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closeUP

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INSIDE

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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)

Eggs, Oats, and Endothelial Function

Regular egg intake has long been thought to increase cardiovascular disease (CVD) risk despite a growing body of evidence to the contrary. Recent large epidemiological studies have found no difference in CVD risk between participants who reported eating one egg per week and those who reported eating one per day. Endothelial function, defined as the ability of an artery to respond appropriately to changes in arterial flow and vasopressure, is a widely-accepted marker of cardiovascular health. Poor endothelial function is often a predictor of coronary heart disease and is associated with other CVD risk factors. Because endothelial function can represent the collective influence of a number of risk factors (including serum lipids) on coronary health, it is used extensively to evaluate the impact of various foods and nutrients on CVD risk. In a recent study, Katz et al. investigated the differential effects of regular consumption of oatmeal and eggs on flow-mediated dilation (FMD) to determine their influence on endothelial function.

Fifty healthy adults (31 men, 19 women) were recruited to participate in this randomized, single-blind, crossover study. To equalize baseline risk among all participants, male subjects were required to be over age 35 and all women were postmenopausal (none were using hormone replacement therapy). Participants were non-smokers with no known CVD or hypercholesterolemia. Potential subjects who were taking vasoactive medications or using high-dose vitamin E or fiber supplements were excluded from the study. Although the participants could not be blinded to treatment assignment, the ultrasonographer did not have access to this information. Since coronary endothelial function is highly correlated with peripheral measurements, the researchers chose to assess endothelial function using brachial artery reactivity studies (BARS), performed by noninvasive ultrasonography.

Following an overnight fast, participants presented for baseline measurements of serum lipids, body weight, and BARS. Participants returned for the subsequent three weeks for baseline postprandial BARS to measure the acute effect of oatmeal and egg intake on vascular reactivity. BARS were performed after ingestion of oatmeal, eggs, and a sausage and cheese breakfast sandwich to compare the two treatments with the effects of a meal high in saturated fat. (Oatmeal was determined in a previous study to improve endothelial function in adults following a high-fat meal challenge.)

Participants were randomly assigned to begin a 6-week treatment period of daily consumption of either 2 eggs or 60 grams of uncooked whole oats. The

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eggs and oats could be prepared using any method, but participants were encouraged to consume them at breakfast. Following the 6-week treatment period, participants fasted overnight and were again admitted to the laboratory for BARS, blood lipid assessment, and weight measurement. BARS consisted of a baseline scan followed by ingestion of 2 hard-cooked eggs or plain oatmeal and more BARS measured 3 hours postprandially. Following a 4-week washout period, participants switched diet assignments and began another 6-week treatment period, after which identical assessments were performed to measure vascular reactivity, serum lipid changes, and body weight.

No statistically significant differences were detected in FMD following acute ingestion of oats, eggs, or sausage/egg sandwich at baseline. Following 6 weeks of egg consumption, there were no significant changes from baseline in pre- or postprandial FMD. Results were similar following the six-week oat challenge, with no significant difference in endothelial function between treatment groups.

With regard to serum cholesterol levels, there were no significant differences in total or LDL cholesterol levels from baseline following six weeks of egg treatment. Following six weeks of oat treatment, total and LDL cholesterol levels had decreased from baseline by 4.8% and 6.6%, respectively. There were no differences between treatment groups on any other lab measurements, including BMI, TAG levels, HDL cholesterol, and systolic blood pressure.

Although regular oat consumption had no apparent influence on endothelial function based on measurements of FMD, eating oats improved serum lipid profiles significantly. Following six weeks of the oat diet treatment, participants experienced a decline in total cholesterol from 203.8±31.5 to 194.0±30.5 mg/dL (P = 0.0017) and in LDL cholesterol from 124.8±25.0 to 116.6±30.8 mg/dL (P = 0.0006). These results support the thought that oat consumption reduces CVD risk by improving blood cholesterol levels.

The authors conclude that although short-term, this research makes some clear statements about regular egg consumption: 1) that six weeks of regular consumption of two eggs per day has no impact on serum cholesterol levels; and 2) that short-term, regular egg consumption does not have an adverse effect on endothelial function. Although further long-term studies will be necessary to confirm the influence of regular egg consumption on serum lipids and endothelial function, the results of this short-term investigation support epidemiological observations that egg intake, independent of dietary fat, is not associated with increased occurrence of CVD. These data also support clinical observations that egg consumption has little or no impact on serum cholesterol levels.

