The Impact of Egg Limitations on Coronary Heart Disease Risk: Do the Numbers Add Up?

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For over 25 years eggs have been the icon for the fat, cholesterol and caloric excesses in the American diet, and the message to limit eggs to lower heart disease risk has been widely circulated. The "dietary cholesterol equals blood cholesterol" view is a standard of dietary recommendations, yet few consider whether the evidence justifies such restrictions. Over 50 years of cholesterol-feeding studies show that dietary cholesterol does have a small effect on plasma cholesterol concentrations. The 167 cholesterol feeding studies in over 3,500 subjects in the literature indicate that a 100 mg change in dietary cholesterol changes plasma total cholesterol by 2.2 mg/dL.

Today we recognize that dietary effects on plasma cholesterol must be viewed from effects on the atherogenic LDL cholesterol as well as anti-atherogenic HDL cholesterol since the ratio of LDL:HDL cholesterol is a major determinant of heart disease risk. Cholesterol feeding studies demonstrate that dietary cholesterol increases both LDL and HDL cholesterol with little change in the LDL:HDL ratio. Addition of 100 mg cholesterol per day to the diet increases total cholesterol with a 1.9 mg/dL increase in LDL cholesterol and a 0.4 mg/dL increase in HDL cholesterol. On average, the LDL:HDL ratio change per 100 mg/day change in dietary cholesterol is from 2.60 to 2.61, which would be predicted to have little effect on heart disease risk. These data help explain the epidemiological studies showing that dietary cholesterol is not related to coronary heart disease incidence or mortality across or within populations.

Key teaching points:

- Based on clinical feeding studies, the average change in plasma total cholesterol is 2.2 mg/dL per 100 mg/day change in dietary cholesterol.
- The plasma cholesterol response to a change in dietary cholesterol is independent of dietary fat type and amount and the baseline plasma cholesterol.
- The plasma lipoprotein cholesterol responses to a 100 mg/day change in dietary cholesterol average 1.9 mg/dL for LDL and 0.4 mg/dL for HDL cholesterol.
- Dietary cholesterol has little effect on the LDL:HDL cholesterol ratio.

INTRODUCTION

In public health the *Precautionary Principle* asserts that "when information about risk is uncertain, it is prudent to assume the worst." Based on this principle, recommendations to limit cholesterol in the diet were initiated in the early 70s in an attempt to reduce coronary heart disease (CHD) risk associated with elevated plasma cholesterol levels. The message was simple, straightforward and easily interpreted: cholesterol in food increases cholesterol in blood which in turn increases heart disease risk. As part of the dietary cholesterol restriction, the recommendation included specific limits on weekly egg consumption, since eggs are a concentrated source of cholesterol in the diet. During the last thirty years restriction of weekly egg intake has become one of the most widely recognized and accepted dietary recommendation for the general public. In fact, the negative view of dietary cholesterol became so well known that in the 1980's it was a major marketing tool for everything from peanut butter to beer to proclaim themselves "cholesterol-free." And eggs became a popular media

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icon for many of the dietary excesses of the population and the image for cholesterol, both dietary and plasma. While the recommendation to reduce dietary cholesterol was based on the best, albeit weak, evidence available at the time, the assumption that dietary cholesterol leads to high blood cholesterol which increases heart disease risk has became a dogma of nutrition and public health policy. Today, few even question the evidence for this relationship, or whether dietary cholesterol restrictions have beneficial effects on CHD risk.

Dietary recommendations which state "no more than" are viewed by many consumers as recommending "as few as possible." Between 1970, when egg restrictions were first proposed, to 1995, per capita egg consumption in the United States decreased from 310 eggs per person per year to 235 [1]. Clearly this 24% reduction in egg consumption occurred not only as a result of public health recommendations to restrict egg consumption, but also from the changing life-style patterns of the population. But as the public became more cholesterol-aware with the efforts of the National Cholesterol Education Program (NCEP), combined with media attention to reports on diet and health from government [2, 3] and health promotion groups [4, 5], and the introduction of the Nutrition Facts Label, consumers became more and more aware of dietary cholesterol restrictions and, in turn, more cholesterol-phobic. For the average consumer, the associations were both straightforward and logical: dietary cholesterol was bad, eggs have high cholesterol, eggs are bad and should be restricted, if not eliminated, from the diet.

