Dietary cholesterol — the role of eggs in the prudent diet

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The recommendation that not more than 300 mg cholesterol be consumed daily to prevent high serum cholesterol levels and coronary heart disease is often used to justify a restriction of egg intake to three or four per week. One egg contains about 200 mg of cholesterol, but eggs are also excellent and relatively inexpensive sources of essential amino acids and certain vitamins. In this paper, the place of eggs in a prudent, cholesterol-lowering diet as a substitute for other animal products, is scrutinised. The extra cholesterol, where considered as the only variable, will increase serum cholesterol levels, but the effect is relatively small. The exclusion of eggs from the diet should be weighed against deprivation of essential nutrients especially in vulnerable groups. While restriction of egg intake in westernised populations seems justifiable, the upper limit of three or four per week may not always be applicable, depending on the overall diet and lipid profile of the individual.

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Dietary advice to the public takes two forms. The recommended dietary allowances (RDAs)[1] define the amount of individual nutrients required for adequate nutritional status of all individuals. Dietary guidelines,[2,3] on the other hand, aim at decreasing the incidence of nutrition-related degenerative diseases, such as coronary heart disease (CHD) in westernised populations. These guidelines form the basis for a so-called prudent diet.

Low-density lipoprotein cholesterol (LDLC) is generally accepted as a major risk factor for CHD, and dietary guidelines therefore aim to reduce LDLC levels. High levels of high-density lipoprotein cholesterol are thought to protect against CHD. Current guidelines promote a reduction in total fat intake, partial substitution of polyunsaturated and mono-unsaturated fatty acids for saturated fatty acids, and an increase in (soluble) dietary fibre. A favourable role for β-carotene and vitamins A, C and E as free radical scavengers has been suggested.[2-4] Among the general public the guidelines can create false hopes, feelings of guilt and even fear of certain foods.[4-6] However, the guidelines, when based on sound scientific evidence and when translated into practical, palatable and effectively protective diets, may help to reduce CHD incidence.

The significance of dietary cholesterol is complex. The advice to restrict cholesterol intake to less than 300 mg per day in American and South African (but not in Canadian and many European) dietary guidelines[2,3] is often interpreted as advice to limit egg consumption to three or four per week. Eggs are not the richest source of dietary cholesterol (Table I), but they are responsible for a major portion of the cholesterol in the Western diet.

Table I. Cholesterol and energy content of foodstuffs[7]

Food (100 g)	Energy (kJ)	Cholesterol (mg)
Eggs		
Egg (boiled)	637	419
Egg white (raw)	221	—
Egg yolk (raw)	1 471	1 238
Milk and products		
Whole milk	257	14
Skim milk	146	2
Cheese (e.g. cheddar, whole milk)	1 685	105
Cheese (e.g. cottage, low fat milk)	433	15
Meat and products		
Bacon (fried, lean)	1 383	67
Beef (lean mince)	917	93
Mutton (leg, roasted fat trimmed)	798	89
Pork sausage (cooked)	1 543	83
Chicken (without skin, cooked)	803	88
Venison (braised)	503	85
Veal (lean, roasted)	718	119
Organ meats		
Liver (sheep, cooked in vinegar)	615	281
Liver (beef, fried)	906	482
Liver (chicken, cooked)	655	631
Brain (sheep/beef)	608	2 043
Kidney (beef, cooked)	601	387
Kidney (sheep, braised)	575	565
Heart (beef, cooked)	733	193
Heart (sheep, cooked)	776	249
Tongue (ox, simmered)	1 185	107
Fish and seafood		
Calamari (fried, oil)	732	260
Caviar	1 053	588
Salmon (steamed)	823	80
White, low-fat fish (grilled)	462	60
Mackerel (canned, water)	760	56
Shrimp/prawns (boiled)	414	195
Tuna (canned, water)	487	30

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In this paper we examine the evidence relating to dietary cholesterol as a CHD risk factor and the place of eggs in a prudent, cholesterol-lowering diet. Extra cholesterol in the diet, when considered as the only variable, will raise serum LDLC, the increase being relatively small. Extra eggs in the diet do not invariably raise LDLC; their effect depends on the foodstuffs that are displaced. Likewise, cessation of egg consumption does not systematically lower LDLC. Eggs contribute essential nutrients to the diet, and removal of eggs from the diet should be weighed against the effect of deprivation of these nutrients.

