N U T R I T I O N

closeUP

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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 health care professionals can receive a **free** subscription for the newsletter by contacting the Egg Nutrition Center.

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Celebrating 25 years of nutrition research and health education (1979-2004)

Bioavailability of Lutein from Eggs

Part of a family of carotenoids thought to benefit eye health, lutein has shown exceptional potential for preventing and possibly treating age-related macular degeneration (AMD). Green vegetables such as spinach are commonly recommended for promotion of eye health because of their high lutein content. Egg yolk contains substantially less lutein, however, lutein from the yolk is thought to be more bioavailable than lutein from other sources because of the highly absorbable lipid matrix in which it is stored. Little research has been done to compare the bioavailability of this important carotenoid from egg yolk vs. other sources.

To address this question, Chung et al. recruited 10 healthy men to participate in a randomized cross-over study designed to compare the bioavailability of lutein from four common sources (egg yolks, spinach, lutein and lutein ester supplements). Blood was drawn two weeks before the study began and on days 1 (baseline), 2, 3, and 10 of each diet period to measure serum carotenoid content, cholesterol concentrations, and TAG levels. For an initial two-week period, participants were instructed to eat diets low in carotenoids to decrease serum levels and to establish a "true" baseline for each subject. Following this period, participants were randomized to one of four 9-day diet treatments [spinach, lutein-enriched egg yolk, lutein supplement (L), lutein ester supplement (LE)]. Each treatment was given once daily with the same test meal—an egg-white frittata—except the egg treatment, for which the frittata was made of three lutein-enriched whole eggs. Each dietary treatment was designed to provide 6 mg lutein/day except the LE supplement dose, which provided only 5.5 mg/day. As mentioned previously, egg yolk does not contain as much lutein as spinach; therefore, lutein-enriched eggs from hens fed a high-lutein diet were utilized to facilitate a fair comparison of bioavailability. Each participant completed all four dietary treatments with a two-week washout period between each treatment.

For each diet regimen, serum lutein increased from day 1 (baseline) to day 2. Serum lutein levels continued to increase steadily until day 10, when levels for all groups had increased significantly from baseline. The egg group experienced the largest increase and was the only group significantly different from the others by day 10. Lutein levels were similar for the spinach group and the L and LE supplement groups at the conclusion of the study. By day 10, serum cholesterol concentrations had increased to 223 ± 12 mg/dL from 183 ± 8 mg/dL at baseline for the egg group only

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Bioavailability, cont...

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(P<0.05). No increased TAG response was observed following the egg treatment.

TAG levels were measured to evaluate intestinal absorption of lutein. The triacylglycerol-rich lipoprotein (TRL) fraction was separated to help differentiate recently-absorbed lutein from endogenous stores. A previous study reported higher TG and TRL responses with high-cholesterol test meals, a condition that would be expected to result in higher lutein responses in these lipid fractions. The egg regimen in the current study did not result in higher TG levels; however, differences in serum lutein levels between groups remained significant after adjustment for serum cholesterol and TG concentrations.

The authors concluded that 1) lutein from egg yolk is more effective in raising serum lutein levels than lutein from spinach or vitamin supplements; 2) both supplement forms of lutein (esterified and non-esterified) raise plasma lutein to the same degree and are similar in effectiveness to spinach.

Although the eggs utilized in this study were enriched above their standard lutein content, the lutein in standard shell eggs comes from the same source (the hen's diet) and would be expected to offer the same high bioavailability as the lutein found in enriched eggs. Lutein-enriched shell eggs might be considered an excellent alternative to lutein supplements.

Because the study cohort was small and consisted only of men, the practical applications of these findings to the general population are limited. Further research is warranted with larger groups and with female cohorts to determine whether these conclusions might be generalized to a wider population.

Chung H, Rasmussen H, Johnson E. Lutein bioavailability is higher from lutein-enriched eggs than from supplements and spinach in men. *J Nutr* 2004;134:1887-1893.

Key messages

- Lutein from egg yolk is more effective in raising serum lutein levels than lutein from spinach or vitamin supplements.
- Both supplement forms of lutein (esterified and non-esterified) raise plasma lutein to the same degree and are similar in effectiveness to spinach.

