

*“The great tragedy of science—  
the slaying of a beautiful  
hypothesis by an ugly fact.”*

—Thomas Huxley

# NUTRITION CLOSE-UP

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*Nutrition Close-Up* is a quarterly publication of the American Egg Board, written and produced by the Egg Nutrition Center. *Nutrition Close-Up* presents up-to-date reviews, summaries and commentaries on the latest research on the role of diet in health promotion and disease prevention, including the contributions of eggs to a nutritious and healthful diet. Nutrition and healthcare professionals can request a free subscription to the newsletter by visiting the Egg Nutrition Center's website at [www.enc-online.org](http://www.enc-online.org).



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## Changes in Plasma Lutein with Egg Intake are Associated with BMI in Postmenopausal Women

It has been well-documented that higher levels of lutein and zeaxanthin (L&Z) pigments in the serum and macular region of the retina are associated with a decreased risk of age-related macular degeneration (AMD). Studies have also shown that the concentrations of these carotenoids generally increase with increased dietary intake of foods rich in these antioxidant pigments. However, individual plasma responses to dietary carotenoid intake vary. For some, increasing dietary intake of lutein and zeaxanthin results in a large increase in serum concentrations. For others, the changes in serum concentrations are minimal. This phenomenon also holds true for dietary cholesterol. While for some, serum LDL and HDL cholesterol concentrations rise modestly when dietary cholesterol intake increases, others experience little or no change. A recent study in healthy young men and women showed that when whole eggs were added to participants' diets, the change in serum lutein levels was positively associated with the serum lipid response to added dietary cholesterol (Clark et al. 2006). In other words, those whose cholesterol levels increased in response to egg intake (responders) experienced a greater increase in serum lutein than those whose serum cholesterol levels did not increase (non-responders). To further clarify the relationship between serum lutein response and cholesterol metabolism, investigators from the University of Connecticut at Storrs tested the effects of egg intake on serum lutein and on serum HDL and LDL cholesterol concentrations in healthy postmenopausal women.

*“...individuals who want to reduce their risk for AMD [might] have to lose weight before they respond to lutein interventions.”*

The study group was comprised of 22 healthy postmenopausal women who had no history of heart disease, kidney disease, or diabetes and were not taking any lipid-lowering medications. The women were randomly assigned to an egg or placebo treatment for 30 days, after which they underwent a 30-day wash-out period and then began the alternate 30-day treatment period. During the egg treatment, the women consumed the liquid equivalent of 3 whole, large eggs daily, which contributed ~640 mg cholesterol and 600 µg of lutein + zeaxanthin. During the placebo treatment period, the women consumed a cholesterol-free egg substitute daily that contributed no lutein or zeaxanthin. Compliance was monitored via seven 24-hour dietary records completed during each treatment period. The women were instructed to maintain their normal eating habits over the course of the study, but to avoid consuming eggs outside of those provided by the study administrators.

Women whose serum cholesterol increased >2.1 mg/dL (>0.06 mmol/L) per 100 mg dietary cholesterol were classified as responders. Those who experienced increases of <1.7 mg/dL (<0.05 mmol/L) per 100 mg dietary cholesterol were classified as non-responders. In this group, 9 women (41%) were classified as responders while 13 (59%) were classified as non-responders. Responders and non-responders were similar with regard to age, BMI, waist circumference, total

*Continued on page 2*

## Changes in Plasma Lutein *continued from page 1*

cholesterol, HDL cholesterol, LDL cholesterol, triacylglycerol concentrations, and baseline dietary cholesterol and lutein + zeaxanthin intake.

Responders experienced statistically significant increases in total, HDL, and LDL cholesterol concentrations from baseline following the egg treatment. Total cholesterol increased from  $176 \pm 52$  mg/dL to  $212 \pm 52$  mg/dL ( $4.56 \pm 1.34$  to  $5.48 \pm 1.35$  mmol/L); HDL cholesterol increased from  $55 \pm 14$  mg/dL to  $60 \pm 19$  mg/dL ( $1.42 \pm 0.37$  to  $1.56 \pm 0.50$  mmol/L); LDL cholesterol increased from  $95 \pm 47$  mg/dL to  $126 \pm 57$  mg/dL ( $2.47 \pm 1.23$  to  $3.26 \pm 1.48$  mmol/L);  $P < 0.05$ . Triacylglycerol concentrations remained unchanged. No changes in serum lipid concentrations were observed in non-responders following egg treatment.

