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# Exploring the Factors That Affect Blood Cholesterol and Heart Disease Risk: Is Dietary Cholesterol as Bad for You as History Leads Us to Believe?[1,2]

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**ABSTRACT**

This paper summarizes presentations given at the 2011 Experimental Biology meetings about the latest research and a paleoanthropological perspective pertaining to the relationship between dietary cholesterol intake and cardiovascular disease risk. For much of the past 50 years, a great deal of the scientific literature regarding dietary fat and cholesterol intake has indicated a strong positive correlation with heart disease. In recent years, however, there have been a number of epidemiological studies that did not support a relationship between cholesterol intake and cardiovascular disease. Further, a number of recent clinical trials that looked at the effects of long-term egg consumption (as a vehicle for dietary cholesterol) reported no negative impact on various indices of cardiovascular health and disease. Coupled with data indicating that the impact of lowering dietary cholesterol intake on serum LDL levels is small compared with other dietary and lifestyle factors, there is a need to consider how otherwise healthy foods can be incorporated in the diet to meet current dietary cholesterol recommendations. Because eggs are a healthful food, it is particularly important that sensible strategies be recommended for inclusions of eggs in a healthy diet. *Adv. Nutr. 3: 711–717, 2012.*

## Introduction

Current dietary cholesterol recommendations range from <200 mg/d for individuals at high risk of cardiovascular disease (CVD)[8] to <300 mg/day for healthy individuals. A recent Institute of Medicine report recommends that dietary cholesterol intake be as low as possible. However, a number of recent studies indicated that the relationship between dietary cholesterol intake, serum lipid levels, and coronary heart disease (CHD) risk is not nearly as strong as reported previously. These recent data have prompted some researchers and public health experts to question current cholesterol recommendations (1) or to at least acknowledge the potential of other dietary covariates (e.g., saturated fat, carbohydrates) that might have a greater impact on CHD risk than dietary cholesterol.

Because eggs are relatively high in cholesterol and low in saturated fat, they have served as a vehicle for delivering cholesterol in a number of clinical trials. The results of these trials indicated, for the most part, a lack of association between egg intake and CHD risk, as indicated by the formation of the less atherogenic, large LDL subfractions with egg ingestion, as well as an increase in plasma HDL cholesterol concentrations that results in the maintenance of the LDL cholesterol (LDL-C)/HDL cholesterol (HDL-C) ratio. Others have speculated that the lack of an effect of eggs on CHD risk may be related to specific antioxidants present in eggs that might reduce risk (2,3).

Many Western countries do not have dietary recommendations for cholesterol intake. Instead, these dietary guidelines focus on reducing saturated fat and *trans* fats because they have more potent effects on risk factors for CVD, primarily

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[8] Abbreviations used: CHD, coronary heart disease; CVD, cardiovascular disease; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol

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the lipid and lipoprotein profile. However, the issue remains controversial. Nevertheless, few, if any, governing bodies advocate for the complete removal of cholesterol from the diet, recognizing, as did the Institute of Medicine in a 2002 report, that "eliminating cholesterol in the diet would require significant changes in patterns of dietary intake, [which]… may introduce undesirable effects and unknown…health risks." Moreover, the Dietary Guidelines Advisory Committee 2010 concluded that the evidence shows that consumption of dietary cholesterol in the amount of 1 egg per day is not harmful and does not result in negative changes in serum lipoprotein cholesterol and triglyceride levels (4). Collectively, the epidemiologic and clinical trial evidence suggests that consumption of 1 egg per day is not associated with risk of CHD or stroke in healthy adults, although consumption of >7 eggs per week has been associated with increased risk in some studies. An important distinction is that among individuals with type 2 diabetes, increased dietary cholesterol intake is associated with CVD risk (4). Future research, including ongoing studies involving cholesterol ingestion by high-risk CVD patients, should shed light on the impact of dietary cholesterol on disease risk and the implications of removing nutritious cholesterol-containing foods from the diet.

