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close UP

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I N S I D E

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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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C-Reactive Protein, LDL Cholesterol and CVD Risk

One of the long-standing arguments for getting plasma LDL levels as low as possible is that a large number of CHD events occur in those with "normal" or "near optimal" LDL levels. As new risk factors for CHD are identified it becomes evident that other factors can make major contributions to CHD risk. Clearly risk reduction interventions need to expand their scope beyond the traditional risk factors of cigarette smoking, hypertension, and hypercholestrolemia. Yet in many cases this is difficult since the importance of the newly identified factors relative to the traditional risk factors are not quantified. A recent study by Ridker et al. has helped address this issue by directly comparing the CVD risk predictive value of plasma C-reactive protein (CRP) levels, a marker of inflammation, with that of LDL cholesterol levels. The investigators measured baseline CRP and LDL cholesterol of 27,939 participants in the Women's Health Study and followed the study subjects for an average of eight years. The mean age for the women in the study was 54.7 years with a mean BMI of 25.9. The average LDL cholesterol concentration in this population was 123.7 mg/dl and the average CRP level was 1.52 mg/L. CRP and LDL levels were minimally correlated (r=0.08).

Seventy-seven percent of first CVD events occurred in women with LDL cholesterol levels below 160 mg/dl and 46% occurred in those with LDL cholesterol values below 130 mg/dl. As shown in the table, analysis of the data indicated that both plasma CRP levels and LDL cholesterol levels were positively related to CVD events after adjustment for other risk factors. These relationships were similar for the 12,139 women using hormone-replacement therapy (HRT) and the 15,745 non-HRT women.

After adjustment for all components of the Framingham risk score, CRP remained a significant, independent predictor of CVD risk in this cohort. This association was true across the entire range of LDL cholesterol levels.

Quintile of C-Reactive Protein

CRP (mg/L)	<0.49	>0.49-1.08	>1.08-2.09	>2.09-4.19	>4.19
Adjusted RR	1.0	1.4	1.6	2.0	2.3

Quintile for LDL Cholesterol

LDL-C (mg/dl)	<97.6	>97.6-115.4	>115.4-132.2	>132.2-153.9	>153.9
Adjusted RR	1.0	0.9	1.1	1.3	1.5

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C-Reactive Protein

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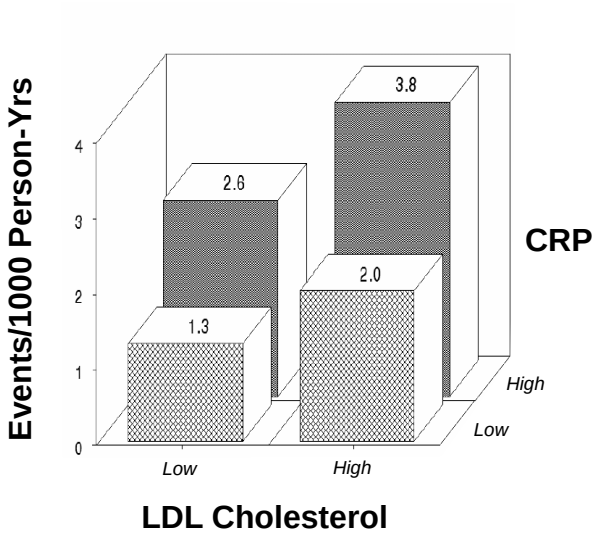
The authors estimated the multivariable-adjusted absolute risk for those below and above the median CRP and LDL cholesterol levels for this study population. As shown in the figure, having a low LDL cholesterol and a high CRP value results in a greater absolute risk than a high LDL cholesterol and low CRP, and the greatest risk occurs when both levels are high. The investigators concluded that "the C-reactive protein level is a stronger predictor of cardiovascular events than the LDL cholesterol level and that it adds prognostic information to that conveyed by the Framingham risk score."

