N U T R I T I O N

closeUP

Volume 21 – Number 2

Summer 2004

INSIDE

Executive Editor:
Donald J. McNamara, Ph.D.

Writer/Editor:
Jenny Heap, M.S., R.D.

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.

enc egg nutrition center
1050 17th St., NW Suite 560
Washington, DC 20036
(202) 833-8850
e-mail: enc@enc-online.org

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

Lutein Intake Slows Age-Related Macular Degeneration

Age-related macular degeneration (AMD), a progressive disease of the retina that impairs central vision (see Figure 1), is the number one cause of vision loss in older adults in the U.S. Ninety percent of all AMD cases are accounted for by atrophic, or "dry," AMD. Although advanced age, female sex, smoking, obesity, and a family history of AMD increase the risk of developing this debilitating disease, dietary intake also plays a role in risk modification. Low fruit and vegetable consumption is associated with increased risk of AMD and supplementation with a combination of antioxidants, zinc, and copper appears to slow the disease's progression. Numerous epidemiological studies have shown a more sprecific relationship between AMD and a low intake of lutein, the carotenoid primarily responsible for macular pigment optical density (MPOD). The lutein antioxidant supplementation trial (LAST) was designed to evaluate the effects of treatment with lutein, both alone and in combination with antioxidants and minerals, on MPOD and disease progression in elderly individuals with atrophic AMD.

Ninety participants (86 M, 4 F) were recruited to participate in a 12-month double-blind, placebo-controlled, supplementation trial. Participants were randomly assigned to one of three treatment groups as follows:
1) **L Group**—received capsules containing 10 mg non-esterified lutein.
2) **L/A Group**—received capsules containing 10 mg non-esterified lutein in addition to 2500 IU vitamin A, 15000 IU beta carotene, 1500 mg vitamin C, 400 IU vitamin D3, 500 IU natural vitamin E, 50 mg vitamin B1, 10 mg vitamin B2, 70 mg vitamin B3, 50 mg vitamin B5, 50 mg vitamin B6, 500 mcg vitamin B12, and a combination of minerals.

*__Figure 1.__ Normal vision (left) vs. impaired central vision typical of AMD (right)

Continued on page 2

Lutein Intake, cont...

Continued from page 1

3) P Group—received a maltodextrin placebo.

Prior to initiation of the trial, dietary status for each participant was evaluated using a food frequency questionnaire (repeated at month 12). Ophthalmic testing was carried out to obtain baseline measures of central vision including MPOD, glare recovery (GR), near- and distance visual acuity (measured in Snellen letters), and contrast sensitivity function (CSF). Participants were also asked to fill out a questionnaire (VFQ-14) for self-evaluation of day-to-day activities, night driving, and glare recovery symptoms. An Amsler grid (Figure 2) was provided with the questionnaire. (The Amsler grid is used as a patient self-assessment tool.) Patients are asked to focus on the center dot and to report whether the lines and/or boxes change shape. For patients with AMD, visual "spots" may appear and lines often appear distorted (metamorphopsia) (Figure 3). Participants were asked to self-administer this test to monitor changes in their vision over time. Ophthalmic testing, the VFQ-14 questionnaire, and the self-administered Amsler grid test were repeated at 4, 8, and 12 months.

No disease progression occurred in any group over the course of the study. Compliance appeared very good for all groups, with nearly every participant taking 92% of their assigned supplement pills. There were no differences between groups at baseline with regard to age, years since AMD diagnosis, smoking status, alcohol or caffeine intake, iris color, multivitamin use, or dietary lutein or iron intake. The only difference between groups at baseline was in the L/A group, which was reported to have slightly lower MPOD. The average BMI for the L/A group was also higher than for the L or P groups. Since lower MPOD has been shown to be associated with obesity, this was consistent with expectations for this sample.