## Current status of knowledge

### Current recommendations

Current dietary recommendations for cholesterol are in the range of 200–300 mg/d. For healthy individuals without diabetes, CVD or hypercholesterolemia, the recommendation typically is <300 mg/d (4,5). For high-risk individuals, the recommendation is <200 mg/d (4,5). The recommendation for dietary cholesterol issued by the Institute of Medicine in the Dietary Reference Intakes for Energy and Macronutrients Report is that dietary cholesterol be "as low as possible" (6). The U.S. dietary recommendations for dietary cholesterol are summarized in **Table 1**.

**Table 1.** U.S. dietary recommendations for cholesterol[1]

| Institute of Medicine, Dietary Reference Intake for Cholesterol, 2002/2005 | Minimize Intake |
|---|---|
| Dietary Guidelines for Americans, 2005 | <300 mg/d |
| Dietary Guidelines for Americans, 2010 | <300 mg/d (<200 mg/d can further help individuals at high risk of CVD) |
| Dietary Guidelines Advisory Committee, 2010 | <300 mg/d. Further reductions to <200 mg/d in persons with or at high risk of CVD or type 2 diabetes |
| National Cholesterol Education Program | |
| ATP I, 1988 | Step 1: <300 mg/d; step 2: <200 mg/d |
| ATP II, 1994 | Step 1: <300 mg/d; step 2: <200 mg/d |
| ATP III, 2002 | <200 mg/d |
| American Heart Association | |
| Dietary Guidelines 2000 | <300 mg/d; <200 mg/d for high-risk patients |
| Dietary Guidelines Revision 2006 | <300 mg/d |
| Guidelines for Women, 2011 | <150 mg/d |
| American Diabetes Association | |
| Nutrition Recommendations, 2006 | Individuals with diabetes: <200 mg/d |
| Nutrition Recommendations, 2008 | Individuals with diabetes: <200 mg/d |

[1] ATP, Adult Treatment Panel; CVD, cardiovascular disease.

These recommendations are based on controlled clinical studies that have shown that dietary cholesterol increases serum total and LDL-C in a dose-response manner (6) and, based on an extensive literature, increasing serum total and LDL-C increases the risk of CVD. Accordingly, for high-risk patients and for all patients with CHD, a serum LDL level of <70 mg/dL is considered a desirable goal.

Although the correlation between serum LDL-C and CVD risk is well accepted, the interindividual response to dietary cholesterol is quite variable. It also is important to note that increases in HDL-C always occur with cholesterol intake even when LDL-C is not increased, as is the case of weight loss or intake of 1 egg per day.

### A theoretical challenge

A theoretical challenge to concerns about adverse health effects of dietary cholesterol derives from the field of paleoanthropology. Human dietary needs did not arise in vacuo; they were adaptations in response to the available food supply. Paleoanthropologists suggest that dietary cholesterol has been in the human diet for millions of years (7–10). Sources included eggs, bone marrow, and organ meats. Stone Age intake of cholesterol is uncertain, but it may well have exceeded current dietary recommendations.

There are many important biological roles for cholesterol that span the spectrum from cell membrane structure to steroid hormone synthesis, bile acid synthesis, and others. The vital role of cholesterol in human metabolism and the well-established place of dietary cholesterol in the native human diet provide a robust theoretical challenge to the view that dietary cholesterol poses a threat to human health. It stands to reason that we are well adapted to the constituents of our native diet in exactly the same way that carnivores are adapted to survive on meat and herbivores on forages.

