

*“When a distinguished but elderly scientist states that something is possible, he is almost certainly right. When he states that something is impossible, he is very probably wrong.”*

—Arthur C. Clark

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# NUTRITION CLOSE-UP

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## Including Eggs in a Reduced-Carbohydrate Diet Improves HDL Levels in Overweight Men

Recent research has demonstrated the value of carbohydrate-restricted diets for overweight/obese individuals with certain risk factor profiles. In addition to reducing body weight and abdominal adiposity, carbohydrate-restricted diets typically improve serum triacylglycerol (TAG) and HDL cholesterol concentrations, all of which are features associated with the metabolic syndrome. The macronutrient composition of eggs (high in protein and extremely low in carbohydrate) makes them ideal for use in carbohydrate-restricted (CR) diets; but what of the additional cholesterol? Are there any drawbacks to including eggs in a CR diet plan?

In a randomized, single-blinded, placebo-controlled study, researchers from the University of Connecticut at Storrs sought to answer this question. Twenty-eight overweight/obese (BMI 26-37 kg/m<sup>2</sup>) men aged 40-70 years were recruited to participate in a 12-week diet trial that would evaluate the results of adding 3 eggs per day to a CR diet. All participants were asked to consume CR diets (consisting of 10-15% of energy from carbohydrate, 25-30% from protein, and 55-60% from fat) for the duration of the study. Each participant was randomly assigned to consume 3 eggs (providing 640 mg/d additional dietary cholesterol, 1.8 g carbohydrate, 19 g protein, 23 g fat, and 1239 kJ) or an equivalent volume of fat-free, cholesterol-

*...these findings demonstrate that carbohydrate-restricted diets can be very successful in reducing risk factors in overweight/obese men who meet the criteria for the metabolic syndrome and that eggs...might be an ideal source of protein for use in [such] diets...*

free egg substitute (providing 2.9 g carbohydrate, 19.8 g protein, 0.3 g fat, and 394 kJ) each day of the study. The egg substitute had the same color and consistency as the liquid whole eggs provided by research coordinators.

Diet records and 12-hour fasting blood samples were collected at baseline, week 6, and week 12. Body composition (measured by dual-energy x-ray absorptiometry [DXA]), blood pressure, and anthropometrics were also measured at these time points. Participants were also asked to maintain their normal activity level throughout the study and provided records of their physical activity at baseline and once each week for the duration of the study. Participants were free-living and were not counseled to limit energy intake.

*Continued on page 2*

The eggs and egg substitute were the only foods provided by the study coordinators. Participants did, however, receive dietary counseling from registered dietitians and were provided with detailed instructions for each dietary intervention, including goals, lists of appropriate (meat, fish, vegetables, some cheese, small amounts of seeds and nuts, low-carbohydrate salad dressing), and inappropriate foods, recipes, sample meal plans, and food record sheets. Participants were instructed not to consume eggs outside of the eggs or egg substitute provided to them by the study coordinators. Compliance was evaluated during weekly follow-up visits.

Dietary analysis showed that calorie consumption dropped significantly for both groups once participants began following the prescribed CR diets ( $P < 0.05$ ). By week 12, participants in the EGG group had reduced their carbohydrate intake to 14.9% from 42.4% (at baseline), while SUB group participants reduced their carbohydrate intake to 19.1% from 41.5% (at baseline). Dietary cholesterol intake differed between groups, as intended. The EGG group increased their cholesterol consumption from  $319 \pm 150$  mg/day (baseline) to  $826 \pm 192$  mg/d ( $P < 0.0001$ ).

Body weight, BMI, waist circumference, and systolic and diastolic blood pressure decreased similarly from baseline for all participants, regardless of group assignment. Abdominal adiposity also decreased similarly for both groups. Total cholesterol, LDL cholesterol, and the LDL:HDL cholesterol ratio remained unchanged over the course of the study for participants in the EGG and SUB groups. TAG levels decreased by 45% for participants in both groups ( $P < 0.001$ ). However, only participants in the EGG group saw increases in HDL cholesterol (from  $47.48 \pm 15.05$  mg/dL [ $1.23 \pm 0.39$  mmol/L] at baseline to  $56.74 \pm 15.05$  mg/dL [ $1.47 \pm 0.39$  mmol/L] at week 12). HDL cholesterol remained unchanged for participants in the SUB group. Fasting plasma glucose concentrations did not change in either group.