Mean MPOD increased from baseline to 12 months by 0.09 (32%) and 0.08 log units (43%) for L and L/A groups, respectively, while MPOD in the P group decreased by 0.03 log units. Near visual acuity improved from baseline by 5.4 Snellen letters for the L group (approximately equivalent to 1 line of visual acuity; P=0.01) and 3.5 Snellen letters for the L/A group (P=0.04). The placebo group showed a non-significant 2.1 Snellen letter decrease in visual acuity.

Average GR times improved 23.7, 34.7, and 22.7 seconds from baseline for groups L, L/A, and P, respectively. Quality of vision, as measured by CSF, improved significantly from baseline for the L and L/A groups, with greater improvement observed in the L/A group. Amsler grid testing results improved only for participants in the L group (P=0.01).

The authors conclude that lutein, alone and with a combination of antioxidant vitamins and minerals, clearly improves MPOD, GR time, and near visual acuity. These data demonstrate the effectiveness of lutein in slowing the progression of AMD and improving central vision in individuals with existing atrophic AMD. More research in this area is critically important, as lutein shows promise as a functional nutrient in promoting MPOD and maintaining retinal health. The potential import of such research to the aging US population is tremendous, given the current prevalence of AMD and anticipated continued growth in this group as more and more "baby boomers" enter their elderly years.

Richer S, Stiles W, Statkute L, et al. Double-masked, placebo-controlled, randomized trial of lutein and antioxidant supplementation in the intervention of atrophic age-related macular degeneration: the Veterans LAST study (Lutein Antioxidant Supplementation Trial). *Optometry* 2004;75(4):216-30.

Key messages

- Supplementation with lutein alone or in combination with antioxidant vitamins and minerals improves macular pigment density, GR time, and near visual acuity.
- Lutein supplementation appears to slow the progression of AMD and improve measures of central vision in individuals with existing AMD.

*__Figure 2__. Amsler grid, normal vision

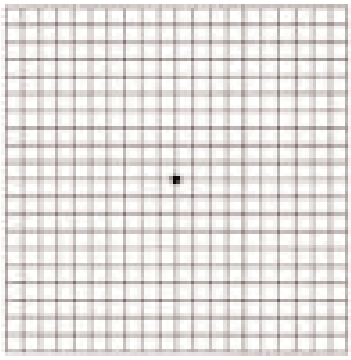
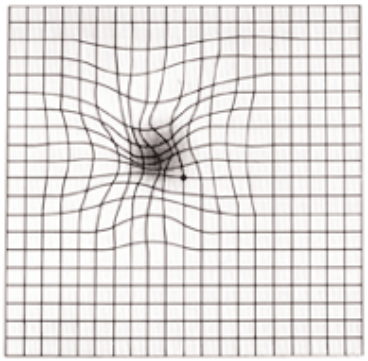


*__Figure 3__. Amsler grid, metamorphopsia



*Images courtesy of the National Eye Institute (NEI), National Institutes of Health (NIH).

Low-Fat, High-CHO vs. Low-Fat, High-Protein Diets for Weight Loss

High-protein, low-carbohydrate (CHO) diets have been criticized from many perspectives. Though short-term studies consistently demonstrate the effectiveness of high-protein diets in producing desired weight loss, health professionals question the suitability of such diets for promoting and maintaining overall health. In spite of well-voiced concerns about the potential adverse effects on serum lipids, kidney function, and bone health, tens of thousands are adopting low-CHO lifestyles and are successfully reaching their formerly elusive weight-loss goals. Recent clinical trials confirm that high-protein diets are at least as effective as their high-CHO counterparts in producing weight loss, but conclusions regarding consequences for blood lipid profiles are controversial.

Much of the concern among healthcare professionals stems from the thought that high-protein diets are, by definition, also high in fat, particularly saturated fat, which is known to raise serum total and LDL cholesterol levels. While many studies show that high-protein diets with varying fat compositions produce no changes in total or LDL cholesterol levels, others show elevations in these lipid markers. It is helpful to note, however, that even high-CHO, low-fat diets have limitations. While low-fat, high-CHO diets generally result in decreased total and LDL cholesterol levels, triacylglycerol and HDL levels tend to worsen with higher CHO intake. Limited data are available regarding the comparative efficacy of high-CHO, low-fat (HCLF) diets vs. high-protein, low-fat (HPLF) diets.