Plasma lutein and zeaxanthin concentrations increased for both responders and non-responders following the egg treatment period, however, for responders, the increase was two-fold that of the non-responders (responders' L&Z levels increased by  $0.27 \pm 0.23$   $\mu\text{mol/L}$ , while non-responders' levels increased by  $0.13 \pm 0.07$   $\mu\text{mol/L}$ ;  $P < 0.05$ ). Baseline plasma lutein concentrations were negatively associated with BMI ( $r = -0.44$ ;  $P < 0.05$ ). Waist circumference was inversely associated with plasma lutein concentrations at baseline ( $r = 0.45$ ,  $P < 0.05$ ) and with the change in plasma lutein concentrations following the egg treatment ( $r = 0.49$ ,  $P < 0.05$ ).

Plasma lutein concentrations following the egg treatment also correlated with the size, but not the number of HDL and LDL particles ( $r = 0.64$ ,  $P < 0.01$ ), indicating that the HDL and LDL particles carried more lutein. Activity of cholesterol ester transfer protein (CETP) and lecithin cholesterol acyltransferase (LCAT), which are instrumental in mediating the transfer of both lutein and cholesterol between lipoproteins, was not associated with changes in lutein concentration and did not differ between treatment groups.

This study supports earlier observations that changes in serum cholesterol and lutein concentrations in response to egg intake are, indeed, related. This relationship can likely be attributed to similar transport mechanisms for cholesterol and these dietary carotenoids. In this cohort of postmenopausal women, BMI and waist circumference were inversely associated with lutein levels at baseline and with changes in plasma lutein following increased egg intake. The authors conclude that those classified as “responders” to dietary cholesterol experience greater increases in plasma lutein and zeaxanthin with egg intake and that “...individuals who want to reduce their risk for AMD [might] have to lose weight before they respond to lutein interventions.” ■

Clark RM, Herron KL, Waters D, Fernandez ML. Hypo- and hyper-response to egg cholesterol predicts plasma lutein and {beta}-carotene concentrations in men and women. *J Nutr*. 2006;136:601-7.

Waters D, Clark RM, Greene CM, Contois JH, and Fernandez ML. Change in plasma lutein after egg consumption is positively associated with plasma cholesterol and lipoprotein size but negatively correlated with body size in postmenopausal women. *J Nutr* 2007, 137:959-963.

### KEY MESSAGES

- Individuals classified as “responders” to dietary cholesterol experienced greater increases in plasma lutein and zeaxanthin with egg intake.
- Serum levels of lutein and zeaxanthin increased to a lesser extent for individuals with higher BMI and waist circumferences.
- Results of this study indicate that weight loss might be necessary for overweight individuals seeking to prevent macular degeneration by increasing their dietary intake of lutein and zeaxanthin.

## SPECIAL FEATURE:

### EGG NUTRITION RESEARCH PRESENTED AT EXPERIMENTAL BIOLOGY 2007

Eggs enhance the anti-inflammatory component of a carbohydrate restricted diet

**Background:** Research has shown that carbohydrate-restricted diets can decrease levels of inflammatory cytokines in addition to reducing body fat and trunk fat.

**Objective:** Evaluate the results of including eggs in a carbohydrate-restricted diet on plasma anthropometrics, blood pressure, body composition, and selected inflammatory markers.

**Methods:** Participants were overweight/obese men (BMI=25-37 kg/m<sup>2</sup>) aged 40-70. The men were randomly assigned to follow one of two diet interventions (egg or egg substitute) for 12 weeks. Those assigned to the egg group ate 3 eggs (providing an additional 640 mg dietary cholesterol) per day. Those assigned to the egg substitute group ate an equivalent amount of egg substitute daily for the 12 weeks.