At baseline, 18 (58% of total) participants were classified as having the metabolic syndrome. Of these, 11 were randomized to the EGG group and 7 to the SUB group. By the conclusion of the study, only 3 (all in the SUB group) still met the criteria for the metabolic syndrome. Overall, these findings demonstrate that CR diets can be very successful in reducing risk factors in overweight/obese men who meet the criteria for the metabolic syndrome and that eggs are appropriate for inclusion in such diets.

These findings further suggest that eggs might be an ideal source of protein for use in CR diets, given that participants in both EGG and SUB groups experienced the same anthropometric and lipid profile improvements and that those in the EGG group actually experienced an increase in HDL cholesterol. ■

Mutungi G, Ratliff J, Fernandez ML, et al. Dietary cholesterol from eggs increases plasma HDL cholesterol in overweight men consuming a carbohydrate-restricted diet. *J Nutr* 2008;138(2):272-6.

## KEY MESSAGES

- Overweight/obese men following carbohydrate-restricted diets experienced reductions in body weight, BMI, abdominal adiposity, waist circumference, triacylglycerol levels, and systolic and diastolic blood pressure.
- Those who were assigned to consume three eggs per day as part of a carbohydrate-restricted diet regimen experienced increased HDL concentrations.
- At baseline, 18 of the 28 participants were classified as having the metabolic syndrome. By the conclusion of the study, only 3 (all in the egg substitute group) still met the criteria for the metabolic syndrome.
- This study demonstrates that carbohydrate-restricted diets can be successful in reducing risk factors in overweight/obese men who meet the criteria for the metabolic syndrome and suggests that eggs are an ideal source of protein for inclusion in such diets.

# Dietary Saturated and *Trans* Fats Associated with Subclinical Atherosclerosis

Although research has shown that specific dietary lipids are associated with changes in cardiovascular disease (CVD) risk factors, their relation to the actual progression of subclinical atherosclerosis has not been investigated. A recent study by Merchant et al., undertaken in a multi-cultural cohort of healthy adults, sheds some light on the impact of specific dietary fats on intimal medial thickness (IMT), an established marker of atherosclerotic progression.

The study participants (n=620) represented a multi-cultural cross-section of Canadian Aboriginal (n=92), South Asian (n=174), Chinese (n=168), and European (n=186) adults who had lived in Canada for at least 5 years. All were between the ages of 35 and 75 years and were free of diagnoses such as angina, cancer, diabetes, cardiovascular disease, hypertension, hypercholesterolemia, kidney, and liver disease. Height, weight, waist, and hip measurements were measured for each participant. Lifestyle characteristics and medical history were obtained through validated questionnaires. Participants completed culture-specific, validated, food-frequency questionnaires to provide information about their typical dietary intake. Carotid IMT was measured in each participant to assess the presence and extent of subclinical atherosclerosis.

After multivariate adjustment, each additional 10 g/d of saturated fat intake correlated with a 0.03 mm greater IMT measurement ( $P<0.01$ ). Likewise, each additional 1 g/d of *trans* fat intake was associated with a 0.03 mm greater IMT measurement ( $P=0.02$ ). Both saturated and *trans* fat intakes were independently associated with IMT, saturated fat being responsible for a 0.03 mm change ( $P<0.01$ ) and *trans* fat being responsible for a 0.02 mm ( $P<0.01$ ) change. The positive associations between saturated fat and *trans* fat intake and IMT were still significant after further adjusting for protein, carbohydrate, polyunsaturated fat, monounsaturated fat, sugar, total fiber, and soluble and insoluble fiber intake.

IMT was not associated with intake of monounsaturated fat or cholesterol. There was an inverse association between the dietary P:S ratio (polyunsaturated:saturated fat ratio) and IMT after multivariate adjustment (-0.06 mm;  $P<0.01$ ). Alcohol intake was also inversely correlated with IMT following multivariate adjustment and it was observed that the relationship between saturated fat intake and IMT was more pronounced in those who rarely or never consumed alcohol (BETA = 0.0066,  $P<0.001$ ) than in those who reported consuming >1 drink/wk (BETA = 0.0001,  $P = 0.79$ ) ( $P$  for interaction = 0.01).