To address this issue, Johnston et al. designed a clinical trial comparing the effects of a low-fat, energy-restricted, high-CHO diet vs. a similar low-fat, energy-restricted diet low in CHO and high in protein. Twenty healthy adults (2 men, 18 women) were recruited to participate. Each was randomly assigned to follow a HPLF or HCLF diet for 6 weeks. The HCLF diet was designed based on the U.S. Dietary Guidelines and consisted of 66% of calories from carbohydrate and 15% of calories from protein. The HPLF dietary treatment consisted of 32% of calories from protein and 41% of calories from CHO and emphasized low-fat animal protein sources. Participants were assigned individual energy intakes based on calculated basal metabolic rate (BMR) minus 25-30% to produce sufficient energy deficit for the desired weight-loss.

Sixteen out of the original 20 participants successfully completed the six-week diet treatment phase (9 HPLF and 7 HCLF participants). Although both diets were designed to provide less than 30% of calories from fat, the HPLF diet treatment was higher both in total fat and saturated fat than the HCLF regimen (28 vs. 21% total fat; 8 vs. 6% saturated fat). Both diets limited refined sugar intake to <10% of total energy and also provided >20g fiber/day. All meals were prepared in the metabolic kitchen. Participants ate lunch Monday through Friday in the lab. Breakfast, dinner, and weekend meals were sent home with participants.

The diet treatment period was preceded by baseline measurements 2 weeks before the trial began. Participants followed an isoenergetic diet based on the US dietary guidelines for 2 days, after which resting energy expenditure (REE) was measured, blood samples were taken for baseline data, and a 24-hour urine collection was completed. On the first day of each week of the feeding trial, participants reported to the research lab for assessment of dietary intake, determination of body composition (utilizing bioelectrical impedance), and self-reported estimation of overall hunger for the previous week (using a 7-point Likert-scale). REE was again measured on the last day of the feeding trial. After four weeks of follow-up, participants returned to the lab for a final assessment of body composition and blood lipids.

Weight loss was similar between groups, with both HPLF and HCLF participants losing an average of 6% body weight. Fat mass also declined, with both groups experiencing a 10% reduction in adipose tissue. Participants in both groups had maintained the weight loss at follow-up, four weeks after the conclusion of the study.

Compliance with dietary requirements was good for both groups, however, two participants dropped out of the study due to out-of-state travel (1 HPLF, 1 HCLF) and two withdrew because of noncompliance due to extreme hunger (both HCLF). With regard to feelings of hunger and satiety, HPLF participants reported feeling more satisfied than did the HCLF participants during the first four weeks of the trial, but both groups reported feeling equally satiated during the last two weeks of the treatment period.

Total cholesterol levels declined for both HPLF and HCLF participants (-5.2%±0.7 vs. -6.2%±0.7, respectively), but the change did not differ significantly between groups. Neither LDL cholesterol, nor the total cholesterol:HDL ratio changed significantly for either group. Changes in triacylglycerol concentrations did not differ between treatment groups. Total cholesterol levels at follow-up did not differ from pre-intervention levels.

Participants in both diet groups experienced a ~24% reduction in plasma insulin concentrations and a ~5% improvement in insulin sensitivity. Plasma

Continued from page 3

glucose concentrations remained unchanged. Creatinine clearance (an indicator of glomerular filtration rate) did not change following either diet treatment, indicating no effect on kidney function. Calcium losses to the urine were higher following the HPLF diet (up 42% from baseline) than the HCLF diet (down 23% from baseline). These data are consistent with those seen in previous clinical trials in which increased protein intake caused greater calcium excretion, particularly when calcium intake also rose. Recent investigations indicate that the increased calcium losses observed with high protein intake are due to higher intestinal calcium absorption (stimulated by the additional protein) and increased calcium consumption, not to bone demineralization.