**Results:** All participants experienced statistically significant reductions in weight, BMI, waist circumference, body fat, and trunk fat ( $P < 0.01$ ). Systolic blood pressure decreased from  $138.8 \pm 17.3$  to  $126.3 \pm 10.6$  mm Hg. Plasma levels of interleukin-8 (IL-8), tumor necrosis factor (TNF)-alpha and monocyte chemoattractant protein 1 (MCP-1) also decreased as a result of the diet interventions. However, the egg intervention resulted in a better response than did the egg substitute intervention ( $P < 0.05$ ).

**Conclusion:** This study suggests that including eggs in carbohydrate-restricted diets might enhance the reduction in inflammatory markers typically achieved by such diet interventions.

Ratliff JC, Mutungi G, Puglisi M, Leite JO, Vaishnav U, Shrestha S, Volek JS, Fernandez ML. University of Connecticut, Storrs, CT. Research supported by a grant from the American Egg Board.

C-reactive protein (CRP) levels are not affected in participants consuming the equivalent of 2 and 4 egg yolks/day while on cholesterol-lowering medication

**Background:** Elevated C-reactive protein (CRP) levels have been associated with an increased risk of cardiovascular disease events and with elevated serum cholesterol levels. Whether dietary cholesterol affects CRP levels is still unknown.

**Objective:** The aim of this study was to determine whether dietary cholesterol has any effect on CRP levels.

**Methods:** A total of 26 older adults (>60 yrs) taking cholesterol-lowering medications completed the 4-phase, 18-week study. In phase 1, participants underwent a 4-week washout phase in which they refrained from consuming eggs. In Phase 2, participants consumed the equivalent of 2 egg yolks per day for 5 weeks. Phase 3 consisted of another 4-week washout period in which no eggs were consumed. In Phase 4, participants consumed the equivalent of 4 egg yolks per day for a total of 5 weeks. CRP levels were measured twice for each phase.

**Results:** CRP levels were not influenced by cholesterol intake levels >1000 mg/day.

**Conclusion:** Statins appear to control serum cholesterol levels even when dietary cholesterol intake is high.

Goodrow EF, Vishwanathan R, Wilson T, Nicolosi R. University of Massachusetts, Lowell. Research supported by a grant from the American Egg Board, Egg Nutrition Center, and the Massachusetts Lions Eye Research Fund

*Continued on page 6*



READ IT AGAIN...  
FOR THE FIRST TIME

1995

The Seven Countries Study: *A closer look at relative vs. absolute risk*

The Seven Countries Study, directed by Dr. Ancel Keys, has been considered a landmark study in modern cardiovascular disease epidemiology. For over a decade, Keys and his research colleagues gathered data from a large cohort of middle-aged men residing in seven countries: the United States, Japan, Italy, Greece, the Netherlands, Finland, and Yugoslavia. The purpose of the study was to assess the relationship between recognized cardiovascular disease risk factors and coronary heart disease (CHD) mortality within various populations.

The study administrators gathered baseline data between 1958 and 1964 from 12,763 men. Major cardiovascular disease risk factors (such as total serum cholesterol, blood pressure, and smoking status) were measured at baseline and again at the five-year and 10-year follow-up. The researchers observed a positive, linear relationship between total serum cholesterol and CHD mortality early in the follow-up phase, however, the association was much weaker for men in the Southern European and Japanese cohorts than for those in the US and Northern European cohorts. The 25-year follow-up provided an opportunity to examine the relationship between changes in serum total cholesterol levels and 25-year CHD mortality in different cultures.

In 25 years, only 56 men (0.4%) were lost to follow-up. Mean baseline serum total cholesterol levels varied between regions, ranging from 160 to 170 mg/dL (4.15 to 4.40 mmol/L) in the Japanese and Serbian cohorts to 200 mg/dL (5.15 mmol/L) in the Southern European cohorts and 240 to 260 mg/dL (6.20 to 6.70 mmol/L) in the US and Northern European cohorts.