Egg Intake and All-Cause Mortality in Japan

Eggs are a culturally ubiquitous food. They hold almost universal appeal, since many, if not all, cultures embrace some tradition involving preparation and eating of eggs. So far, a reliable body of research conducted in the United States has accumulated showing that there is no association between egg intake and heart disease risk in Americans. However, the effect of egg consumption on cardiovascular disease and CVD mortality is relatively unknown in other populations. To determine the effect of egg intake on serum cholesterol and all-cause mortality in an aging Japanese population, Nakamura et al. used information gathered in the National Integrated Project for Prospective Observation of Non-communicable Disease and Its Trends in the Aged (NIPPON DATA).

In this prospective study, subjects from randomly-selected health districts in Japan completed a survey of health, lifestyle, and dietary components and were followed for 14 years (1980-1994). Each participant completed a physical examination, medical history, blood testing, and a lifestyle questionnaire (including smoking status and drinking habits). Blood pressure, height, and BMI were measured at baseline and dietary intake information was obtained from a food-frequency questionnaire. Participants were asked to rank their usual egg consumption as follows: >2 eggs/day, ~1/day, ~1/2 /day, ~1-2/week, or seldom (<1 egg/week).

Only 1.3-6.1% of participants fell in the extreme categories of >2 eggs/day and <1 egg/week. The women in these categories were significantly older, on average, than those in the other categories of egg consumption. A positive dose-response relationship between egg consumption and age-adjusted total cholesterol was observed in the women (P=0.0001), while for the men, cholesterol levels were similar across all categories of intake. Multivariate adjusted all-cause mortality was higher for women in the 1 egg/day category (1.0) than for those in the 1-2 eggs/week category (0.78, P=0.02); however, relative risk of death from stroke, IHD (ischemic heart disease), and cancer were similar across consumption categories for women. All-cause mortality did not differ between women who reported eating 1 egg/day and those who reported eating >2 eggs/day. Relative risk of death (all-cause, IHD, cancer, stroke) did not differ across egg consumption categories for men.

Because lower all-cause mortality was observed in the 1-2 eggs/week group, the authors conclude that limiting egg consumption might provide some health benefits for women in this Japanese population; however, it is interesting to note that although higher egg intakes were associated with slightly increased plasma cholesterol concentrations, deaths from IHD were not similarly related. The authors acknowledge that the design of this study left ample room for inaccuracy and confounding factors. For example, the dietary information used to generate these results consisted of a single, unrepeated food frequency questionnaire completed 14 years before the conclusion of the study. Food frequency questionnaires can be effective in

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evaluating overall intake of certain nutrients, but the one used in this study did not provide data on intake of total energy, saturated fat, cholesterol, or polyunsaturated fat, all of which were potentially confounding dietary factors. In addition, the cause of death was unknown for nearly 50% of the participants in each egg intake group. Further research with larger participant numbers in the extreme categories of egg intake (>2 eggs/day and <1 egg/week) and more comprehensive dietary information is needed to clarify the relationship between egg intake and all-cause mortality in this demographic of Japanese women.

Nakamura Y, Okamura T, Tamaki S, et al. Egg consumption, serum cholesterol, and cause-specific and all-cause mortality: the National Integrated Project for Prospective Observation of Non-communicable Disease and Its Trends in the Aged, 1980 (NIPPON DATA80). *Am J Clin Nutr* 2004;80:58-63.

Egg Intake and LDL Atherogenicity

Extensive research in the area of cholesterol and heart disease has made it clear that saturated fat and *trans*-fat impact blood cholesterol levels to a greater extent than dietary cholesterol. Cholesterol intake has been shown to raise LDL cholesterol levels minimally (<1%) in most individuals who are not cholesterol sensitive. While LDL cholesterol has been shown to be an independent predictor of heart disease risk, a growing body of research is showing that all LDL particles are not created equal. Two predominant LDL particle subclasses exist. Pattern A is characterized by large, cholesterol ester-rich particles. Pattern B is characterized by small, dense particles. The smaller, denser LDL particles of the Pattern B subclass appear to enter more easily into the arterial wall, are more vulnerable to oxidation, and bind more readily to proteoglycans; thus, they are thought to be more atherogenic than their larger counterparts. It is thought that a predominance of the pattern B subclass increases CHD risk 3-fold and atherogenicity appears to rise with decreasing LDL diameter. It has been shown that dietary saturated fat and *trans*-fat raise LDL cholesterol levels. Dietary cholesterol has also been shown to raise serum cholesterol in some individuals, although to a lesser extent. Understanding the influence of dietary fat and cholesterol from different sources on LDL atherogenicity could prove valuable in medical nutrition therapy. Very little research has addressed the influence of dietary cholesterol from eggs on the atherogenicity of LDL particles.