These findings not only help clarify the roles of saturated and *trans* fats in promoting CVD risk, but also provide evidence that cholesterol intake is not associated with the progression of subclinical atherosclerosis—a finding that supports the growing body of research showing that cholesterol intake is not independently associated with CVD risk. The observations regarding the protective roles of polyunsaturated and monounsaturated fats are not surprising, given the research available to date. Of note, moderate to high alcohol consumption seemed to weaken the relationship between saturated fat intake and IMT, suggesting that alcohol intake might help blunt the negative effects of saturated fat in the diet. ■

Merchant AT, Lelemen LE, de Koning L, et al. Interrelation of saturated fat, *trans* fat, alcohol intake, and subclinical atherosclerosis. *Am J Clin Nutr* 2008;87:168-74.

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EAD IT AGAIN...  
FOR THE FIRST TIME

1965

## Reducing Dietary Cholesterol is not the Solution: *Have we known it all along?*

“For the purpose of controlling the serum [cholesterol] level, dietary cholesterol should not be completely ignored but attention to this factor alone accomplishes little.” Such was the conclusion of Ancel Keys, Joseph T. Anderson, and Francisco Grande following a 1965 review of data from five cholesterol feeding studies. The studies had been designed to elucidate the effects of varying levels of dietary cholesterol intake on plasma cholesterol levels. Their findings mirror much of what has been observed in cholesterol research to date, specifically that even great reductions in cholesterol intake can be expected to make only nominal differences in total serum cholesterol concentrations. Keys, Anderson, and Grande first reviewed one of their own studies, in which 22 physically healthy men followed 4 diets that differed in cholesterol content (50 mg, 380 mg, 520 mg, and 1460 mg/day) but were similar in all other respects (providing 2625 kcals, on average). The average change in serum cholesterol following each diet assignment was 0, 16, 14, and 27 mg/dL (0, 0.4, 0.4, and 0.7 mmol/L), respectively. At the time, the average daily intake of cholesterol among men in the US, Great Britain, and Northern Europe was estimated to be 150-350 mg/1000 kcals. Thus, the diet containing only 50 mg cholesterol per day would have represented a drastic reduction in cholesterol intake, while the one containing 1460 mg cholesterol per day would have represented a huge increase. Yet, even these drastic changes in dietary cholesterol intake made relatively little difference in serum cholesterol concentrations.

Beveridge et al. conducted a formula-based cholesterol feeding trial in students. Participants were given an identical, virtually fat-free formula diet for 8 days, after which they were randomly assigned to formula diets containing 30% fat and varying levels of cholesterol, ranging from 13 to 3441 mg/day. Because it generally takes several weeks to see the full effect of dietary changes on serum cholesterol levels, the change in serum chole-

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*“For the purposes of controlling the serum [cholesterol] level, dietary cholesterol should not be completely ignored but attention to this factor alone accomplishes little...efforts to reduce cholesterol in the diet can produce an effect on the serum but unless the effort is heroic, the change in the serum will be small.”*

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sterol levels in this experiment was adjusted based on the assumption that only 70% of the full dietary effect was observed after 8 days. The changes ranged from 0 mg/dL (0 mmol/L) in the group with the lowest intake (13 mg/day) to 57 mg/dL (1.5 mmol/L) in the group with the highest intake (3441 mg/day).

Connor et al. conducted a similar cholesterol feeding trial using 4 formula diets differing in cholesterol content (cholesterol-free vs. cholesterol-rich) and fatty acid

composition. The statistics were corrected to account for differences in fatty acid content. Those following the cholesterol-free diet experienced no change in serum cholesterol, while those following the cholesterol-rich formula (providing ~725 mg cholesterol/day) experienced an increase of 26-35 mg/dL (0.7-0.9 mmol/L).

In another trial conducted by Steiner et al., 5 participants were given a diet containing 3000 mg cholesterol per day and providing 40% of energy from olive oil. After 5 weeks of follow-up, the diet resulted in an average increase of 50 mg/dL (1.3 mmol/L).

As previously noted, the average daily cholesterol intake for men in the U.S., Great Britain, and Northern Europe at the time this review was written was estimated to be 150-350 mg/1000 kcals. The authors note that the change in serum cholesterol as a function of changes in dietary cholesterol in these studies was consistent with what would have been expected based on predictive equations used at the time. Using these equations, it was estimated that an individual could reduce their total cholesterol level by 24 mg/dL (0.6 mmol/L) if all cholesterol were removed from a diet that typically provided 250 mg/1000 kcals per day (the ordinary intake for the time). While this change would be clinically significant, the authors note that it would be nearly impossible to achieve except on a strict vegetarian diet and that a reasonable “low-cholesterol” diet would not result in this magnitude of serum cholesterol reduction. “But,” they wrote, “a 50 per cent decrease in dietary cholesterol will produce an average decrease in the serum of only about 7 mg/dL [0.2 mmol/L].”