This data was examined both within and across cultural cohorts, providing an interesting perspective on relative vs. absolute risk in this population of middle-aged men. Within all but the Japanese cohort, CHD mortality rose with increasing quartiles of serum cholesterol (see Table 1). The incremental change in relative risk (RR) per 20 mg/dL (0.50 mmol/L) increase in serum cholesterol was calculated for each cohort and fell between 1.10 and 1.15.

These incremental increases in CHD mortality risk were strikingly similar between cultural cohorts, ranging from 1.14 in Serbia to 1.21 in the US. The RRs for the highest compared to the lowest quartiles of serum total cholesterol were ~1.5 to 2.3 within cultures. Absolute CHD mortality rates, however, differed greatly across cultural cohorts. For example, for this population of middle-aged men, a cholesterol level of 210 mg/dL (5.45 mmol/L) represented varying mortality rates

Table 1: Adjusted RRs per quartile of cholesterol intake

COHORT	CHOLESTEROL QUARTILE				$\chi^2$ TREND P
	1 (LOW)	2	3	4 (HIGH)	
Northern Europe	1.0	1.11 (0.84-1.45)	1.34 (1.03-1.74)	2.03 (1.59-2.59)	<0.001
United States	1.0	1.09 (0.79-1.51)	1.39 (1.03-1.89)	2.34 (1.77-3.11)	<0.001
Southern Europe (Inland)	1.0	1.21 (0.84-1.74)	1.50 (1.06-2.12)	1.52 (1.07-2.15)	<0.01
Southern Europe (Mediterranean)	1.0	1.03 (0.57-1.85)	1.35 (0.78-2.33)	1.66 (0.98-2.80)	<0.05
Serbia	1.0	1.43 (0.69-2.96)	1.88 (0.94-3.73)	1.86 (0.92-3.76)	$\leq$ 0.05
Japan	1.0	1.51 (0.60-3.84)	0.89 (0.31-2.57)	1.13 (0.42-3.02)	$\leq$ 0.05

across cultural cohorts: 4-5% in Japan and Mediterranean Southern Europe, 10% in Southern Europe (Inland), 12% in the US, and 15% in Northern Europe. Figure 1 further illustrates the striking disparity in absolute risk between cultural cohorts, CHD mortality risk for Northern European men in the lowest cholesterol quartile (average: 190 mg/dL [4.90 mmol/L]) was nearly double that of Southern European (Mediterranean) men in the highest cholesterol quartile (average level: 250 mg/dL [6.45 mmol/L]).

What is most remarkable about these findings is that although CHD mortality risk rose with increasing serum cholesterol levels within individual cultural cohorts (*relative risk*), the discrepancies in *absolute risk* between these cohorts could not be predicted by serum cholesterol; neither could these differences be explained by variation in age, smoking status, or systolic blood pressure. The findings suggest that an exclusive focus on reducing blood cholesterol levels will not decrease CHD mortality risk in Northern Europe and the US to the more desirable levels characteristic of Mediterranean Europe. They indicate, rather, that other factors are clearly involved in determining CHD mortality risk.

