect for adding n-3 fatty acids from fish or plant sources to the diet, without reducing the total fat intake. Added to this, dietary consumption and long-term follow-up data from almost 85,000 female nurses enrolled in the Nurses’ Health Study have just concluded that among women higher consumption of fish and separately omega-3 fatty acids is associated with a lower risk of CHD (Hu et al., 2002).

Several scientific studies have demonstrated that moderate fat diets, where the fat is supplied by monounsaturated fatty acids (MUFAs) and where saturated fatty acids (SFAs) are kept low, will reduce total cholesterol levels and more significantly reduce levels of the harmful low density lipoprotein (LDL) cholesterol fraction in much the same way as diets low in total fat. In fact, low-fat diets can reduce levels of the protective high-density lipoprotein (HDL) cholesterol fraction in the blood. In contrast, diets where saturated fat is replaced with MUFAs and polyunsaturated fatty acids (PUFAs) help to reduce levels of LDL-cholesterol, without adversely affecting the HDL fraction, so helping to maintain normal blood cholesterol levels. The beneficial effects of increased MUFA intake have been recently debated at a workshop convened by the British Food Standards Agency (Sanderson et al., 2002). Current UK dietary recommendations for SFA are to reduce intakes to less than 10 per cent of energy. However, this target has not been achieved, with current intakes at 15 per cent of energy. The workshop agreed that a more effective public health strategy for reducing CHD risk may be to replace SFA with MUFAs as this may prove more efficacious in achieving the target of less than 10 per cent energy as SFA. The protective effects of olive oil, a significant source of oleic acid, the main MUFA in the diet, are now well publicised (Berry et al., 1995; Bonanome et al., 1992). Furthermore, over the last decade it has been recognised from scientific studies that diets that include nuts are also cardioprotective (Kris-Etherton et al., 1999a). Nuts are rich sources of unsaturated fat, particularly monounsaturated, and they are low in saturated fat. Additionally nuts have other potentially protective components including magnesium, vitamin E, fibre, arginine and a range of phytochemicals including resveratrol.

A review of nut studies calculates that, irrespective of the amounts of fat and nuts in the diet, cholesterol-lowering diets that include nuts can reduce total cholesterol by approx. 4-16 per cent and LDL-cholesterol by approx. 9-20 per cent. This compares to a 3-14 per cent reduction in total cholesterol achieved with the American Step I diet (low fat, <30% energy, low SFA, 8-10 per cent) in normocholesterolaemic adults (Kris-Etherton et al., 1999b).

Published results from five major epidemiological studies conclude that frequent nut consumption reduces CHD risk and a review by Kris-Etherton et al. (2001) illustrates these results. The studies under review included The Adventist Health Study, Iowa Women’s Study, Nurses’ Health Study Physicians’ Health Study and the Cholesterol and Recurrent Events (CARE) Study. Her review concludes that consuming a 1oz/30g serving of nuts more than once per week significantly decreases relative risk of CHD in men and women. Moreover, there appears to be a dose-response relationship where risk of CHD is inversely associated with frequency of nuts consumed from less than one to more than five servings per week. This association is true for men and women and for non-fatal MI, fatal MI and sudden cardiac death, even after taking account of confounding factors. These results suggest that there is an independent protective effect of nut consumption on CHD risk where consuming more than 5 1oz/30g servings per week is associated with a 18-51 per cent reduction in CHD risk in all subjects (or a 25-39 per cent reduction in the cohort of subjects that characterise the US adult population, including the very elderly and those with coronary disease).