Katz DL, Evans MA, Nawaz H, et al. Egg consumption and endothelial function: a randomized controlled crossover trial. *Int J Cardiol* 2005;99:65-70.

Key messages

- Flow-mediated dilation remained stable following daily consumption of 2 eggs or 60 g whole oats over a 6-week period. Neither the egg breakfast, nor the oatmeal breakfast had any effect on endothelial function.
- Daily consumption of 2 eggs for breakfast for a period of 6 weeks did not raise total or LDL cholesterol levels.
- Total and LDL cholesterol levels were reduced significantly following 6 weeks of oat consumption.

COMMON ABBREVIATIONS

BMI: body mass index (kg/m^2)
CHD: coronary heart disease
CHO: carbohydrate
CVD: cardiovascular disease
HDL: high density lipoprotein
LDL: low density lipoprotein
Lp(a): lipoprotein (a)
MUFA: monounsaturated fatty acids
PUFA: polyunsaturated fatty acids
PVD: peripheral vascular disease
RR: relative risk
SFA: saturated fatty acids
TAG: triacylglycerol
VLDL: very low density lipoprotein

Choose Your Diet, Pick Your Poison

In today's climate of "extreme" makeovers, invasive body sculpting, and one-size-fits-all diet marketing, healthcare professionals have been justifiably outspoken against less-traditional, commercial weight-loss diets. The lack of research supporting the efficacy and long-term safety of such regimes has caused many to wonder whether effective methods are safe, and whether safe methods are likewise effective. Although most of these "fad" diets (as the title so aptly implies) disappear not long after they are launched, a few have stood the test of time. The Journal of the American Medical Association (JAMA) recently published the first side-by-side comparison of four of the most important commercial contenders—the Atkins diet, the Ornish diet, the Zone diet, and the more traditional Weight Watchers eating plan. With weight loss at 1 year as the main outcome measure, the four diet plans were also compared with respect to changes in heart disease risk factors, adherence rates, and short-term safety.

Dansinger et al. recruited 160 obese adults between the ages of 22 and 72 to be randomized to one of four popular diet regimens (n=40 for each diet group). Adults with BMI >27 and <42 who also had at least one cardiac risk factor (elevated fasting glucose, dyslipidemia, high blood pressure, or on medications to treat such symptoms) were qualified to participate in the study. Participants were randomly assigned to follow one of four popular diets (Atkins, Ornish, Zone, or Weight Watchers) for a period of two months.

Participants assigned to the Atkins diet were instructed to consume <20 g carbohydrate daily, gradually increasing to 50 g/day. Those assigned to the Zone diet were to consume a macronutrient balance of 40% carbohydrate, 30% fat, and 30% protein. Those on the Ornish regimen were instructed to consume a vegetarian diet containing no more than 10% of calories from fat, while those following the Weight Watchers plan were to stay within a pre-determined point limit (each point being worth ~50 calories) based on body weight (24-32 points/day for most).

During the initial 2-month period, participants met in small classes to receive diet instruction and support, including educational materials and the official diet cookbooks. They were encouraged to adhere to their assigned diet to the best of their ability during this initial 2-month period, after which they were instructed to follow the diet according to their level of interest. All participants were encouraged to take a daily multivitamin, to exercise at least 60 minutes per week, and to avoid outside commercial support.

Follow-up assessments were conducted at baseline, 2, 6, and 12 months, in which blood samples were obtained for measurement of serum total cholesterol, HDL cholesterol, triglycerides, glucose, insulin, high-sensitivity C-reactive protein, and creatinine levels. LDL-cholesterol was calculated. Total protein, nitrogen, and creatinine levels were also monitored.

Participants in the four groups did not differ significantly with respect to baseline demographics, weight measures, CVD risk factors, or serum glucose, insulin, cholesterol, or C-reactive protein levels. Although there were no significant differences between any of the diets in terms of attrition rates at one year, there was a tendency toward higher discontinuance rates among participants assigned to follow the more extreme diet regimens (P=0.08; 48% for the Atkins diet and 50% for the Ornish diet) as compared to the more traditional diets (35% for the Zone diet and 35% for Weight Watchers.) No adverse events were reported and there was no evidence of renal impairment for any of the participants.