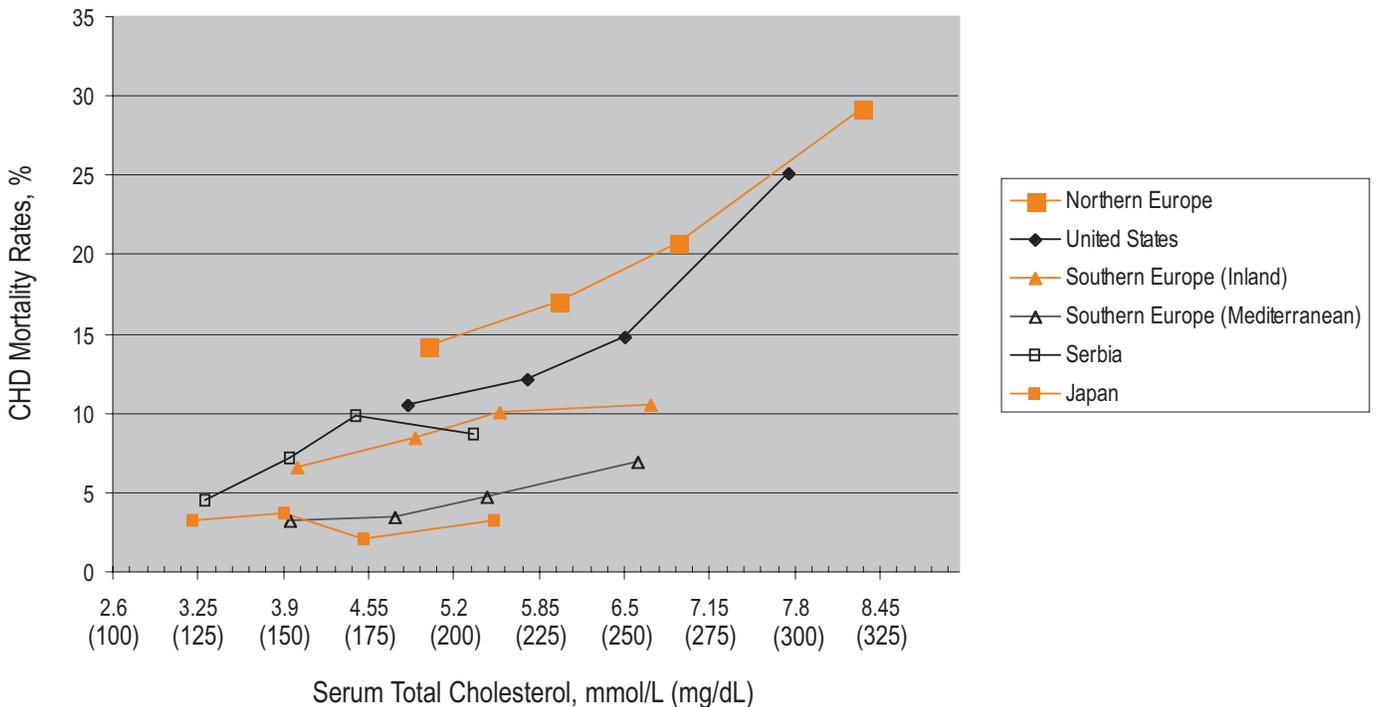
The authors suggest that since dietary patterns differ greatly between the cultural cohorts studied, nutritional factors might

be responsible, in large part, for the observed discrepancies in risk between cultures. For example, according to the dietary information collected from a small sample of men in this study, dietary patterns characteristic of the US and Northern Europe included more meat, less fish, fewer fruits and vegetables, and less alcohol than intake patterns characteristic of the Mediterranean. There was also a notable difference between these diets with respect to fatty acid content. While men in Mediterranean Europe consumed predominantly monounsaturated fat, men in the US and Northern Europe consumed more saturated fat. Antioxidant and flavonoid intake was also much higher in Southern Europe (both Inland and Mediterranean) and Japan than in the US and Northern Europe.

In addition to dietary influences, other factors might help account for differences in absolute CHD mortality risk. These include genetic and other biological risk factors (such as HDL cholesterol levels) that were not addressed in this research. ■

Verschuren WM, Jacobs DR, Bloemberg BP, et al. Serum total cholesterol and long-term coronary heart disease mortality in different cultures: Twenty-five-year follow-up of the Seven Countries Study. *JAMA* 1995;274(2):131-136.

Figure 1: Twenty-five-year CHD mortality rates by baseline cholesterol quartile



## Balancing and communicating risks and benefits associated with egg consumption— a relative risk study

**Background:** The American Heart Association (AHA) and the National Cholesterol Education Program (NCEP) recommend restricting egg consumption based on the hypothesis that egg intake will result in an increase in blood cholesterol levels, which have been associated with increased risk for coronary heart disease (CHD) risk. However, several studies have demonstrated that dietary cholesterol from eggs has little, if any, effect on serum cholesterol concentrations or on the risk for CHD after adjustment for other potential risk factors. Further, eggs provide many essential nutrients such as high quality protein, B vitamins and folate, fat-soluble vitamins such as A, D, and E, and minerals such as iodine, zinc, calcium, and iron. Eggs also contain lutein and zeaxanthin, which are thought to be instrumental in preventing the development of cataracts and age-related macular degeneration. They are also rich sources of choline, a nutrient thought to play a vital role in proper memory development. Therefore, recommendations to restrict egg consumption to avoid CHD risk may be misguided, considering 1) the evidence that egg intake has little to no effect on serum cholesterol levels, and 2) the valuable nutrients eggs provide at such low cost to consumers.