This study explains a number of issues related to CHD risk and raises a number of important questions which have significant impact on how CVD risk is evaluated and the therapies applied. An obvious question is, What is the appropriate treatment for a woman with low LDL cholesterol but with high CRP levels? Previous studies suggested that statin therapy is effective for individuals with this condition; however, this is not a group normally treated with statin drugs. Perhaps most importantly these data provide values for the distribution of CRP levels in the population for future comparisons and risk estimations. The results also indicate that a single cutoff point is applicable regardless of HRT status.

Ridker PM, Rifal N, Rose L, et al. Comparison of C-reactive protein and low-density-lipoprotein cholesterol levels in the prediction of first cardiovascular events. *N Eng J Med* 2002;347:1557-65.



Key messages

- Plasma LDL cholesterol levels and plasma C-reactive protein levels are independent predictors of CVD risk in women.
- The C-reactive protein level is a stronger predictor of CVD events than the LDL cholesterol level.
- Patients with low LDL cholesterol and high C-reactive protein levels are at greater risk than those with high LDL cholesterol and low C-reactive protein.
- The C-reactive protein level adds to the prognostic value of the Framingham risk score.

Fish and Dementia

Many people can remember the days when fish was popularly known as "brain food". And while that view is often considered an old wife's tale, it seems there is some scientific evidence that fish can in fact help ward off the risk of dementia. Using data from the PAQUID Study of cognitive and functional aging, Barberger-Gateau et al. found that those who ate fish or seafood at least once a week had a significantly lower risk of being diagnosed as having dementia. The study population of 1674 people aged 68 and over was followed for seven years. During that time period 170 new cases of dementia, including 135 cases of Alzheimer's disease, were diagnosed. Participants who ate fish or seafood at least once a week had a significantly lower risk of being diagnosed as having dementia with an age and sex adjusted hazard ratio of 0.66 (95% confidence interval 0.47 to 0.93). For Alzheimer's disease the hazard ratio was 0.69 (95% CI 0.47-1.01). There was no significant association between meat consumption and risk of dementia. So while the question of whether eating fish will make you smarter remains uncertain, these data indicate that eating fish can help you stay as smart as you are.

Barberger-Gateau P, Letenneur L, Deschamps, V et al. Fish, meat, and risk of dementia: cohort study. *BMJ* 2002;325:932-933.

Soyfoods, Isoflavones and CHD Risk

Many studies suggest that soyfood consumption lowers plasma cholesterol levels and CHD risk, yet the identity of the active component in soyfood responsible for this benefit remains elusive. Isoflavones associated with soy protein have been implicated in plasma LDL lowering, but isoflavones by themselves have not been shown to lower serum lipids. In order to better understand the effects of soyfoods and isoflavones on CHD risk factors, Jenkins et al. investigated the effects of soy-protein foods with high- versus low-isoflavone content on plasma lipids, plasma homocysteine, LDL oxidation, and blood pressure in 41 hyperlipidemic men (n=23) and women (n=18).

The investigators used a randomized crossover study design consisting of three 1 month long dietary trials, a dairy- and egg-protein control period and 2 soy-protein periods (50-52 g/day), one with low (10 mg/day) and one with high (73 mg/day) isoflavone soy-protein content. The basal diet was low fat (16-18% en), low SFA (<4.5% en) and low cholesterol (<75 mg/day). The soy-protein foods were provided by the researchers.

While both soy-protein diets lowered plasma lipids compared to the control diet, there were no significant differences in plasma lipid responses between the low- and high-isoflavone soy phases. Intake of the soy-protein diets resulted in lower plasma LDL cholesterol levels (-9%), reduced total:HDL cholesterol (-2%) and LDL:HDL cholesterol (-5%) ratios, decreased plasma homocysteine levels (-7%), less oxidized LDL (9-20%), and an overall lower estimated CHD risk (-10%). Soy-protein intake was also related to reduced systolic blood pressure in men. The data indicate that there was a range of small beneficial effects associated with replacing exchanging animal-protein with soy-protein in an NCEP Step II diet irrespective of the isoflavone content.