At the one-year follow-up, all four of the diets had resulted in a significant reduction in daily calorie intake (P<0.05; 138 for Atkins, 251 for Zone, 244 for Weight Watchers, and 192 for Ornish) which did not differ significantly between diets (P=0.70). Similarly, all diets resulted in statistically significant weight reductions at one year compared to baseline, but the amount of weight lost did not differ between diets (P=0.40). Changes in body weight and waist size were similar between men and women (P = 0.30). Based on self-report, adherence decreased similarly over time for each of the four groups. Approximately 25% of participants in each group had maintained an effective level of adherence throughout the study, resulting in a sustained 1-year weight reduction of >5% baseline body weight, while 10% had reduced their weight >10% from baseline. Weight loss was strongly associated with self-reported dietary adherence (r=0.60; P<0.001). Exercise levels increased for participants in all diet groups (P<0.05) and did not differ between groups.

Cardiac risk factors had improved at 1 year for all diets. C-reactive protein levels had decreased by ~15% to 20% for all diets at the 1-year follow-up (statistically significant for all but the Zone diet; P=0.09). Changes in total/HDL cholesterol ratio, C-reactive protein, and insulin levels mirrored changes in weight and were similar for all diets.

In conclusion, all four of the dietary regimens evaluated in this study resulted in statistically significant weight loss and improvements in cardiac risk factors. The fact that the diets did not differ from each other in terms of the extent of these changes supports the concept that weight reduction, regardless of the dietary means of achieving it, is effective in improving cardiovascular disease risk. Adherence to positive dietary changes along with increased physical activity appear to be the key components in achieving and maintaining weight loss.

Dansinger ML, Gleason JA, Griffith JL, et al. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction. *JAMA* 2005;293:43-53.

Saturated Fat Intake Slows Atherogenesis in Postmenopausal Women

Cardiovascular disease is the leading cause of death among women, claiming nearly half a million lives each year. Despite the results of multiple studies showing that dietary changes affect men and women differently, the quest for optimal nutrient intake for reducing CVD risk has traditionally focused on men. In addition, nutrition guidelines for reducing CVD risk have concentrated on decreasing saturated fat intake, but have not taken into account the differential effects of replacing saturated fat with carbohydrate, protein, polyunsaturated fat, or monounsaturated fat. To address this issue, Mozaffarian et al designed an investigational study to examine the effects of dietary macronutrient intake on 3-year atherosclerotic progression in postmenopausal women.

Data for this analysis were taken from a study originally designed to assess the effects of hormone replacement on the progression of coronary atherosclerosis. The participants included in this study were postmenopausal women not receiving hormone replacement therapy who had at least one coronary stenosis (>30% reduction in luminal diameter). Women provided information on health status, medical history, and physical activity, and underwent lab testing including quantitative coronary angiography and serum lipoproteins at baseline. Food frequency questionnaires were also completed at baseline to assess nutrient intake.

Coronary angiography was repeated after an average of 3.1 years. The primary outcome assessed in this study was change in coronary luminal diameter at follow-up. Statistical models allowed for the evaluation of isocaloric macronutrient substitution. Total fat intake was low in this population, possibly as a result of dietary modification due to known pre-existing coronary disease. Although current smokers were more likely to have a higher intake of saturated fat (SFA), SFA intake was correlated to higher HDL, HDL_2, and HDL_3 concentrations, higher apo A-I, lower TG concentrations, and lower ratios of total to HDL cholesterol.

With regard to luminal diameter, higher intakes of SFA were associated with slower progression of coronary atherosclerosis. This observation remained significant after adjustment for potentially confounding demographic and lifestyle factors. Participants in the first quartile of intake experienced a 0.22 mm decline in luminal diameter, while those in the second and third quartiles experienced decreases of 0.1 mm and 0.07 mm, respectively (P=0.002). Participants in the highest quartile of SFA intake experienced an average increase of 0.01 mm in luminal diameter (P<0.001). Expressed as percentages, participants in the lowest quartile of SFA intake experienced a mean increase in luminal narrowing of 8.0%, while those in the second, third, and fourth quartiles experienced mean increases of 3.6%, 2.7%, and 0%, respectively (P for trend = 0.002).