Dietary cholesterol, serum cholesterol and CHD

Metabolic ward and controlled studies have demonstrated that an increase in dietary cholesterol as the only dietary variable, taken either as crystalline cholesterol, egg yolk or whole egg, raises serum total cholesterol (TC).[8] The mean change in TC levels of groups of subjects can be predicted by the Keys equation,[9] $\Delta Chol = 1,5\ (Z_2-Z_1)$, where $\Delta Chol$ is the change in serum TC in mg/dl (1 mg/dl = 0,0259 mmol/l), Z_2 is the square root of the new cholesterol intake in mg/4 200 kJ (1 kJ = 0,238 kcal), and Z_1 is the square root of the previous cholesterol intake. The square root expression in the formula indicates that the effect of adding or removing cholesterol depends on the amount of other cholesterol-rich foods already present in the diet. When total cholesterol intake is high, the effect of a given change in intake will be smaller than at lower levels of cholesterol intake. Thus, according to the equation, the removal of 200 mg cholesterol (equivalent to the amount of cholesterol in one egg) should cause a fall in TC of 0,19 mmol/l in subjects whose diets initially provide 300 mg cholesterol per day, and only 0,08 mmol/l when diets provide 1 200 mg cholesterol per day.

Another issue that complicates the proposed relationship between dietary cholesterol and CHD somewhat is the between-subject variability in serum TC response to changes in cholesterol intake. This has led to the concept of hypo- and hyper-responders[10,11] to dietary cholesterol. The true width of the responsiveness distribution (i.e. after correction for intra-individual fluctuations of serum cholesterol) upon an increase in cholesterol intake (from 100 to 750 mg/day) may be rather small. Katan *et al.*[12] calculated that 16% of their population had a cholesterolaemic response either less than half of the mean response or more than 150% of the mean. Two per cent showed no increase at all. Thus, an 88-year-old man who had a normal serum TC level despite eating 25 eggs per day for at least 15 years[13] should possibly be considered a rare and exceptional hyporesponder.

Hyporesponsiveness to dietary cholesterol is probably caused by an individually determined low efficiency of cholesterol absorption.[14] In general, there are two compensatory mechanisms that protect against the development of hypercholesterolaemia after cholesterol ingestion. The major mechanism is inhibition of *de novo* cholesterol synthesis in the body. With a cholesterol intake of about 400 mg per day, whole-body cholesterol synthesis is about 600 mg per day but drops with larger cholesterol intakes. Another compensatory mechanism that is triggered after cholesterol feeding, is enhanced hepatic conversion of cholesterol into bile acids, leading to increased loss of cholesterol via the faeces in the form of bile acids. The two mechanisms maintain low hepatic cholesterol concentrations so that hepatic LDL-receptor activity does not become depressed and clearance of LDLC from the blood continues at a high rate.

Eggs, serum cholesterol and CHD

Although egg yolk is a convenient source of dietary cholesterol for experimental trials, eggs are more than just cholesterol. A 50 g egg contains about 6 g protein and 5 g fat.[7] Of the fat, 36% is in the form of saturated fatty acids, 48% are mono-unsaturated and 16% are polyunsaturated fatty acids.[7,15] Fats rich in saturated fatty acids raise serum cholesterol while oils rich in polyunsaturated fatty acids lower it. Keys *et al.*[16,17] integrated the results of various studies on human subjects, and derived an equation to predict the cholesterolaemic effect of dietary fats. Gram for gram, saturated fatty acids are twice as potent in elevating serum cholesterol as polyunsaturated fatty acids are in bringing about a decrease. Mono-unsaturated fatty acids were considered neutral, i.e. isocaloric substitution of carbohydrates for mono-unsaturated fatty acids had no effect on serum cholesterol.[17] Obviously, replacement of saturated with mono-unsaturated fatty acids will cause a decrease in serum cholesterol. The cholesterol-elevating effect of saturated fatty acids is limited to fatty acids containing 12 (lauric acid), 14 (myristic acid) or 16 (palmitic acid) carbon atoms.[9]