Two caveats to consider in this study are that the hyperlipidemic study subjects were consuming a very low-fat, low-cholesterol diet and that the shift from animal-protein to soy-protein was substantial with exchange of 55% of the total protein intake. It is unclear whether the observed effects of soy-protein could be achieved by taking soy as a supplement or whether it is necessary to substitute soy-protein for animal-protein in the diet. Research is also needed to determine whether these effects of soyfoods would be comparable in subjects consuming less restrictive diets.

In another study of the effects of soy on plasma lipoproteins, Lichtenstein et al. tested the independent effects of 25 g/4.2 MJ soy protein ± soy isoflavones (46-52 mg/4.2 MJ) relative to animal protein ± soy isoflavones on lipoprotein responses in 42 mildly hypercholesterolemic subjects. The experimental design involved a 42 day feeding period for each of the 4 diets in a 2 x 2 factorial design. The fatty acid profiles of the diets were similar as were the other constituents.

Twenty-five grams of soy protein per 4.2 MJ resulted in small but significant reductions in plasma total cholesterol (-5.0 mg/dl) and LDL cholesterol (-3.9 mg/dl) with a 1.2 mg/dl increase in HDL cholesterol. Soy-derived isoflavones had little effect on plasma lipoprotein levels. The authors concluded that "daily consumption of relatively high levels of soy protein (>50 g/day), at least double the level that would allow foods to qualify for a health claim, would be predicted to confer little benefit to normocholesterolemic subjects or those with borderline high LDL cholesterol levels."

Both of these studies indicate that many of the commercially available products which contain soy protein ± soy isoflavones at the level allowable for a health claim (6.25 g per serving) would have little beneficial effect on plasma cholesterol levels and that overreliance on use of soy protein products to lower an elevated plasma cholesterol level would have little benefit in terms of CVD risk reduction. Whether soy foods provide benefits other than plasma cholesterol lowering, and at what levels of intake, remains to be determined.

Jenkins DJ, Kendall CW, Jackson CC, et al. Effects of high- and low-isoflavone soyfoods on blood lipids, oxidized LDL, homocysteine, and blood pressure in hyperlipidemic men and women. *Am J Clin Nutr* 2002;76:365-372.

Lichtenstein AH, Jalbert SM, Adlercreutz H, et al. Lipoprotein response to diets high in soy or animal protein with and without isoflavones in moderately hypercholesterolemic subjects. *Arterioscler Thromb Vasc Biol* 2002;22:1852-58.

Cholesterol Absorption Inhibitors

Plant sterol/stanol containing margarines have become a widely used treatment for high plasma cholesterol levels. These sterols/stanols lower plasma LDL levels by inhibiting intestinal cholesterol absorption, which in turn lowers plasma LDL cholesterol levels through an increase in hepatic LDL receptor expression and a parallel release of the feedback control mechanisms, resulting in an increase in endogenous cholesterol synthesis. A new class of specific cholesterol absorption inhibitors have been shown effective in lowering plasma cholesterol levels in animal models and in humans. One of these inhibitors, ezetimibe, acts at the brush border of the small intestine and inhibits the uptake of dietary and biliary cholesterol into the enterocyte. Sudhop et al. carried out a series of studies of ezetimibe to determine effects on cholesterol absorption and metabolism in humans.

The investigators used a randomized, double-blind, placebo-controlled, 2-period, cross-over trial in 18 male subjects with mild hypercholesterolemia. Ezetimibe was given at a dosage of 10 mg/day for 2 weeks and metabolic measurements were done during the last week of each period. Cholesterol absorption rates were significantly decreased by 54%, from 49.8% to 22.7%, with drug treatment. Plasma total cholesterol levels were reduced 13% and LDL cholesterol concentrations by 22%. Ezetimibe treatment had no effect on plasma HDL cholesterol or triacylglycerol levels.

Ezetimibe treatment significantly increased fecal neutral sterol excretion (unabsorbed dietary and biliary cholesterol) which resulted in an increase in endogenous cholesterol synthesis rates. As shown in the table, the increased excretion of cholesterol is primarily from biliary cholesterol, which averaged 2100 mg/day whereas dietary cholesterol averaged 310 mg/day. The increase in endogenous cholesterol synthesis (832 mg/day) was slightly greater than the decrease in reabsorbed biliary cholesterol (570 mg/day) plus absorbed dietary cholesterol (80 mg/day).