Now that we have three decades of research on the dietary cholesterol - blood cholesterol question, it is appropriate to re-evaluate the basic assumptions involved in dietary cholesterol and egg restrictions. What is the evidence that cholesterol and egg restrictions significantly lower plasma cholesterol levels and CHD risk?

EGGS, DIETARY CHOLESTEROL AND PLASMA CHOLESTEROL

Dietary Cholesterol Restrictions

The scientific basis for dietary cholesterol restrictions are based on three lines of evidence: animal feeding studies, epidemiological surveys and clinical trials [4]. Both animal feeding studies and epidemiological survey data have confounding factors which make extrapolation to human health and disease prevention extremely complicated. Animal feeding studies are problematic due to the heterogeneity of plasma cholesterol responses to dietary cholesterol, with some animals being extremely sensitive while others are almost completely resistant to any effects of dietary cholesterol on plasma levels. Another complicating factor in animal studies is that in many studies it was necessary to feed pharmacological doses of dietary cholesterol to induce hypercholesterolemia and an atherogenic plasma profile in many species. Primate studies, for example, can require cholesterol intakes equal to a human intake of 2 to 3 grams per day to induce hypercholesterolemia and atherosclerosis [6]. In addition, most animal species have HDL particles as the major plasma lipoprotein carrier of cholesterol in sharp contrast to humans with LDL cholesterol as the major fraction. The combination of species variability in response to cholesterol, the use of non-physiological amounts of dietary cholesterol to induce hypercholesterolemia, and the distinctly different plasma lipoprotein profile make extrapolation of the findings from animal feeding studies to determine disease risk prevention in humans very complicated.

Data from epidemiological surveys have often found a positive relationship between dietary cholesterol and CHD mortality rates across populations [7] but not within populations [8–13]. The observation of a relationship between dietary cholesterol and CHD mortality in cross-cultural studies is almost always based on a simple regression analysis. The interpretation of this relationship is complicated by the co-linearity of cholesterol and saturated fat calories in the diet, and dietary saturated fat is consistently found to be positively correlated with CHD incidence. In contrast, a number of recently reported epidemiological surveys used multivariate analysis to determine relationships and have reported that when saturated fat and dietary fiber are included in the analysis, dietary cholesterol is no longer significantly correlated with CHD mortality [9, 10, 14–17]. Prospective studies in large numbers of participants have consistently reported a null relationship between dietary cholesterol and CHD morbidity and mortality when using multivariate analysis [9, 10, 15, 16]. In a study of 80,082 women followed for 14 years and 37,851 men followed for eight years, Hu *et al.* [18], reported that the relative risk of CHD was the same whether the participants consumed fewer than one egg a week or more than one egg a day. [See review by Kritchevsky and Kritchevsky in this issue.]

There has never been a clinical trial of the effects of dietary cholesterol on atherosclerosis. The primary evidence that dietary cholesterol contributes to CHD risk is based on the effects of dietary cholesterol on plasma total cholesterol levels, which have served as a surrogate marker for heart disease risk. This review reports an analysis of the data from cholesterol feeding trials in humans to evaluate the effects of dietary cholesterol on plasma total cholesterol levels as well as plasma lipoprotein cholesterol levels and the factors which could affect dietary cholesterol-induced changes in plasma cholesterol levels and heart disease risk. The evidence for heterogeneity in the plasma cholesterol response to dietary cholesterol is also evaluated, including estimates of the relative plasma cholesterol changes in hyper-responders and hypo-responders. Finally, the effects of dietary cholesterol, and egg, restrictions on plasma cholesterol levels and CHD risk in the population are evaluated.