**Objective:** A multi-factorial risk apportionment technique was used to determine the contribution of egg intake to overall CHD risk relative to other risk factors including BMI, dietary [intake], smoking status, alcohol intake, lipid profile, activity level.

**Methods:** Estimates of egg contribution to dietary cholesterol and nutrition were developed based on consumption data from nationwide food consumption survey. A report card to communicate the relative risks of egg cholesterol and the nutritional benefits were explored in this paper.

**Results:** The contribution of egg intake to any given individual's

CHD risk is modified by the presence or absence of other risk factors. Assuming that serum cholesterol changes by 4.5 mg/dL for every 100 mg increase in daily cholesterol intake (McNamara, 2000), the contribution of eggs to CHD risk for a male with the following risk factors would be 0.8%.

Risk factors assumed in this model were as follows:

- ◆ Relatively poor diet
- ◆ Current smoker
- ◆ BMI >25
- ◆ Exercises < 3.5 hours/week
- ◆ Drinks <5g alcohol per day
- ◆ High-normal blood pressure
- ◆ LDL 130-159 mg/dL (3.37-4.12 mmol/dL)
- ◆ HDL 35-59 mg/dL (0.91-1.53 mmol/dL)

**Conclusion:** Given that the majority of males in the US over age 25 have two or more modifiable risk factors, consumption of 1 egg per day probably accounts for 0.86% of CHD risk (in this population). The authors conclude that "...targeting eggs and egg cholesterol as a means to reduce CHD risks for US males 25+ is not expected to yield a meaningful reduction in the CHD Egg Nutrition Center, and the Massachusetts Lions Eye Research Fundburden."

Tran NL, Barraj L, Mink P, McNamara DJ. Exponent, Washington, DC. Research supported by a grant from the American Egg Board.

## Different Strokes for Different Folks

Cultural differences are a wonderful part of what makes travel so very interesting and enlightening. One would think, however, that science would cross cultural barriers and that facts would be facts, no matter what border one happened to cross. But such is not the case when it comes to nutrition and nutrition policy.

There are actually places in this world where some of the dietary dogmas so venerated in the US are cast aside as fundamentally unsound and unproven. This is especially exasperating to some of our senior nutritional scientists who have vested their careers in promulgating these dogmas. They simply cannot understand why scientific bodies representing other nations would choose not to follow along. How dare they question (let alone refute with scientific evidence) what has been anointed by a “consensus” of US scientists? [Of course, one should remember that a consensus is simply a common statement from a faction that does not allow dissent from the group opinion.] In some parts of the world, the voices of dissent (rarely heard in this country) are the majority, and debate actually leads to different opinions and interpretations.

This becomes especially intriguing considering what happens to eggs at the US-Canada border. When I stand in the US, eggs are considered marred by their cholesterol content. According to US labeling regulations, cholesterol is a “negative nutrient” and because eggs contain > 60 mg per serving, they are subjected to restrictions in terms of the positive things that can be said about them. In the US, the “no more than 300 mg per day of dietary cholesterol” recommendation continues to be a meaningful restriction for many consumers and their health care providers, and the recommendation to limit egg consumption remains part of the standard dietary advice. Now, I take one step over the border into Canada, and—POOF!—no 300 mg per day cholesterol restriction, no “negative nutrient” label, and—get this—eggs carry the Health Check, a labeling symbol created by the Heart and Stroke Foundation of Canada to help consumers identify heart-healthy foods.