The investigators also analyzed plasma indicators of cholesterol absorption and synthesis, and the observed changes were consistent with the sterol balance measurements.

These data provide clear evidence that the cholesterol absorption inhibitor ezetimibe effectively lowers the fractional cholesterol absorption rate in humans along with a decrease in plasma LDL cholesterol levels and an increase in endogenous cholesterol synthesis. Combined usage of the cholesterol synthesis inhibiting statin drugs along with an effective cholesterol absorption inhibitor such as ezetimibe has the potential to effectively lower plasma cholesterol levels. The combination would be effective because the statin drug would reduce the absorption inhibitor-mediated increase in endogenous cholesterol synthesis. Clearly, the major effectiveness of the cholesterol absorption inhibitor is to block reabsoption of endogenous biliary cholesterol which is the predominant source of intestinal cholesterol in humans.

Sudhop T, Lutjohann D, Kodal A et al. Inhibition of intestinal cholesterol absorption by ezetimibe in humans. *Circulation* 2002;106:1942-1948.

Parameter	Placebo	Ezetimibe
Cholesterol intake (mg/day)	307	313
Fractional absorption (%)	49.8	22.7
Absorbed dietary (mg/day)	153	71
Fecal neutral sterol excretion (mg/day)	999	1718
Fecal biliary cholesterol excretion (mg/day)	846	1647
Biliary cholesterol secretion (mg/day)	1990	2222
Cholesterol synthesis (mg/day)	931	1763
Plasma total cholesterol (mg/dl)	214	186
LDL cholesterol (mg/dl)	142	111

Lutein Levels and Macular Pigment Density

Studies have shown that a low macular pigment (MP) density is a risk factor for age-related macular degeneration (AMD). The carotenoids lutein and zeaxanthin are the predominant pigments in the macular region, and epidemiological studies suggest that those with the highest dietary intakes of lutein and zeaxanthin have a reduced risk of AMD. However, there are a number of uncertainties regarding the relationship between plasma and adipose tissue levels of these carotenoids and MP density. For example, studies have found a significant positive correlation between adipose tissue lutein concentrations and MP density in men but a negative correlation in women. Broekmans et al. have reported a cross sectional study of the associations between MP density with serum and adipose lutein in 376 men (n=177) and women (n=199).

The study subjects were selected from a larger study population in the Netherlands based on their intake of fruits and vegetables, either in the highest quintile or lowest tertile of intakes. Blood samples were obtained from all participants and adipose samples from 89 men and 98 women. MP density was measured by spectral fundus reflectance.

Lutein concentrations in serum and adipose tissue were higher in women than in men (0.19 vs 0.16 mol/L and 0.47 vs 0.32 mol/kg wet wt.). The correlations between serum lutein (short-term intake) and adipose lutein (long-term intake) were 0.46 in men and 0.53 in women. Surprisingly, MP density was 13% higher in men than in women. In men, but not in women, MP density was positively correlated with serum lutein and zeaxanthin and with adipose tissue lutein. There was a significant positive correlation in both men and women between plasma vitamin C and MP density. The average MP density in men was 0.35 and the data indicated that a serum lutein increment of 0.1 mol/L was associated with a 0.064 higher MP density (18%).

The authors proposed a number of interpretations of the data. They note that this group had a narrow range of serum carotenoids as compared to other populations, possibly due to a lower intake of lutein-containing fruits and vegetables in this Dutch group. They also suggest that the observed gender differences, which have been reported by other investigators, might result from more effective adipose tissue competition for plasma carotenoids in women than in men. It could also be argued that in men there is a more effective deposition of plasma lutein in the macular region of the eye. These data could also explain, in part, why males have a lower AMD risk than females. Clearly, dietary intervention studies are needed to investigate the influence of lutein intake on MP density and AMD risk in men and women. But given these results, there is supportive evidence that intake of lutein-containing and vitamin C-containing foods have potential benefits in lowering AMD risk.