Upon the isocaloric replacement of dietary fats, the omission determines the resulting serum cholesterol change as much as does the addition. Keys' formula indicates that two-thirds of the lowering of serum cholesterol seen after consumption of polyunsaturated fatty acids at the expense of saturated fatty acids is attributable to the saturated fatty acids removed, and only one-third to the polyunsaturates added. When food substitution occurs, changes in cholesterol intake ($\Delta Z = Z_2 - Z_1$) also have to be accounted for. The following Keys' formula can be used: $\Delta Chol$ (mg/dl) $= 1,2\ (2\Delta S - \Delta P) + 1,5\Delta Z$,[9] where $\Delta Chol$ is the change in serum TC in mg/dl, and ΔS, ΔP and ΔZ are the changes in dietary saturated and polyunsaturated fatty acids and cholesterol, respectively. This formula does not take into account carbohydrates, proteins and mono-unsaturated fatty acids because these macronutrients are defined as neutral. However, when these nutrients are substituted for isocaloric amounts of saturated fatty acids, serum cholesterol will decrease. The Keys' formula indicates that the effect of fatty acids and cholesterol content in the diet are independent of one another. This may not be so and animal experiments suggest that the hypercholesterolaemic effect of dietary cholesterol is enhanced by a high saturated fat intake.[18]

Table II illustrates how the effect on serum cholesterol of changes in diet is the net effect of addition and omission of foodstuffs. The effect of cholesterol consumption in the form of eggs depends on the overall change in the diet. Keys' formula predicts that if 500 mg of cholesterol (an amount of cholesterol equivalent to that in two eggs) is added each day to a moderate-cholesterol diet (24 mg of cholesterol/MJ; 240 mg/day) without changes in protein or fat intake, the serum cholesterol level will rise by on average 0,29 mmol/l

Table II. The effect on serum cholesterol of egg consumption, when different foods are replaced while energy intake is kept constant*

	Composition of the resulting diet				
	Fatty acids (% of energy)			Cholesterol	Predicted serum
	Total	Saturated	Polyunsaturated	(mg/MJ)	cholesterol change (mmol/l)
None (basal diet)	38	15	6,5	24	
Two eggs, isocalorically replacing cheese and meat	37	13	6,7	71	+0,1
Two eggs, isocalorically replacing toast and jam	42	16	6,8	76	+0,4

* The predicted change in serum cholesterol was calculated from Keys' formula.[9] The basal diet provided 10 MJ (2 400 kcal) per day.

(11,3 mg/dl) or about 6%. The extent of the actual serum cholesterol change upon consumption of whole eggs, however, depends on what foods the eggs replace in the diet.

The fact that the effect of egg consumption on serum cholesterol is also determined by the omission of foodstuffs to maintain energy balance, explains that in various uncontrolled egg feeding trials and also in epidemiological studies egg intake was found not to affect serum cholesterol.[8] In the Framingham study[19] there was no association between egg consumption, serum cholesterol and CHD.

Table III. Nutritive contribution of one egg (50 g) to the daily nutrient allowances of a 70 kg, 25 - 50-year-old man

Nutrient	RDA[1]	50 g egg[7]	Contribution (%)
Energy (kJ)	9 - 10 000	319	3,2 - 3,55
Protein (g)	56	6,3	11,25
Total fat (g)[2,3]	71-79	5,2	6,5 - 7,25
SATFA (g)[2,3]	23,7 - 26,3	1,51	5,7 - 6,4
MUFA (g)[2,3]	23,7 - 26,3	2,01	7,6 - 8,5
PUFA (g)[2,3]	23,7 - 26,3	0,65	2,5 - 2,7
Cholesterol (mg)[2,3]	< 300	209,5	69,8
Calcium (mg)	800	19,5	2,4
Iron (mg)	10	0,9	9,0
Magnesium (mg)	350	4,5	1,3
Phosphorus (mg)	800	96	12
Zinc (mg)	15	0,58	3,8
Copper (mg)	1,5 - 3,0	0,06	2,0 - 4,0
Manganese (mg)	2,0 - 5,0	0,03	0,6 - 1,5
Vitamin A (RE)	1 000	33	3,3
Thiamin (mg)	1,5	0,07	4,3
Riboflavin (mg)	1,7	0,20	11,8
Niacin (mg)	19	0,05	0,26
Vitamin B_6 (mg)	2,0	0,02	1,05
Folate (µg)	200	23	11,5
Vitamin B_{12} (µg)	2,0	0,95	47,5
Pantothenic acid (mg)	4,0 - 7,0	0,87	12,4 - 21,8
Biotin (µg)	30 - 100	10,85	10,9 - 36,2
Vitamin D (µg)	5,0	3,97	79,4
Vitamin E (mg)	10,0	1,65	16,5