To address this question, researchers at the University of Connecticut recruited 40 men and 51 premenopausal women to participate in a two-month dietary intervention trial evaluating the effects of egg consumption on plasma cholesterol and LDL particle characteristics. Baseline lifestyle factors (such as smoking status, physical activity, alcohol consumption, weight, and blood pressure) were measured prior to study initiation. Participants were randomly assigned to the egg or placebo regimen, which they adhered to for 30 days. At the conclusion of the 30 days, blood samples were obtained for each participant and lifestyle factors were again assessed for accurate interpretation of the blood lipid profiles. Following a 3-week washout period, each participant switched diet groups and completed another 30-day diet period on the egg or placebo regimen. During the egg regimen participants consumed the liquid equivalent of 3 eggs per day (adding 640 mg/d cholesterol to the background diet). During the placebo regimen, participants consumed an equal weight of cholesterol- and fat-free egg substitute. Participants were instructed to maintain a background diet <300 mg cholesterol, <30% calories from total fat, and <10% calories from saturated fat. Plasma lipids, CETP and LCAT (markers of reverse cholesterol transport), and insulin level were measured at baseline and following each 30-day treatment and the washout period.

Cholesterol responsiveness was classified according to the extent to which total plasma cholesterol changed with the additional cholesterol intake. An increase of 2.2-2.5 mg/dL (TC) was considered a normal response to a 100 mg increase in daily cholesterol intake. Individuals who experienced an increase of >2.5 mg/dL (TC) per 100 mg/d (translating into an increase of 16 mg/dL for the experimental group) were classified as hyperresponders. Those whose total cholesterol increased by <2.2 mg/dL per 100 mg additional cholesterol intake (or <14 mg/dL) were classified as hyporesponders.
According to 7-day food records, all participants adhered to the NCEP I guidelines when self-selecting food intake. Average cholesterol intake was 764 ± 67 mg/d during the egg regimen, significantly greater than the 167 ± 118 mg/d reported during the placebo period. Average percentages for total and saturated fat intakes were also higher during the egg period than the placebo period.

Researchers selected 28 (14 F, 14 M) hyperresponders and 26 (13 F, 13 M) hyporesponders for further evaluation of blood lipids and LDL subtype classification. Of these participants, 29 were found to have the pattern B (smaller LDL) phenotype, while 25 were found to have the pattern A (larger LDL) phenotype. LDL pattern phenotype did not change significantly for the group as a whole following the egg period, however, women were more likely than men to be classified with the type A phenotype (larger LDL particles). This is not surprising, since of the 91 participants who completed the study, 70% of men were classified as having the B phenotype vs. only 37% of women. In fact, the only significant difference in LDL particle phenotype was between male and female hyperresponders (females had larger LDL particles than males.) Regardless of response category or treatment regimen, women had higher

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CETP activity than men. Body mass index (BMI) measurements were higher for men than for women, as were insulin levels (13.0 ± 5.7 uU/L vs. 9.0 ± 7.3 uU/L). Insulin levels did not differ between treatment groups.

Hyperresponders experienced a statistically significant increase in LDL cholesterol levels following the egg regimen. There was no change in plasma LDL levels following either diet regimen for hyporesponders. Hyperresponders had significantly higher levels of the largest LDL subclass (p=<0.05) than hyporesponders following the egg regimen. Activity levels of CETP and LCAT were also elevated in hyperresponders following the egg regimen.

It has been shown in previous studies that size and composition are significant factors in determining the atherogenicity of LDL particles. The predominance of pattern B subclass LDL is associated with a 3-fold increase in the risk of CHD. Individuals with the more atherogenic pattern B subclass also appear more susceptible to the deleterious effects associated with low-fat/high-carbohydrate diets (higher TAG and lower HDL-C concentrations) and one study has suggested that low-fat/high-carbohydrate diets actually favor a predominance of smaller, denser, LDL particles.