Broekmans WM, Berendschot TT, Klopping-Ketelaars IA, et al. Macular pigment density in relation to serum and adipose tissue concentrations of lutein and serum concentrations of zeaxanthin. *Am J Clin Nutr* 2002;76:595-603.

Antioxidant Vitamin Intake, Plasma Levels and Carotid Atherosclerosis

Recent studies on the association between antioxidant vitamins and atherosclerosis have been inconsistent and inconclusive. Iannuzzi et al. studied 307 women from the Progetto Atena prospective study in Naples to determine whether intake and plasma concentrations of antioxidant vitamins were associated with the presence of plaques in the common carotid artery and carotid bifurcation. The investigators used a semi-quantitative food-frequency questionnaire to determine average intake of antioxidant vitamins (vitamins A, C and E) over the past year. None of the women took dietary supplements containing antioxidants. Plasma levels of vitamin A, vitamin E and carotenoids were determined by established methods. Carotid artery atherosclerosis was determined by high-resolution ultrasound measurements of intima-media thickness (IMT) of the common carotid artery and the carotid bifurcation.

The investigators found a significant inverse trend between vitamin E intake and plasma vitamin E:plasma cholesterol and IMT values at the carotid bifurcation. These findings did not apply for the common carotid nor were intakes or plasma levels of the other antioxidant vitamins related to IMT values.

While the researchers did find that a low intake of vitamin E was associated with an increased risk of plaques at the carotid bifurcation, they could not rule out the possibility that vitamin E intakes and plasma levels simply reflect a healthier overall diet resulting in lower atherosclerotic risk. However, the finding that the other antioxidant vitamins were not related would argue against this possibility. The authors also noted that supplements were not a source of vitamin E in this study. The questions would be whether the vascular protection by vitamin E occurs only during the early stages of lesion development, and whether vitamin E supplements might only be effective in individuals with inadequate intakes or low plasma levels. What the study does indicate is that low vitamin E intake is associated with higher risk of early atherosclerosis in middle-aged women.

Iannuzzi A, Celentano E, Panico S, et al. Dietary and circulating antioxidant vitamins in relation to carotid plaques in middle-aged women. *Am J Clin Nutr* 2002;76:582-587.

It's All in the Genes

Two recent studies have provided more evidence that genetics makes major contributions to not only our steady state levels of plasma cholesterol but also our responses to dietary interventions. Gylling and Miettinen reported a study of the influence of heredity on cholesterol absorption and synthesis rates in probands with high and low cholesterol absorption. These investigators used a combination of plasma markers for cholesterol absorption and synthesis as well as fecal steroid balance data to measure cholesterol absorption and synthesis in 37 siblings of hypercholesterolemic probands with low (n=6) and high (n=7) absorption of cholesterol. The investigators found that the plasma levels of cholesterol absorption markers and cholesterol synthesis markers were negatively related indicating that those with the lowest fractional absorption had higher rates of endogenous cholesterol synthesis. Comparisons of the plasma markers for absorption and synthesis showed that these metabolic parameters exhibited intrafamily patterns of inheritance. High absorbers had an absorption efficiency of 48.7% and an endogenous cholesterol synthesis rate of 608 mg/day while low absorbers had fractional cholesterol absorption rates of 36.9% and an endogenous synthesis of 1546 mg/day. The largest difference between the two groups was the size of the intestinal biliary cholesterol pool which was increased by almost 70% in the low absorbers. In both the probands and siblings, plasma HDL cholesterol levels were significantly lower in the low versus high absorbers. In contrast, LDL cholesterol levels were similar indicating that rates of LDL synthesis and/or removal were effectively regulated in both groups.