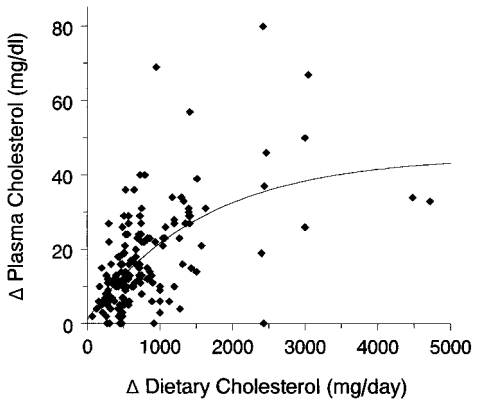

Fig. 1. Relationship between mean change in dietary cholesterol (mg/day) and mean change in plasma cholesterol levels (mg/dL) for 167 cholesterol feeding studies published between 1960 and 1999.

Cholesterol Feeding Studies: Effects on Plasma Cholesterol Levels

There are 167 published cholesterol feeding studies in 3,519 subjects dating back to 1960. The studies selected for this analysis were limited to those which used a cross-over design with cholesterol intake being the sole experimental variable. These studies were carried out in various patient types (normocholesterolemic, hypercholesterolemic, young to elderly, men and women) under differing experimental conditions (metabolic ward conditions, controlled feeding design, and free-living outpatients) and incorporated a wide range of background diets (low to high total fat, low to high P:S ratio, etc.). Most notable, these studies used dietary cholesterol challenges ranging from physiological, with addition of a hundred to three hundred milligrams of dietary cholesterol, to the pharmacological, with additions of 3–5 grams of cholesterol to the test diets. Fig. 1 shows the relationship between changes in cholesterol intake and changes in plasma cholesterol level for the 167 published studies. The figure shows clearly that plasma cholesterol levels do increase as dietary cholesterol increases. The figure also shows that the plasma cholesterol response to dietary cholesterol becomes progressively attenuated at very high cholesterol intakes presumably due to the decreased fractional absorption of cholesterol with higher intakes [19].

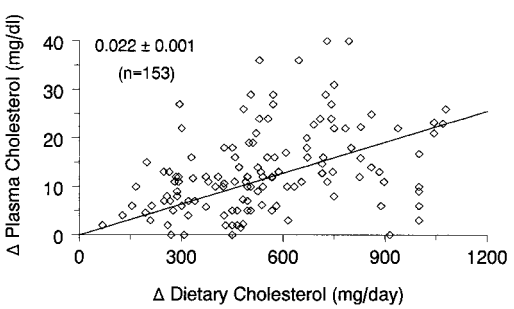

Fig. 2. Relationship between mean change in dietary cholesterol (mg/day) and mean change in plasma cholesterol levels (mg/dL) for 153 cholesterol feeding studies using a dietary cholesterol challenge of less than 1200 mg/day published between 1960 and 1999.

Table 1. Predicted plasma cholesterol response to a 100 mg/day change in dietary cholesterol

Reference:	△Plasma Cholesterol (mg/dL)
Hegsted *et al.* 1965 [23]	4.5
Keys *et al.* 1965 [24]	2.5
Keys 1984 [25]	2.5
Hegsted 1986 [26]	4.0
NIH 1988 [27]	4.0
McNamara 1990 [20]	2.2
Hegsted *et al.* 1993 [28]	2.7
Hopkins 1992 [29]	2.5
McNamara 1995 [30]	2.5
Clarke *et al.* 1997 [21]	2.5
Howell *et al.* 1997 [22]	2.2
McNamara 2000	2.2

