

*“Cholesterol is poisonous, so
Never, never eat it.
Sugar, too, may murder you
There's no way to beat it.
And FATTY FOOD may do you in,
Be certain to avoid it.
Some food was rich in vitamins
But processing destroyed it.
So let your life be ordered
By its documented facts,
And die of malnutrition . . .*

But with ARTERIES intact.”

—David Kritchevsky

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Egg Intake Improves Macular Pigment Density

Age-related macular degeneration (AMD) is a debilitating, progressive eye disease faced by nearly 15% of the population aged 75 and over and is the single largest cause of legal blindness in the U.S. This condition is caused by a partial breakdown of the retinal pigment epithelium and results in damage to the macula (the portion of the retina responsible for central vision). Because it impairs central vision, AMD severely limits the ability of affected individuals to read, drive, and perform other daily tasks. Research has demonstrated that increased blood levels of lutein and zeaxanthin (dietary carotenoids that accumulate in the macula) might reduce the risk of this eye disease and that lutein supplementation might be effective in slowing the progression of AMD. Studies have also shown that increased dietary intake of fruits and vegetables high in lutein and zeaxanthin improve macular pigment levels (or macular pigment optical density—MPOD).

Egg yolk is recognized as a highly bioavailable source of lutein and zeaxanthin. Probably due to the lipid matrix of the yolk, lutein and zeaxanthin from egg yolk have been shown to be more bioavailable than from spinach or supplements. Previous research in an elderly population with a mean age of 62 y showed that consumption of 1.3 eggs for a period of 4.5 weeks significantly increased plasma lutein and zeaxanthin concentrations and was accompanied by a modest increase in LDL cholesterol concentrations.

To determine the effect of egg supplementation on serum lipid and carotenoid levels in an older population, Goodrow et al. recruited 33 men and women (mean age 79 y) for participation in an 18-week cross-over study. Potential participants who had been taking lutein or zeaxanthin supplements or who were on cholesterol lowering medications were excluded from the study.

The study consisted of four phases. During Phase I (4 weeks), participants were asked to limit their intake of foods containing lutein and zeaxanthin and to avoid eating eggs and foods made with eggs as a main ingredient. Phase II consisted of a 5-week intervention period in which participants were assigned to the no-egg (egg substitute) or 1 egg/day treatment. Those in the 1 egg/day treatment group were instructed to simply add one egg to their normal diet every day. Phase III was a 4-week washout period in which eggs, egg-containing foods, and foods containing lutein and zeaxanthin were to be avoided by all participants. Phase IV was a 5-week cross-over period in which the groups initially assigned to the 1 egg/day treatment were asked to follow the no-egg protocol, and vice versa. Two fasting blood samples were drawn during each phase of the study to measure serum carotenoid and lipid levels.

Analysis of the background “normal” diet of participants revealed that only 5 lutein and zeaxanthin-containing foods were typically consumed—orange juice, lettuce, yellow corn, broccoli,

Macular pigment optical density improved significantly and linearly in women whose diets were supplemented with six carotenoid-enriched eggs per week.

Continued on page 2

and onions—and that these were consumed in such small amounts that they contributed insignificant amounts of these carotenoids. During the intervention phases, one egg per day added approximately 143 ± 28 μg lutein, 94 ± 18 μg zeaxanthin, and 210 ± 9 mg cholesterol per day to participants' diets.

By the end of the 5-week egg intervention, participants' serum levels of lutein and zeaxanthin had increased from baseline by 26 and 38%, respectively ($P < 0.05$). Serum total cholesterol, LDL cholesterol, HDL cholesterol, and triacylglycerol concentrations did not differ from baseline after completing the 5-week egg intervention.

These findings are significant in light of the fact that most previous studies examining the effects of egg intake on serum carotenoid levels used carotenoid-enriched eggs, while Goodrow et al. chose to use unenriched eggs. These observations demonstrate that the addition of one egg a day (a common, inexpensive and highly bioavailable food source of lutein and zeaxanthin) to the regular diet of elderly individuals is effective in raising serum carotenoid levels without negatively affecting the lipid profile.