An exoneration of dietary cholesterol raises the question: what was the basis for implicating dietary cholesterol as a CVD risk factor in the first place? The answer highlights important distinctions between the modern and Stone Age food supplies. In a modern context, cholesterol and other dietary factors likely to influence both serum lipids and the risk of coronary disease, salient among them saturated fat, tend to covary. Both cholesterol and saturated fat are found in dairy products and processed foods containing them and in meat. Eggs and seafood, sources of cholesterol but not of considerable amounts of saturated fat, are smaller contributors to the typical modern diet. The linkage of specific foods with CVD risk in the absence of the total diet context is problematic. For example, the impact of dietary

**Table 2.** Changes in LDL-C, HDL-C, and LDL-C/HDL-C ratio after a cholesterol challenge[1]

| Population (*n*) | Duration, *wk* | Cholesterol, *mg/d* | LDL-C | HDL-C | LDL/HDL | Ref. |
|---|---|---|---|---|---|---|
| Children (54) | 4 | 518 | ↑ | ↑ | ↔ | 14 |
| Women (51) | 4 | 640 | ↑ | ↑ | ↔ | 15 |
| Men (28) | 12 | 640 | ↔ | ↑ | ↔ | 17 |
| Men/women (42) | 12 | 215 | ↑ | ↑ | ↓ | 3 |
| Men/women (34) | 4 | 640 | ↔ | ↑ | ↔ | 16 |
| Men/women (56) | 12 | 250 | ↔ | ↑ | ↓ | 18 |
| Men/women (45) | 12 | 400 | ↔ | ↔ | ↔ | 19 |

[1] HDL-C, HDL cholesterol; LDL-C, LDL cholesterol.

cholesterol on serum lipid levels is reduced when saturated fatty acid intake is low (11).

In the Stone Age context, dietary cholesterol was more reliably distinct from saturated fat. Dairy was not consumed until the advent of agriculture, ~12,000 y ago, and was not consumed by many cultures for millennia thereafter. Humans likely adapted to an intake of dietary cholesterol unencumbered by adverse associations and then encountered such associations only in a modern context. Dietary cholesterol was then likely indicted by association.

## A growing body of literature

A growing body of epidemiological research questions the association between dietary cholesterol and serum lipids, when account is adequately taken of other dietary variables (11,12). More important still are prospective, population-based studies that, when similarly scrupulous about variation in other dietary components, find no association between cholesterol intake in general, or egg intake in particular, and the risk of CVD (13).

Recent reports derived from clinical interventions established that increases in cholesterol intake result in increases in both LDL-C and HDL-C in those subjects who respond to dietary cholesterol challenges (~25% of the population), whether they are children (14), young adults (15), or elderly individuals (16). Further, there are specific circumstances in which dietary cholesterol results in increases in only HDL-C, whereas no increases in LDL-C are observed, as is the case with weight loss interventions (11,17), intake of only 1 egg per day (3), or other factors (18). Recent findings on lipoprotein responses to dietary cholesterol challenges in a variety of populations are summarized in **Table 2**.

Egg intake also has resulted in the formation of fewer atherogenic lipoproteins including increases in large LDL (15,19) and large HDL particles (20). It is well-known that small LDL particles become more readily oxidized and can more easily penetrate the arterial wall where they are taken up by macrophages, leading to the formation of foam cells and the initiation of the atherosclerotic process (21). Large HDL particles are associated with increased reverse cholesterol transport (22). Thus, the generation of these lipoprotein particles (large LDL and large HDL particles) by egg intake suggests increased protection against atherosclerosis.

Further, the Lipid Research Clinics Prevalence Follow-up Study (23), which examined both men and women ($N$ = 4546) reported no important relationships between CHD deaths and dietary cholesterol intake. Several other studies (24–26) also failed to find an association between CHD incidence and egg intake, and recent reports indicated a lack of correlation between egg intake and risk of CHD or stroke (27,28).

## Growing knowledge of the benefits of HDL-C

With respect to the implications of altered lipoprotein levels on CVD risk, recent research in the area of HDL biology reveals the breadth and benefit of HDL's contribution to wellness, with some surprises. Since the mid-1970s, circulating HDL-C has been widely accepted as a negative risk factor for CVD. The traditional function attributed to HDL has been its ability to remove excess cholesterol from peripheral tissues, mainly cholesterol-laden macrophages in atheromatous plaques, and to deliver cholesterol in the form of cholesteryl esters back to the liver for catabolism or excretion into bile by a process called the reverse cholesterol transport pathway. A simple consequence of this pathway is that the more circulating HDL one has, the greater the potential for removal of excess cholesterol and thus a lower risk of the development of atherosclerosis and cardiovascular events. There are strong experimental and recent clinical data in support of the importance of the reverse cholesterol transport pathway (29); however, there is also substantial interest in alternative HDL functions. Recent studies of alternative HDL functions provided possible mechanistic links for HDL in a diversity of pathways, including roles for

