

Eggs and dietary cholesterol – dispelling the myth

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Summary

Dietary cholesterol in foods such as eggs has only a small and clinically insignificant effect on blood cholesterol, especially when compared with the much greater effects of dietary saturated fatty acids on blood cholesterol. These facts are now well established and, as a consequence, recommendations from major food and health bodies concerning dietary cholesterol have been relaxed in the UK and elsewhere in recent years. However, in the minds of the public, cholesterol in the diet, specifically from eggs, continues to be viewed with suspicion and that view is still reflected in the advice of some professionals.

Introduction

The popular misconception that eggs are ‘bad for your blood cholesterol’ and therefore ‘bad for your heart’ persists among many people and still continues to influence the advice of some health professionals. This myth prevails despite strong evidence to show that the effects of cholesterol-rich foods on blood cholesterol are small and clinically insignificant. It also prevails in the face of the incontrovertible fact that changes in dietary saturated fatty acids (SFA) influence the level of circulating low density lipoprotein (LDL)-cholesterol to a much greater extent than the dietary cholesterol in foods. The misunderstanding of the relationship between dietary and blood cholesterol originated in part from the erroneous belief that the cholesterol we eat converts directly into blood cholesterol, but also from the strong dietary messages about egg restriction that emanated primarily from the United States (US) in the 1970s. The ubiquitous American egg white omelette options on menus that are otherwise dominated by high saturated fat and energy-dense choices should be viewed as a nutritional contradiction to cardiovascular health. In spite of the lifting of restrictions on egg consumption by major food and health advisory bodies in the UK, Europe and else-

where, the mythology lives on against a background of escalating obesity and advice to lower SFA intake – a climate in which the egg, as a protein-rich, nutrient-dense food that is not high in energy or SFA, can make a valuable contribution to a healthy balanced diet.

Nutrient composition of eggs

Eggs are a nutrient-dense food that, contrary to popular opinion, are not high in SFA or in energy. The nutrient composition of a raw egg is shown in Table 1. Eggs are rich in cholesterol (391 mg per 100 g in a raw egg; approximately 225 mg in a medium-sized egg) (FSA 2002). However, the total fat and SFA content is not high and the fat in eggs is predominantly unsaturated (44% monounsaturated; 11% polyunsaturated). An egg is also relatively low in energy (896 kJ/214 kcal per 100 g; approximately 335 kJ/80 kcal in a medium-sized egg) and is a valuable source of many essential micro-nutrients and a rich source of high quality protein (FSA 2002). The protein content of the diet has long been associated with increased satiety and there is some evidence that the high protein content of eggs may contribute to greater satiety than, for example, white bread and ready-to-eat breakfast cereals (Holt *et al.* 1995). There is also emerging evidence from the US that eggs could help to promote weight loss in overweight and obese subjects by increasing feelings of satiety and reducing short-term energy intake (Vander Wal *et al.* 2005).

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Table 1 Selected nutrient content of raw hens' eggs

	Per 100 g	Per medium egg (average 58 g)
Energy kJ/kcal	627/151	324/78
Protein (g)	12.5	6.5
Carbohydrate (g)	trace	trace
Fat (g)	11.2	5.8
Saturated fatty acids (g)	3.2	1.7
Monounsaturated fatty acids (g)	4.4	2.3
Polyunsaturated fatty acids (g)	1.7	0.9
Cholesterol (mg)	391	227
Retinol (μ g)	190	98
Vitamin D (μ g)	1.3	0.9
Riboflavin (mg)	0.47	0.24
Folate (μ g)	50	26
Vitamin B ₁₂ (μ g)	1.8	1.3
Phosphorus (mg)	200	103
Iron (mg)	1.9	1.0
Zinc (mg)	1.3	0.7
Iodine (mg)	53	27
Selenium (μ g)	11	6

Source: FSA (2002).

Origins of the dietary cholesterol misconception

The misconception surrounding the effects of dietary cholesterol on health originates from the early part of the 20th century, when experimental models for coronary atherosclerosis were developed by feeding cholesterol or cholesterol-rich foods such as butter to animals, particularly the rabbit or rat; it is important to note that many of these regimens were therefore also high in SFA. Such studies led to the somewhat simplistic conclusion that because cholesterol was the key component of the classic atherosclerotic lesion, dietary cholesterol must be central to the aetiology of the disorder in both animals and humans (see Mann 2001). Extrapolation from these animal models to humans is notably unreliable because of the unphysiological quantities of fat fed to animals adapted to low fat, high carbohydrate diets and marked variations in lipoprotein physiology, response to diet and susceptibility to coronary disease between different animal species (McNamara 2001; Lee & Griffin 2006).