These data indicate that energy-restricted diets, whether high in protein or high in CHO, can be equally effective in promoting weight loss. The authors conclude that the weight loss observed in this 6 week dietary treatment trial was due to calorie restriction, not to macronutrient distribution, and that higher protein intake during energy restriction appears to promote satiety. This research further indicates that high-protein diets do not negatively affect kidney function in the short term. Longer-term studies with larger population samples must be completed before these conclusions can be generalized to larger populations.

Volek JS, Sharman MJ, Gomez AL, DiPasquale C, et al. Comparison of a very low-carbohydrate and low-fat diet on fasting lipids, LDL subclasses, insulin resistance, and postprandial lipemic responses in overweight women. *JACN* 2004;23(2):177-184.

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

High Dairy Protein Intake:

Influence on Urinary Calcium Losses and Bone Turnover During Weight Loss

Weight loss has typically been associated with increased bone resorption and reduced bone mineral density (BMD). Because increased urinary calcium losses have been reported with high protein intake, researchers and clinicians have long questioned whether utilizing high-protein diets for weight loss might compromise bone health. It has been suggested that bone resorption during weight loss should be countered by increased dietary calcium. To clarify the interaction between high-protein calorie-restricted diets, calcium intake, and bone metabolism, Bowen et al. examined the differential effects of mixed-protein and dairy-protein intake on calcium excretion and markers of bone turnover in 50 overweight adults following either a high mixed-protein or a high dairy-protein diet for weight loss.

Sixty overweight adults (BMI 27-40), aged 20-65 years, were recruited to participate in this study. Each was assigned to undergo one of two high-protein, energy restricted diets for 12 weeks followed by an additional 4-week "energy balance" phase. The dietary treatments were isocaloric and consisted of the same macronutrient distribution,

Continued from page 4

differing only in protein source. The high dairy-protein diet (DP) emphasized dairy foods such as milk, cheese, and yogurt to provide ample calcium, while the high mixed-protein diet (MP) focused on lean meats, fruit, eggs, almonds, and legumes.

Ten participants withdrew from the study for personal reasons, employment commitments, or illness. Only two were excluded for non-compliance. Based on similarly elevated urinary urea:creatinine (Cr) excretion between study groups, dietary compliance appeared high in both cohorts. Protein intake among participants was ~1.2 g/kg body weight, of which 5% was from dairy sources for the MP group, compared to 62% for the DP group. Calcium intake for DP participants was 3.7 times that of MP participants. Macronutrient distribution remained similar between groups throughout the intervention. Participants in both groups lost an average of 10% of baseline body weight during the energy restriction phase (DP; -9.0±0.6 kg, MP; -9.3±0.7 kg), followed by weight stabilization during the four weeks of energy balance.

Urinary calcium losses are often reported to rise with increased protein intake. The cause of this increased calcium excretion is not known; however, recent research indicates that the calcium losses are due to greater calcium absorption in the gut, which is promoted by augmented protein intake, and not attributed to bone resorption. Contrary to what would have been expected according to previous studies, urinary calcium excretion decreased 33% from baseline following both interventions (-1.13±0.3 mmol/d, P = 0.004).

The 33% decrease in urinary calcium excretion for both groups was unexpected given the high protein content of these dietary interventions. In general, protein from cheese, fish, and meat promotes acid production and reduces blood pH, causing increased urinary calcium excretion. Alkali production, which can be promoted by eating foods such as fruits, vegetables, and non-cheese dairy, can blunt urinary calcium losses. The authors suggest that both dietary interventions may have resulted in sufficient alkali to produce this unexpected result. Another explanation for the observed reduction in calcium excretion may be related to increased intake of phosphorus from animal protein sources. Because it promotes calcium reabsorption in the kidney, phosphorus prevents calcium loss in the urine. The best sources of phosphorus are high-protein foods such as milk, meat, poultry, fish, eggs, nuts, and legumes. Both the DP and MP diets were relatively high in these phosphorus-rich foods, which may further explain why urinary calcium losses decreased.