**TABLE 3.** Outcome variables after 6 wk of treatment: eggs vs. oatmeal[1]

| | Mean ± SD | | |
|---|---|---|---|
| Variable | Baseline | Egg | Oatmeal |
| BMI, kg/$m^2$ | 28.7 ± 7.2 | 28.1 ± 5.8 | 28.5 ± 5.7 |
| Total cholesterol, *mg/dL* | 203.8 ± 31.5 | 205.3 ± 35.6 | 194.0 ± 30.5[1] |
| HDL, *mg/dL* | 52.6 ± 14.6 | 51.2 ± 15.1 | 53.3 ± 16.5 |
| LDL, *mg/dL* | 124.8 ± 25.0 | 129.1 ± 32.2 | 116.6 ± 30.8 |
| Triglycerides, *mg/dL* | 135.6 ± 77.3 | 126.6 ± 72.8 | 122.5 ± 75.7 |
| Reference diameter, cm | 0.31 ± 0.07 | 0.44 ± 0.07 | 0.43 ± 0.07 |
| % Diameter change 60 s to baseline | 11.0 ± 9.5 | 8.3 ± 6.3 | 6.6 ± 8.0 |
| systolic blood pressure, *mm Hg* | 129.8 ± 11.7 | 124.9 ± 10.9 | 124.1 ± 12.5 |

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[1] $P < 0.05$ compared with baseline value.

**Table 4.** Endothelial function after 6 wk of treatment: eggs vs. oatmeal[1]

| Treatment | Preprandial | Postprandial | % Change |
|---|---|---|---|
| Egg | | | |
| Reference diameter, *cm* | 0.43 ± 0.08 | 0.43 ± 0.08 | |
| Hyperemic diameter at 60 s, *cm* | 0.47 ± 0.08 | 0.44 ± 0.07 | |
| Flow-mediated vasodilation | 8.66 ± 9.69 | 8.32 ± 6.33 | −0.96[2] |
| Oatmeal | | | |
| Reference diameter, *cm* | 0.43 ± 0.07 | 0.43 ± 0.07 | |
| Hyperemic diameter at 60 s, *cm* | 0.46 ± 0.08 | 0.47 ± 0.07 | |
| Flow-mediated vasodilation | 6.98 ± 8.45 | 6.56 ± 7.99 | −0.79[3] |

[1] Values are mean ± SD.
[2] $P > 0.05$ adjusting with preprandial (paired *t* test).
[3] $P > 0.05$ compared with different treatments (ANOVA).
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HDL in improving diabetes and glucose metabolism (30,31), reducing inflammation (32,33), attenuating oxidative stress (34,35), reducing platelet aggregation (36), protecting against microbial infection (37), enhancing cardiac differentiation (38), and promoting ischemia-induced angiogenesis (39). Many of these described beneficial effects are due to HDL-induced gene expression changes in cells and likely represent a tonic effect of HDL on cell function. In addition to the transcriptional response to HDL, multiple groups have demonstrated that HDL stimulates the release of nitric oxide from endothelial cells through the activation of endothelial nitric oxide synthase (34,40).