Any evaluation of the effects of dietary cholesterol on plasma total and lipoprotein cholesterol levels must take into account the wide range of dietary cholesterol challenges employed in the various studies and the fact that with the feeding of massive doses of any nutrient there are potential risks of disturbances in multiple physiological regulatory mechanisms, as well as the potential to overwhelm those mechanisms. For the purpose of this analysis, studies using dietary cholesterol challenges greater than 1200 mg/day have been excluded from the analysis since at levels above this intake cholesterol fractional absorption decreases. In order to normalize all studies to a comparable intake, the intakes were adjusted for body weight to 70 kg [20] and the plasma cholesterol response normalized for a 100 mg/day change in dietary cholesterol. The 153 reported studies which fit these criteria exhibit a linear relationship between △ cholesterol intake and △ plasma cholesterol with a slope of 0.022 mg/dL per mg/day cholesterol (Fig. 2). The dose adjusted plasma cholesterol response is △ 2.2 mg/dL per △100 mg/day dietary cholesterol (±0.13, 95% CI 1.9–2.5). These data are consistent with two recently reported meta-analyses of the effects of dietary lipids on plasma cholesterol levels by Clarke *et al.* [21] and Howell *et al.* [22], which reported plasma cholesterol changes of △ 2.5 mg/dL and △ 2.2 mg/dL per △ 100 mg/day dietary cholesterol, respectively. The results from these recent analyses are consistent with other predictive equations dating back to 1965 (Table 1).

Table 2. Whole body cholesterol metabolism in a 70 kg adult consuming 400 *versus* 300 mg of cholesterol per day

Total body cholesterol		145g	
Plasma cholesterol		8g	
Dietary cholesterol	0.40g		0.30g
Absorption (60%)	0.24g		0.18g
Cholesterol synthesis	0.92g		0.95g
Total cholesterol input	1.16g		1.13g
Metabolic requirements	0.25g		0.25g
Excess	0.91g		0.88g

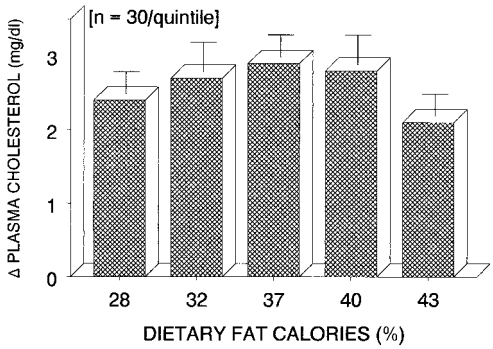

Fig. 3. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol for each quartile of dietary fat calories (%). Data presented as mean±SEM for n=30 per quartile.

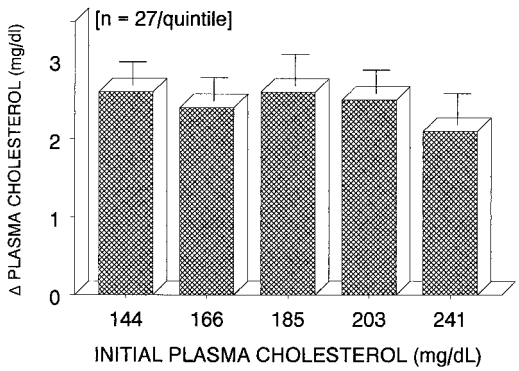

Fig. 5. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol for each quartile of initial plasma cholesterol (mg/dL). Data presented as mean±SEM for n=27 per quartile.

The relatively modest changes in plasma cholesterol in response to changes in dietary cholesterol are readily explained when the total body dynamics of exogenous and endogenous cholesterol are considered quantitatively. As shown in Table 2, dietary cholesterol has a relatively small mass contribution to whole body cholesterol metabolism in humans [19]. Total body cholesterol content is approximately 150 g with one-third of the cholesterol localized in the brain and central nervous system. At a plasma cholesterol level of 200 mg/dL an adult would have 8 g of cholesterol in the plasma compartment. Cholesterol absorption averages 60% with a inter-individual range from 20% to 80%. Endogenous cholesterol synthesis ranges between 11 and 13 mg/kg-day in men, women and children and is feedback regulated by dietary cholesterol [31, 32]. As can be seen from the data in Table 2, a change in dietary cholesterol of 100 mg/day results in an actual change in cholesterol metabolism of 30 mg/day, or less than 3% of the total mass of cholesterol metabolized daily in the body. The precision of the feedback mechanisms regulating endogenous cholesterol synthesis is one factor which accounts for the variability of responses between individuals to a change in dietary cholesterol [33].