The influence of dietary cholesterol on LDL particle atherogenicity is valuable in evaluating the appropriateness of current dietary guidelines. The data provided by this dietary intervention demonstrate that the influence of dietary cholesterol on LDL particle size (and thus, atherogenicity) is heavily modulated by sex differences and responsiveness to dietary cholesterol. Data from the present study indicate that additional dietary cholesterol (up to 640 mg/d) appears to have no effect on lipid levels or LDL atherogenicity in hyporesponsive individuals. The data further suggest that for hyperresponders, as LDL cholesterol levels increase, the LDL pattern subclass shifts toward larger, less-atherogenic particles. CETP activity levels also increased following the egg regimen. An increase in CETP activity (when not accompanied by decreasing HDL concentrations) is considered protective against atherogenesis. The authors conclude that egg intake is not contraindicated for healthy men and premenopausal women and does not increase LDL particle atherogenicity.

Herron K, Lofgren I, Sharman M, et al. High intake of cholesterol results in less atherogenic low-density lipoprotein particles in men and women independent of response classification. *Metabolism* 2004;6:823-30.

Fat Intake and CVD Risk in Type 2 Diabetes

Dietary fat has undergone, perhaps, one of the greatest paradigm shifts in nutrition history. The value of limiting total fat intake has been questioned, reviewed, and revised in recent years with increasing evidence that the various classes of fats play different—even contradictory—roles in atherogenesis. Researchers have long questioned the differential roles of dietary saturated fat, *trans*-fat, mono- and polyunsaturated fats and cholesterol on CVD risk. Many questions have recently been clarified by large, longitudinal, epidemiological studies, the major findings being that saturated and *trans*-fats have a greater impact on blood cholesterol levels and are more atherogenic than dietary cholesterol. These results have been inconsistent, however, in adult diabetic populations. Recent studies suggest that individuals with type 2 diabetes might have greater sensitivity to saturated fat, trans-fat, and cholesterol than nondiabetic adults in the population, putting them at higher risk for CVD. Hu et al. evaluated data from diabetic participants in the Nurses Health Study in an effort to clarify the effects of different dietary lipids on CVD risk within this demographic.

Of the 121,700 female nurses who participated in the Nurses Health Study (NHS) (from 1976-1996), 5,674 had type 2 diabetes mellitus at baseline (prevalent cases) or were diagnosed over the course of the study (incident cases). (Data from those women who had diabetes at baseline had been excluded from the original statistical analysis.) All participants were aged 30-55y with no history of myocardial infarction (MI), angina, coronary revascularization, stroke, or cancer. Food frequency questionnaires were used to obtain diet information from participants in 1980, 1986, 1990, and 1994. At baseline, each participant completed a questionnaire assessing medical history and lifestyle. This questionnaire was repeated every 2 years to monitor changes and document CVD events, including fatal CHD, nonfatal MI, and stroke. This cohort was followed for a total of 22 years (follow-up was extended to 1998).

Women were categorized into quintiles of fat intake (as % of total energy), cholesterol intake (mg/1000 kcals), ratio of polyunsaturated to saturated fat (P:S), and the Keys score (expressing the predicted magnitude of change in serum cholesterol based on saturated fat, polyunsaturated fat, and cholesterol intake). Multivariate analysis included total caloric intake, percentage of energy derived from protein and fats, along with potentially confounding factors such as alcohol intake, smoking status, family history of MI, vitamin E supplementation, multivitamin consumption, intake of dietary fiber, physical activity, diabetes medication, BMI, and postmenopausal status.

Age-adjusted multivariate analysis (including adjustment for other dietary lipids) showed that saturated fat (P for trend=0.03) and cholesterol intake (P for trend=0.01) were positively related to CVD risk, while *trans*-fat and polyunsaturated fat intake were not. It was

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estimated that replacing 5% of calories from carbohydrates with equivalent energy from saturated fat would increase risk by 29% (P=0.003). Additionally, according to these data, increasing cholesterol intake by 200 mg/1000 kcals would be expected to increase risk by 37% (RR: 1.37; P=0.003). It should be noted that for a woman with a daily intake of ~2000 kcals, the increase in dietary cholesterol would be 400 mg per day. In this study population, the highest quintile of cholesterol intake was 596 mg/2000 kcals, the only quintile with a significantly increased risk.