Although the classically described inverse association between CVD and HDL is attributed to circulating HDL levels, a recent study suggests that HDL efflux capacity also presents a strong inverse correlation with CVD, independent of circulating HDL levels (41). There have been numerous clinical approaches and drug trials aimed at increasing HDL-C levels and very few, if any, aimed at increasing HDL efflux capacity. Nevertheless, simply increasing HDL levels may not provide cardiovascular benefits if other parts of the pathway, such as cholesterol efflux, lipoprotein transfer, lipolysis, and hepatic delivery, are compromised (42,43). It is also important to point out that increased cardiovascular risk can occur in the context of increased HDL-C. Mutations to hepatic lipase and cholesteryl ester transport protein, for example, both result in elevated HDL-C levels and an increased risk of CVD (42,43)

In summary, the field of lipoprotein biology is rapidly racing forward to identify novel and alternative functions of HDL. Future advances in this area will likely translate not only into a better understanding of HDL biology but the true implications of dietary cholesterol on health and disease risk as well.

**Table 5.** Lipids and biometrics after 6 wk of treatment: eggs vs. egg substitute in hyperlipidemic adults[1]

| Lipid panel | Eggs | Egg Substitute | *P* Value[2] |
|---|---|---|---|
| Total cholesterol, mg/dL | | | |
| Baseline | 244 ± 24 | 244 ± 24 | 1.00 |
| 6 wk | 239 ± 27 | 227 ± 27 | |
| Change | −5 ± 21 ($P = 0.10$) | −18 ± 18 ($P < 0.01$) | <0.01 |
| LDL *mg/dL* | | | |
| Baseline | 168 ± 17 | 168 ± 17 | |
| 6 wk | 165 ± 24 | 154 ± 24 | |
| Change | −2 ± 19 ($P = 0.30$) | −14 ± 20 ($P < 0.01$) | 0.01 |
| HDL, *mg/dL* | | | |
| Baseline | 52 ± 15 | 52 ± 15 | 1.00 |
| 6 wk | 51 ± 14 | 50 ± 13 | |
| Change | −1 ± 11 ($P = 0.53$) | −2 ± 10 ($P = 0.03$) | 0.63 |
| Triglycerides, *mg/dL* | | | |
| Baseline | 132 ± 52 | 132 ± 52 | |
| 6 wk | 118 ± 47 | 116 ± 50 | 1.00 |
| Change | −14 ± 37 ($P = 0.54$) | −18 ± 43 ($P = 0.03$) | 0.83 |
| Total cholesterol to HDL cholesterol ratio | | | |
| Baseline | 5.0 ± 1.3 | 5.0 ± 1.3 | 1.00 |
| 6 wk | 5.0 ± 1.2 | 4.8 ± 1.3 | |
| Change | −0.06 ± 0.66 ($P = 0.54$) | −0.21 ± 0.82 ($P = 0.11$) | 0.38 |
| Body composition | | | |
| Weight, *kg* | | | |
| Baseline | 81 ± 19 | 81 ± 19 | 1.00 |
| 6 wk | 82. ± 18 | 82 ± 18 | |
| Change | 0.4 ± 2.3 ($P = 0.33$) | 0.7 ± 2.4 ($P = 0.08$) | 0.52 |
| BMI, $kg/m^2$ | | | |
| Baseline | 29.2 ± 4.5 | 29.2 ± 4.5 | 1.00 |
| 6 wk | 29.3 ± 4.3 | 29.5 ± 4.5 | |
| Change | 0.2 ± 0.8 ($P = 0.18$) | 0.4 ± 0.9 ($P = 0.04$) | 0.56 |

[1] Values are mean ± SD. *P* values obtained by repeated-measures ANOVA except as otherwise stated. *P* values in parentheses indicate within-group *P* values.
[2] *P* value obtained by Student's *t* test. Change = 6 wk − baseline.
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**TABLE 6.** Endothelial function after 6 wk of treatment: hyperlipidemic adults[1]