However, an important question still remains...If supplementation with egg yolk increases serum levels of lutein and zeaxanthin, does it increase the concentration of these carotenoids in the macula as well? Another recent study by Wenzel et al. addressed this question in a cohort of younger females, aged 24–59y. Twenty-four healthy, non-smoking females were assigned to 1 of 3 treatment groups (EGG1, EGG2, or control) for a period of 12 weeks. Women in the EGG1 group were instructed to consume 6 carotenoid-enriched eggs (each containing ~ 331 μg of lutein+zeaxanthin) per week over the course of the study. Women assigned to the EGG2 group were instructed to consume the same number of carotenoid-enriched eggs (each containing ~ 964 μg of lutein+zeaxanthin) each week during the study period. Those assigned to the control group were instructed to take 1 gelatin capsule (placebo) per day over the 12-week course. Serum lipids, serum carotenoids, and macular pigment optical density (MPOD—a measure of carotenoid levels in the macula) were measured at baseline and following weeks 4, 8, and 12 of the intervention period.

Age, BMI, serum lipoprotein, and serum carotenoid concentrations were similar between the three treatment groups at baseline. MPOD did not correlate with serum lutein or zeaxanthin levels at baseline, neither did total cholesterol concentration correlate with lutein, zeaxanthin, or MPOD. However, HDL cholesterol levels did correlate with combined serum lutein+zeaxanthin ($r = 0.40$, $P = 0.04$) and with MPOD ($r = 0.44$, $P = 0.03$). Baseline MPOD did differ between the EGG1 and EGG2 groups, with the EGG2 group reporting a higher MPOD ($P < 0.05$).

It is interesting to note that while serum total cholesterol and triacylglycerol (TAG) levels increased significantly in the control group ($P = 0.042$ and $P = 0.02$, respectively), these measures did not change for the women in the EGG1 or EGG2 group. HDL cholesterol was unchanged for all groups, leaving the EGG1 and

EGG2 groups with stable total:HDL cholesterol (TC:HDL-C) ratios, while the ratio of the control group actually increased following a cubic trend ($P = 0.001$). LDL cholesterol levels did not change for any group over the course of the study. TAG concentrations did not change for the EGG1 or EGG2 groups, but increased for women in the control group ($P = 0.02$).

The authors note that three participants (2 in the EGG1 group and 1 in the EGG2 group) discontinued treatment 2 months into the intervention because their total cholesterol levels had increased $\sim 17\%$ from baseline. HDL cholesterol levels for each of these participants also increased by $\sim 17\%$ from baseline, leaving their TC:HDL-C ratio unchanged at 3.2.

Serum lutein concentrations did not increase significantly in any of the treatment groups. However, serum zeaxanthin levels increased from baseline for both EGG groups (EGG1: $P = 0.04$; EGG2: $P = 0.01$). MPOD increased significantly and linearly from baseline in both EGG1 and EGG2 participants ($P = 0.001$ and $P = 0.04$, respectively). MPOD did not increase for the control group.

It has been established that individuals generally fall into one of two categories with regard to physiological response to cholesterol feeding and are typically described as hyporesponders or hyperresponders. The authors note that based on the definition provided previously by Herron et al. (hyperresponder = change in serum cholesterol > 0.06 mmol/L for each additional 100 mg/d dietary cholesterol, which is the average response observed across studies), 7 of the 16 participants assigned to the supplementary egg groups were hyperresponders. They represent $\sim 43\%$ of the study population, similar to the percentage found by Herron et al. to be hyperresponsive to dietary cholesterol. They also note that the increase in combined serum lutein + zeaxanthin from baseline in this hyperresponsive group was 8-fold greater than the increase observed in the hyporesponders.

A number of interesting observations warrant further investigation in this area. First, although serum levels were not consistently predicted by lutein and zeaxanthin intake in this study, MPOD improved significantly and linearly in women whose diets were supplemented with six carotenoid-enriched eggs per week. Second, serum cholesterol levels were less predictable. Mean serum total cholesterol increased for the control group, while it remained stable in the egg feeding groups. Third, serum lutein and zeaxanthin increased to a greater extent in participants classified as hyperresponders than in those classified as hyporesponders.