Both polyunsaturated fat (P for trend = 0.04) and carbohydrate (P for trend = 0.001) were associated with atherosclerosis progression. Compared to the lowest quartile of carbohydrate intake, the highest quartile was associated with a 19 mm greater decline in luminal diameter. When replacing SFA, carbohydrate was associated with greater decreases in luminal diameter (P = 0.04) and approached significance in its association with atherosclerotic progression when replacing monounsaturated fat. Notably, higher carbohydrate intake was associated with atherosclerotic progression only in women whose reported dietary intake yeilded an average glycemic index ≥55.1 (above the median). There was no significant association in women for whom the average glycemic index value fell below the median (≤55.1). Monounsaturated fat, total fat, and protein intakes were not associated with atherosclerosis progression.

The association of SFA intake with slowed progression was stronger in women who reported a lower intake of monounsaturated fat (P for interaction = 0.04); however, the association was weaker in women taking lipid-lowering medication (P for interaction = 0.008). Changes in BMI, blood glucose, or serum lipid levels over the course of the study did not affect the observed associations.

The major SFAs are lauric, myristic, palmitic, and stearic. Stearic acid is the largest single contributor, comprising ~25% of total SFA intake. When the data were analyzed based on type of SFA, each 1% increase in calorie intake from stearic acid was associated with a 0.11 mm smaller decline in luminal diameter, while each 1% increase in calorie intake from lauric, myristic, or palmitic acid was associated with a 0.05 mm smaller decline in luminal diameter.

According to the associations observed in this study, atherosclerotic progression in postmenopausal women appears to be promoted by carbohydrate and polyunsaturated fat intake and slowed by higher consumption of SFA. Although they do not correspond with current dietary recommendations, these observations do not necessarily contradict the findings of previous research. Results from the Nurses' Health Study (the only large-scale study to evaluate the influence of dietary lipids on CHD risk in women) indicated that after adjustments for polyunsaturated fat intake along with other nutrients, SFA intake was not significantly associated with CHD events in healthy pre- and postmenopausal women. The authors speculate that these results might

have mirrored those of the current study if atherosclerotic progression (instead of CHD events) had been monitored and if the subjects had been postmenopausal women with some degree of coronary stenosis. The authors conclude that SFA intake slows the progression of atherosclerosis in postmenopausal women with existing coronary stenosis, and that this effect may be even more pronounced when the background diet is high in carbohydrates (especially refined carbohydrates) or low in monounsaturated fat. Further research in this area is clearly needed to replicate and expand upon these findings.

Mozaffarian D, Rimm EB, Herrington DM. Dietary fats, carbohydrate, and progression of coronary atherosclerosis in postmenopausal women. *Am J Clin Nutr* 2004;80;1175-84.

Influence of Lifestyle Factors on C-Reactive Protein Levels

Elevated serum lipids are not the only indicators of increased heart disease risk. High serum LDL cholesterol, a long-time foe, now shares the spotlight with low HDL cholesterol concentrations, high plasma homocysteine, smoking, high blood pressure, physical inactivity, diabetes, and the metabolic syndrome as harbingers of heart disease risk. Discovered in 1930, C-reactive protein (CRP) is a relative newcomer on the CVD risk factor scene. Whether CRP is an independent risk factor for CVD has not yet been established; however, the majority of research studies suggest that elevated CRP levels are associated with increased risk for heart attack, even among individuals with normal serum lipids. Thus, evaluating lifestyle components that influence CRP levels is potentially critical as it relates to CVD prevention.

To address this issue, Fredrikson et al. investigated the association between CRP levels and various lifestyle and dietary factors. Data from 760 individuals (342 men and 418 women), ages 49-70, were utilized in this study. Diet history, alcohol consumption habits, smoking status, physical activity, and information on education level and occupation were obtained at baseline. Blood pressure, serum lipids, and CRP levels were also measured at baseline.