As noted above, cholesterol feeding studies have involved a variety of experimental conditions, and from different reports there have been suggestions that associated dietary and physiological factors influence the plasma cholesterol response to dietary cholesterol. Using data from the 167 published cholesterol feeding studies, it is possible to determine whether the type and/or amount of fat in the diet influences the response to dietary cholesterol and whether hypercholesterolemic individuals are more responsive to dietary cholesterol compared to normocholesterolemic subjects. Studies with the variable of interest were analyzed according to quartile distributions of the variables to determine effects on the dose adjusted plasma cholesterol response to a dietary cholesterol challenge. The results of these analyses are shown in Figs. 3–7. Dietary fat calories had no significant effect on the plasma cholesterol response to dietary cholesterol (Fig. 3), and there is no evidence that dietary fat type influences the change in plasma cholesterol with changes in dietary cholesterol (Fig. 4). As shown in Fig. 5, the plasma cholesterol response to a 100 mg/day increase in dietary cholesterol does not differ between hypo-cholesterolemic and hyper-cholesterolemic individuals. Based on these data there is no evidence that individuals with elevated plasma cholesterol levels are more sensitive to the plasma cholesterol raising effects of dietary cholesterol. Baseline dietary cholesterol intake does appear to significantly alter the plasma response to dietary cholesterol, with the response being greater

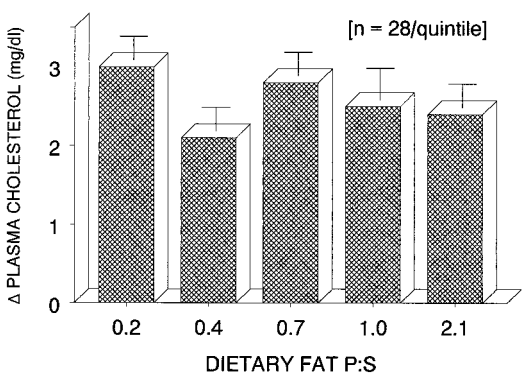

Fig. 4. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol for each quartile of dietary fat P:S ratio. Data presented as mean±SEM for n=28 per quartile.

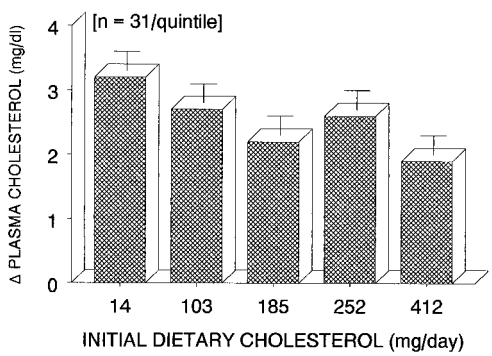

Fig. 6. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol for each quartile of initial dietary cholesterol (mg/day). Data presented as mean±SEM for n=31 per quartile.

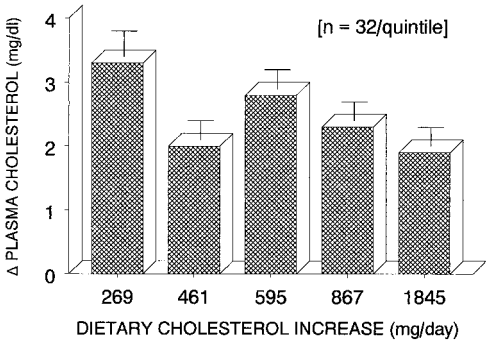

Fig. 7. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol for each quartile of dietary cholesterol increase (mg/day). Data presented as mean±SEM for n=32 per quartile.

when the initial cholesterol intake is near zero (Fig. 6). As can be seen in Fig. 6, with addition of cholesterol to a baseline diet containing 100 mg/day or more cholesterol, the dose-adjusted plasma cholesterol response is fairly constant across intake levels. The plasma cholesterol response to dietary cholesterol becomes attenuated as the challenge dose increases (Fig.7), probably due to the decreased ability of the body to maintain a constant fractional absorption rate [19].