Since then, data from large prospective cohort and intervention studies have established an indisputable link between raised LDL-cholesterol and increased risk of coronary heart disease (CHD). However, it is erroneous to assume that this relationship is simply cause and effect and that small changes in LDL-cholesterol always translate into clinically significant changes in CHD risk.

An overview of the evidence

A plethora of ecological and clinical studies that examined the effects of dietary cholesterol (usually as eggs) on serum cholesterol levels were published in the latter half of the 20th century; again many of these studies were subject to confounding by the presence of large amounts of SFA in the experimental diets and used extreme contrasts of cholesterol (>1000 mg per day). In reviewing these studies, Mann (2001) concluded that although cross-population and longitudinal studies showed associations between dietary cholesterol and CHD, the results most likely reflected the close association between dietary cholesterol and SFA, which tend to exist together in the same foods.

Lee and Griffin (2006) also reviewed these studies, together with the more rigorous studies undertaken in the 1990s (Ginsburg *et al.* 1994, 1995; Knopp *et al.* 1997), in which the background diet was low in both total fat and SFA. They concluded that while dietary cholesterol can increase serum cholesterol, both LDL- and high density lipoprotein (HDL-) cholesterol is increased. They observed that the effect is apparent at cholesterol intakes of less than 400 mg per day, but that it is small and the potential adverse impact of raised LDL-cholesterol on CHD risk is countered by the potential beneficial effects of increases in HDL-cholesterol. A meta-analysis that examined the effects of dietary cholesterol intake from eggs on the total to HDL-cholesterol ratio (an accepted marker of CHD risk) demonstrated that the ratio was adversely influenced by cholesterol intake (Weggemans *et al.* 2001). An unexpected finding in this study was that the greatest adverse effect was in subjects with the lowest ratios (*i.e.* those with the lowest risk of CHD), a result that was difficult to explain and raises doubt as to the validity of the overall conclusion. More recently, a small randomised, controlled, parallel study examined the combined effects of weight loss by energy restriction and increased dietary cholesterol (two eggs per day) on circulating LDL-cholesterol and demonstrated no increase in total plasma or LDL-cholesterol when accompanied by moderate weight loss (Harman *et al.* 2008). Similarly, another recent small study of 28 overweight and obese male subjects, this time on a carbohydrate-restricted diet and consuming either three eggs a day or an egg substitute, found that the egg consumers responded with no change in LDL-cholesterol but with a significant increase in HDL-cholesterol levels compared with those not eating eggs (Mutungi *et al.* 2008). There were substantial differences in the relative amounts of total fat and carbohydrate in the two weight loss regimes used in

these studies that might account for the different metabolic findings between them. However, together these studies add further weight to the suggestion that eggs can be consumed daily as part of an otherwise healthy diet that is relatively low in SFA without adverse effects on serum cholesterol.

Despite observations that dietary cholesterol can increase serum cholesterol, it is important to note that there is no consistent evidence from the 30 or more years of prospective studies that dietary cholesterol or specifically egg consumption has an independent association with risk of heart disease (Kritchevsky & Kritchevsky 2000; McNamara 2000, 2001; Kritchevsky 2004; Lee & Griffin 2006). Only a limited number of studies have specifically explored egg consumption and cardiovascular risk. Combined prospective survey data from the Health Professionals' Follow-up Study and the Nurses' Health Study (over one million subjects) indicated that there was no difference in cardiovascular risk (CHD or stroke) between people who consumed less than one egg a week or more than one egg a day (Hu *et al.* 1999). Similarly, a Japanese study of over 90 000 middle-aged men and women failed to find any relationship between egg consumption and CHD risk (Nakamura *et al.* 2006); however, the paucity of dietary intake data collected in this study limits its value. Although a re-analysis of data from the Physicians' Health Study (Djoussé & Gaziano 2008) reported an increase in all-cause mortality associated with egg intakes of seven or more per week, no dietary intake data were reported in this study and hence an analysis of SFA contribution to risk was not performed; the authors' conclusions were dismissed in an accompanying editorial that highlighted statistical flaws in the study (Robert 2008). It has been suggested that people with diabetes may respond differently to dietary cholesterol (Hu *et al.* 1999) and hence may need to be more cautious about dietary cholesterol intake (BNF 2005). However, there is also evidence suggesting that dietary cholesterol and eggs have significantly less impact in insulin-resistant subjects (Lee & Griffin 2006).