In general, higher BMIs were associated with higher CRP levels, with a 40% difference between those with BMIs >27.6 kg/m^2 and those with BMIs below 23.1 kg/m^2. Participants with type 2 diabetes (P=0.005) and high total cholesterol/HDL cholesterol ratios also had higher CRP concentrations. HDL cholesterol levels were inversely related to CRP levels. Participants with HDL concentrations above 62 mg/dL had CRP levels 88% lower than those with HDL concentrations below 42 mg/dL. CRP levels were 40% higher for smokers compared to non-smokers/former smokers.

Alcohol consumption, physical activity, and dietary intake of total energy, total fat, saturated fat (SFA), monounsaturated fat (MUFA), polyunsaturated fat (PUFA), PUFA/SFA ratio, omega-6, and omega-3 fatty acids were not significantly related to CRP concentration in the participant group as a whole. When women were analyzed separately, lower CRP levels were weakly associated with higher intakes of total fat ($r = -0.13$, $P = 0.011$), SFA ($r = -0.13$, $P = 0.011$), MUFA ($r = -0.13$, $P = 0.010$), PUFA ($r = -0.14$, $P = 0.007$), and n-3 PUFA ($r = -0.14$, $P = 0.004$). Low intakes of vitamin C were related to higher CRP levels. A weak, inverse association was observed between CRP and intake of total carbohydrates ($r = -0.07$, $P < 0.05$); however, when differentiated from total carbohydrate, no relationship was detected between sugar intake and CRP.

Based on these observations, the researchers conclude that CRP levels are more consistently associated with markers of the metabolic syndrome (history of type 2 diabetes, high BMI, dyslipidemia) than with dietary or lifestyle factors. This observation supports the findings of previous studies demonstrating the anti-inflammatory effects of HDL and the proinflammatory effects of high triglycerides and hyperglycemia. Among the lifestyle factors evaluated, smoking appeared to be the most influential. Any observed associations between CRP levels and dietary intake were weak, if significant, but may have been stronger with a larger study population.

Fredrikson G, Hedblad B, Nilsson J, et al. Association between diet, lifestyle, metabolic cardiovascular risk factors, and plasma C-reactive protein levels. *Metabolism* 2004; 53:1436-1442.

Alpha-linolenic Acid and Vascular Inflammation

Omega-3 fatty acids, especially those from fatty fish, have long been promoted as key ingredients in a cardioprotective diet arsenal. These long-chain fatty acids have been shown to suppress the production of proinflammatory cytokines, inhibit lymphocyte proliferation, and to down-regulate gene expression of VCAM-1, E-selectin, and ICAM-1 (serum cell adhesion molecules thought to participate in the formation of atherosclerotic lesions). Proinflammatory endothelial responses are receiving greater attention as vascular inflammation is increasingly seen as a major factor in atherogenesis. Alpha-linolenic acid (ALA) is a plant-derived omega-3 fatty acid that has shown some promise as a cardioprotective agent; however, whether ALA is instrumental in reducing inflammation and attenuating pro-atherogenic pathways has not been established.

To evaluate the influence of ALA on vascular inflammatory markers, serum lipids and lipoproteins, and CVD risk factors, Zhao et al. administered a controlled feeding, crossover study in which 20 men and 3 postmenopausal women with moderate hyper-cholesterolemia were randomly assigned to follow one of three diets for a period of six weeks: an average American diet (AAD); a diet high in PUFA and ALA (ALA diet); or a diet high in PUFA and linoleic acid, an omega-6 fatty acid thought to play a role in promoting vascular inflammation (LA diet). The fatty acid composition of the AAD, which served as the control diet, was 13% saturated fat (SFA), 13% monounsaturated fat (MUFA), and 9% polyunsaturated fat (PUFA). The ALA and LA diets, both containing ~16-17% total energy from PUFA, contained ~8% SFA. The LA diet contained 12.6% LA and 3.6% ALA, having an LA:ALA ratio of 4:1, while the ALA diet contained 10.5% LA and 6.5% ALA, reducing the ratio to 2:1. All three of the experimental diets contained comparable amounts of MUFA (13%; from walnuts and walnut oil), 35% fat, 50% carbohydrate, 15% protein, and 300 mg cholesterol. Flaxseed was used as a primary source of omega-3 fatty acids in the ALA diet. Following each 6-week diet period, fasting blood draws were taken. Participants had a <3 week washout period between each diet assignment.