Conclusions drawn from the Health Professionals Follow-up Study and the Nurses' Health Study indicate that for healthy men and women, egg intake is not associated with CVD risk. More specifically, men and women who reported eating at least 1 egg/day had the same CVD risk as those who reported eating <1 egg/week. Therefore, egg intake should not be contraindicated for healthy men and women. Questions remained, however, with regard to the influence of egg intake on CVD risk in diabetic participants. This was the only group for which CVD risk increased across rising quintiles of reported egg consumption (*JAMA* 1999;281:1387-1394). The authors concluded that, "Most epidemiological studies have not found a significant association between dietary cholesterol and CVD risk in the general population. However, dietary cholesterol may be more detrimental to diabetics than to nondiabetics." The researchers remain uncertain of the mechanism that might increase risk to those with diabetes, but they hypothesized that it might be due to "heightened insulin resistance and dyslipidemia among diabetic patients."

These data suggest that diabetic women might be more sensitive to dietary saturated fat and cholesterol than non-diabetic women. Increased intakes of saturated fat and cholesterol are associated with greater CVD risk in this population. These results also indicate that replacing saturated fat with monounsaturated fat might be more effective than replacing saturated fat with carbohydrates in reducing CVD risk.

Tanasescu M, Eunyoung C, Manson J, Hu F. Dietary fat and cholesterol and the risk of cardiovascular disease among women with type 2 diabetes. *Am J Clin Nutr* 2004;79:999-1005.

Early-Pregnancy Protein Intake and Size at Birth

Healthy babies start with healthy pregnancies. While extensive research has shown the important role of specific micronutrients in pregnancy outcomes (folic acid supplementation, for example, has been shown to dramatically reduce the incidence of neural tube defects), little is known about the influence of macronutrient ratios during pregnancy. It has been suggested that in babies born small for gestational age, physiological adjustments made by the fetus to compensate for insufficient maternal calorie or nutrient intake predispose the unborn child to adult-onset disorders such as type 2 diabetes and cardiovascular disease. The influence of differing intakes of carbohydrate, protein, and fat during pregnancy has not been thoroughly evaluated.

To address this question, Moore et al. studied the early- and late-pregnancy macronutrient intakes of 557 pregnant Caucasian women between the ages of 18 and 41. A total of two dietary interviews were conducted using semiquantitative food frequency questionnaires, the first prior to 16 weeks gestation (early pregnancy) and the second between wk 30 and 34 of pregnancy. During the first interview, height and weight were recorded for each expectant mother. During the second, weight was recorded a second time to determine the extent of maternal weight gain. At birth, each baby was weighed and measured. A ponderal index was calculated based on weight and length. The placenta was also weighed at birth.

Data from 429 cases were considered reliable (based on complete birth data and realistic reported dietary intake) and were used for further evaluation. After adjustment for gestational age and potential confounding factors such as maternal age, height, prepregnancy weight, primiparity (first birth), smoking, alcohol consumption, and recreational drug use, the percentage of total calories from protein during early pregnancy (<16 weeks gestation) was positively associated with birth weight, placental weight, and ponderal index (P<0.05). There was a negative correlation between carbohydrate intake and ponderal index (P=0.04). Neither maternal weight gain, nor prepregnancy BMI had a significant influence on the results. Nutrient intake in late pregnancy did not appear to have any significant influence on birth characteristics.

The authors concluded that protein intake in early pregnancy is positively associated with birth size and that this association is independent of both total energy intake and maternal weight gain. They conjecture that early pregnancy might be a nutritionally critical time since the placenta develops during this period. Previous studies have shown detrimental effects of very high protein intake during pregnancy, indicating that the benefits of higher protein consumption may be lost beyond a certain intake level. While these data must be interpreted carefully, they shed important light on a unique situation in which macronutrient composition seems to exert an influence independent of total energy intake.

Moore V, Davies M, Willson K, et al. Dietary composition of pregnant women is related to size of the baby at birth. *J Nutr* 2004;134:1820-1826.

Vitamin D Deficiency Associated with Diabetes Risk

Although vitamin D is known mainly for its role in calcium metabolism, insufficient intake of this important nutrient has been associated with the development of type 1 diabetes, hypertension, and even some cancers. Although the association between vitamin D deficiency and diabetes is not clear, the mechanism appears to be related to insulin sensitivity and possibly to ß cell function. To clarify this relationship, Chiu et al. examined the influence of plasma vitamin D concentrations on insulin sensitivity and ß cell function in a glucose-tolerant adult population.