It is also interesting to note the wide variation in individual responsiveness to egg feeding. While Wenzel et al. reported no increase in mean serum cholesterol levels with egg feeding in a group of young to middle-aged women, they identified 3 individuals (who did not complete the study) whose total and HDL levels increased significantly following egg feeding and 7 who were classified as hyperresponders. In contrast, Goodrow et al. did not

observe an increase in serum lipids following egg feeding in a population of older adults. Further investigation utilizing larger sample sizes should help clarify the role of age and gender in cholesterol feeding responses. ■

Goodrow EF, Handelman G, Nicolosi RJ, et al. Consumption of one egg per day increases serum lutein and zeaxanthin concentrations in older adults without altering serum lipid and lipoprotein cholesterol concentrations. *J Nutr* 2006;136:2519-2524.

Wenzel AJ, Nicolosi RJ, Handelman GJ, et al. A 12-wk egg intervention increases serum zeaxanthin and macular pigment optical density in women. *J Nutr* 2006;136:2568-2573.

Herron KL, Vega-Lopez S, Fernandez ML, et al. Pre-menopausal women, classified as hypo- or hyperresponders, do not alter their LDL/HDL ratio following a high dietary cholesterol challenge. *J Am Coll Nutr* 2002;21:250-258.

KEY MESSAGES

- Adding one egg a day to the regular diet of elderly individuals significantly raises serum carotenoid levels without affecting the serum lipid profile.
- Women consuming six carotenoid-enriched eggs per week experienced a significant, linear increase in macular pigment density.

Dietary Lipids Unrelated to Ovarian Cancer

Cancer risk is thought to be influenced by a number of factors including genetic inheritance, environmental carcinogens, body weight, physical activity, and dietary intake.

Since genetic inheritance is largely thought of as a non-modifiable risk factor, there has been great interest in finding and documenting lifestyle factors—particularly dietary variables—that influence cancer risk. Because dietary fat and cholesterol are thought to increase circulating estrogen levels, researchers have questioned whether their intake might increase the risk of breast and ovarian cancers.

Since previous research examining fat and cholesterol intake and ovarian cancer risk have yielded conflicting results, scientists at the Harvard School of Public Health undertook a meta-analysis of 12 cohort studies based in North America and Western Europe. In addition to examining cholesterol intake alone, the researchers also analyzed the influence of egg consumption on cancer risk. While cholesterol and egg consumption have been associated with higher ovarian cancer risk in a few studies, egg intake has been associated with a decreased risk of breast cancer in others.

The pooled analysis included data from 553,217 women, including 2,132 with identified cases of ovarian cancer. All of the studies involved obtained individual food and nutrient intakes using a food frequency questionnaire at baseline. All included an assessment of total fat, SFA, MUFA, PUFA, and cholesterol intake and all but one study assessed egg consumption, specifically.

Neither total fat, nor cholesterol intake appeared to modify ovarian cancer risk. When women consuming >45% of energy from fat were compared with those who reported consuming 30 to 35%, no association was found between total fat intake and ovarian cancer risk (pooled multivariate RR = 1.08, 95% CI: 0.86-1.34). This observation also held for those consuming <25% vs. 30 to 35% of total calories from fat. There was no association between cholesterol intake and incidence of ovarian cancer. Egg intake was like-

wise found to be unrelated to ovarian cancer risk when intakes of >50 g/day (equivalent of one large egg) were compared with intakes of <6.25 g/day (pooled multivariate RR = 1.18, 95% CI: 0.89-1.57, p-value, test for trend = 0.52). When analyzed as a continuous variable, there was a slight, but nonsignificant, association between higher egg intake and ovarian cancer risk (pooled multivariate RR for a 50 g/day increment = 1.11, 95% CI: 0.99-1.24).

When women with the highest intakes were compared to those with the lowest, only weak positive associations were found between animal fat and SFA intake and ovarian cancer (pooled multivariate RR = 1.23, 95% CI: 0.98-1.55, p value for trend = 0.12). Evaluations of linearity showed that an increased risk for ovarian cancer was only detectable at intake levels above 21% of calories from SFA .