Nuts include almonds, brazils, cashews, hazelnuts, macadamia nuts, pecans, pistachios, walnuts and legume peanuts. Dietary data for the five studies under review
were collected from food frequency questionnaires and results for specific nuts have not been evaluated except in the Nurses’ Health Study (Hu et al., 1998). This group reported results for nuts and separately for peanuts for which dietary data had been collected in 1986 and 1990. The results demonstrated that peanuts specifically were inversely associated with CHD risk, by a similar magnitude to all nuts combined. Typically, peanuts account for about 68 per cent of total nut consumption in the USA, the site for all five of these epidemiological studies (USDA, Center for Nutrition Policy and Promotion, 2000). These results indicate therefore that peanuts contribute significantly to the overall protective effects seen by nuts as a whole. Since the contribution made by nuts to the UK diet is currently far lower than in the USA, these results might suggest that increasing nut consumption in the UK could make a specific, positive contribution to reducing risk of CHD. Further research would be required to validate this.

Over the last decade, more than 11 clinical studies have been conducted to test the effects of different nuts, including peanuts and peanut butter, on the blood lipid and lipoprotein levels. Two studies have looked specifically at peanuts. O’Byrne et al., (1997) compared a typical low-fat diet with a peanut-supplemented, low-fat diet, where subjects received between 35–68g peanuts daily depending on their total energy needs. The study group of free-living, post-menopausal, hypercholesterolaemic women in the USA was instructed by a registered dietitian. By six months, LDL- and total-cholesterol levels reduced significantly by 12 per cent and 10 per cent respectively from baseline in those on the peanut-supplemented diet, whereas those on the low-fat diet saw no change in these variables. Furthermore, triglyceride levels remained unchanged in the peanut-supplemented group but increased in the low-fat group by 14 per cent. It has been suggested, however, that the preferred results from the peanut-supplemented group may have been influenced by the fact that this group at baseline had higher LDL and total-cholesterol levels.

A further study by Kris-Etherton et al. (1999a) used a well-designed, randomised, double-blind, five-period crossover study to compare the average American diet with four cholesterol-lowering diets: a low-fat, low saturated-fat (American Step II) diet; and three moderate-fat, high monounsaturated-fat diets based on olive oil, peanut oil, and peanuts/peanut butter. The diet composition and lipid results are illustrated in Table I. Although all four cholesterol-lowering diets successfully reduced total and LDL-cholesterol, the high MUFA diets effectively reduced triacylglycerol levels whereas the more traditional low fat, low saturated fat diet markedly increased triacylglycerol levels. HDL-cholesterol was little affected by the high MUFA diets whereas the reduction in HDL observed with the low-fat, low saturated-fat diet was also significant. The authors estimated the change in risk for CHD that could be expected to occur from following each of these diets, using published factors for LDL-, HDL and triacylglycerol levels. Collectively this suggested that the low-fat, low saturated-fat diet would be expected to decrease risk for CHD by approx 12 per cent, whereas the moderate-fat, high-MUFA diets would decrease risk more favourably by 25 per cent (olive oil); 16 per cent (peanut oil) and 21 per cent (peanut/peanut butter). The purpose of including different sources of peanuts in this study design was to uncover any potential for a beneficial effect from the non-fatty acid constituents in peanuts. Any such effects would not be observed with the peanut oil or olive oil diets. These results are suggestive of an effect, although more research would be required to demonstrate this.

Several feeding studies have now been undertaken to test the effects of different nuts, including peanuts, on plasma lipids and lipoproteins (O’Byrne et al., 1997; Colquhoun et al., 1996; Rajaram et al., 2001). In 1999 Kris-Etherton et al. reviewed the nine nut studies then available and compared the actual cholesterol responses (total, HDL, LDL) achieved with the nut-containing diets within these studies to the predicted responses derived using accepted predictive equations (Mensink and Katan, 1992; Hegsted et al., 1993; Yu et al., 1995). This analysis indicates that nuts have a greater effect on total and LDL-cholesterol than can be predicted based on the fatty acid profiles of nuts alone. Indeed, the magnitude of the effect, according to Kris-Etherton et al. (1999a), is at least 25 per cent greater than would be predicted. In other words, it seems that there are one or more non-fat components of nuts that provide protection against CHD when consumed on a regular
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Table I Summary of results from study of low-fat and moderate-fat cholesterol-lowering diets on risk of CHD compared to average US diet