In another study, Nicklas and colleagues reported that the apolipoprotein E genotype has a significant effect on the plasma lipoprotein cholesterol response to dietary interventions in obese postmenopausal women. It has been known for over two decades that lipid responses to a dietary intervention are highly variable between individuals. There is evidence that there is a gene-sex interaction on the effects of the apo E genotype on lipid responses to dietary manipulations. In this study of 18 apo E4+ and 61 apo E4- women, the investigators compared the effects of initiation of an AHA Step I diet on plasma lipids. Both groups had similar reductions in plasma LDL cholesterol concentrations. Apo E4- women decreased HDL levels by 17% and increased triglyceride levels by 20% on the AHA diet whereas apo E4+ women had a smaller decrease in HDL (8%) and no change in triglycerides. The decrease in HDL was most pronounced for HDL2 which is the more protective HDL subfraction. The LDL:HDL ratio increased by 16% in the apo E4- group and did not change in the apo E4+ women. The investigators raise a serious concern based on the results of this study by stating "Since a low HDL-C concentration is a particularly strong predictor of a higher risk for CHD in women, consumption of a low-fat diet may place some women at a higher risk for CHD."

Gylling H, Miettinen TA. Inheritance of cholesterol metabolism in probands with high and low cholesterol absorption. *J Lipid Res* 2002;43:1472-76.

Nicklas BJ, Ferrell RE, Bunyard LB, et al. Effects of apolipoprotein E genotype on dietary-induced changes in high-density-lipoprotein cholesterol in obese women. *Metabolism* 2002;51:853-858.

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

Editorial

The Credibility is in the Doing

There are 100,000 advocacy groups in the legislative city, and this commentary applies to a number of them. Before you talk the talk to me about the horrors of pesticides, herbicides, and fungicides; the global risks from genetically modified crops; and sing the "be organic or die" hymn in my face, kindly walk the walk and live by your words. There should be a federal law that anyone who beats up on agriculture should have at least once in their life survived on a home vegetable garden. The educational benefits would be phenomenal. Just think, all those folks who rant and rave against this or that would actually learn something about the foods they eat. Just one year of having to grow their own food, and of course live that year on what they grew, would be a real world education for those whose knowledge base of agriculture is which row in the supermarket has their favorite organic, non-GM, eco-friendly, caffeine-free $22.00 per pound coffee.

Now I realize the risk involved in this effort. It could be devastating for those black-thumbed individuals who routinely kill every houseplant they ever touched. To actually start with a seed and end up with a meal is not an easy process and those who have never been closer to agriculture than a petting farm or a farmer's market would no doubt find it a daunting task. But the challenge would be worth the effort since then, when those who survive the year open their mouths to voice their doom and gloom comments about big, bad farmers and their terrible production practices, they might actually have a sliver of credibility, they've actually grown something themselves.

So let me tell them what they've got in store for the year. First, you need to get the ground ready. No, just spreading seeds on the ground will not feed you during the winter. Pick the right spot; enough sunshine, good drainage, and then fence it in to keep out some of your major competition. Now prepare the garden: raking, tilling, and then conditioning with topsoil, compost, and manure. Hopefully the ground isn't so wet or so frozen solid that you cannot work it until spring, then it might be too late. Next decision, what to grow? Need to grow foods throughout the early, middle and late growing seasons as well as getting in those storage vegetables for the winter. Timing is key. What goes in when and when will it be ready? What can go into the garden before the last frost and what needs to wait? What varieties are naturally resistant to diseases yet still productive enough for you to live on? Decisions, decisions, decisions.

Okay, after reading seed catalogs and a gardening web site or two, you're ready to buy the seeds and get the seedlings started. Oh, I forgot to warn you about this part. See, some plants need more time to bear fruit than the last frost-first frost interval in many parts of the country, so you start the seeds indoors and then transplant outdoors when it's safe. So if you want tomatoes in July and August you better start your seeds in February. Same for peppers, eggplants, summer and winter squash, cucumbers, and a few others. By April you can get the lettuce and greens, herbs, spinach, peas, beets, turnips, kale and other spring vegetables seeded while you're also taking care of those seedlings growing and needing feeding and transplanting and TLC. And while all this is going on don't forget about storing up for winter; get ready for canning and freezing what you can. And if you want to have anything to eat start watching out for the competition: rabbits, moles, deer, slugs, insects, molds, viruses and uncooperative weather patterns. By May get the late crops in the ground: carrots, onions, beans, and all those seedlings you've nurtured for the last three months. And now the lesson really begins! Flea beetles devastate your eggplants, squash beetles destroy your squash plants, cucumber beetles transfer viral wilt to kill your plants. Insects, bacteria, and viruses join forces with rodents, birds and creepy, crawly things in a classic battle for survival. And then your real antagonist enters the battle: mother nature. Too hot for this plant, too cold for that one. Too dry for tomatoes, too wet for beans. Believe me, the weather is never, ever just right.