Step back across the border and—voila! Egg intake should be limited, cholesterol-free equals “healthy,” (no matter what else a food might contain), and the beneficial nutrients found in eggs must be hidden behind a warning about their cholesterol content.

Step back across the border and the egg’s nutritional contributions can actually be promoted.

“But,” you protest, “that’s Canada. They just don’t like to follow the United States’ lead.” Sorry, the same holds true in Australia, New Zealand and Ireland. In Australia, eggs carry the “tick” from the Australian Heart Foundation, and in Ireland, roadside billboards sponsored by the Irish Heart Foundation proclaim, “An egg a day is okay!” So while everyone has access to the same body

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*“[The] dietary guidelines in the United States are the only ones that...recommend a quantitative threshold for cholesterol intake.”*

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of science, they certainly do not have the same interpretation of what that science means. Could there be something we’re missing in this debate? Or have we just been there for so long that it’s an intractable position? Must be nice to be able to evaluate the evidence without preconceived opinions. But after almost forty years of telling the same story, will US thought leaders thwart a time-honored tradition for the sake of being “scientific?”

A report from the Life Sciences Research Office (LSRO) titled, “The Scientific Evidence and Approach Taken to Establish Guidelines for Cholesterol Intake in Australia, Canada, the United Kingdom, and the United States” ([http://www.lsro.org/articles/cholest\\_egg\\_rpt.html](http://www.lsro.org/articles/cholest_egg_rpt.html)), states that “Among the current national and international guidelines reviewed in this LSRO report, all agree on the necessity to reduce saturated fat but dietary guidelines in the United States are the only ones that also recommend a quantitative threshold for cholesterol intake.” But then everyone else could be wrong and we’re the only ones right. Or! No, no, no! The thought is just too outrageous! There is no way we could have been wrong for so many years...is there? We must always remember the words of Thomas Huxley—“The great tragedy of science—the slaying of a beautiful hypothesis by an ugly fact...” and then be open to those “ugly facts.” ■

*Donald J. McNamara, Ph.D  
Executive Editor, Nutrition Close-Up*



## ARE YOU UP-TO-DATE ON CHOLINE?

By now, you're probably aware that choline is not only an essential nutrient, but one that is vitally important in the arsenal of protective prenatal nutrients. Adequate choline intake supports proper fetal brain development and has been shown to prevent some birth defects...But did you know that according to NHANES data, only 10% of the US

population consumes enough choline to reach Adequate Intake levels? This means that far too many woman of childbearing are falling short of the 450 mg/day recommended to support a healthy pregnancy and the 550 mg/day needed during breastfeeding.

To keep you up-to-date on the emerging science surrounding this important nutrient, the Egg Nutrition Center has developed a brochure for you and your patients.

Please call us at 202-833-8850 to order your complimentary copies or visit our website ([www.enc-online.org](http://www.enc-online.org)) to download a printable online version of the brochure.

### How much choline is needed during pregnancy and lactation?

An Adequate Intake (AI) of choline is:  
 • 425mg/day for an adult non pregnant woman  
 • 450mg/day during pregnancy  
 • 550mg/day for breastfeeding women

Many prenatal vitamins do not currently contain choline, so the best source of choline remains from food.

Food source	mg Choline/100g	whole serving size	mg Choline/serving
Egg (over)	553.7	1 egg	553.7
Egg	265.0	2	530.0
Chicken liver	196.3	2.0oz	392.7
Cheeseburger	21.0	1 sandwich	42.0
Bulbini	85.4	2 oz	170.8
Chicken	78.7	2 oz	157.4
Wendy's hamburger	16.4	1 patty	32.8
Progresso Pesto	26.4	1.5oz	39.6
Wendy's green	12.1	1/2 cup	18.15
2% milk	16.4	1 cup	32.8
Yogh	27.3	1/2 cup	32.8
Wendy's	85.7	1/2 pizza (7 oz)	157.4
Green & Cheese Burrito	22.8	1 piece	22.8
Broccoli	16.7	10 cups	167.0

Revised 11/11, Department of Agriculture, Database for the Choline Content of Foods in Trade, 2004

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