In summary, no significant associations were found between intake of total fat, MUFA, PUFA, TFA, animal fat, vegetable fat, eggs, or cholesterol and ovarian cancer risk. Several previous studies have found an association between fat intake and estrogen production, which supports the theory that dietary fat might increase the risk of ovarian cancer. One reason that this meta-analysis might have failed to detect the association, if one exists, is that most of the studies included a one-time food-frequency questionnaire that was not capable of taking into account any dietary changes over time. The authors also suggest the possibility that the window of greatest dietary influence might fall within an earlier stage of life. Overall, however, these findings indicate that dietary intakes of total fat, cholesterol, and eggs are unrelated to endometrial cancer risk. Further research is needed to clarify the influence of high SFA intake. ■

Genkinger J, Hunter DJ, Willett W, et al. A pooled analysis of 12 cohort studies of dietary fat, cholesterol and egg intake and ovarian cancer. *Cancer Causes and Control* 2006;17:273-285.

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FOR THE FIRST TIME

1981

Cholesterol Responsiveness Varies Between Individuals

It's hard to believe that in the 1980s, cholesterol research was still considered "young" by today's standards. Whereas today clinicians take into consideration a number of interacting serum markers when assessing cardiovascular health, at that time scientists were just beginning to recognize that the distribution and composition of plasma lipoproteins might influence CVD risk. Prior to the early '80s, research had focused almost exclusively on total serum cholesterol as the marker of concern and very little human research existed even in this field of study. It was during this time that Flaim et al. undertook an egg feeding trial in a small group of male university students to examine the influence of cholesterol intake on serum lipids and lipoproteins beyond serum total cholesterol. Because scientific views on dietary cholesterol and its effects on serum lipids were largely based on evidence from animal studies, this controlled human feeding study provided valuable insight. It was the first of its kind to examine the effects of cholesterol from eggs on different subtypes of serum cholesterol, and was, likewise, among the first to recognize the variation in response between individuals.

Flaim et al. followed 24 normolipidemic male university students for 5 weeks to determine the effects of increased dietary cholesterol from eggs on serum lipids. Plasma total cholesterol levels were determined for each participant at baseline, after which the participants were ranked by cholesterol level and matched with similarly-ranked controls. Both participants and controls were fed a "common American diet" for a period of 4 weeks. Controls consumed ~400 mg of cholesterol from non-egg sources. Participants consumed a similar diet that provided an additional 1000 mg of cholesterol per day from 4 whole eggs. The protein, carbohydrate, fat, and fatty-acid contributions were similar between diets.

During the fifth week, all study participants returned to their normal, unsupervised dietary habits. Because all meals were prepared, served, and eaten in the metabolic kitchen, compliance was very good. All but one subject completed the study.

Six fasting blood samples were drawn over the course of the study, one at baseline, one weekly during the experimental period, and one at the conclusion of week 5.

There were no statistically significant differences between participants and controls with regard to plasma total cholesterol levels over the course of the study. The researchers noted, however, that individual responses to the high cholesterol diet varied considerably. While 7 of the 12 (categorized as nonresponders) did not experience any increase in plasma total cholesterol levels with the additional 1000 mg dietary cholesterol, 5 of the 12 (categorized as responders) experienced a significant increase following week 1 of the experimental period. In 3 of these 5, however, total cholesterol levels returned to baseline levels by the end of week 4 (intermediate responders). Only 2 of the 5 responders experienced sustained increases in total serum cholesterol levels. Table 1 illustrates the changes in serum total cholesterol, HDL, LDL, and VLDL cholesterol levels over the course of the study for 3 participants consuming the high-cholesterol diet, one representing each response category.

HDL cholesterol levels were consistently higher for those consuming the high-cholesterol diet, but did not differ significantly between treatment groups except at week 3 (54 ± 3 mg/dl for the high-cholesterol diet group vs. 44 ± 3 mg/dl for the lower-cholesterol diet group; $p < 0.05$). Mean LDL and VLDL levels did not differ significantly between or within treatment groups, neither did the relative distribution of

HDL, LDL, or VLDL cholesterol levels. Triacylglycerol levels fluctuated inconsistently within both groups over the course of the study and increased significantly within the high-cholesterol group only at weeks 3 and 5.