Serum levels of specific fatty acids indicated that compliance to the assigned diets was good. CRP levels, which indicate the presence of inflammation, were lower following both the ALA and LA diets compared to the control diet (75% lower for ALA; $P<0.01$ and 45% lower for LA; $p<0.08$). Interestingly, participants with lower CRP levels experienced greater decreases in LDL cholesterol concentrations following the ALA and LA diets (compared to control) than those with higher CRP levels (-0.51 ± 0.06 vs. -0.36 ± 0.06 mmol/L; $P=0.068$). Both ICAM-1 and E-selectin were significantly lower following the LA and ALA diets and VCAM-1 was lower following the ALA diet when compared with the control AAD ($P=0.01$).

Serum total cholesterol, LDL cholesterol, TG, and apo B levels were reduced following the LA (10.9, 12.3, 18.4, and 9.4% lower, respectively) and ALA diets (10.8, 11.0, 18.4, and 9.7% lower, respectively) compared to the AAD ($P<0.05$), but did not differ from each other. Intake of the ALA diet resulted in a statistically significant decrease in HDL cholesterol levels and apo AI, however, the TC:HDL cholesterol ratio also decreased significantly following both the LA and ALA diet periods.

These results indicate that diets high in both LA and ALA effectively reduce serum lipid and lipoprotein levels. Although both LA and ALA appear to improve cardiovascular risk factors related to inflammation, intake of the ALA diet more markedly reduced CRP levels and resulted in a more favorable reduction in markers of endothelial activation, VCAM-1 and E-selectin, than during the LA diet. The observation that the LA diet, which was higher in ALA than the AAD, also reduced ICAM-1 and E-selectin concentrations, is significant in light of previous in-vitro findings that diets high in LA promote inflammatory endothelial responses. The LA diet used in this study also contained ALA, leading the researchers to conclude that adding ALA to diets high in omega-6 fatty acids (such as LA) might counteract some of the proinflammatory effects of omega-6 lipids.

Zhao G, Etherton T, Martin K, et al. Dietary alpha-linolenic acid reduces inflammatory and lipid cardiovascular risk factors in hypercholesterolemic men and women. *J Nutr* 2004; 134:2991-2997.

Editorial: *What the Public Asks...Believe it or Not!*

Having a web site (www.enc-online.org) devoted to eggs results in a large number of very interesting and curious consumer questions. Here are some examples, unedited and uncorrected. (And I swear I am not making these up—real e-mails from real people.) Should you need answers to any of them, just send us an e-mail.

Questions:

"I would like to know what food group the egg is from. I have heard that it is part of the meat group, but others have said it is dairy."

"I am doing this diet that requires equal amount of protein to carbs. The carbs is no problem for me, but as for protein, for the past few weeks, I have been eating 6-8 eggs a day. Is that dangerous?"

"My mother-in-law says she buys eggs from a person who dates each egg. She claims that if you eat that egg within 2 days of the date on the egg, it has no cholesterol in it. Do you think there is any truth to this?"

"I have heard that the bulk of the protein in eggs is in the whites. Is that true?"

"I have to do an assignment for school and i was just wondering who was the first person to think of eating an egg?"

"I have a question about egg protein. I am in a heated descision about where the protein comes from in the egg. I say it comes from the yolk. While my frined says its the egg whites that have the protein. I have looked but found nothing the helps me."

"I'm just wondering if you know the difference between brown and white eggs so if you could email me back straight"

"Is there such a thing as a blue egg. Or does it have to be colored to be that way"

"Recently some guys at work told me that that little bit of white "stuff" inside the egg is actually rooster sperm. I didn't believe them but they insist it's true."

"My boys' teching intern (science) tells them that in unrefrigerated eggs there's an enzyme or something that helps break down cholesterol in the egg; thus you'd consume less. however, in the refrigerated egg, this compound is destroyed so in effect, you'd get a higher dose of cholesterol with refrigerated eggs than fresh eggs. i've never heard of such."