No changes were detected in BMD following the intervention, probably due to the short duration of the study. However, markers of both bone formation and resorption were elevated from baseline following the MP intervention, indicating that bone turnover was greater for MP participants. The elevation in urinary pyridinoline (Pyr):Cr, a marker of bone resorption, increased from baseline and was similar for both intervention groups. Levels of deoxypyridinoline (Dpr):Cr, (a more specific marker of bone resorption than Pyr:Cr, which also indicates tendon breakdown) also increased for both groups, but the rise was significantly greater in MP participants. The authors speculate that this could have been due to the concurrent decrease in calcium intake in the MP group. Elevations in these urinary markers of bone resorption are consistent with previous studies examining bone turnover during energy restriction and weight loss. Plasma osteocalcin, a marker of bone formation, remained stable throughout the DP intervention, but increased significantly from baseline in the MP diet group ($P<0.001$). Thus, markers of bone formation and resorption indicate that the DP regimen provided some protection against bone turnover during weight loss.

The authors conclude that weight loss on energy-restricted, high mixed-protein diets results in increased urinary markers of bone resorption as well as increased plasma markers of bone formation. These data further indicate that for individuals on high-protein diets, emphasizing dairy foods, and thus increasing calcium intake, may ameliorate bone turnover during weight loss.

Bowen J, Noakes M, Clifton P. High dairy protein, high-calcium diet minimizes bone turnover in overweight adults during weight loss. *J Nutr* 2004;134;568-573.

Very Low Carbohydrate Diets:

Effects on CVD Risk Factors in Normolipidemic Overweight Women

Although recent studies examining the effectiveness of very low-carbohydrate (CHO) diets have found them equal or superior to traditional low-fat diets in inducing weight loss, relatively few studies have addressed comparative CVD risk factor outcomes. This information is critical since most very low-CHO diet regimens tend to be high in saturated fat, which has long been known to increase serum cholesterol levels. Because serum lipids and other markers of CVD risk often improve with weight-loss, independent of dietary intake, results from weight-loss studies must be interpreted carefully. Short-term adherence to very low-CHO diets not intended for weight loss has been shown to reduce triacylglycerol (TAG) and insulin levels, and to increase HDL-C, LDL-C, and LDL particle size, thus reducing overall CVD risk. However, whether the effects of energy-restricted, very low-CHO diets vs. traditional low-fat, energy-restricted diets differ with regard to CVD risk factors is not known. Volek et al. addressed this issue in a recent study designed to compare the effects of a short-term, very low-CHO diet with those of a short-term, traditional low-fat diet on CVD risk factors in overweight women.

Thirteen women classed as overweight or obese (percentage body fat >30%), but otherwise healthy, were recruited to participate. The women were sedentary or moderately active with a mean age of 34.0±8.6 years and a mean BMI of 29.6±4.0 kg/m^2. All participants had normal serum lipid profiles at baseline.

To control for the independent effects of weight loss, diets were designed to induce similar weight reduction in both study groups (500 kcal/day deficit). Calorie intake levels were calculated for individuals based on resting energy expenditure from indirect calorimetry measurements and activity level. The low-fat diet regimen consisted of 25% of total calories from fat (<10% calories from saturated fat and <300 mg cholesterol/day), 20% of total calories from protein, and 55% of total calories from carbohydrate. During the low-fat regimen, participants were encouraged to eat foods such as whole grains, fruits and vegetables, fruit juices, low-fat dairy products, and lean meat. The very low-CHO regimen consisted of 60% calories from fat, 30% calories from protein, and 10% calories from carbohydrate. Beef, poultry, fish, oils, nuts, seeds, peanut butter, some vegetables, salads with low-CHO dressing, cheese, eggs, protein powder, and some low-CHO commercial beverages and snacks were typically eaten during this phase. No restrictions were made on type of fat, source of fat, or on cholesterol intake during the very low-CHO phase. Participants were provided with a daily multivitamin to be taken throughout both dietary interventions.