These findings suggest that in young, healthy males already consuming a typical American diet providing a moderate amount of cholesterol, the addition of 1000 mg of cholesterol per day from whole eggs makes no appreciable difference in serum lipids. These data support the findings of several other studies demonstrating that when cholesterol was added to the diets of those already consuming a moderate amount of cholesterol, the additional cholesterol made no difference in plasma total cholesterol levels.

Perhaps the most notable of the findings reported by Flaim et al. was that individual responses to the additional dietary cholesterol varied so greatly. Subsequent research has confirmed that in a given population, individuals typically fit into one of

two response categories—non-responders (hyporesponders) and hyper-responders. Although this finding has been replicated many times over and this intraindividual variation has long been recognized by scientists in the field of lipid research, public health policy still seems to embrace one-size-fits-all recommendations for the population in general. Continuing research is needed to clarify the relative roles of other factors such as weight control, physical activity, and genetic influences on lipoprotein cholesterol profiles and CHD risk. ■

Flaim E, Ferreri LF, Thye FW, Hill JE, Ritchey SJ. Plasma lipid and lipoprotein cholesterol concentrations in adult males consuming normal and high cholesterol diets under controlled conditions. *Am J Clin Nutr* 1981;34:1103-1108.

TABLE 1: EXAMPLES OF SERUM LIPID CHANGES BY RESPONSE CATEGORY

RESPONSE CATEGORY	WEEK	CHOLESTEROL (mg/dl)			
		Total	HDL	LDL	VLDL
Non-Responder	0	134	43	84	7
	1	142	52	84	7
	2	142	49	90	3
	3	127	46	70	11
	4	131	44	82	5
Intermediate Responder	0	132	43	84	6
	1	161	53	100	8
	2	144	57	77	9
	3	138	51	66	21
	4	137	48	84	5
Responder	0	138	30	106	2
	1	180	38	138	3
	2	175	44	127	3
	3	178	40	121	17
	4	183	38	128	17

Choline Intake May be Insufficient for Many Adults in the U.S.

Choline—the “new” essential nutrient—is a relatively recent arrival in terms of nutrition research. After all, recommended daily intake levels for the nutrient were not established until 1998. Because of its potentially broad applications, however, its addition to the list of essential nutrients has generated a great deal of research interest. Choline participates in a number of critical biological functions. It helps keep homocysteine levels in check, serves as a precursor to phospholipids and neurotransmitters essential for proper nerve signaling, and is critical for proper brain and memory development and function. Therefore, its potential research implications range from brain development in the neonate to prevention of Alzheimer’s disease in the elderly.

To lay the groundwork for further research, it is necessary to determine the range of choline intake in the American population and determine whether this range is clinically important in terms of physiological need. Two questions needed to be answered. Do choline deficiencies exist in the population? If so, are they of clinical importance?

Because data on typical dietary intake levels in the United States have been scarce, Cho et al. examined intake data and laboratory information from the Framingham Offspring Study to describe current intake levels and to investigate the clinical importance of variations in intake within a largely Caucasian population in Framingham, Massachusetts. To evaluate the clinical importance of variations in choline intake, Cho et al. examined the influence of choline on total homocysteine concentrations (tHcy). Because choline, like folate, betaine, and vitamins B-6 and B-12, is a methyl group donor, the authors hypothesized that reported choline intake (as assessed by a food frequency questionnaire) would be associated with tHcy levels. (Because betaine is a metabolic product of choline and can act in a similar manner, betaine intake was also measured and assessed.)

Data from 1960 men and women (children of Framingham Heart Study participants) who participated in the Framingham Offspring Study and were free of heart disease were used in the analysis. Information regarding dietary intake was obtained from semiquantitative food frequency questionnaires and fasting blood samples were obtained from each participant to determine tHcy concentrations.