Likewise, when evaluating the effects of increasing dietary cholesterol, it must be acknowledged that (as observed in these studies) the addition of large amounts of cholesterol to diets already high in cholesterol has a relatively small impact on serum concentrations. The authors state that “such considerations indicate that efforts to reduce cholesterol in the diet can produce an effect on the serum but unless the effort is heroic, the change in the serum will be small.”

Today we have a better understanding of the effects of dietary cholesterol on plasma cholesterol and its effects on both LDL and HDL cholesterol levels. The estimated 24 mg/dL (0.6 mmol/L) decrease if all cholesterol were removed from the diet (in 1965 estimated to be 625 mg/day per 2500 kcals) would decrease LDL cholesterol by about 17 mg/dL (0.4 mmol/L) and HDL cholesterol by 7 mg/dL (0.2 mmol/L) with no change in the LDL:HDL ratio. Keys later recognized that dietary cholesterol had little, if any, effect on plasma cholesterol levels and considered it a non-issue, but by then the restrictions had been put in place and have remained there ever since. ■

Keys A, Anderson JT, Grande F. Serum cholesterol response to changes in the diet II. The effect of cholesterol in the diet. *Metabolism* 1965;14:759-65.

# Low Protein Intake Might Increase Risk of Age-Related Sarcopenia

Changes in body composition are considered a normal part of the aging process. Most men and women will experience a loss of lean mass (LM) and increased fat stores as they age. Sarcopenia—the age-related loss of skeletal muscle—poses a special threat since it leads to functional impairment that can significantly impact quality of life. It is thought that insufficient dietary protein intake might increase a person's susceptibility to this age-related muscle atrophy, and further, that adequate intake of protein of high biological value might be protective.

From a larger, prospective investigation—the Health, Aging, and Body Composition (Health ABC) Study—researchers from Wake Forest University gathered information from a 2066-person random sample of white and black Medicare-eligible, community-dwelling participants aged 70-79 years. At the time of study enrollment, participants were free of life-threatening illnesses and reported no difficulty in walking a quarter of a mile, climbing up 10 steps, or performing basic daily activities. Body composition was measured by dual-energy x-ray absorptiometry (DXA) at baseline. The measurement was repeated three years later to assess changes in lean mass (LM) and appendicular LM (aLM, which represents skeletal muscle). Dietary protein intake was assessed for each participant at baseline using an interviewer-administered food-frequency questionnaire. To determine the biological value of the protein consumed, researchers tracked not only total protein consumption, but whether the protein was of animal or vegetable origin.

The mean protein intake was ~0.9 g/kg/d for both men and women in the study. Over 3 years of follow-up, total LM declined by 0.68±1.94 kg and aLM by 0.48±1.08 kg, on average. Participants who lost >3% of their body weight (28.8%) lost an average of 1.30±1.06 kg in aLM. Those who gained >3% of their body weight (21.7%) gained an average of 0.32±0.88 kg in aLM, while those who maintained their baseline weight (49.5%) lost 0.35±0.81 kg aLM.

After adjusting for potential confounders, both total protein and animal protein intake were significantly associated with changes in LM [BETA (SE): 5.31 (1.64) and 5.26 (1.65), respectively;  $P<0.01$ ]. These associations were slightly weakened after further adjusting for changes in fat mass (FM), but remained significant. Intake of vegetable protein was not associated with LM or aLM after adjusting for all potential confounders, including changes in FM.

Higher protein intakes seemed to help conserve LM and aLM. Participants in the highest quintile of protein consumption lost 43% less LM ( $P$  for trend<0.01) and 39% less aLM than did participants in the lowest quintile. Of those who lost weight over the course of the study, participants in the top three quartiles of

protein intake tended to maintain more aLM than those in the lowest quintile ( $P$  for trend<0.05). Among those who gained weight, participants in the highest quintile of protein intake acquired more aLM than those in the lowest ( $P$  for trend<0.05). Each of these associations were weakened, but remained significant after adjusting for changes in FM.