Cholesterol Feeding Studies: Effects on Plasma Lipoprotein Cholesterol Levels

An assumption made in studies of dietary cholesterol effects on plasma cholesterol levels is that changes in plasma total cholesterol reflected changes in the relative risk of CHD, since plasma total cholesterol concentrations serve as a surrogate marker for CHD risk. Early studies of the effects of dietary cholesterol on plasma cholesterol concentrated on measuring total plasma cholesterol levels and an increase in serum cholesterol was a priori associated with an increase in CHD risk. Over the years, as techniques for measuring specific lipoprotein cholesterol fractions became more widely available, investigators have quantitated the effects of dietary factors such as dietary cholesterol on specific lipoprotein fractions in the fasting and postprandial states.

In 1997 Clarke *et al.* [21] reported data from a meta-analysis of controlled, metabolic ward cholesterol feeding studies showing that dietary cholesterol increased plasma total cholesterol by increasing both LDL cholesterol and HDL cholesterol levels. These authors estimated that a 100 mg/day reduction in dietary cholesterol would lower plasma total cholesterol by 2.5 mg/dL with a reduction in LDL cholesterol of 1.9 mg/dL and in HDL cholesterol of 0.4 mg/dL. Using the database for all studies reporting the effects of dietary cholesterol on plasma lipoprotein cholesterol levels (n=69), the estimated responses to a 100 mg/day change in dietary cholesterol are 2.36 mg/dL for total cholesterol (95% CI 2.00–2.73), 2.07 mg/dL for LDL (95% CI 1.71–2.42) and 0.44 mg/dL for HDL (95% CI 0.34–0.55).

Hypo-Responders and Hyper-Responders

The data presented above represent the average plasma cholesterol response predicted for changes in dietary cholesterol in a population. Numerous studies have shown that there is a large degree of heterogeneity of responses to dietary cholesterol and that within the population there are hypo-responders and hyper-responders [34–37]. It has been estimated that between 15% and 25% of the population is sensitive to dietary cholesterol, whereas the remainder has an attenuated plasma cholesterol response to dietary cholesterol. Analysis of the available data indicates that the dose adjusted response to a 100 mg/day dietary cholesterol challenge in hyper-responders is 3.9±0.6 mg/dL (n=10, 95% CI 2.5–5.3), compared to a response of 1.4±0.2 mg/dL (n=13, 95% CI 1.0–1.9) in hypo-responders (p=0.0002). The difference in the plasma cholesterol response between hypo- and hyper-responders to dietary cholesterol is almost threefold. As might be expected, the plasma lipoprotein responses to dietary cholesterol in hypo-responders and hyper-responders differ primarily in the LDL cholesterol response (Fig. 8), with the LDL response being 0.76±0.25 (n=5) *versus* 2.84±0.66 (n=5) mg/dL per 100 mg/day in hypo- *versus* hyper-responders (p=0.0185). The plasma HDL cholesterol change with a 100 mg/day change in dietary cholesterol were 0.50±0.14 (n=5) and 0.69±0.16 (n=5) in hypo- and hyper-responders, respectively (p>0.05).

Studies attempting to characterize hyper-responders have suggested that individuals with apoE-4 [38–40], combined hyperlipidemia [41], the apolipoprotein A-IV-2 allelle [42] and specific apo B promoter gene polymorphisms [39] have increased sensitivity to dietary cholesterol. Studies have also shown that excess body weight is associated with decreased sensitivity to dietary cholesterol [43]. As shown in Fig. 9, individuals with combined hyperlipidemia have a greater plasma total and LDL cholesterol response to addition of two eggs per day to the diet compared to subjects with hypercholesterolemia [41]. In a similar manner, data indicate that the dose-adjusted plasma cholesterol response to dietary cholesterol is greater in individuals with body mass index (BMI) values of <24, compared to those with higher BMI values (Fig. 10) [43]. This differential effect of body weight may relate to

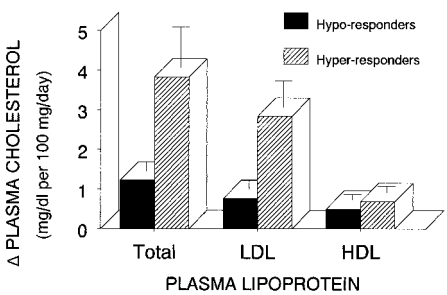

Fig. 8. Plasma total, LDL and HDL cholesterol changes per 100 mg/day change in dietary cholesterol in hypo-responders and hyper-responders. Data presented as mean±SEM.