After adjustment for caloric intake, the mean choline intake for the study participants was 313 ± 61 mg/d, with 50% of the choline coming from food sources of phosphatidylcholine, including red meat, chicken, eggs, fish, and shellfish. The mean daily intake was 312 mg/d for men and 314 mg/d for women. Neither men, nor women met the recommended AI levels of 550 mg/d for men and 425 mg/d for women. The mean intake level for betaine was 208 ± 90 mg/d. Intakes of dietary choline and betaine were only weakly correlated because individuals obtained these nutri-

ents from different food sources. Animal products, including red meat, poultry, milk, and eggs provided most of the dietary choline. The correlation between choline and folate, vitamin B-6, and vitamin B-12 was also only moderate.

Higher intakes of choline and betaine were associated with lower tHcy levels, as expected, and the combined intake levels were predictive of tHcy concentrations. The multivariate geometric mean tHcy concentrations were 10.9 and 9.9 $\mu\text{mol/L}$ for the lowest and highest quintiles of combined choline/betaine intake, respectively (P for trend < 0.0001).

To test whether combined choline/betaine intake is more physiologically important when folate intake is low, the researchers evaluated the influence of choline/betaine intake on tHcy concentrations by three levels of folate intake. As expected, combined choline/betaine intake was a significant predictor of tHcy concentrations only when folate intake was < 250 $\mu\text{g/d}$ (P for interaction < 0.0001).

These findings suggest that for the average American Caucasian population, variations in choline intake are physiologically important and that neither men nor women consume the recommended levels of choline. The observations of Cho et al. also indicate that consumption of foods rich in choline might help make up for deficits in folate intake for physiological processes that require methyl group donation. This would be important not only for those seeking to keep homocysteine levels in check, but also for women not meeting the recommended folate intake levels during pregnancy. Another recent case-control study (Shaw et al. 2004) demonstrated that higher levels of periconceptional choline and betaine intake were associated with a reduced risk of neural tube defects, independent of folate intake. Multivariate odds ratio for the highest vs. lowest quartile of choline intake was 0.51; 95% CI: 0.25, 1.07 (Shaw et al. 2004). Therefore, meeting the recommended intake of choline during the periconceptional phase might provide additional “insurance” against neural tube defects.

Cho E, Zeisel S, Willett WC, et al. Dietary choline and betaine assessed by food-frequency questionnaire in relation to plasma total homocysteine concentration in the Framingham Offspring Study. *Am J Clin Nutr* 2006;83:905-11.

Shaw GM, Carmichael SL, Yang W et al. Periconceptional dietary intake of choline and betaine and neural tube defects in offspring. *Am J Epidemiol* 2004;160:102-9.

Letter to the Editor from Dr. David Kritchevsky 11/08/2006

Dear Don,

Your notes in "Nutrition Close-Up" (1) brought back memories of the public firestorm precipitated on the publication of "Toward Healthful Diets" (2) in 1980 by the Food and Nutrition Board of the National Academy of Science (of which I was a member). We had become concerned about the contradictory and misleading nutrition information available to the American public. The 24 page pamphlet contained recommendations for healthful diets based on the best available science of that time. The recommendations were:

- Select a nutritionally adequate diet from the foods available, by consuming each day appropriate servings of dairy products, meats or legumes, vegetables and fruits, and cereal and breads.
- Select as wide a variety of foods in each of the major food groups as is practicable in order to ensure a high probability of consuming adequate quantities of all essential nutrients.
- Adjust dietary energy intake and energy expenditure so as to maintain appropriate weight for height; if overweight, achieve appropriate weight reduction by decreasing total food and fat intake and increasing physical activity.
- If the requirement for energy is low (e.g., reducing diet), reduce consumption of foods such as alcohol, sugars, fats, and oils, which provide calories but few other essential nutrients.
- Use salt in moderation; adequate but safe intakes are considered to range between 3 and 8 g of sodium chloride daily.

Sound familiar? It should, it's not too different from recent Dietary Guidelines for Americans. What caused the violent reaction? Although we had written, "for persons with a positive family history of heart disease and other risk factors, such as obesity, hypertension and diabetes, concentration of blood lipids and lipoprotein fractions should be determined and, if any are abnormal, therapy should be undertaken under a physician's guidance," we had not included a special statement on cholesterol!