And did you plant enough for winter storage? Turnips, parsnips, winter squash, potatoes, etc. Enough canned vegetables? Enough in the freezer for survival? Did you get enough tomato sauce prepared? The non-growing season can last a long time. And while you're taking care of this business, better get the winter garden in: lettuce, spinach, kohlrabi, collard greens, kale, turnips, and various greens. Watch out for those slugs, they're storing up for winter too. A few caterpillars can eat a kale plant in a night so get out there with a flash light and pick those little suckers. Remember, this is healthy growing, no chemicals, all organic. It's you against them!

So after a year of real education I'll be happy to listen to those who want to tell me what to grow and how to grow it and who should grow it and what I should eat. I'll listen, but since I learn these lessons every year in my own garden, I'm pretty sure I'll still not agree with them since I know that my home grown, juicy, organic beets cost me a whole lot more time and money than those in the grocery store. Oh I enjoy them, but I also know that we cannot feed the world that way. I can hardly feed my wife and myself during a bad year, and the cost easily gets out of hand.

There might be 100,000 advocacy groups in the federal city but, the question is, How many actually know what they're talking about?

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

If You Doubt the Findings, Question the Credibility!

A report at the recent American Heart Association annual meeting held in Chicago this November resulted in a media display of scientific bickering the likes of which rarely are seen by the public. Westman and colleagues from Duke University reported a study comparing the Atkins Diet to the AHA Step I diet in 120 overweight subjects followed for 6 months. On the Atkins diet the subjects lost 31 pounds and on the AHA diet they lost 20 pounds. HDL levels increased on the Atkins diet and LDL levels didn't change much on either diet. The media loved it and headlines appeared around the country. The backlash was right on schedule and the usual suspects responded with "too small, too short, no long term evidence of value, not a fair comparison, funded by the Atkins Foundation, etc."

More and more studies are being reported which indicate that the high protein, low CHO diet isn't the blood cholesterol raising, heart disease promoting cuisine it was predicted to be. Two other presentations at the AHA meeting also reported positive benefits from the Atkins diet. Clifton and coworkers from Australia reported that a high protein, energy restricted diet, as compared to a standard protein, energy restricted diet, resulted in greater preservation of lean tissue mass in hyperinsulinemic females along with a greater decrease in plasma triglycerides and glucose. They concluded that "an energy restrictive high protein diet may reduce the risk of cardiovascular disease and delay the onset of type 2 diabetes in subjects with hyperinsulinemia." O'Brien et al. found that there was a greater reduction in inflammatory markers for CVD with a low CHO diet than with a low fat diet. C-reactive protein and serum amyloid A levels were reduced with intake of a low CHO diet in these obese subjects. In addition, the subjects lost more weight on the low CHO diet. The investigators concluded that "for short periods of time, a LC [low CHO] diet is more efficacious, both in causing weight loss and in reducing serum inflammatory markers, than is a calorically-matched LF [low fat] diet."

After years of the scientific community telling the Atkins diet proponents to do some research, they are, and those that told them to do it aren't happy with the results. Fact is, nothing is really proven at this point but there is a consistency to the findings suggesting that there might be more than one healthy "scientifically supported" dietary approach to weight loss. The trick seems to be to find a low calorie dietary pattern people can understand and maintain. The precise values for protein, fat and carbohydrates probably matter less than whether people can adhere to it.

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