<table>
<thead>
<tr>
<th>Diet analysis</th>
<th>Average American</th>
<th>Low-fat, low saturated-fat</th>
<th>Olive Oil</th>
<th>Peanut oil</th>
<th>Peanuts and peanut butter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate$^a$</td>
<td>50</td>
<td>59</td>
<td>50</td>
<td>50</td>
<td>47</td>
</tr>
<tr>
<td>Total Fat$^a$</td>
<td>34</td>
<td>25</td>
<td>34</td>
<td>34</td>
<td>36</td>
</tr>
<tr>
<td>SFA$^a$</td>
<td>16</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>MUFA$^a$</td>
<td>11</td>
<td>12</td>
<td>21</td>
<td>17</td>
<td>18</td>
</tr>
<tr>
<td>PUFA$^a$</td>
<td>7</td>
<td>6</td>
<td>6</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>Cholesterol mg/d</td>
<td>400</td>
<td>200</td>
<td>200</td>
<td>200</td>
<td>200</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood Lipid results</th>
<th>Approximate % responses to each diet compared to the average American diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>0 −10 −10 −10 −10</td>
</tr>
<tr>
<td>LDL-cholesterol</td>
<td>0 −14 −14 −14 −14</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>0 −4 0 0 0</td>
</tr>
<tr>
<td>Triacylglycerol</td>
<td>0 +15 to 21 −13 −13 −13</td>
</tr>
</tbody>
</table>

Notes: $^a$ As per cent of total energy

Source: Modified from Kris-Etherton et al. (1999)

basis. The authors point out, however, that specific research is still required to directly test this figure (Kris-Etherton et al., 1999b). A recent study has begun to unravel the mechanisms by which nuts offer protection against CHD. Albert et al. (2002) examined the associations between nut consumption and risk of sudden cardiac death together with other CHD end points in a cohort of 21,454 US male physicians followed up for 17 years as part of the Physician’s Health Study. The authors found that regular consumption of two or more portions of nuts (2oz/week) by middle-aged men reduced risk specifically of sudden cardiac death by 47 per cent, whereas nut consumption was not associated with significantly reduced risk of non-sudden coronary heart disease death or non-fatal myocardial infarction. Taking all fatal coronary heart disease events, consuming two or more portions of nuts per week was associated with a 30 per cent reduced risk compared to those who rarely or never consumed nuts. These results were significant even after controlling for known cardiac risk factors and other dietary habits. The authors conclude that this distribution of benefit points to nuts playing a role in reducing fatal ventricular arrhythmias. There are several components of nuts that could have such a role, based on observational and experimental studies. These include the α-linolenic acid content of nuts, which, although low in nuts compared to oily fish, has been shown to have antiarrhythmic effects even in very small amounts (Albert et al., 2002). Nuts are also rich sources of potassium, magnesium and vitamin E, which all have antiarrhythmic potential (Kris-Etherton et al., 1999b).

Summary

It is now acknowledged that as well as modifying the fat component of the diet, other dietary constituents can confer additional protection from CHD. In addition to nuts having a fatty acid make-up that has been shown to protect against CHD effectively, nuts also contain a useful number of non-fat cardioprotective constituents including dietary fibre, plant protein, micronutrients (copper, magnesium, vitamin E), plant sterols and phytochemicals. The significance of each of these constituents in protecting our health through regular nut consumption is not yet fully realised. The range of nutrients and bioactive substances present in peanuts known to independently protect against CHD provides several mechanisms through which they can offer protection and as such can be beneficial to health. The full extent of this role is yet to be discovered, although sufficient evidence is now available to confirm that peanuts should have a place within a heart healthy diet.

For nutritional information, latest research updates and healthy recipes visit The American Peanut Council Web site at: www.peanutsusa.org.uk/
References