| Variable | Egg ($n$ = 36) | Egg Substitute ($n$ = 36) | $P$ Value[2] |
|---|---|---|---|
| Endothelial function | | | |
| Flow-mediated dilation, % | | | |
| Baseline | 5.6 ± 3.9 | 5.8 ± 3.9 | 0.78 |
| 6 wk | 5.3 ± 4.1 | 6.9 ± 4.0 | |
| Change | −0.1 ± 1.5 ($P$ = 0.80) | 1.0 ± 1.2 ($P$ < 0.01) | <0.01 |
| Adjusted change[3] | −0.2 ± 1.3 ($P$ = 0.35) | 0.9 ± 1.4 ($P$ < 0.01) | <0.01 |
| Stimulus-adjusted response measure | | | |
| Baseline | 0.08 ± 0.10 | 0.06 ± 0.06 | 0.39 |
| 6 wk | 0.08 ± 0.11 | 0.09 ± 0.09 | |
| Change | 0.01 ± 0.05 ($P$ = 0.54) | 0.03 ± 0.06 ($P$ < 0.01) | 0.07 |

[1] Values are mean ± SD; $P$ value obtained from repeated-measures ANOVA except as otherwise stated; $P$ values in parentheses indicate within-group $P$ values.
[2] $P$ value obtained by Student's $t$ test; change = 6 wk − baseline.
[3] Obtained from generalized linear models, controlling for age, blood pressure, LDL, and BMI.
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## Endothelial function and cholesterol intake

An important tool for assessing cardiovascular risk is endothelial function testing (44). A dynamic measure of vascular responses, ultrasound-based methods of assessing endothelial function provide a means of gauging the cumulative influence of multiple moieties in circulation on the behavior and health of the cells lining arteries and arterioles. There is thus a case to be made that endothelial function is potentially a summative measure of overall cardiac risk status and at least a valuable addition to standard risk measures (45). The ever-expanding footprint of research in this area in the cardiology literature attests to its importance.

Katz et al. recently reported 2 endothelial function studies examining the influence of egg ingestion, and thus dietary cholesterol intake, on cardiac risk status. The first study (46) assessed the effects of daily ingestion of 2 eggs for 6 wk in 50 healthy adult men and women. A randomized, single-blind, crossover design was used, with oatmeal as a positive control. No adverse effects of egg ingestion were seen on serum lipids (Table 3) or endothelial function (Table 4).

A second study (47) extended similar methods to 40 adults with hyperlipidemia, comparing eggs with cholesterol-free egg substitute. In this at-risk population for which restriction of dietary cholesterol (i.e., <200 mg/d is recommended), no harmful effects of daily intake of 2 eggs were seen on either lipids (Table 5) or endothelial function (Table 6).

## Magnitude of dietary effects and global response

Table 7 (48) shows that the approximate LDL-C reduction when dietary cholesterol is decreased to <200 mg/d is 3–5%. As is apparent, other dietary interventions elicit a greater cholesterol-lowering response, i.e., decreasing saturated fat to <7% of total energy intake, losing 10 lb of body weight, and incorporating plant sterols/stanols into the diet.

Table 8 (49) presents recommendations made by global heart organizations for dietary cholesterol. It is evident that the recommendations are less stringent than those made in the United States (50). In fact, as noted, some countries have no recommendations for dietary cholesterol. The European Union, Korea, India, Canada, and New Zealand, among other countries, do not have a recommendation for cholesterol intake in their dietary guidelines, based on several lines of evidence including 1) the lack of effect of egg intake and CHD risk; 2) the consistent increases observed in HDL-C under numerous dietary interventions that include dietary cholesterol; 3) the formation of less atherogenic or protective lipoproteins with egg consumption; and 4) the additional benefits of eggs for memory and protection against macular degeneration.

## Potential implications of removing cholesterol from the diet

There is, finally, a larger public health context in which the issue of cholesterol restriction looms large. Dietary recommendations, when adopted, have implications for both what is taken out of the diet and what is put in. Advice to restrict cholesterol may contribute to increased consumption of questionable alternatives, such as simple sugars, other highly refined carbohydrates, and saturated and *trans* fats. The net effect of advice to limit cholesterol intake on overall diet quality is not well established in the current food environment and could well be adverse.