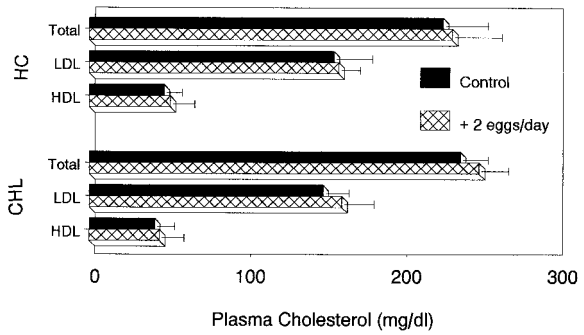

Fig. 9. Changes in plasma total and lipoprotein cholesterol levels (mg/dL) in subjects with hypercholesterolemia (HC) or combined hyperlipidemia (CHL) with intake of a control diet or a control diet plus 2 eggs per day as reported by Knopp et al. [41].

the fact that endogenous cholesterol synthesis is a function of body weight [19] and individuals with low BMI values would be predicted to have lower rates of synthesis and reduced feedback regulatory responses.

Even though dietary cholesterol has a relatively small effect on plasma total cholesterol levels, some have proposed that dietary cholesterol is atherogenic, even when plasma cholesterol levels are unchanged due to effects on postprandial lipoproteins [44, 45]. One proposed effect of dietary cholesterol is generation of atherogenic postprandial lipoproteins. Five reports have indicated that dietary cholesterol neither alters the post-prandial lipoprotein profile nor the efficacy of plasma to facilitate cholesterol efflux from cells. Ginsberg *et al.* [46, 47], Clifton and Nestel [48] and Knopp *et al.* [41] reported that dietary cholesterol had no negative effects on the pattern of post-prandial lipoproteins and that there were no significant increases in any candidate atherogenic particles with either acute or long term cholesterol feeding. Blanco-Molina *et al.* [49] reported that cholesterol feeding to humans increased plasma-induced cholesterol efflux from cells in culture. These data are consistent with other reports in the literature which did not find a dietary cholesterol induced production of atherogenic lipoprotein particles or inhibition of reverse cholesterol transport. To date, no studies using physiological intakes of dietary cholesterol have reported generation of atherogenic post-prandial lipoprotein particles.

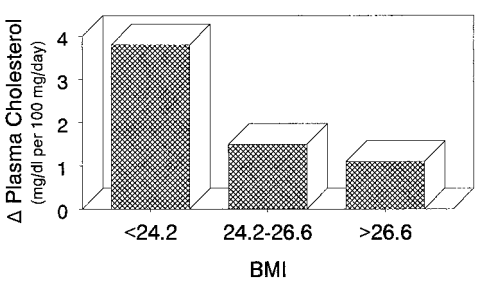

Fig. 10. Changes in plasma cholesterol (mg/dL) per 100 mg/day change in dietary cholesterol as a function of BMI [43].

Eggs and Plasma Cholesterol

In 1998 a series of Letters to the Editor in the *American Journal of Clinical Nutrition* discussing the relationship between eggs and CHD [50–52], there was the statement that "It is a reasonable inference that the sizable decline in per capita egg consumption in the United States in recent decades, and hence in per capita total cholesterol intake, has been one important component of the improved dietary patterns leading to a fall in mean serum cholesterol concentration in the adult population from ≈6.08 mmol/L (235 mg/dL) in the 1950s to ≈5.30 mmol/L (205 mg/dL) in the 1990s, and to the concomitant sustained marked reductions in mortality rates from CHD, all cardiovascular diseases, and all causes." [50].