Potential participants were screened for abnormal glucose tolerance and hypertension, after which 126 healthy, glucose-tolerant, normotensive participants were enrolled to participate in the study. Seventy three women and 53 men made up this diverse study cohort, which was 27% Asian American, 8.7% African American, 42.9% white, and 21.4% Mexican American. Average age for participants was 26 ± 6y, BMI was 24.7 ± 4.2, and waist-to-hip ratio was 0.795 ± 0.071. Plasma glucose, insulin, and lipid concentrations were determined for each participant, along with fasting plasma levels of vitamin D (25(OH)D). Participants with 25(OH)D less than 20 ng/mL were considered vitamin D deficient.

The hyperglycemic clamp technique was used to evaluate insulin sensitivity and ß cell function. Each participant underwent a continuous infusion of a dextrose solution adjusted to maintain serum glucose at 180 mg/dL over the course of 2 hr. A first-phase insulin response (1stIR) was measured based on plasma insulin concentrations during the first 10 min. of infusion. A second-phase insulin response (2ndIR) was measured based on serum insulin concentrations during the last hour of the clamp process. Insulin sensitivity index (ISI) was calculated based on average plasma insulin and average glucose infusion rates obtained during the last hour of the insulin clamp study. ß cell function was evaluated indirectly based on 1st and 2nd IR.

Vitamin D levels fell below the 20 ng/mL mark for 47 (37%) of the 126 participants. Vitamin D status was associated with ethnic group, with 54% of African Americans, 47% of Asian Americans, 41% of Mexican Americans, and 26% of white participants categorized as deficient.

Participants at risk for the metabolic syndrome were defined as those who had two or more of the following risk factors: waist circumference >102 cm (men) or >88 cm (women); serum triacylglycerol concentration >150 mg/dL; HDL-cholesterol concentration <40 mg/dL (men) or <50 mg/dL (women). (Plasma glucose concentration >110 mg/dL and blood pressure >130/85 mmHg are also considered risk factors, however, potential participants who were hypertensive or hyperglycemic were excluded from this study.) Of those categorized as vitamin D deficient (47), 14 (30%) were at risk for the metabolic syndrome, vs. only 9 (11%) of those with normal 25(OH)D levels. This suggests that vitamin D deficiency might play a role in the development of the metabolic syndrome.

Vitamin D status was also associated with BMI. Ethnicity (P=0.0086) and BMI (P=0.0033) accounted for 15% of the variance in 25(OH)D concentration. Vitamin D status was an independent predictor for BMI, with lower vitamin D levels being associated with higher BMI (r=-0.2517). 25(OH)D concentrations were inversely associated with fasting plasma glucose concentrations and glucose levels at 60-, 90-, and 120-minutes postchallenge, indicating a negative effect of low circulating 25(OH)D on ß cell function. There was a positive correlation between 23(OH)D concentration and ISI (P<0.0001, r= 0.4600). In combination, 25(OH)D, sex, BMI, diastolic blood pressure, age, and ethnicity accounted for 42% of the variation in ISI. 1st and 2nd phase insulin responses (1st and 2nd IR) were inversely related to ISI (P<0.0001, r=-0.5860; P<0.0001, r=-0.7612), and thus, to 25(OH)D concentrations. Vitamin D level was an independent predictor for BMI, ISI, and serum glucose concentrations at baseline (fasting) and 60, 90, and 120 minutes.

These findings support those of previous studies showing vitamin D deficiency to be associated with insulin resistance and reduced ß cell function. Although a great deal of animal research is already available showing the importance of vitamin D for normal insulin secretion, the observations of Chiu et al. shed new light on the role of vitamin D in glucose metabolism and disease risk. The authors hypothesize from these data that increasing circulating vitamin D from 10 to 30 ng/mL might improve insulin sensitivity by 60%, which could be sufficient to reduce the burden on ß cells and restore normal glucose tolerance. They also suggest that vitamin D therapy might be more effective than either metformin or troglitazone, two common pharmaceuticals used to improve insulin sensitivity. The potential of vitamin D to improve insulin sensitivity and ß cell function warrants further research in human populations.

Chiu K, Chu A, Go V, Saad M. Hypovitaminosis D is associated with insulin resistance and ß cell dysfunction. *Am J Clin Nutr* 2004;79:820-825.