The aforementioned Institute of Medicine report on Dietary Reference Intakes for Energy and Macronutrients (6) states that "Because cholesterol is unavoidable in ordinary diets, eliminating cholesterol in the diet would require significant changes in patterns of dietary intake. Such adjustments

**Table 7.** Approximate and cumulative LDL-C reduction achievable by dietary modification[1]

| Dietary Component | Dietary Change | Approximate LDL-C Reduction, % |
|---|---|---|
| Major interventions | | |
| Saturated fat | <7% of calories | 8–10 |
| Dietary cholesterol | <200 mg/d | 3–5 |
| Weight reduction | Lose 10 lb | 5–8 |
| Other LDL-C lowering options | | |
| Viscous fiber | 5–10 g/d | 3–5 |
| Plant sterol/stanol esters | 2 g/d | 6–15 |
| Cumulative estimate | | 20–30 |

[1] LDL-C, LDL cholesterol.
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**Table 8.** Recommendations by "heart organizations" for dietary cholesterol

| Australia | National Heart Foundation (2001) | Individuals at Risk or With Plasma Cholesterol >5.0 mmol/L (193 mg/dL) | Limit Cholesterol-Rich Foods Such as Egg Yolks and Offal |
|---|---|---|---|
| Canada | Canadian Consensus Conference on Cholesterol (1988) | Individuals known to have a high incidence of hyperlipidemia, such as CHD, a family history of CHD, and early CHD or who had known risk factors | Restrict intake of foods high in cholesterol such as organ meats and egg yolks; <300 mg/d |
| Canada | Heart and Stroke Foundation (2011) | | No specific recommendation |
| United Kingdom and Europe | European Heart Network and Eurodiet Working Party | Individuals who need lowering of blood cholesterol | No recommendation; it was concluded that cholesterol intakes in Europe are on average well within the limit (<300 mg/d) |
| WHO | Study group on diet, nutrition and prevention of noncommunicable diseases (2003) | Population approach to reducing cardiovascular disease risk | <300 mg/d |

may introduce undesirable effects (e.g., inadequate intake of protein and certain micronutrients) and unknown and unquantifiable health risk."

The current intake of dietary cholesterol in the United States of ~278 mg/d has been relatively constant since 1989–1991 (51). A major source of cholesterol in the U.S. diet is eggs and egg-mixed dishes, contributing ~24.6% to total daily intake. Therefore, it is instructive to assess the potential impact of removing eggs from the diet as a part of an overall low-cholesterol diet. Eggs are a very good source of important nutrients, providing high-quality protein, carotenoids, essential fatty acids, and many vitamins and minerals (e.g., vitamins A, E, D, and K, calcium, iron, phosphorus, zinc, thiamin, vitamins B-6 and B-12, folate, pantothenic acid, niacin, riboflavin, magnesium, copper, manganese, selenium, and potassium). Eggs also are one of the few sources of choline in the diet. According to Fernandez (52), the lack of association of egg intake and CHD appears to be related to specific nutrients and antioxidants present in eggs including the carotenoids lutein and zeaxanthin as well as vitamin E. Further, the large lipoprotein particles derived from egg intake have been closely associated with the increased transport of plasma carotenoids (53).

Obviously, eggs are a good source of many nutrients and inclusion in the diet facilitates achieving nutrient adequacy, a key recommendation of contemporary dietary guidance. Moreover, eggs are an inexpensive food and provide affordable nutrition for all population groups, especially for those of low-income status.

## Conclusions

The current epidemiological evidence indicates that dietary cholesterol (at current intakes) does not increase the risk of heart disease in healthy individuals. Clinical studies have shown that two thirds or more of the population do not have a considerable increase in plasma cholesterol after a dietary cholesterol challenge for extended periods of time, whereas in those who do respond, both LDL-C and HDL-C increase, and therefore they maintain their LDL-C/HDL-C ratio. Many countries have issued dietary guidelines that do not have recommendations for dietary cholesterol. Given that eggs are a good source of many nutrients, there is a growing awareness about how consumers can incorporate eggs into a healthy diet that meets current food-based dietary recommendations. Thus, there may be a need to reconsider the recommendations for dietary cholesterol for healthy populations.


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