Changing recommendations concerning dietary cholesterol and egg intake

Health advisory bodies in the US have historically issued more stringent recommendations on egg consumption than in the UK. From the late 1960s, people at greater risk of familial hyperlipidaemias, who were on cholesterol-lowering diets, were advised to greatly restrict egg intake and from 1970, all US consumers were warned about egg consumption. One interpreta-

tion of this precautionary warning to the US population could be that if the evidence is equivocal then let us assume the worst (McNamara 2001; Klein 2006). Since 2000, the American Heart Association (AHA) has removed specific reference to eggs in their dietary recommendations for heart health, although they have retained the recommendation to restrict cholesterol intake to below 300 mg per day (Krauss *et al.* 2000; AHA 2008). The USDA Dietary Guidelines for Americans also advise daily cholesterol intakes of below 300 mg for healthy individuals and below 200 mg per day for people with elevated LDL-cholesterol levels, but again make no specific references to eggs (USDA 2005). On the other hand, the US Institute of Medicine (IOM) Food and Nutrition Board continues to recommend minimisation of cholesterol within the bounds of a nutritionally adequate diet (IOM 2005); as Klein (2006) observes, this is based on the IOM's inability to set a no-observed-adverse-effect level for dietary cholesterol.

In the UK, a precautionary approach was also adopted by the Department of Health Committee on Medical Aspects of Food Policy (COMA) when it reviewed dietary advice in relation to cardiovascular disease. COMA recommended that average dietary cholesterol intake should not rise beyond the average value of 245 mg per day, but acknowledged that the effects of dietary cholesterol were quantitatively less than that of SFA (DH 1994). This approach was echoed in the advice of the British Heart Foundation (BHF), which at that time recommended that although dietary SFA intake was more important for plasma cholesterol reduction, it was 'probably wise' to limit egg consumption to about three per week, a number still quoted by some health professionals.

The more recent realisation of the lack of evidence on which to base recommendations for the restriction of dietary cholesterol or eggs has led to a slow but sure relaxation of policy on egg consumption in most countries, although not universally in the US. In the UK, the BHF places emphasis on the reduction of SFA intake, stressing the fact that dietary sources of cholesterol such as eggs, offal and seafood such as prawns do not usually contribute greatly to circulating cholesterol levels. This charity no longer suggests a limit on the number of eggs consumed and in fact uses eggs in recipes that encourage a healthy approach to eating and weight control (BHF 2008). Similarly, the FSA sets no restrictions on the number of eggs that should be eaten within an otherwise varied diet, again placing far greater emphasis on the reduction of dietary SFA intake to reduce blood cholesterol levels and recommending eggs as a good choice in the context of a healthy balanced diet (FSA 2008a,

2008b). However, individuals who have familial hypercholesterolaemia, an inherited susceptibility to high blood cholesterol levels associated with a greatly increased risk of premature development of CHD that affects about one in 500 people in the UK, may be particularly sensitive to dietary cholesterol intake and are currently still advised to restrict egg consumption to two to three per week (Heart UK 2008).

The European Heart Network (2002) also emphasises the importance of dietary SFA and *trans* fatty acids in relation to the lowering of blood cholesterol and does not specifically recommend restriction of dietary cholesterol or eggs. Similarly, the Irish Heart Foundation (2008) emphasises SFA reduction and promotes the consumption of eggs in the context of a healthy, varied diet. Although the Heart Foundation (Australia) previously advised dietary cholesterol and egg restriction, neither this body nor the National Health and Medical Research Council of Australia (NHMRC) make any specific recommendation about dietary cholesterol (NHMRC 2006; Heart Foundation (Australia) 2008).

Conclusions

There is no doubt that a raised concentration of serum LDL-cholesterol increases the risk of CHD. Although evidence from various studies indicates that increasing dietary cholesterol intake can raise serum LDL-cholesterol levels, many of the earlier studies were confounded by the presence of SFA in the experimental diets. The overall effects are small and clinically insignificant in comparison with the established LDL-raising effects of SFA. Studies indicate that HDL-cholesterol levels also increase in response to egg-feeding, thereby to some extent attenuating the effects of egg intake on LDL-cholesterol. Evidence from prospective cohort studies suggests that this relatively small increase in circulating cholesterol does not correspond with an increase in CHD risk. In recognition of these facts, advice in relation to eggs as a source of dietary cholesterol has changed in recent years. Most health and heart advisory bodies in the UK, Europe and elsewhere no longer set limits on the number of eggs people should eat, provided they are consumed as part of a healthy diet that is not high in SFA. This is a message that health professionals need to communicate to the public.

The egg is a nutrient-dense food, a valuable source of high quality protein and essential micronutrients that is not high in SFA or in energy. In the current difficult financial climate, eggs can play a useful role as a relatively inexpensive source of nutrition for all and especially for people on low incomes. The high protein

content of eggs may help with weight maintenance or loss, a significant factor in the context of the current fight against obesity. It is high time that we dispelled the mythology surrounding eggs and heart disease and restored them to their rightful place on our menus where they can make a valuable contribution to healthy balanced diets.

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