The authors conclude that a higher intake of total protein and animal protein does, indeed, appear to conserve lean body mass (LM) and skeletal muscle (aLM) in older, community-dwelling, adults. Lower protein intake was also associated with greater loss of LM in participants who lost weight over the course of the study. An association between vegetable protein intake and LM was not found in this study, but the authors note that the range of intake was greater for animal than for vegetable protein, which might have “limited [the] ability to detect a significant association between vegetable protein and changes in LM.”

These findings are particularly interesting as they relate to the current RDA for dietary protein intake for aging adults, which now stands at 0.8 g/kg/day. (The average intake among study participants was ~0.9 g/kg/day.) It is well known that this recommendation was based, in large part, on short-term nitrogen balance studies, not long-term body-composition studies. Maintaining nitrogen balance might do little, if anything, to preserve lean tissue. The authors conclude that “although the differences in LM over the 3-year follow-up were small, if compounded over greater lengths of time, they may result in substantial differences in LM... These results suggest that low protein intake may be a modifiable risk factor for sarcopenia among older adults.” ■

Houston DK, Nicklas BJ, Kritchevsky SB, et al. Dietary protein intake is associated with lean mass change in older, community-dwelling adults: The Health, Aging, and Body Composition (Health ABC) Study. *Am J Clin Nutr* 2008;87:150-5.

## Give Me a Break—Perverse News From Good Science

The headline was a stunner: “Fast Food: The Fast Track to Organ Damage” followed by the line “Welcome to Fast Foods! How can we destroy your internal organs?” I love studies that can be so easily interpreted for the public and fit such succinct and informative headlines. (I also love being boiled in oil!) The study the journalist was referring to was actually entitled, “Fast-food-based hyper-alimentation can induce rapid and profound elevation of serum alanine aminotransferase in healthy subjects.” [article available online at <http://gut.bmj.com/cgi/rapidpdf/gut.2007.131797v1>]

To really appreciate this one you first must look at the experimental design: 18 slim, healthy Swedish men and women took on a fast food diet, eating meals from popular chains twice a day for four weeks while refraining from exercise (no more than 5,000 steps per day). The goal: to double daily caloric intake and boost body weight by 10 to 15 percent and then observe the effects on the liver. Now, please note that the control group does nothing—eats normally and exercises normally and doesn’t gain weight. Now that’s a control group!

People on the fast-food/low exercise regimen gained an average of more than 14 pounds within 4 weeks (0.5 lbs per day), and one person gained more than 26 pounds in just two weeks, according to researchers. At the end of the experiment, blood tests showed evidence of liver damage in the subjects on the fast food diet, leading the media to proclaim that “the liver is also at risk when you roll up to the drive-through window.”

This one reminds me of that scientific gem of a documentary entitled *Super Size Me* by that prominent nutritional scientist, What’s His Name. Let me see...eat 5,000 calories a day from a fast food restaurant for 30 days and gain 24.5 pounds...and you don’t feel good? Amazing! Now the question is, if I consumed that many calories from any source—not just fast food—would I also gain weight and feel bad? Don’t know. Nobody has tested that one yet. We just send them off to eat, eat, eat and sit, sit, sit and blame the fast food, not the calories or the lack of activity or the combination of the two. To the media, fast food is bad; and any study that proves them right is a good study. Reminds me of a few scientists I know!

The authors of the study were much more rational than the media. Their conclusion: “Hyper-alimentation per se can induce profound ALT elevations in less than 4 weeks.” Okay, documented

by the study and appropriately expressed. And more importantly, the study actually does have some clinical applicability—“We conclude that chronically or intermittently elevated ALT can be of purely nutritional origin, particularly when found in the absence of liver steatosis. The fact that 14 out of 18 participants had pathological ALT levels after just 1 week clearly displays that elevated ALT levels after a short over-indulgent holiday can be caused not only by alcohol ingestion, but also by a higher caloric intake than usual combined with a sedentary behaviour. We suggest that in the clinical evaluation of subjects with elevated ALT physicians should include not only questions about alcohol intake, but also explore whether recent excessive food intake has occurred.”

Note the absence of any reference to “fast food” by the scientists who actually did the research. Only media hype could twist it to play on everyone’s fears and phobias. Give me a break! ■

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*Executive Editor, Nutrition Close-Up*



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