Editorial: *The Conflicts of Science: What's Bad is Maybe Good, and What's Good is Maybe Bad.*

Amazing how an eight-page newsletter can have four reports at one time about eggs, dietary cholesterol and health, with opposite conclusions. The perfect example of why media loves researchers, something controversial to talk about, and consumers hate them, because they never seem able to make up their minds or state a simple conclusion. Five years ago a major study from the Harvard School of Public Health reported that CVD risk was the same whether one ate less than one egg a week or an egg a day (Hu et al. JAMA 1999;281:1387-1394). So naturally, someone had to publish a study that says the opposite (or else the media would get bored with too much good news).

Of the six research publications reviewed in this issue of Nutrition Close-Up, two can be considered positive towards eggs and two are negative. Pretty much the way a lot of research ends up, some good news, some bad news. The consumer's perspective pretty much depends on which ones get the media attention. In these cases, the headlines were "An egg a day may be too much for some women," "New research shows that eggs may provide best bioavailable source of lutein" and "Eggs have no impact on dangerous cholesterol." Take your pick and draw your conclusions.

Battling headlines is nothing new (think presidential election) but it sure can be confusing. Do eggs cause harm or health? Should I or shouldn't I? And the scientific community (very much like the political community) seems indisposed to presenting anything within context. If it fits my theories I agree, if not, I disagree, and please do not confuse me with data. So let us take the studies and consider the data.

A few uncertainties in the study by Nakamura et al. need consideration before we conclude that "an egg a day may be too much for some women."

1. Dietary data were collected once in 1980 via a self-administered food frequency questionnaire. Can one assume that the dietary patterns remained constant over the subsequent 14 years of the study (1980-94)?

2. Within the context of the dietary analyses, there was no adjustment for total caloric intake, saturated and polyunsaturated fat intake, or other sources of dietary cholesterol. Other studies indicate that while plasma cholesterol levels increased significantly in this population between 1980 and 1989, there was a decrease in the mortality rate from coronary heart disease.

3. The available mortality data for stroke, IHD, and cancer account for only 52% of deaths. 48% of the deaths were due to unknown causes. The finding that total, but not cause-specific deaths, were related to egg consumption leaves unclear what relationships exists between egg consumption and mortality.

4. The actual values for deaths per 1000 patient years (TPY) in the various egg consumption groups were virtually identical, as were the age-adjusted relative risks. It is only after multivariate analysis with correction for multiple variables (including, for unknown reasons, total cholesterol levels) that a significant effect is noted.

5. It is unclear from the patient classification pattern how patients were defined since one group is > 2 eggs/day (n=69) and the next group is 1 egg/day (n=1393). What was the classification for those whose weekly egg consumption was between 7 and >14 per week?

6. The relative risk values for those who ate an egg a day, half an egg a day or seldom ate eggs were the same; only those eating 1-2 per week had a lower relative risk. The absence of a dose-response relationship is concerning.

7. None of these findings were observed in the male study participants. Are eggs a problem only for Japanese women?

8. The findings are in conflict with those of Hu et al. (JAMA 1999) in over 80,000 women followed for 14 years with multiple food frequency analyses over the follow-up period and statistical adjustments for saturated fat intake.

9. If egg intake was related to increased serum cholesterol in this cohort, then why was an elevated incidence of heart disease not observed in the 1 egg per day group? And why should any effect on mortality occur exclusively in women?

The report by Tanasescu et al. is consistent with data from this group's earlier (JAMA 1999) published analysis of egg intake and heart disease risk — egg intake increases heart disease risk in patients with diabetes. Whether this is due to diabetes-induced changes in lipid and lipoprotein metabolism is unclear. (Were any of the women in the Nakamura et al. study diabetic?)

Herron et al. reported data from an outpatient study showing that egg intake does not have a negative impact on the plasma lipoprotein profile. Even in subjects who do experience an increase in their LDL levels with egg intake, it is the less atherogenic particles that increase along with HDL cholesterol levels. As reported by Chung et al., egg intake does increase another lipid in the plasma, namely lutein, which is considered beneficial in the promotion of eye and skin health and has been associated with atherosclerosis prevention and decreased risk for some

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cancers. Less risk, more benefit.

So are eggs good, bad or indifferent? The evidence is becoming more persuasive that a high cholesterol intake (including eggs) can have a negative impact on the CVD risk profile of those with diabetes. At the same time, emerging evidence of increased lutein bioavailability speaks to the benefits of egg consumption; and there is little if any compelling evidence that egg intake increases CVD risk. In the end, all we can say is what has been said so many times before — "more research needs to be done."

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