Participants received weekly dietary counseling. Dietary compliance was assessed and body weights were measured during these weekly sessions. Blood samples were obtained on two separate days before and after each four-week dietary intervention and were analyzed for determination of blood cholesterol and TAG levels, oxidized LDL, lipoprotein particle size, glucose, and insulin levels. Oral fat tolerance testing was also administered after each dietary treatment period.

For both dietary phases, compliance appeared to be good. During the low-fat regimen, participants maintained 21% of total calories from fat. Based on reported use of urinary reagent strips, all participants maintained ketosis throughout the low-CHO diet phase and maintained 9% of total calories from carbohydrate based on self-reported intake. Weight loss was significantly greater for participants during the low-CHO regimen than during the low-fat regimen (-2.96±1.45 kg vs. -1.06±2.07 kg). However, no data were provided for actual calorie reduction from baseline for either group, so it is not possible to deduce whether weight loss was due to improved diet compliance with higher fat and protein intake, to greater energy restriction, or due to some unknown factor.

Although total, LDL, and HDL cholesterol levels were significantly lower following the low-fat diet period, the total cholesterol:HDL cholesterol ratios were similar following both diet periods, indicating that both dietary interventions similarly affected overall CVD risk. Although the very low-CHO diet did not decrease LDL cholesterol levels, it prevented the drop in HDL cholesterol seen with the low-fat regimen. The authors submit that, based on previously-measured changes in blood lipids associated with weight loss alone, total, LDL, HDL cholesterol, and TAG would have decreased by 5.7, 2.3, 0.8, and 3.9 mg/dL on the low-CHO diet and by 2.1, 0.8, 0.3. and 1.4 mg/dL on the low-fat diet. Discrepancies between actual and expected changes in blood lipids associated with weight loss indicate that the changes in blood lipids observed in this study were due primarily to dietary modifications.

In previous, similar studies conducted by Volek et al., men with unfavorable lipid profiles experienced improvements in CVD risk factors (such as decreased TAG levels, increased HDL cholesterol levels and increased LDL particle size distribution) proportional to the extent of baseline dyslipidemia following a very low-CHO diet regimen. Those with the worst status benefited most from the very low-CHO treatment. The authors speculate that similar improvements were not seen in this cohort of women because none were dyslipidemic at baseline.

Continued from page 6

There were no significant changes in oxidized LDL from baseline following either study. Glucose and insulin levels and insulin resistance were slightly, but significantly, lower following the very low-CHO diet, however, the authors question the clinical relevance of this small improvement. The only difference in lipoprotein particle size between diet interventions was VLDL, which was lower following the very low-CHO diet. Oral fat tolerance was not significantly different following either diet regimen.

The authors conclude that both low-fat and very low-CHO, calorie-restricted diets similarly affect CVD risk factors in overweight, normolipidemic women. These results warrant further research in this group. Larger samples and longer-term diet interventions are needed to substantiate these conclusions and to clarify questions of safety and long-term efficacy.

Johnston CS, Tjonn SL, Swan PD. High-protein, low-fat diets are effective for weight loss and favorably alter biomarkers in healthy adults. *J Nutr.* 134:586-591, 2004.

Editorial: *"It's Not Our Fault! Government and Industry Did It." The Not-So-Subtle Art of Science Spin.*

In a recent article in *Food Chemical News* (3 May 2004, Vol 46 #12), members of the FDA Food Advisory Committee responded to the question, "Does the current scientific evidence suggest a relationship between total fat intake and risk of coronary heart disease?" with a resounding no. They were quoted as stating there is essentially no relationship between dietary fat and CHD and that the emphasis on total fat was distracting attention from saturated and trans fat. And then, the article stated "that most panel members regarded the low-fat fad as an example of what can happen when industry and regulators embrace a scientific concept too quickly and fail to ponder the possible consequences." Embrace! Too quickly! Give me a break! More like jammed down their throats until they were made to choke.