Based on data from the meta-analysis of cholesterol feeding studies, it is possible to quantitate the effects of the decrease in per capita egg consumption on the average plasma cholesterol level in the population. The decrease in per capita egg consumption from 321 eggs per year in 1960 to 235 eggs per year in 1995 equates to an average decrease of 1.65 eggs per week which, at a cholesterol content of 215 mg/large egg, lowers the average dietary cholesterol intake by 355 mg/week or 51 mg/day. A 51 mg/day decrease in dietary cholesterol would be predicted to lower the mean plasma total cholesterol level by 1.1 mg/dL (51 mg/day × △ 0.022 mg/dL per mg/day). And that 1.1 mg/dL decrease occurs due to a 0.9 mg/dL decrease in LDL cholesterol and a 0.2 mg/dL fall in HDL cholesterol. Based on these data, the 27% decrease in per capita egg consumption from 1960 to 1995 accounts for only 3% of the 30 mg/dL fall in the average cholesterol level in the population during this time period.

Eggs and LDL:HDL Cholesterol Ratio

Based on the results of the meta-analysis by Clarke *et al.* [21], the average effects of adding an egg a day to the diet on plasma LDL cholesterol, HDL cholesterol and the LDL:HDL ratio can be estimated. As shown in Table 3, addition of an egg a day to the diet increases plasma LDL by 4.1 mg/dL and HDL by 0.9 mg/dL with changes in the LDL:HDL ratio ranging from

Table 3. Theoretical changes in plasma lipoprotein cholesterol levels and LDL:HDL cholesterol ratio with addition of 1 egg per day to the diet

	Cholesterol (mg/dL)		LDL:HDL Ratio	
	LDL	HDL	LDL:HDL	% Change
Baseline	130	50	2.60	
+ 1 egg/day	134	51	2.63	1.2%
Baseline	150	50	3.00	
+ 1 egg/day	154	51	3.02	0.7&
Baseline	170	50	3.40	
+ 1 egg/day	174	51	3.41	0.3%

0.03 in the low LDL model to 0.01 in the high LDL model. These modest changes in the LDL:HDL cholesterol ratio with addition of an egg a day to the diet are consistent with the epidemiological study reports that egg consumption is not related to CHD incidence [18, 53]. Based on the available data, egg restrictions would be predicted to have little effect on plasma cholesterol levels or on CHD risk.

SUMMARY AND CONCLUSION

Clinical studies of the effects of dietary cholesterol on plasma lipids and lipoproteins carried out over the last thirty years clearly show that dietary cholesterol does have a small, but measurable effect on plasma cholesterol levels. The plasma cholesterol response to dietary cholesterol is independent of dietary fat type and amount and independent of the baseline plasma cholesterol level and dietary cholesterol increment. The plasma cholesterol response is greater when cholesterol is added to a cholesterol-free diet, but the changes are similar when cholesterol is added to a diet containing 100 mg or more of cholesterol a day. The mean plasma cholesterol response to dietary cholesterol averages 2.2 mg/dL per 100 mg/day. The dietary cholesterol induced increase in plasma total cholesterol results from increases in both LDL cholesterol and HDL cholesterol levels, with little change in the LDL:HDL cholesterol ratio.

There is considerable heterogeneity of responses between humans with the majority of individuals being classified as hypo-responders (1.4 mg/dL per 100 mg/day) and a minority exhibiting hyper-responder characteristics (3.9 mg/dL per 100 mg/day). Both hypo-responders and hyper-responders increase plasma total cholesterol in response to dietary cholesterol by increasing both LDL cholesterol and HDL cholesterol concentrations, but the LDL cholesterol increase is significantly higher in the hyper-responders.

The results of clinical trials, as well as epidemiological survey data, demonstrate that egg consumption has little relationship to hypercholesterolemia or CHD incidence. Recommendations that egg consumption be restricted by the general population are not supported by the experimental data and, as noted by the reviews published in this issue of the journal, limits a valuable and affordable source of high quality nutrition from the diet. The nutrient density of eggs and their role in a heart healthy diet need to be reconsidered in light of the changing dietary patterns in the population and the increase in obesity in the American public. There is no scientific justification for population-wide specific limits on weekly egg consumption, and such restrictions only serve to divert attention away from effective dietary and life-style interventions to lower CHD risk.

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