"My daughter and I are having a "discussion" about how to tell if eggs are still good or have outlived their shelf/fridge life. I say that if you place an egg in water and it sinks to the bottom, that it is still fine. She maintains that if it floats, it's fine. Would you please let me know by return email which is which?"

"We have eight hens we raised from chicks that have just started laying in the last couple of weeks. I recently heard from a cousin that fresh eggs are lower in cholesterol than store-bought, and that the longer eggs sit around the higher the cholesterol content. Is this true? Are my hens sitting on golden (dietarily speaking) eggs?"

"I reached your website in hopes of finding the "why" to something my doctor told me. He said that the cholesterol in eggs is less when they are prepared with the yolks runny. Is this true? If so, why? I think he also said that the total protein of the egg increases if the egg is prepared with the yolk runny."

"I WAS JUST WONDERING WHY CHICKEN IS SO MUCH LOWER IN CHOLESTEROL THAN AN EGG."

"Is it true eggs that have various colored shells, not including brown, have less cholesterol? Is it true that eggs produced by chickens that never scratch the ground have lower or no cholesterol?"

"What is the egg, a vegetable, a meat, or a fruit? It is a Fruit, the fruit of a chicken."

"Is the 213mg of cholesterol per egg the good or the bad kind?"

"We have a question: Are eggs naturally white or brown? or white eggs are bleached?"

"I've been eating 6 whole eggs/day . I'm doing this to increase my protein level, since I work out 5-6 times per week. I was wondering where I can find information on the nutritional content of the yolk and of the white, separately. My cholesterol level is 168 and I was wondering if, I should only be eating the whites."

"Do eggs have amino acids, and if so, which ones and how much?"

"I was told by my doctor that hard boiled eggs have no cholesterol, can this be true?"

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

Toxoplasmosis and Pregnancy: *Clarification of information regarding eggs*

A recently released study published in the American Journal of Obstetrics and Gynecology included the recommendation that all pregnant women be screened for infection caused by *Toxoplasma gondii*. This is an important disease prevention issue since pregnant women are more susceptible to toxoplasmosis infection and a serious infection can have devastating effects during pregnancy.

Recent news reports stemming from this study incorrectly suggested that raw or undercooked eggs are a source of the parasite. This was an erroneous assumption based solely on the fact that study investigators questioned participants about their exposure not only to known sources of *Toxoplasma gondii*, but also to uncooked and unpasteurized foods including raw eggs.

The fact that participants were questioned about their consumption of raw eggs does not imply that eggs are a source of the parasite. Table eggs are not known to contain *Toxoplasma gondii* and have never been associated with toxoplasmosis. The Centers for Disease Control and Prevention state on their website that "Chicken, other fowl, and eggs almost never contain toxo. However, you should still cook these foods until well done because of the risk for other diseases." (www.cdc.gov/hiv/pubs/brochure/oi_toxo.htm)

Adding to potential confusion is the fact that *Toxoplasma gondii*, like other parasites, produces oocytes—commonly referred to as "eggs"—which are often present in the feces of infected animals. Any reports mentioning toxoplasmosis in conjunction with "eggs" (oocytes) might lead consumers to confuse these parasite "eggs" with chicken eggs; therefore, it is very important that the difference be clarified.

It is very important that pregnant women are aware of ways to reduce their risk of developing toxoplasmosis in addition to avoiding other infections such as foodborne illnesses. Pregnant women need to take extra precautions with the food that they eat. Even though eggs are not associated with toxoplasmosis, as with any animal food product, it is good food safety practice to always cook eggs thoroughly before eating.

Eggs are an important food that should be included in the diets of pregnant and lactating women. Eggs contain high quality protein in addition to nutrients that are beneficial to a growing fetus and newborn such as lutein, zeaxanthin, and choline in addition to many vitamins and minerals.

For more information on Toxoplasmosis and food safety, view the following websites:

www.cdc.gov/ncidod/dpd/parasites/toxoplasmosis
www.fightbac.org
www.foodsafety.gov
www.enc-online.org

Boyer KM, Holfels E, Roizen N, et al. Risk factors for *Toxoplasma gondii* infection in mothers of infants with congenital toxoplasmosis: Implications for prenatal management and screening. *Am J Obstet Gynecol* 2005;192:564-71.

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