I'm always amazed at the ability some have to blame the other guy. Thirty-five years ago it was the same scientists sitting around debating the diet-heart disease question and the diet-cancer question who, based on some rather simplistic epidemiological data and questionable animal studies, decided that dietary fat was bad and that the answer to our chronic diseases was to eat less fat and cholesterol. Somehow, someway the magic number was set at less than 30% of calories from fat and the "experts" then proceeded to convince the McGovern Committee that, since they were the experts, they knew the answers. Contrary views were not only met with derision and accusations of "old fuddyness" but also that definitive label of contempt, "a pawn of industry." [Check out the history of the National Academy of Sciences' "Toward Healthful Diets" report which was trashed for focusing on excess weight and the importance of caloric balance rather than the low-fat, low-cholesterol mantra.] Even the AMA was beat up for being too cautious with its dietary recommendations (*JAMA* 1979; 242:43-48). "In time, when our knowledge of the relationships, if any, of specific food components to the development of chronic diseases reaches maturity, it may be feasible to make more refined recommendations. Until then, the AMA recommends that the American public give primary emphasis to the achievement and maintenance of the most desirable body weight and further recommend that this be accomplished through the combination of dietary control and exercise."

The marginal success of the Lipid Research Clinics (LRC) Trial lead to the Cholesterol Consensus Conference resulting in the National Cholesterol Education Program (NCEP) without so much as an "I beg your pardon?" to those who weren't "made guys" in the "lipid mafia." A consensus is easy when only those with a single view are allowed expression. And those who publicly questioned the scientific basis and rationale of this rush to dietary nirvana were simply considered out of date and antiquated by their colleagues who, in a non-mean spirited way, closed them out of committees and panels and symposiums and eventually, out of funding. Clearly any scientist who failed to see the certainty of the evidence wasn't much of a scientist and would eventually, if allowed to continue, ruin all the conversions of the new faithful to the low-fat doctrine. Clearly they had to get the dietary heretics out of the picture so as not to "confuse the public" ("government speak" for why meaningful changes in nutrition policy are unattainable).

So the American Heart Association (AHA) dietary guidelines became the NCEP dietary guidelines became the FDA Nutrition Facts Label guidelines became the US Dietary Guidelines. Of course this process was relatively easy to achieve since each committee usually consisted of the "usual suspects." With the size of the scientific community in this country, isn't it amazing that the same people seem to be found on all these committees, whether it

Editorial cont...

Continued from page 7

be AHA, NCEP, IOM-NAS, FDA, Dietary Guidelines Advisory Committee, and just about every symposium dealing with diet and heart disease. I know a few folks who have served on every one of these at one time or another spouting the same low-fat, low-cholesterol rhetoric with little or no debate. A clear example of real diversity of scientific opinions helping to establish government policy! But now we blame the government for rushing too fast when all they heard was "low fat, low cholesterol; low fat, low cholesterol" and the nagging persistence of the food police to do something about those evil purveyors of unhealthy, perchance deadly, fatty foods (ah yes, the old "heart attack on a plate").

So what did the food industry do? They did what everyone was screaming at them to do! The plea to market low-fat, low-cholesterol foods came from the health advocates, the government, the media, the food police, and the consumer. So they put the low-fat foods on the shelves and on the menus and, surprise, the market just wasn't there for "sawdust and woodchip" snacks and fast foods (remember the McLean burger? Border Lights?). So let's be fair to the food industry, they are in the business of selling food and, if that food doesn't sell, they better do something about it. That something was sugar. Take out the fat, add the sugar, sell the product. (For the consumer advocates who live on some distant planet, it's called marketing.) And, for the judgmental out there, it was marketing in response to the demand of the scientists, government, and consumers for low-fat, low-cholesterol products. How could industry be so irresponsible! They gave us what we asked for and were willing to buy. So now we blame government and industry for jumping too quickly on the low-fat bandwagon. I suggest a check of history to find out exactly who was pushing that wagon—making sure you got on, or got out of the way.

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

Egg Nutrition Center • 1050 17th Street, NW Suite 560 • Washington,DC 20036

closeup NUTRITION

Non Profit
Organization
US Postage Paid
Permit #293
Merrifield, VA