SATFA = saturated fatty acids; MUFA = mono-unsaturated fatty acids; PUFA = polyunsaturated fatty acids; RE = retinol equivalents.

The nutritive value of eggs

The contribution of one 50 g egg to the daily nutrient allowance[1] of a 70-kg adult man is given in Table III. In addition to protein of high biological quality (the ten essential amino acids form 53% of the protein[15]), eggs also contain significant amounts of iron, phosphorus, riboflavin, folate, vitamin E and especially vitamins D and B_{12}. In particular, vitamin D status may be compromised in elderly subjects who stay indoors most of the time[20] and vitamin E status in subjects on a low-fat diet.[21] Because of the high nutrient density (concentration of nutrients expressed on the basis of energy content), eggs could also form a valuable part of the diet of children, the ill, and individuals at risk of malnutrition.

The place of eggs in the prudent diet

An effective prudent diet should take all dietary factors known to influence serum cholesterol into account.[4] It should also aim at achieving and maintaining optimal body weight.[2,3] These prerequisites should be translated by nutritionists and dietitians into practical and palatable diets. In recommendations to the public, emphasis should be placed on the concept of moderation, realistic expected benefits, and the principle that all foods are allowed, but amounts may be restricted, especially of those foods containing large amounts of saturated fatty acids.[5] Recommendations should be based on overall nutritive content, and not on cholesterol content alone. The use of fats and oils in the cooking and dressing of food should be limited.

The following is an example of a daily menu of a high-fibre (4 - 6 g dietary fibre per 1 000 kJ),[22] low-fat diet (20% of total energy as fat, 80 - 120 mg cholesterol), providing adequate amounts of all essential nutrients: (*i*) one portion of a low-fat animal product (80 - 100 g cooked product) which may be meat, eggs, chicken or fish. One egg may replace 50 g of meat, chicken or fish; (*ii*) one portion (100 - 150 g cooked product) of a fibre-rich plant protein food which may be dried beans, dried peas or lentils; (*iii*) two portions (500 ml) of skimmed milk or equivalent amount of energy as yoghurt or cottage cheese; (*iv*) at least six portions of wholegrain cereal foods such a porridge, bread, pastas, breakfast cereals, samp, rice, etc. The number of portions and portion sizes will be determined by energy requirements. Only 15 g butter or margarine or oil should be used; (*v*) at least five portions (approximately 100 g per portion) of vegetables and fruit, including fresh dark green or yellow products; and (*vi*) moderate amounts of sugar and alcohol are allowed (except when serum triglyceride levels are high), but adjustments in energy intake from other sources would have to be made.

Not all individuals will comply with, or need, a diet in which fat contributes only 20% of total energy intake. Most guidelines[2,3] recommend that the present fat intake of Western populations, which represents 35 - 40% of energy, should be reduced to about 30% of energy. This could be achieved by adding extra animal protein food or spread on bread to the daily menu given above, or by using some fat and oil in the preparation of food. For example, an 8 000 kJ diet in which fat contributes 20%, 25% or 30% of total energy, will have to contain not more than 42 g, 53 g or 63 g fat respectively. Since fat is an integral part of many foods, low-fat products should be chosen and the minimum of added fats used.

Effective compliance with this prudent diet may be improved when more emphasis is placed on the total diet and not on specific foods. As stated recently,[23] it is obvious that a single food taken in normal portions cannot determine the quality of a complete diet. The message to the public should be that there is no such thing as a good or a bad food, and that only diets or eating patterns can be considered good or bad.

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