Appearance of "Toward Healthful Diets" unleashed a virtual Niagara of vitriol. The guidelines did not satisfy the zealots of cholesterol avoidance. Almost all discussion in the press focused on our omission of specific guidelines for cholesterol with few words devoted to the rest of the publication. We had ignored the popular belief that dietary cholesterol was equivalent to coronary disease. The gist of the newspaper stories was that we had sold out to 'Big Industry'. There was even a Herblock cartoon. We were attacked with a vehemence that only the truly righteous can summon.

There have been many changes of focus in the heart disease field since 1980. Coronary disease is now considered to appear as a result of injury or inflammation (a view first expressed by Virchow c. 1850). As you, and others have pointed out, (3,4) the average cholesterol responses to 100 mg/day change in dietary cholesterol are between 2.2 and 2.5 mg/dl. A report published by the Royal Society of Medicine Forum on Food and Health published in 2000 (5) concluded that it was no longer appropriate to view dietary cholesterol as a major cause of hypercholesterolemia and coronary heart disease and less stringency concerning cholesterol and intake was warranted.

Recognition of the importance of the presence of individual compounds such as choline, lutein, zeaxanthin polyphenols, etc. have made foods, originally shunned because of their fat content, desirable. A life-long dietary pattern appears to be the major factor in the development of diet related disease. One meal is a minor contributor. The only people who died after a single meal were those who dined with the Borgias.

Great advances are taking place in assessment of individual physiological response to specific nutrients and may eventually permit individualized nutritional treatments for specific problems. Every new observation leads to new hypotheses and technical approaches. Only our view of dietary cholesterol remains much as it did in 1950.

References:

1. McNamara DJ. Something Old, Something new (Nothing borrowed, nothing blue). *Nutrition Close-Up* 2006;23:7.
2. Food and Nutrition Board. "Toward Healthful Diets:" National Academy of Science, Washington, DC. 1980. 24 pp. Board members: AE Harper, Chair; H Kamin, Vice-Chair; RB Alfin-Slater; SH Chafkin; GK Davis; RL Hall; GC Harrison; V Herbert; OC Johnson; D Kritchevsky; RA Neal; RE Olson; GM Owen; WB Robinson; IH Rosenberg.
3. Howell WH, McNamara DJ, Tosca MA, Smith BT, Gaines JA. Plasma lipid and lipoprotein responses to dietary fat and cholesterol: a meta-analysis. *Am J Clin Nutr* 1997;65:1747-1764.
4. McNamara, DJ. Dietary Cholesterol and Atherosclerosis. *Biochim Biophys Acta* 2000;1529:310-320.
5. Gray J. Conference Report. Dietary cholesterol as a cardinal risk factor: myth or reality? *Nutrition Bulletin* 2000;25:365-367.

EDITOR'S COMMENT

I received the above letter from my long time friend and colleague, Dr. David Kritchevsky, on 8 November 2006. I am sad to report that David died 20 November 2006. Over the years I have had the good fortune to know some of the greats in the diet-heart disease field but no one ever combined the high standards, the skepticism, the personality, and the humor that Dr. Kritchevsky contributed to the field. I first knew Dr. Kritchevsky from his 1958 book, *Cholesterol*, which in 1966 was already a classic and must reading for those interested in cholesterol research. Whether it was research on dietary fat and heart disease and cancer risk, dietary fiber and health, effects of conjugated linoleic acid (CLA), or fostering directions for public health, David's contributions were abundant and often contrarian. To this productive and outstanding scientist David added a large dose of the comic one-liners so valued at conferences (well, appreciated by those who agreed with David while those on the other side felt more the sting of common sense). And if a piano was around you could be sure that David would delight the crowd with his repertoire of songs written especially for the diet-chronic disease crowd ("How are things in my aorta?", "I wish I had a big grant" to name just a couple, along with the cholesterol biosynthetic pathway song so useful to biochemistry students everywhere).

I will sorely miss David's phone calls when he had comments to make about the editorials in *Nutrition Close-Up*. I will miss his humor and charm and common sense so appreciated when we did have the chance to get together. The nutrition community has lost a leader who understood that science is important but people are more important, and that common sense isn't so common. He will be missed. ■

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

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